MICROBIOLOGY OF ENDODONTICS AND ASEPSIS IN ENDODONTIC PRACTICE

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Microorganisms cause virtually all pathoses of the pulp and the periradicular tissues. To effectively treat endodontic infections, clinicians must recognize the cause and effect of microbial invasion of the dental pulp space and the surrounding periradicular tissues. Once bacterial invasion of pulp tissues has taken place, both nonspecific inflammation and specific immunologic response of the host have a profound effect on the progress of the disease. Knowledge of the microorganisms associated with endodontic disease is necessary to develop a basic understanding of the disease process and a sound rationale for effective management of patients with endodontic infections. Although the vast majority of our knowledge deals with bacteria, we are now aware of the potential for endodontic disease to be associated with fungi and viruses.[1–4] The topics of this chapter are directed toward the role of microorganisms in the pathogenesis of endodontic disease with recommendations for treatment of endodontic infections. Owing to much recent controversy over the "theory of focal infection," an update on this issue will be presented first.

THEORY OF FOCAL INFECTION REVISITED

In 1890, W. D. Miller associated the presence of bacteria with pulpal and periapical disease. In 1904, F. Billings described a "focus of infection" as a circumscribed area of tissue infected with pathogenic organisms. One of his students was E. C. Rosenow, who in 1909 described the "Theory of Focal Infection" as a **localized or generalized infection caused by bacteria traveling through the bloodstream from a distant focus of infection.** In 1910, a British physician, William Hunter, presented a lecture on the role of sepsis and antisepsis in medicine to the faculty of McGill University. He condemned the practice of dentistry in the United States, which emphasized restorations instead of tooth extraction. Hunter stated that the

restorations were "a veritable mausoleum of gold over a mass of sepsis." He believed that this was the cause of Americans' many illnesses, including pale complexion, chronic dyspepsias, intestinal disorders, anemias, and nervous complaints.

Soon pulpless teeth (teeth with necrotic pulps) and endodontically treated teeth were also implicated. Weston Price began a 25-year study on pulpless and endodontically treated teeth and their association with focal infection. With expansion of the theory, many dentists and physicians became "100 Percenters," who recommended the extraction of all pulpless and endodontically treated teeth. The dental literature contained numerous testimonials reporting cures of illnesses following tooth extraction. These reports were empirical and without adequate follow-up. However, they wrongfully supported the continued extraction of teeth without scientific reason. In many cases, the diseases returned, and the patients had to face the additional difficulty of living with mutilated dentitions.

In the 1930s, editorials and research refuted the theory of focal infection and called for a return to constructive rather than destructive dental treatment rationale.[5,6] The studies by Rosenow and Price were flawed by inadequate controls, the use of massive doses of bacteria, and bacterial contamination of endodontically treated teeth during tooth extraction. In 1939, Fish recognized four zones of reaction formed in response to viable bacteria implanted in the jaws of guinea pigs.[7] He described the bacteria as being confined by polymorphonuclear neutrophil leukocytes to a zone of infection. Outside the zone of infection is the zone of contamination containing inflammatory cells but no bacteria. Next, the zone of irritation contained histocytes and osteoclasts. On the outside was a zone of stimulation with mostly fibroblasts, capillary buds, and osteoblasts. Fish theorized that removal of the nidus of infection would lead to resolution of the

infection. This theory became the basis for successful root canal treatment

Today the medical and dental professions agree that there is no relationship between endodontically treated teeth and the degenerative diseases implicated in the theory of focal infection. However, a recent book entitled *Root Canal Cover-up Exposed* has resurrected the focal infection theory based on the poorly designed and outdated studies by Rosenow and Price.[8] This body of research has been evaluated and disproved. Unfortunately, uninformed patients may receive this outdated information and believe it to be credible new findings. To further confuse the issue, recent epidemiologic studies have found relationships between periodontal disease and coronary heart disease, strokes, and preterm low birth rate.[9,10] It must be kept in mind that epidemiologic research can identify relationships but not causation. Further research may show that periodontal disease constitutes an oral component of a systemic disorder or has etiologic features in common with medical diseases. They may occur at the same time without necessarily indicating a cause-effect relationship.

Endodontic infections can spread to other tissues. An abscess or cellulitis may develop if bacteria invade periradicular tissues and the patient's immune system is not able to stop the spread of bacteria and bacterial by-products. This type of infection/inflammation spreads directly from one anatomic space to an adjacent space. This is not an example of the theory of focal infection, whereby bacteria travel through the circulatory system and establish an infection at a distant site.

Practitioners are well aware of the relationship between bacteremias caused by dental procedures (especially tooth extraction) and infective endocarditis. This is an example of focal infection that is not related to the classic theory of focal infection. A bacteremia associated with a dental procedure introduces bacteria into the circulation. It does not arise because of the mere presence of an endodontically treated tooth. Studies have shown that the incidence and extent of a bacteremia are related to the amount of bleeding (trauma) produced by a dental procedure.[11–14] These studies have shown that nonsurgical endodontic procedures produce a relatively low incidence of bacteremia when compared to tooth extraction. Simple tooth extraction produces an extensive bacteremia 100% of the time.[12,15] Endodontic therapy should be the treatment of choice instead of tooth extraction for patients believed to be susceptible to infective endocarditis following a bacteremia.

A recent study found the frequency of bacteremia associated with **nonsurgical** root canal instrumentation

to be from 31 to 54%.[16] If the endodontic instrument was confined to inside the root canal 1 mm short of the apical foramen, the incidence of bacteremia was 4 in 13 (31%). If the instruments (sizes 15, 20, and 25) were deliberately used to a level 2 mm beyond the apical foramen, the incidence of bacteremia was 7 in 13 (54%). Ribotyping with restriction enzymes showed identical characteristics for the clinical isolates from the root canals and for the bacteria isolated from the blood. This typing method suggests that the microorganisms recovered from the bloodstream during and after endodontic treatment had the root canal as their source. However, to show a causal relationship between an oral infection and systemic disease, it is not adequate to show only a potential relationship via a bacteremia. Hard evidence is needed to show that the organism in the nonoral site of infection actually came from the oral cavity. If possible, Koch's postulates should be fulfilled to establish a causal role of the microorganism from the oral cavity.

Successfully completed root canal therapy should not be confused with an untreated infected root canal system or a tooth with a periradicular abscess that may be a source of bacteremias. In addition, numerous bacteremias occur every day as a result of a patient's normal daily activities. Endodontics has survived the theory of focal infection because of recognition by the scientific community that successful root canal treatment is possible without endangering systemic health.

ENDODONTIC INFECTIONS

Colonization is the establishment of microbes in a host if appropriate biochemical and physical conditions are available for growth. Normal oral flora is the result of a permanent microbial colonization in a symbiotic relationship with the host. Although the microbes in the normal oral flora participate in many beneficial relationships, they are opportunistic pathogens if they gain access to a normally sterile area of the body such as the dental pulp or periradicular tissues and produce disease. The steps in the development of an endodontic infection include microbial invasion, multiplication, and pathogenic activity. Much of the pathogenic activity is associated with host response.

Pathogenicity is a term used to describe the capacity of a microbe to produce disease, whereas virulence describes the degree of pathogenicity. Bacteria have a number of virulence factors that may be associated with disease. They include pili (fimbriae), capsules, extracellular vesicles, lipopolysaccharides, enzymes, short-chain fatty acids, polyamines, and low-molecular-weight products such as ammonia and hydrogen sulfide. Pili may be important for attachment to sur-

faces and interaction with other bacteria in a polymicrobial infection. Bacteria including gram-negative black-pigmented bacteria (BPB) may have capsules that enable them to avoid or survive phagocytosis.[17]

Lipopolysaccharides are found on the surface of gram-negative bacteria and have numerous biologic effects when released from the cell in the form of **endotoxins.** The endotoxin content in canals of symptomatic teeth with apical rarefactions and exudate is higher than that of asymptomatic teeth.[18] Endotoxins have been associated with periapical inflammation and activation of complement.[19,20]

Enzymes are produced by bacteria that may be spreading factors for infections or proteases that neutralize immunoglobulins and complement components.[21–24] The enzymes in neutrophils that degenerate and lyse to form purulent exudate also have an adverse effect on the surrounding tissues.

Gram-negative bacteria produce **extracellular vesicles** (Figure 3-1). They are formed from the outer membrane and have a trilaminar structure similar to the outer membrane of the parent bacteria. These vesicles may contain enzymes or other toxic chemicals. It is believed that these vesicles are involved in hemagglutination, hemolysis, bacterial adhesion, and proteolytic activities.[25,26] Because these vesicles have the same antigenic determinants on their surface as their parent bacteria, they may protect the bacteria by combining with and neutralizing antibodies that would have reacted with the bacteria.

Anaerobic bacteria commonly produce short-chain fatty acids including propionic, butyric, and isobutyric

acids. As virulence factors, these acids may affect neutrophil chemotaxis, degranulation, chemiluminescence, and phagocytosis. **Butyric acid** has been shown to have the greatest inhibition of T-cell blastogenesis and to stimulate the production of interleukin-1, which is associated with bone resorption.[27]
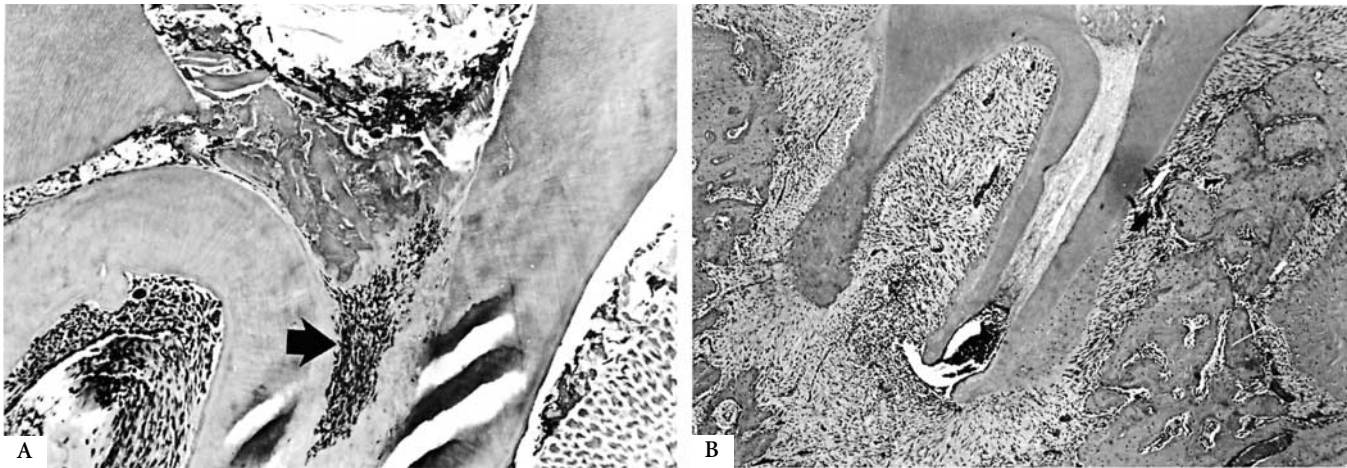
Polyamines are biologically active chemicals found in infected canals.[28] Bacteria and host cells contain polyamines. Putrescine, cadaverine, spermidine, and spermine are involved in the regulation of cell growth, regeneration of tissues, and modulation of inflammation. The amount of total polyamines and putrescine is higher in the necrotic pulps of teeth that are painful to percussion or with spontaneous pain.[28] When a sinus tract was present, a significantly greater amount of cadaverine was detected in the pulp space.[28] Although some correlations between some virulence factors and clinical signs and symptoms have been shown, an absolute cause and effect relationship has not been proven.

ASSOCIATION OF MICROBES WITH PULPAL DISEASE

Antony van Leewenhoek, the inventor of single-lens microscopes, was the first to observe oral flora.[29] His description of the "animalcules" observed with his microscopes included those from dental plaque and from an exposed pulp cavity. The father of oral microbiology is considered to be W. D. Miller. In 1890, he authored a book, *Microorganisms of the Human Mouth*, which became the basis for dental microbiology in this country. In 1894, Miller became the first researcher to associate the presence of bacteria with pulpal disease.[30]

The true significance of bacteria in endodontic disease was shown in the classic study by Kakehashi et al in 1965.[31] They found that no pathologic changes occurred in the exposed pulps or periradicular tissues in germ-free rats (Figure 3-2, A). In conventional animals, however pulp exposures led to pulpal necrosis and periradicular lesion formation (Figure 3-2, B). In contrast, the germ-free rats healed with dentinal bridging regardless of the severity of the pulpal exposure.[31] Thus, the presence or absence of microbial flora was the major determinant for the destruction or healing of exposed rodent pulps.

Invasion of the pulp cavity by bacteria is most often associated with dental caries. Bacteria invade and multiply within the dentinal tubules (Figure 3-3). **Dentinal tubules range in size from 1 to 4 μm in diameter,** whereas the majority of bacteria are less than 1 μm in diameter. If enamel or cementum is missing, microbes may invade the pulp through the exposed tubules. A

Figure 3-1 Extracellular vesicles are shown between *Prevotella intermedia* cells (×20,000 original magnification).
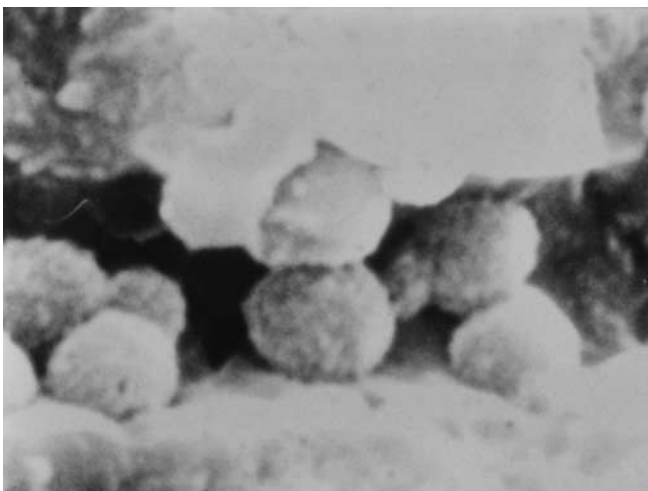
Figure 3-2   Role of bacteria in dentin repair following pulp exposure. **A,** Germ-free specimen obtained 14 days after surgery, with food and debris in the occlusal exposure. Nuclear detail of surviving pulp tissue (**arrow**) can be observed beneath the bridge consisting of dentin fragments united by a new matrix. **B,** Intentional exposure of first molar in control rat (with bacteria 28 days postoperatively). Complete pulp necrosis with apical abscess. A reproduced with permission from Kakehashi S, Stanley HR, Fitzgerald RJ. Oral Surg 1965;20:340. **B** reproduced with permission from Clark JW, Stanley HR. Clinical Dentistry. Hagerstown (MD): Harper & Row; 1976;4:10.

tooth with a vital pulp is resistant to microbial invasion. Movement of bacteria in dentinal tubules is restricted by viable odontoblastic processes, mineralized crystals, and various macromolecules within the tubules. Caries remains the most common portal of entry for bacteria and bacterial by-products into the pulpal space. However, bacteria and their **by-products** have been shown to have a direct effect on the dental pulp even without direct exposure.[32–34] These studies demonstrated inflammatory reactions opposite the exposed dentinal tubules. Although the inflammatory

Figure 3-3   Coccal forms of bacteria seen in the cross-section of a fractured dentinal tubule (×15,000 original magnification).

reactions could result in pulpal necrosis, the majority of pulps were able to undergo healing and repair.[32–34]

Following trauma and **direct exposure** of the pulp, inflammation, necrosis, and bacterial penetration are no more than 2 mm into the pulp after 2 weeks.[35] In contrast, a necrotic pulp is rapidly invaded and colonized. Peritubular dentin and reparative dentin may impede the progress of the microorganisms. However, the "dead tracts" of empty dentinal tubules following dissolution of the odontoblastic processes may leave virtual highways for the microbes' passage to the pulp cavity. Microbes may reach the pulp via direct exposure of the pulp from restorative procedures or trauma injury and from pathways associated with anomalous tooth development.

It is believed that the egress of irritants from an infected root canal system through tubules, lateral or accessory canals, furcation canals, and the apical foramina may directly affect the surrounding attachment apparatus. However, it is debatable whether periodontal disease directly causes pulpal disease.[36–39] The presence of pulpitis and bacterial penetration into exposed dentinal tubules following root planing in humans has been demonstrated.[40] Langeland et al. found that changes in the pulp did occur when periodontal disease was present, but pulpal necrosis occurred only if the apical foramen was involved.[38] Recently, Kobayashi et al. compared the bacteria in root canals to those in periodontal pockets.[41] The authors believe that bacteria concurrent in both areas suggest that the sulcus or periodontal pocket is the source of

the bacteria in root canal infections. To differentiate an abscess of periodontal origin from that of endodontic origin, the enumeration of spirochetes has been recommended.[42] Abscesses of periodontal origin contained 30 to 58% spirochetes, whereas those of endodontic origin were 0 to 10% spirochetes.

Anachoresis is a process by which microbes may be transported in the blood or lymph to an area of inflammation such as a tooth with pulpitis, where they may establish an infection. The phenomenon of anachoresis has been demonstrated in animal models both to nondental inflamed tissues and inflamed dental pulps.[43–45] However, the localization of bloodborne bacteria in instrumented but unfilled canals could not be demonstrated in an animal mode.[46,47] Infection of unfilled canals was possible only with overinstrumentation during the bacteremia to allow bleeding into the canals.[47] Anachoresis may be the mechanism through which traumatized teeth with intact crowns become infected.[48] The process of anachoresis has been especially associated with bacteremias and infective endocarditis.

Once the dental pulp becomes necrotic, the root canal system becomes a "privileged sanctuary" for clusters of bacteria, bacterial by-products, and degradation products of both the microorganisms and the pulpal tissue.[49–51]

PULPAL INFECTION

Polymicrobial interactions and nutritional requirements make the cultivation and identification of all organisms from endodontic infections very difficult. Prior to 1970, very few strains of strict anaerobes were isolated and identified because of inadequate anaerobic culturing methods. **The importance of anaerobic bacteria** in pulpal and periapical pathoses has been revealed with the development of anaerobic culturing methods and the use of both selective and nonselective culture media. However, even with the most sophisticated culturing methods, there are still many microorganisms that remain uncultivable. The bacteria in an infected root canal system are a restricted group compared to the oral flora.

Most of the bacteria in an endodontic infection are strict anaerobes. These bacteria grow only in the absence of oxygen but vary in their sensitivity to oxygen. They function at low oxidation-reduction potentials and generally lack the enzymes superoxide dismutase and catalase. Microaerophilic bacteria can grow in an environment with oxygen but predominantly derive their energy from anaerobic energy pathways. Facultative anaerobes grow in the presence or absence of oxygen and usually have the enzymes superoxide dismutase and catalase. Obligate aerobes require oxygen for growth and possess both superoxide dismutase and catalase.

Most species in endodontic infections have also been isolated from periodontal infections, but the root canal flora is not as complex.[41] Using modern techniques, five or more species of bacteria are usually isolated from root canals with contiguous apical rarefactions. The number of colony-forming units (CFUs) in an infected root canal is usually between 10^2 and 10^8. A positive correlation exists between an increase in size of the periapical radiolucency and both the number of bacteria species and CFUs present in the root canal.[52,53]

The dynamics of bacteria in infected root canals have been studied in monkeys.[51,54,55] After infecting the monkey root canals with indigenous oral bacteria, the canals were sealed and then sampled for up to 3 years. Initially, facultative bacteria predominated; however, with increasing time, the facultative bacteria were displaced by anaerobic bacteria.[51,54,55] The results indicate that a selective process takes place that allows anaerobic bacteria an increased capability of surviving and multiplying. After almost 3 years (1,080 days), 98% of the cultivable bacteria were strict anaerobes.

The root canal system is a selective habitat that allows the growth of certain species of bacteria in preference to others. Tissue fluid and the breakdown products of necrotic pulp provide nutrients rich with polypeptides and amino acids. These nutrients, low oxygen tension, and bacterial by-products determine which bacteria will predominate.

Antagonistic relationships between bacteria may occur. Some metabolites (eg, ammonia) may be either a nutrient or a toxin, depending on the concentration. In addition, bacteria may produce bacteriocins, which are antibiotic-like proteins produced by one species of bacteria to inhibit another species of bacteria. When Sundqvist et al. cultured intact root canals, 91% of the organisms were strict anaerobes.[56] When Baumgartner et al. cultured the apical 5 mm of root canals exposed by caries, 67% were found to be strict anaerobes.[57] **A polymicrobial ecosystem seems to be produced that selects for anaerobic bacteria over time.** Gomes et al.[58,59] and Sundqvist[50,60] used odds ratios to show that some bacteria tend to be associated in endodontic infections. This suggests a **symbiotic relationship** that may lead to an increase in virulence by the organisms in that ecosystem. Clinicians may consider chemomechanical cleaning and shaping of the root canal system as total disruption of that microbial ecosystem.

Although no absolute correlation has been made between any species of bacteria and severity of endodontic infections, several species have been implicated with

some clinical signs and symptoms. Those species include BPB, *Peptostreptococcus*, *Peptococcus*, *Eubacterium*, *Fusobacterium*, and *Actinomyces*.[53,56,58,61–72] Table 3-1 shows the percentage of incidence of bacteria isolated from intact root canals from five combined studies.[53,56,73–75] Table 3-2 shows the taxonomic changes that have taken place with the bacteria formerly in the genus **Bacteroides**.

Studies of endodontically treated teeth requiring **re-treatment** have shown a prevalence of facultative bacteria, especially **Streptococcus faecalis,** instead of strict anaerobes.[76–80] In addition, fungi have been shown to be associated with failed root canal treatment.[1,80,81] Infection at the time of refilling and the size of the peri-

apical lesion were factors that had a negative influence on the prognosis for re-treatment.[80]

Black-pigmented bacteria have been associated with clinical signs and symptoms in several studies.[53,56,58,61,62,65–70,72] Unfortunately, taxonomic revision based on deoxyribonucleic acid (DNA) studies has made the interpretation of previous research results based on conventional identification of the bacteria at the very least confusing and in many cases impossible. Conventional identifications of microbes based on Gram stain, colonial morphology, growth characteristics, and biochemical tests are often inconclusive and yield presumptive identifications. Sundqvist described some of the taxonomic changes that have affected those species of bacteria often cultured from root canals.[82]

Previously, *Prevotella intermedia* was the species of BPB most commonly isolated from endodontic infections. In 1992, isolates previously thought to be *P. intermedia* were shown to be a closely related species now known as *P. nigrescens*.[83] Recent studies have demonstrated that *P. nigrescens* is actually the BPB

Table 3-1 Bacteria Cultured and Identified from the Root Canals of Teeth with Apical Radiolucencies

Bacteria	Incidence (%)
Fusobacterium nucleatum	48
Streptococcus sp	40
Bacteroides sp*	35
Prevotella intermedia	34
Peptostreptococcus micros	34
Eubacterium alactolyticum	34
Peptostreptococcus anaerobius	31
Lactobacillus sp	32
Eubacterium lentum	31
Fusobacterium sp	29
Campylobacter sp	25
Peptostreptococcus sp	15
Actinomyces sp	15
Eubacterium timidum	11
Capnocytophaga ochracea	11
Eubacterium brachy	9
Selenomonas sputigena	9
Veillonella parvula	9
Porphyromonas endodontalis	9
Prevotella buccae	9
Prevotella oralis	
Proprionibacterium propionicum	8
Prevotella denticola	6
Prevotella loescheii	6
Eubacterium nodatum	6

*Nonpigmenting species.
Other species isolated in low incidence included *Porphyromonas gingivalis, Bacteroides ureolyticus, Bacteroides gracilis, Lactobacillus minutus, Lactobacillus catenaforme, Enterococcus faecalis, Peptostreptococcus prevotii, Eienella corrodens*, and *Enterobacter agglomerans*.
Adapted from Sundqvist G.[82]

Table 3-2 Recent Taxonomic Changes for Previous Bacteroides Species

Porphyromonas—black-pigmented (asaccharolytic *Bacteroides* species)
    Porphyromonas asaccharolyticas (usually nonoral)
    *Porphyromonas gingivalis**
    *Porphyromonas endodontalis**

Prevotella—black-pigmented (saccharolytic *Bacteroides* species)
    Prevotella melaninogenica
    Prevotella denticola
    Prevotella loescheii
    *Prevotella intermedia**
    Prevotella nigrescens[†]
    Prevotella corporis
    Prevotella tannerae

Prevotella—nonpigmented (saccharolytic *Bacteroides* species)
    *Prevotella buccae**
    Prevotella bivia
    Prevotella oralis
    Prevotella oris

 Prevotella oulorum

Prevotella ruminicola

*Studies have associated species with clinical signs and symptoms.
[†]Most commonly isolated species of black-pigmented bacteria from endodontic infections.

most commonly isolated from both root canals and periradicular abscesses of endodontic origin.[84–86] Another study associating BPB with endodontic infections found BPB in 55% of 40 intact teeth suffering necrotic pulps and apical periodontitis. Sixteen of the 22 teeth in the sample "were associated with purulent drainage or an associated sinus tract."[87] Future studies will likely use molecular methods to detect and identify the microbes using extracted DNA.

PULPAL PATHOGENESIS

Because of the polymicrobial nature of periodontal and endodontic disease, a modification of Koch's postulates has been recommended by Socransky.[88,89] This recommendation states that the humoral response to the organism should be suggestive of its role in the disease. Jontell et al. have demonstrated the presence of dendritic cells in the pulp that activate T lymphocytes, which, in turn, direct other immunocompetent cells to mount a local immune response.[90–92] Hahn and Falkler have shown the production by the pulp of immunoglobulin (Ig)G specific for bacteria in deep caries.[93] In addition, they found an increase in the ratio of T helper lymphocytes and B lymphocytes to T suppressor cells in response to approaching caries.[93] In general, the presence of a mononuclear cell infiltrate (lymphocytes, macrophages, and plasma cells) is indicative of an immune response. Bacterial antigens activate both T and B cells. This response may be stimulated by viable bacteria or soluble bacterial components. Lipopolysaccharides cause polyclonal stimulation of B cells and induce macrophage activation.

PERIRADICULAR INFECTIONS

Today we know that serious odontogenic infections, beyond the tooth socket, are much more common as a result of endodontic infections than as a result of periodontal disease.[94] The seriousness of an infection beyond the apex of a tooth depends on the number and virulence of the organisms, host resistance, and anatomic structures associated with the infection. Once the infection has spread beyond the tooth socket, it may localize or continue to spread through the bone and soft tissue as a diffuse abscess or cellulitis.

The terms abscess and cellulitis are often used interchangeably in common clinical use. An **abscess** is a cavity containing pus (purulent exudate) consisting of bacteria, bacterial by-products, inflammatory cells, numerous lysed cells, and the contents of those cells. **Cellulitis** is a diffuse, erythematous, mucosal, or cutaneous infection that may rapidly spread into deep facial spaces and become life threatening. As a diffuse celluli-

tis matures, it may contain foci of pus consistent with an abscess. The relationship of specific species of bacteria or aggregates of bacteria with the pathogenesis of endodontic abscesses/cellulitis has not been established. Endodontic infections occur when opportunistic pathogens gain access to the normally sterile dental pulp and produce disease. Infections of the root canal system may spread to the contiguous periradicular tissues. Endodontic abscesses are invariably polymicrobial, and several strains of bacteria are cultured from each infection.[66,70,95–100]

The microorganisms identified in periradicular infections (abscesses) of endodontic origin are similar to bacteria isolated and identified from within the root canal system.[53,56,58,61–72] Only a few strains of bacteria isolated from oral abscesses will produce an abscess in pure culture.[17,101–105] A recent study showed that *Fusobacterium nucleatum, Peptostreptococcus anaerobius,* and *Veillonella parvula,* but not any strains of BPB could produce abscesses in pure culture in a mouse model.[101] **In mixed culture** with *F. nucleatum,* the BPB *Prevotella intermedia* and **Porphyromonas gingivalis** were significantly more abscessogenic than *F. nucleatum* in pure culture.[101] This supports the concept of **synergistic relationships between bacteria in an endodontic infection.** *Porphyromonas gingivalis* has also been shown to express collagenase as a potential **virulence factor**. *Porphyromonas endododentalis,* however, does not appear to possess this same collagenase gene, *prtC.*[106]

Whether asymptomatic chronic apical periodontitis lesions (periapical granulomas) are sterile has been controversial since the beginning of the 1900s.[107–111] It was generally believed that bacteria usually stayed confined to the root canal system of an infected tooth except when associated with an abscess or cellulitis. It was believed that "a granuloma is not an area in which bacteria live, but in which they are destroyed.".[108] Since then, histologic studies have demonstrated intraradicular organisms, plaque-like material at the root apex, intracellular organisms in the body of the inflammatory lesions, and extracellular bacteria within the body of the lesions[1,112–114] (Figures 3-4 to 3-7).

In an elegant study by Nair using both light and electron microscopy, both intracellular and extracellular bacteria were observed within the body of four granulomas and one radicular cyst. Whereas these 5 teeth were **symptomatic** and clinically diagnosed as acute periapical inflammation, 25 other teeth that were **asymptomatic** did not have identifiable extracellular bacteria.[115]

Recently, several investigators have demonstrated the presence of bacteria by culturing lesions diagnosed
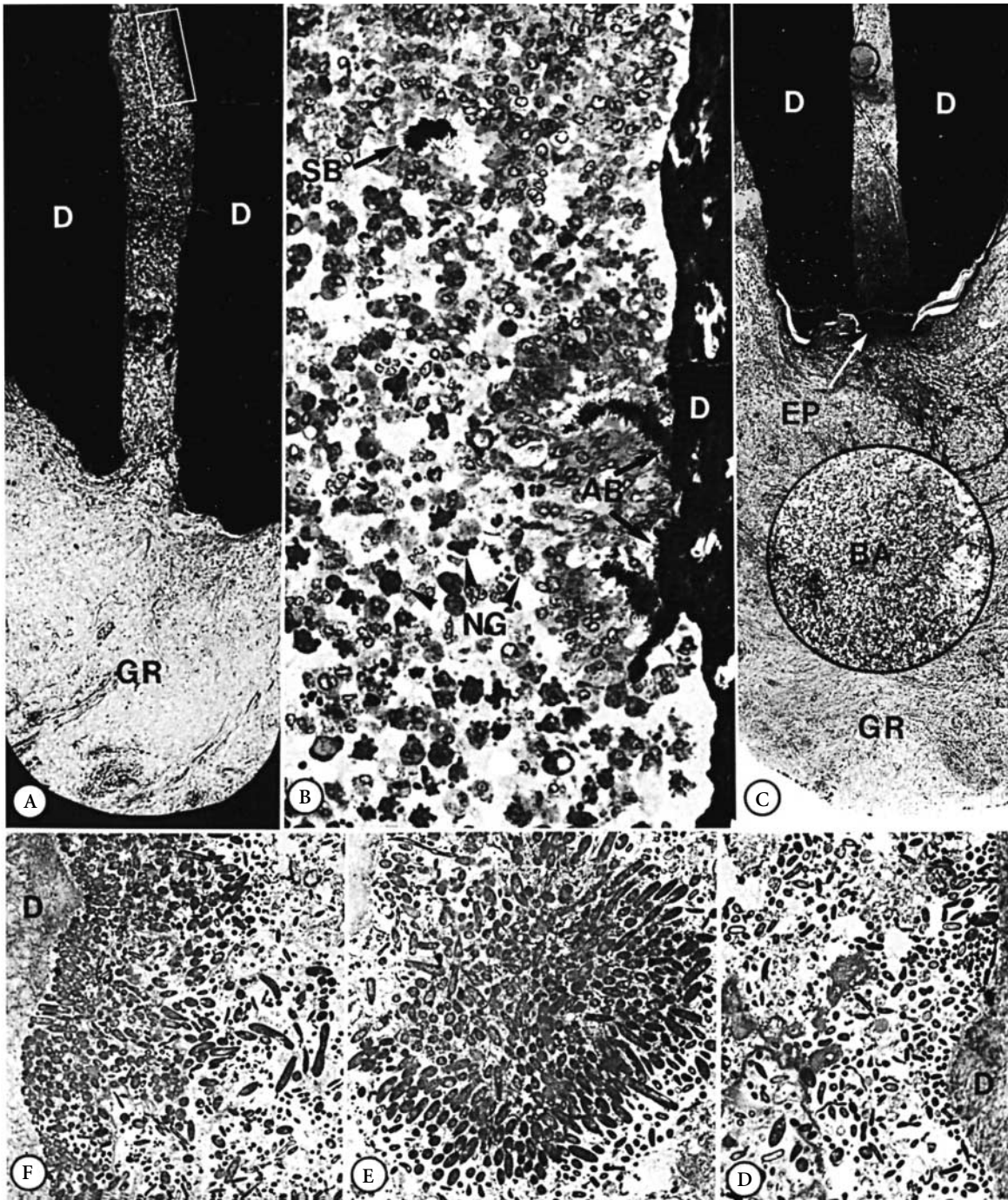
Figure 3-4 The endodontic flora in the radicular third of periradicularly affected human teeth. The flora appears to be blocked by a wall of neutrophils (NG in **B**) or by an epithelial plug (EP in **C**) in the apical foramen. Note the dense aggregates of bacteria sticking to the dentin wall (AB in **B**) and similar ones (SB in **B**) along with loose collections of bacteria (inset in **C**) remaining suspended in the root canal among neutrophils. A cluster of an apparently monobacterial colony is magnified in **D**. Electron micrographs show bacterial condensation on the surface of the dentin wall, forming thin (**E**)- or thick (**F**)-layered bacterial plaques. The rectangular demarcated portion in **A** and the circular one in **C** are magnified in **B** and the inset in **C**, respectively. GR = granuloma; D = dentin. Reproduced with permission from Nair PN.[115]
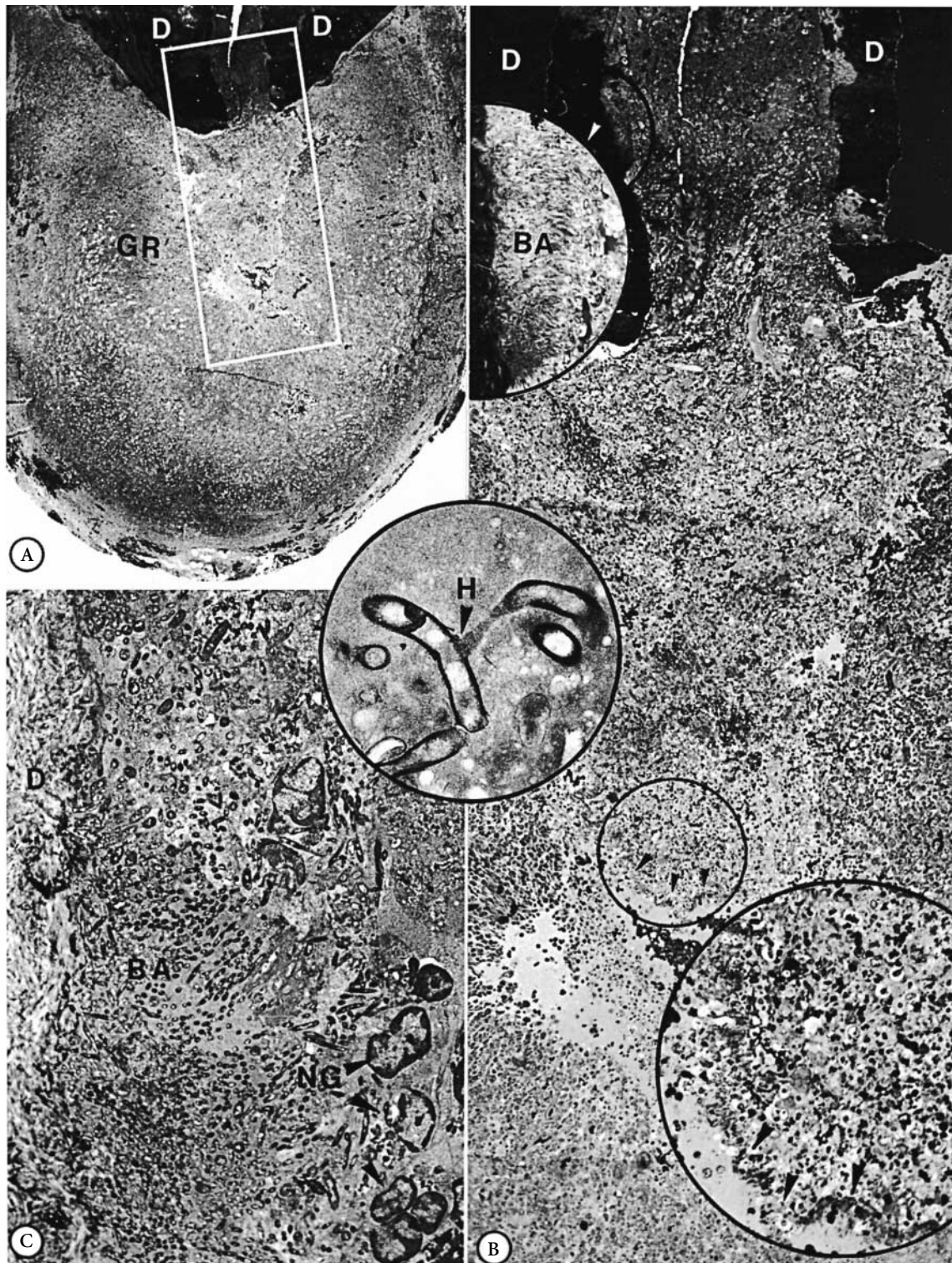
Figure 3-5   A radicular plaque invading a resting granuloma. (The rectangular demarcated area in **A** is magnified in **B**.) The well-encapsulated granuloma (GR in **A**) shows the bacterial front (**arrowheads** in **B** and lower inset) deep within the body of the lesion. Note the funnel-like area of tissue necrosis immediately in front of the apical foramen (**A** and **B**) and the plaque-like bacterial condensation (BA in **B** and upper inset) along the root dentin. This plaque is electron microscopically shown in **C**. The middle inset shows a high magnification of a branching or hyphal-like structure found among the plaque flora. D = dentin; NG = neutrophilic granulocytes. Reproduced with permission from Nair PN.[115]
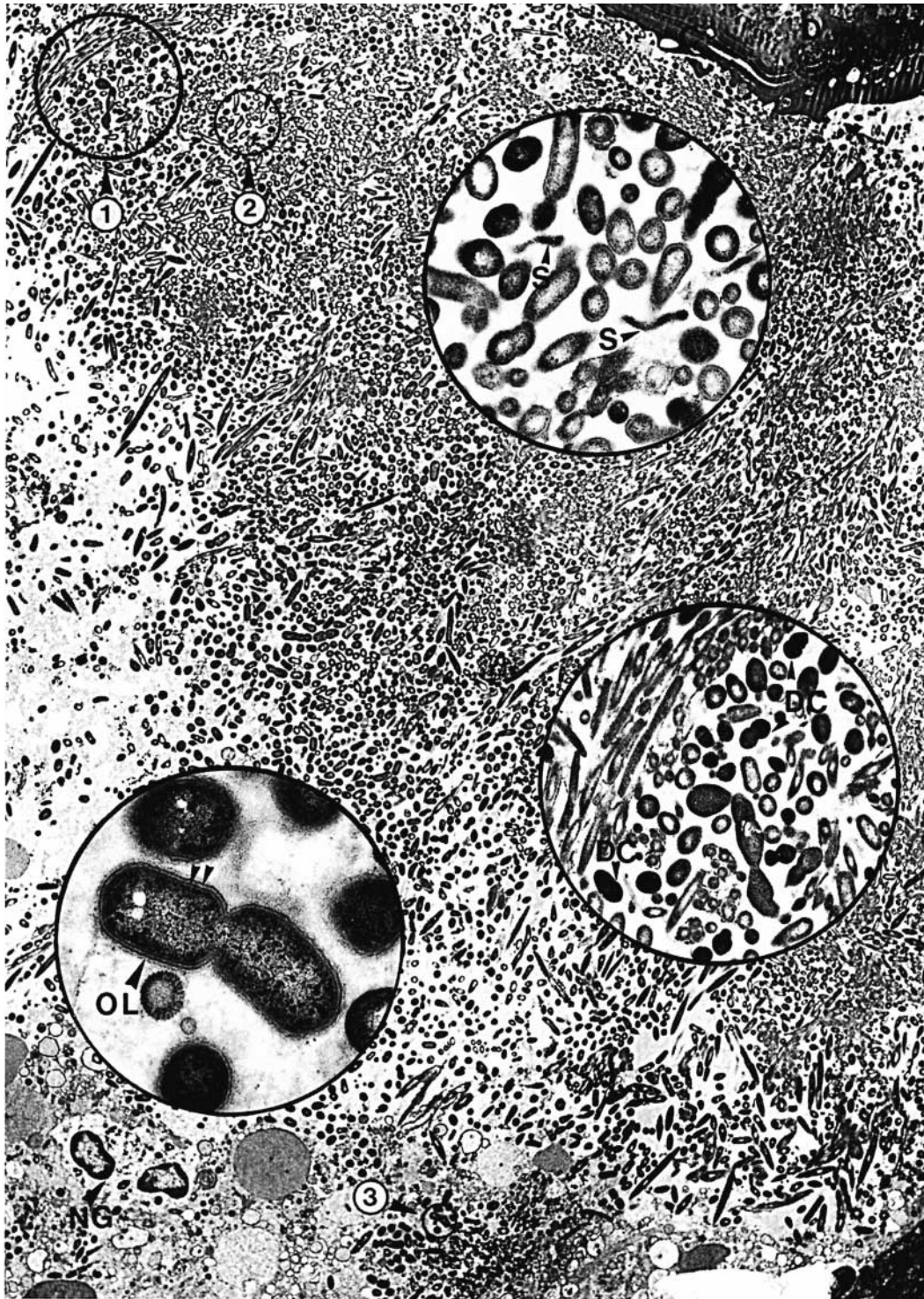
Figure 3-6   A massive periradicular plaque associated with an acute lesion. Note the mixed nature of the flora. Numerous dividing cocci (DC, middle inset), rods (lower inset), filamentous bacteria, and spirochetes (S, upper inset) can be seen. Rods often reveal a gram-negative cell wall (**double arrowhead**, lower inset), some of them showing a third outer layer (OL). The circular areas 1, 2, and 3 are magnified in the middle, upper, and lower insets, respectively. D = dentin; C = cementum; NG = neutrophils. Reproduced with permission from Nair PN.[115]
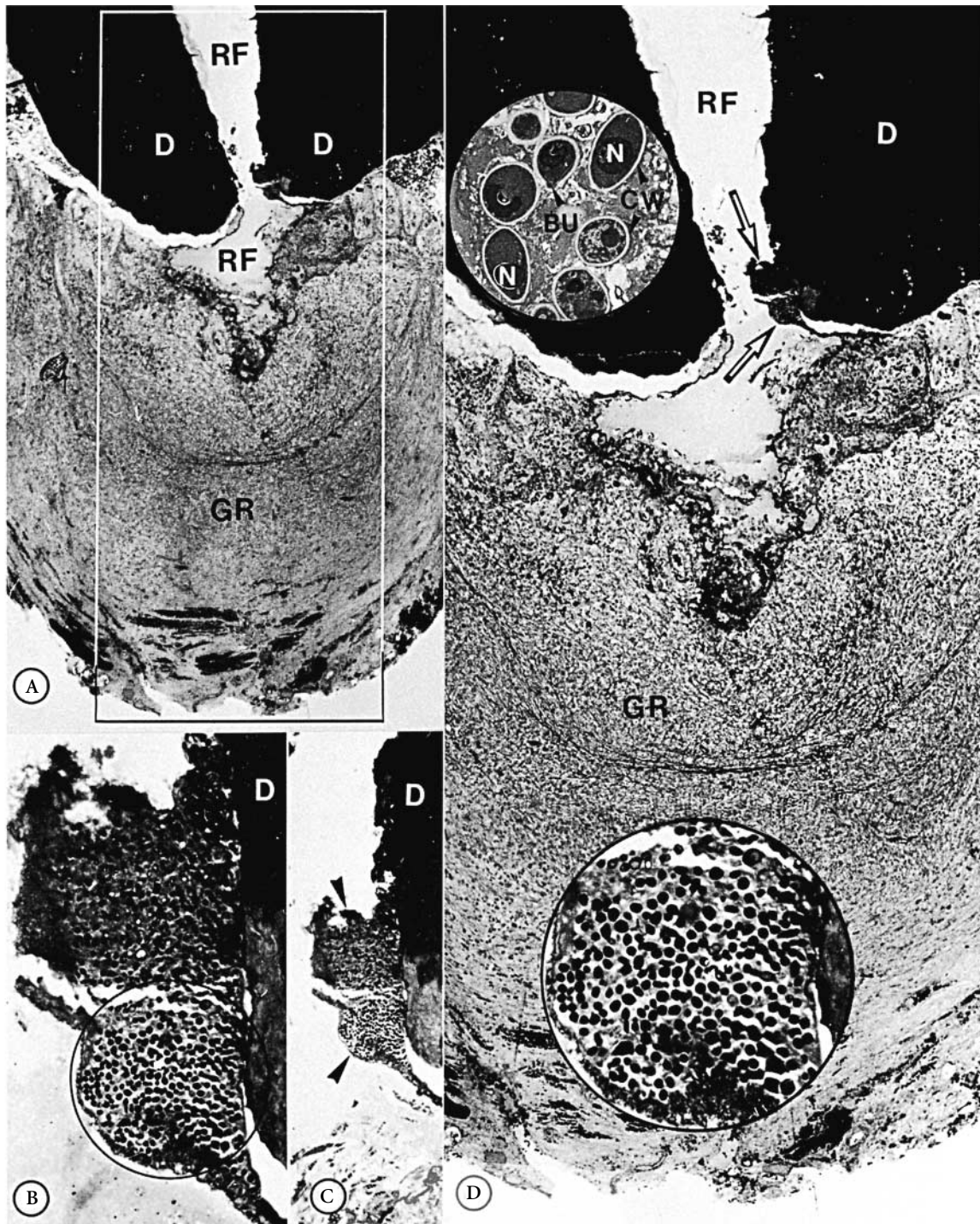
Figure 3-7   Presence of fungus in the root canal and apical foramen of a root-filled (RF in **A** and **D**) tooth with a therapy-resistant periradicular lesion (GR in **A** and **D**). The rectangular demarcated area in **A** is magnified in **D**. Note the two clusters of microorganisms located between the dentinal wall (D) and the root filling (**arrows** in **D**). Those microbial clusters are stepwise magnified in **C** and **B**. The circular demarcated area in **B** is further magnified in the lower inset in **D**. The upper inset is an electron microscopic view of the organisms. They are about 3 to 4 μm in diameter and reveal distinct cell wall (CW), nuclei (N), and budding forms (BU). Reproduced with permission from Nair PN.[1]

as chronic apical inflammation.[113,114,116,117] There is the possibility of microbial contamination from communication of the apical tissues with bacteria located in the apical foramen or the oral cavity or during a surgical procedure and collection of the microbial sample.

Depending on the host's resistance and the virulence of the bacteria, invasion of the periradicular tissues may occur from time to time. Perhaps asymptomatic periradicular inflammatory lesions (granulomas) may contain invading bacteria and even abscesses (microabscesses) not clinically detectable. If the opportunistic organism is successful in invading and establishing an infection, a clinically apparent abscess and possibly a cellulitis may develop (phoenix abscess). Further research is needed to clarify this aspect of endodontic infections.

PERIADICULAR PATHOGENESIS

Research has shown that periradicular inflammatory tissue is capable of an **immunologic response to bacteria**. Studies using an enzyme-linked immunosorbent assay (ELISA), radioimmunosorbent tests, and radial immunodiffusion assays have detected IgG, IgA, IgM, or IgE in fluids of explant (tissue) cultures of endodontic periapical lesions.[118–122] A DOT-ELISA was used to show that BPB (*P. intermedia, P. endodontalis,* and *P. gingivalis*) were the bacteria most reactive with IgG produced by explant cultures of periapical lesions.[120] An ELISA has also been used to show an increase in serum IgG reactive with *P. intermedia* in patients with periodontal disease or combined endodontic-periodontal disease.[123] Recently, exudates from root canals associated with symptomatic periapical lesions were shown to contain higher concentrations of β-glucuronidase and interleukin-1β.[124] Those with severe involvement had higher IgG, and those with a sinus tract or swelling contained higher concentrations of IgM.

Numerous studies have quantitatively analyzed the **lymphocytes** and their subsets in periapical lesions.[125–134] Periapical lesions associated with untreated teeth have a denser inflammatory cell infiltrate than periapical lesions associated with treated teeth.[135] No associations were seen between the histologic diagnosis, clinical signs and symptoms, or radiographic size of the lesions. Most studies have shown that the majority of **lymphocytes** in periapical lesions associated with untreated teeth are **T cells**. However, with treated teeth, Alavi et al. found that half of all inflammatory lesions associated with endodontically treated teeth had more B than T cells.[135] In a rat model, Stashenko and Wang showed that T helper cells outnumber T suppressor cells during lesion expansion up

to 15 days.[136] After 15 days, the lesion expansion slows, and T suppressor cells outnumber T helper cells. They believe that T helper–mediated activities may involve bone destruction and lesion expansion. Others believe that lymphocyte proportion may shift in response to population shifts in microorganisms.[55,60,137]

The periapical inflammatory responses that occur following bacterial infection of the root canal system result in the formation of granulomas and cysts with the resorption of surrounding bone. **Interleukin-1** and **prostaglandins** have been especially associated with periapical bone resorption. Research is showing that these inflammatory responses are very complex and consist of several diverse elements.[138] Prostanoids, kinins, and neuropeptides are endogenous mediators responsible for intermediate-type responses that include vasodilatation, increased vascular permeability, and leukocyte extravasation. Bacteria and their by-products produce nonspecific immune responses including neutrophil and monocyte migration/activation and cytokine production. Chronic apical periodontitis also involves specific T lymphocyte– and B lymphocyte–mediated antibacterial responses. Figure 3-8 shows some of the interactions believed to be associated with bone resorption.

TREATMENT OF ENDODONTIC ABSCESSES/CELLULITIS

The vast majority of infections of endodontic origin can be effectively managed without the use of antibiotics. Systemically administered antibiotics are not a substitute for proper endodontic treatment. Chemomechanical débridement of the infected root canal system with **drainage through the root canal** or by incision and drainage of soft tissue will decrease the bioburden so that a normal healthy patient can begin the healing process. Antibiotics are not recommended for healthy patients with a symptomatic pulpitis, symptomatic apical periodontitis, draining sinus tract, or localized swelling of endodontic origin or following endodontic surgery.[139,140]

An **antibiotic regimen** should be **prescribed** in conjunction with proper endodontic therapy when there are systemic signs and symptoms or a progressive/persistent spread of infection. The presence of a fever (>100°F), malaise, cellulitis, unexplained trismus, and progressive swelling are all signs and symptoms of systemic involvement and the spread of infection (Table 3-3). Under these circumstances, an antibiotic is indicated in addition to débridement of the root canal harboring the infection and drainage of any accumulated purulence.
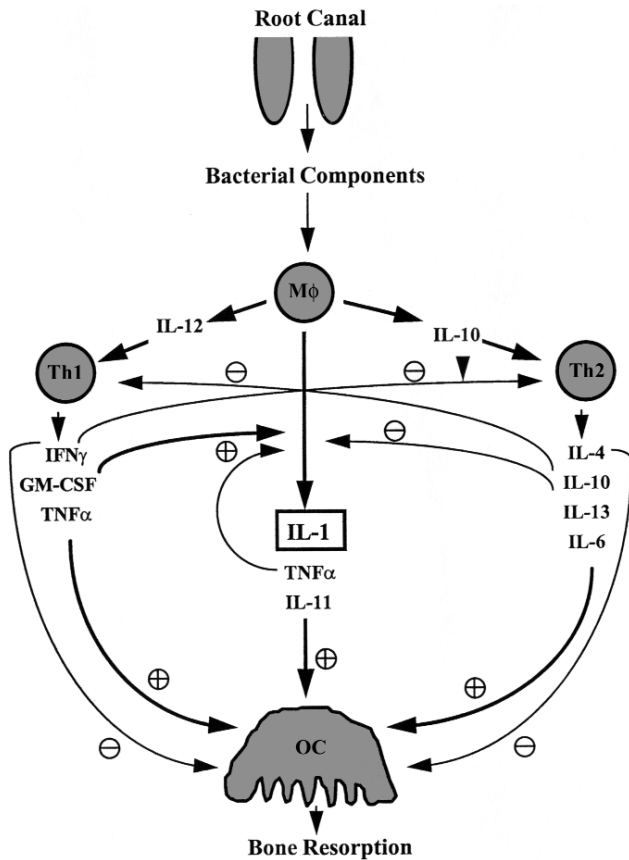
Figure 3-8 Regulation of periapical bone destruction by the cytokine network. GM-CSF = granulocyte-macrophage colony-stimulating factor; TNFa = tumor necrosis factor alpha; IL = interleukin; Th1, Th2 = T helper cell subsets; IFN = interferon; Mφ = macrophage; OC = osteoclast. Heavy lines = stimulation; light lines = inhibition. Reproduced with permission from Stashenko P and Wang SM.[136]

Patients with serious endodontic infections should be closely followed on a daily basis. The patient's condition will usually rapidly improve once the source of the infection is removed. Because of the lack of circulation, systemically administered antibiotics are not effective against a reservoir of microorganisms within an infected root canal system. Likewise, a minimum inhibitory concentration of an antibiotic may not reach a space filled with pus because of poor circulation. The antibiotic moves via a diffusion gradient through the edematous fluid and purulent exudate that accumulates in an anatomic space. **Pus** contains mainly neutrophils with some other inflammatory cells, cellular debris, bacteria, bacterial by-products, enzymes, and edematous fluid. An **incision for drainage** will allow drainage of the purulent material and improve circulation to the area.

Empirical selection of an antibiotic (antimicrobial agent) must be based on one's knowledge of which bacteria are most commonly associated with endodontic infections and their antibiotic susceptibility.[139–143] The clinician must be thoroughly familiar with the antibiotic and inform the patient of the benefits, possible side effects, and possible sequelae of failing to take the proper dosage. The antibiotic should generally be continued for 2 to 3 days following resolution of the major clinical signs and symptoms of the infection. Following treatment of the source of the infection and adjunctive antibiotic therapy, significant improvement in the patient's status should be seen in 24 to 48 hours. A loading dose is important to provide an initial adequate therapeutic level of antibiotic. An adequate maintenance dose is recommended to prevent the selection of resistant bacteria.

Penicillin VK is the antibiotic of choice because of its effectiveness against both facultative and anaerobic microorganisms commonly found in polymicrobial endodontic infections.[141–144] However, up to 10% of the population may be allergic, so a careful history of drug hypersensitivity is important. **Amoxicillin** has an increased spectrum of activity that includes bacteria not routinely associated with infections of endodontic origin.

Erythromycin has traditionally remained the alternative choice for patients allergic to penicillin, but it is **not effective against anaerobes** associated with endodontic infections. **Clarithromycin** and **azithromycin** are macrolides like erythromycin, with some advantages over the latter. They have a spectrum of antimicrobial activity that includes facultative bacteria and some anaerobic bacteria associated with infections of endodontic origin. They also have less gastrointestinal upset than erythromycin.

Table 3-3   Indications for Adjunctive Antibiotics (Antimicrobial Therapy)

Systemic involvement
Fever > 100°F
Malaise
Lymphadenopathy
Trismus
Progressive infections
Increasing swelling
Cellulitis
Osteomyelitis
Persistent infections

For serious infections when the patient is allergic to penicillin, **clindamycin** is effective against both facultative and strict anaerobic bacteria associated with endodontic infections. It is well distributed throughout the body, especially to bone, where its concentration approaches that of plasma.

Metronidazole is a synthetic antimicrobial agent with **excellent activity against anaerobic bacteria**; however, it is ineffective against facultative bacteria. It is a valuable antimicrobial agent in combination with penicillin when penicillin alone has been ineffective.

When antibiotics are prescribed in conjunction with débridement of the root canal system and drainage of purulence, significant improvement should be seen within 24 to 48 hours. If the infection is not resolving, the diagnosis and initial treatment should be reviewed. If another source of the infection is not found or if additional attempts for drainage are unsuccessful, the **addition of metronidazole (250 mg every 6 hours) to penicillin is indicated**. Because metronidazole is effective only against anaerobic bacteria, penicillin should be continued to treat any facultative bacteria present.

For a more detailed discussion of the role of antibiotics in endodontics, the reader is referred to chapter 18.

PROPHYLACTIC ANTIBIOTICS FOR MEDICALLY COMPROMISED PATIENTS

Prophylactic antibiotic coverage may be indicated for medically compromised patients requiring endodontic treatment. The American Heart Association (AHA) has made major changes in their updated recommendations.[145] Their guidelines are meant to aid practitioners but are not intended as the standard of care or as a substitute for clinical judgment. The incidence of endocarditis following most procedures on patients with underlying cardiac disease is low. A reasonable approach for prescribing prophylactic antibiotics considers the degree to which the underlying disease creates a risk for endocarditis, the apparent risk for producing a bacteremia, adverse reactions to the prophylactic antibiotic, and the cost-benefit aspect of the regimen.[145]

The incidence of bacteremia has been shown to be low during root canal therapy; however, a transient bacteremia can result from the extrusion of the microorganisms infecting the root canal beyond the apex of the tooth.[11,12,14] In addition, care must be taken when positioning rubber dam clamps and accomplishing other dental procedures that may produce bleeding with an accompanying bacteremia. Medically compromised dental patients who are at risk of infection should receive a regimen of antibiotics that either follows the recommendations of the AHA or an alternate regimen determined in consultation with the patients' physicians.[145] Table 3-4 gives the antibiotic regimens recommended for dental procedures.[145] It is believed that the antibiotics **amoxicillin, ampicillin, and penicillin V are equally effective against alpha-hemolytic streptococci; however, amoxicillin is recommended** because it is better absorbed from the gastrointestinal tract and provides higher and more sustained serum levels.[145]

Table 3-4   Prophylactic Regimens for Dental Procedures

Situation	Agent	Regimen
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g; children: 50 mg/kg orally 1 h before procedure
Unable to take oral medications	Ampicillin	Adults: 2.0 g IM, or IV; children: 50 mg/kg IM or IV 30 min before procedure
Allergic to penicillin	Clindamycin	Adults: 600 mg; children: 20 mg/kg orally 1 hr before procedure
	or cephalexin or cefadroxil	Adults: 2.0 g; children: 50 mg/kg orally 1 h before procedure
	Azithromycin or clarithromycin	Adults: 500 mg; children: 15 mg/kg orally 1 h before procedure
Allergic to penicillin and unable to take oral medications	Clindamycin	Adults: 600 mg; children 20 mg/kg IV within 30 min before procedure
	Cefazolin	Adults: 1.0 g; children: 25 mg/kg IM or IV within 30 min before procedure

From Dajani A et al.[145]

For more complete details concerning antibiotic prophylaxis, the reader is referred to chapter 18 and to the reports by Strom et al.[146] and Durack.[147]

COLLECTION OF A MICROBIAL SAMPLE

Adjunctive antibiotic therapy for endodontic infections is most often prescribed empirically based on our knowledge of the bacteria most often associated with endodontic infections. At times, culturing may provide valuable information to better select the appropriate antibiotic regimen. For example, an immunocompromised/immunosuppressed patient (not immunocompetent) or patients at high risk of developing an infection (eg, history of infective endocarditis) following a bacteremia require close monitoring. These patients may have an infection caused by bacteria usually not associated with the oral cavity. Other examples include a seemingly healthy patient who has persistent or progressive symptoms following surgical or nonsurgical endodontic treatment.

An aseptic microbial sample from a root canal is accomplished by first isolating the tooth with a rubber dam and disinfecting the tooth surface and rubber dam with sodium hypochlorite or other disinfectant. Sterile burs and instruments must be used to gain access to the root canal system. Antimicrobial solutions should not be used until after the microbial sample has been taken. If there is drainage from the canal, it may be sampled with a sterile paper point or aspirated into a syringe with a sterile 18- to 25-gauge needle. Any aspirated air should be vented from the syringe into a sterile gauze. The aspirate should either be taken immediately to a microbiology laboratory in the syringe or injected into pre-reduced transport media. To sample a dry root canal, a sterile syringe should be used to place some pre-reduced transport medium into the canal. A sterile endodontic instrument is then used to scrape the walls of the canal to suspend microorganisms in the medium.

To prevent contamination by "normal oral flora," a microbial sample from a soft tissue swelling should be obtained before making an incision for drainage. Once profound anesthesia is achieved, the surface of the mucosa should be dried and disinfected with an iodophor swab (The Purdue Frederick Company, Norwalk, Conn.). A sterile 16- to 20-gauge needle is then used to aspirate the exudate. The aspirate should then be handled as described above. If purulence cannot be aspirated, a sample can be collected on a swab after the incision for drainage has been made, but great care must be taken to prevent microbial contamination with normal oral flora. After collecting the specimen on a swab, it should be quickly placed in pre-reduced medium for transport to the laboratory.

Good communication with the laboratory personnel is important. The sample should be Gram-stained to demonstrate which types of microorganisms predominate. The culture results should show the prominent isolated microorganisms and not just be identified as "normal oral flora." Antibiotics can usually be chosen to treat endodontic infections based on the identification of the prominent microorganisms in the culture. With persistent infections, susceptibility testing can be undertaken to establish which antibiotics are the most effective against resistant microbial isolates. At present, it may take 1 to 2 weeks to identify anaerobes. In the future, molecular methods will be used to rapidly detect and identify known opportunistic bacteria.

ROOT CANAL DÉBRIDEMENT AND INTRACANAL MEDICATION

The goal of clinical treatment is to completely disrupt and destroy the bacteria involved in the endodontic infection. Endodontic disease will persist until the source of the irritation is removed. The microbial ecosystem in an infected root canal has been directly linked to both acute and chronic inflammation.[31,138]

Root Canal Débridement

Root canal débridement includes the removal of the microorganisms and their substrates required for growth. Chemomechanical cleaning and shaping of the root canal system remove a great deal of the irritants, but total débridement is impeded because of the complex root canal systems with accessory canals, fins, cul-de-sacs, and communications between the main canals. The last decade has seen the development and use of several innovative methods and materials to aid in root canal débridement. The ability of nickel-titanium instruments to remain centered in canals has facilitated the use of the step-down method of instrumentation without significant concern for ledge formation or canal transportation.[148,149] In addition, the step-down method removes debris as progress is made toward the apex, so irritating debris is not carried apically and extruded into the periapical tissues.[150] The step-down method also enlarges the coronal portions of the canal so that there is a larger reservoir for an irrigant. Numerous irrigants have been used and studied, but sodium hypochlorite (0.5 to 5.25%) remains the most popular irrigant in the United States. Sodium hypochlorite, in concentrations of 0.5 to 5.25%, has the ability to dissolve organic pulpal debris in areas not reached by endodontic instruments.[151–155] It is also an excellent antimicrobial.[73,75,156]

When sodium hypochlorite is alternated as an irrigant with 15% ethylenediaminetetraacetic acid (EDTA), both the instrumented and the noninstrumented surfaces of a root canal are chemically débrided.[157] Sodium hypochlorite reacts with organic tissue to facilitate cleaning; however, this reaction inactivates the agent and decreases its antibacterial capacity. Thus the **irrigant in the canal should be frequently replenished** to maintain the most optimum activity of sodium hypochlorite. Both 0.5% and 5% sodium hypochlorite have been shown to be effective antimicrobials in clinical studies.[158,159] However, **5% sodium hypochlorite is more effective than 0.5 sodium hypochlorite as a solvent of necrotic tissue.**[160] Research has shown that the combined use of 15% EDTA and 5.25% sodium hypochlorite was more efficient as an antimicrobial than 5.25% NaOCl by itself for irrigating infected root canals.[158] The irrigants must be passively introduced into the canal without wedging the needle and inadvertently infusing the irrigant into the periapical tissues, where they will produce pain and tissue injury.[161,162] Use of a needle with a slot at the tip or side opening helps to prevent wedging of the needle. Sonic and ultrasonic devices may be used to improve the efficacy of irrigation.[163–167]

Intracanal Antisepsis

Residual microorganisms left in the root canal system following cleaning and shaping or microbial contamination of a root canal system between appointments have been a concern. If root canal treatment is not completed in a single appointment, antimicrobial agents are recommended for intracanal antisepsis to prevent the growth of microorganisms between appointments. The access opening in the tooth must also be sealed with an effective interappointment filling to prevent microbial contamination by microleakage from the oral cavity. Despite the controversy over culturing root canals, most clinicians agree that healing is more likely in the absence of bacteria.[168–170]

A recent study used modern microbiologic techniques, with teeth root-filled at a single appointment and evaluated for clinical success.[76] Initially, all 55 single-rooted teeth were infected. After instrumentation and irrigation with 0.5% sodium hypochlorite, bacteria could still be cultivated from 22 of the 55 root canals. Periapical healing was followed for up to 5 years. Complete healing occurred in 94% of those teeth that had negative cultures but only 68% of those with positive cultures at the time of root canal obturation.[76] These findings suggest the importance of eliminating bacteria from the root canal system before obturation.

In the past, numerous antimicrobial agents have been used that were antigenic and cytotoxic and provided relatively short-term antisepsis.[171–175] These included traditional phenolic and fixative agents such as camphorated monochlorophenol, formocresol, eugenol, metacresylacetate, and halides (iodine-potassium iodide). A reliance on mechanical instrumentation and aversion to the use of cytotoxic chemicals have led to a lack of use of an intracanal dressing by many clinicians, a practice that allows remaining bacteria to multiply between appointments.

The current **intracanal dressing of choice is calcium hydroxide**. Although not characterized as an antiseptic, studies have shown calcium hydroxide to be an effective antimicrobial agent.[158,176–180] Other studies have shown it to be an effective interappointment dressing over several weeks.[181,182] When mixed into a paste with water, calcium hydroxide's solubility is less than 0.2%, with a pH of about 12.5. Some of its antibacterial activity may be related to the absorption of carbon dioxide that starves capnophilic bacteria in the root canal.[183] The Saunders group in Dundee was disappointed, however, in the lack of antibacterial activity of calcium hydroxide against the anaerobes *P. gingivalis* and *Peptostreptococcus micros*.[184]

On the other hand, calcium hydroxide has been shown to hydrolyze the lipid moiety of bacterial lipopolysaccharides, making them incapable of producing such biologic effects as toxicity, pyrogenicity, macrophage activation, and complement activation.[177] Lipopolysaccharides have been shown to be present in the dentinal tubules of infected root canals.[18,185] Obliterating the canal space with calcium hydroxide, during treatment, may minimize the ingress of tissue fluid used as a nutrient by microorganisms.[186] Removal of the smear layer facilitates the diffusion of calcium hydroxide into the dentinal tubules.[187] But smear layer or not, a Brazilian group was disappointed in the inability of calcium hydroxide to destroy bacteria in infected dentinal tubules,[188] whereas four root canal sealers appeared to be quite effective against tubuli bacteria, AH-26 being the best.[189] Moreover, zinc oxide–eugenol sealer was found to be more effective in inhibiting the growth of *Streptococcus anginosus* than three of the calcium hydroxide–containing sealers.[190]

Actinomyces israelii, a species of bacteria isolated from periapical tissues, has been reported to not respond to conventional endodontic therapy.[63,191,192] Recently, however, both sodium hypochlorite and calcium hydroxide have been shown to be highly effective in killing *A. israelii*.[193] The optimal treatment of **periapical actinomycosis** is endodontic surgery that removes the likely cause, enables microscopic confirmation, and

has a high chance of success without prescribing antibiotics.[63,191,193] In classic forms of actinomycosis involving invasion and spread of *A. israelii* in the periradicular tissues, antibiotic treatment is justified. When actinomycosis cannot be controlled by surgery, **antibiotic therapy is justified** and optimized by prescribing for an extended period of 6 weeks with **amoxicillin or cephalexin**.[193]

Calcium hydroxide has been shown to have some efficacy in the dissolution of pulp tissue in vitro and may increase the ability of sodium hypochlorite to dissolve remaining organic tissue at subsequent appointments.[160,194] The tissue-dissolving property seems to work equally well in aerobic and anaerobic environments.[195] Some commercial preparations of calcium hydroxide come packaged in syringes, or the powder may be mixed with water or glycerin to form a thick paste. The paste is carried into the pulp chamber with a plastic instrument, amalgam carrier, or syringe and then carried down the canals using a lentulo, prefitted pluggers, or counterclockwise rotation of endodontic files. Calcium hydroxide is easily removed from the canal system at the next appointment using endodontic files and irrigation.

When exposed to carbon dioxide in an open container, some calcium hydroxide is slowly converted into inactive calcium carbonate. In a closed container, it is quite stable, with only 1 to 2% being converted after several months.[196] A good temporary filling that is several millimeters thick to prevent microleakage is important between appointments.[197–201] Calcium hydroxide has also been shown to decrease the amount of microbial contamination under temporary fillings.[202]

Another root canal medicament has more recently been introduced in Germany, a liquid medication known as camphorated chloroxylenol (ED84), which is claimed to be as effective as a "temporary root canal dressing for a duration of 2 days" and to be nontoxic to tissue.[203]

ASEPSIS IN ENDODONTIC PRACTICE

Endodontics has long emphasized the importance of aseptic techniques using sterilized instruments, disinfecting solutions such as sodium hypochlorite, and rubber dam barriers. In the past decade, numerous articles have been written regarding the exposure of dental personnel to infectious diseases.[204–209] In 1979, Crawford discussed guidelines for contamination control with respect to sterilization and disinfection in endodontic practice.[204] More recently, further recommendations have been made to prevent transmission of infectious diseases.[205–209] Interestingly, the basic tenets still apply today, but with many additions. The list of

identified risks to health care professionals has increased tremendously. The Occupational Safety and Health Administration (OSHA) regulations have had a profound impact on the practice of dentistry.

Traditionally, hepatitis B has been the benchmark disease on which infection control has been based.[205] In an office that treats approximately 20 patients per day, the personnel can expect to encounter 1 active carrier of hepatitis B virus (HBV) every 7 working days. In addition, one can expect exposure to 2 patients with oral herpes and an unknown number of patients infected with human immunodeficiency virus (HIV). It is generally accepted that the potential for HBV transmission in the dental environment is greater than that for HIV.[210] Immediate exposure is one critical factor, but HBV and tubercle bacilli have been shown to survive on inanimate surfaces beyond 7 days, thus illustrating the longevity of the pathogens. Hepatitis B virus is also highly infectious, with as little as 0.00001 mL of contaminated blood capable of transmitting the disease. Human immunodeficiency virus has been recovered from 1 to 3 days after drying under certain conditions.[211]

Human immunodeficiency virus and HBV infections have raised the concern of the profession and the public alike. Health care workers worry about acquiring HIV from patients, and patients worry about being exposed to diseases in dental offices. Much attention has been aroused by the highly publicized case in Florida in which a dentist may have infected at least five of his patients before he himself died from acquired immune deficiency syndrome (AIDS).[212]

The transmission route of HIV/HBV in this two-way street is primarily through the exchange of blood. Percutaneous injury to dentists is the most direct patient-to-dentist transmission method. Infected dentists, in turn, can then unknowingly infect other patients.[213]

Percutaneous injuries to dentists are caused by burs (37%), syringe needles (30%), sharp instruments (21%), orthodontic wires (6%), suture needles (3%), scalpel blades (1%), and other objects (2%). In recent years, however, needlestick injuries have dropped dramatically.

Oral surgeons suffer the highest percutaneous injury rate and endodontists the lowest. The average dentist performs about 3,000 invasive procedures a year—37% of all procedures. The percutaneous injury rate ranges from 3.16 (general practice) to 3.43 (specialties) "sticks" per year, any one of which could be disastrous.[213] Proper sterilization and infection control procedures in dental offices have become important issues for the public, the dental profession, and government agencies such as OSHA.

INFECTION CONTROL

The basic theorems of asepsis in general dentistry apply to endodontics with little variance. Figure 3-9 illustrates the major aspects of infection control in the dental environment. Each of these areas is reviewed in this chapter.

Objectives

In the development of any program, including one for contamination control, certain goals should be formulated. The American Dental Association (ADA) Council on Dental Therapeutics has recommended the following[206]:

1. Decrease the number of pathogenic microbes to the level where normal body resistance mechanisms can prevent infection.
2. Break the cycle of infection from dentist, assistant, and patient and eliminate cross-contamination.
3. Treat all patients and instruments as though they could transmit an infectious disease.
4. Protect patients and personnel from infection and protect all dental personnel from the threat of malpractice.

Even though these are general objectives, they provide a framework for the development of a contamination control program.

Terminology

The following terms apply to the topic of infection control:

1. **Sterilization:** The process that **destroys** all types and forms of microorganisms, including viruses, bacteria, fungi, and bacterial endospores. Major methods of sterilization include steam autoclave, dry heat, chemical vapor under pressure, ethylene oxide gas, and immersion in liquid chemical disinfectants/ sterilizers.

Figure 3-9 Major aspects of infection control in dentistry. (Courtesy of Dr. James A. Cottone, University of Texas Health Science Center at San Antonio, Texas.)

2. **Disinfection:** A process that is less lethal than sterilization. Three levels of disinfection are differentiated, depending on the type and form of microorganism destroyed.
   - **High-level disinfection:** A process that can kill some, but not necessarily all, bacterial spores. It is tuberculocidal, and if the disinfectant is capable of destroying bacterial spores, it is labeled sporicidal.
   - **Intermediate-level disinfection:** A process that is capable of killing *Mycobacterium tuberculosis,* HBV, and HIV. It may not be capable of killing bacterial spores.
   - **Low-level disinfection.** A process that kills most bacteria, some fungi, and some viruses. It does not kill *M. tuberculosis* or bacterial spores.
3. **Bactericidal:** A process or an agent that destroys (kills) bacteria.
4. **Bacteriostatic:** A process or an agent that inhibits growth or multiplication of bacteria.
5. **Contamination:** The introduction of an infectious agent into an area.
6. **Biomedical waste:** Generally any waste that is generated or has been used in the diagnosis, treatment, or immunization of human beings or animals, in research pertaining thereto, or in producing or testing a biologic agent, or that may contain infectious agents and may pose a substantial threat to health. This does not include hazardous waste.[214]
7. **Biohazardous waste:** Depending on regional regulations, this may include or exclude any of the following[214]:
   - **Laboratory waste,** including specimen cultures from medical and pathologic laboratories, culture dishes, and dishes and devices used to transfer, inoculate, and mix cultures or material that may contain infectious agents and may pose a substantial threat to health. All nonsterilized cultures are presumed biohazardous.
   - **Specimens** sent to a laboratory for microbiologic analysis are presumed biohazardous.
   - **Surgical specimens,** including human or animal parts or tissues removed surgically or by autopsy, are presumed biohazardous.
   - **Recognizable fluid and blood elements** and regulated body fluids and containers and articles contaminated with blood elements or regulated body fluids.
   - **Sharps,** including all objects or devices having acute rigid corners, edges, or protuberances capable of cutting or piercing and including, but not limited to, hypodermic needles, blades, and slides. [This would be likely to include endodontic instruments.]
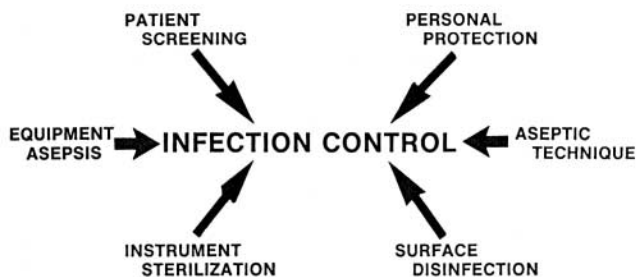
8. **Medical solid waste:** Empty specimen containers, bandages or dressings containing nonliquid blood, surgical gloves, treated biohazardous waste, and other materials that are not biohazardous.[214]

PATIENT EVALUATION

The identification of patients with transmissible diseases and of those belonging to high-risk groups is essential before treatment begins.[205,207,208,215] The Ad Hoc Committee on Infectious Diseases of the American Association of Public Health Dentists has listed diseases of concern to dental personnel[207] (Table 3-5). Populations at high risk of contracting hepatitis B are listed in Table 3-6. According to the Centers for Disease Control and Prevention (CDC), however, because the medical history and examination cannot reliably identify all patients with bloodborne pathogens, blood and body fluid precautions should be consistently used for all patients.[210] The concept stresses that all patients should be assumed to be infectious for HIV and other bloodborne pathogens.[216] Unfortunately, the medical history is only an adjunct to the patient's background and cannot be considered a totally inclusive source of information.

In the daily practice of endodontics, one must **frequently re-evaluate** the patient's medical history, at least on a yearly basis. With the recent advances in the treatment of medically compromised patients, a greater number of patients will enter the office with immunocompromised conditions, cardiovascular susceptibilities, and a host of other physical limitations that may require special attention. Consultation with attending physicians is most important in proper care of such patients.

CLASSIFICATION OF INSTRUMENT STERILIZATION

Spaulding's classification for instruments has been cited as a methodology for instrument sterilization.[217] The categorization of instruments depends on the contact with different tissue types to determine whether sterilization or disinfection is required. The categories are as follows:

1. **Critical items:** Instruments that touch sterile areas of the body or enter the vascular system and those that penetrate the oral mucosa. Examples are scalpels, curettes, burs, and files. Because of their potential for harboring microorganisms, dental handpieces also must be sterilized.[218] Instruments in this category

Table 3-5   Transmissible Diseases of Concern to Dental Providers

Hepatitis (types A, B, non-A/non-B) (hepatitis B virus)
Acquired immune deficiency syndrome (human immunodeficiency virus)
Syphilis
Gonorrhea
Influenzas
Acute pharyngitis (viral or streptococcal)
Pneumonias
Tuberculosis
Herpes
Chickenpox
Infectious mononucleosis
Rubella
Rubeola
Mumps

Table 3-6   Groups at High Risk of Contracting Hepatitis B

Health care personnel

Selected patients and patient contacts
Patients and staff in hemodialysis units and hematology/oncology units
Patients requiring frequent or large-volume blood transfusions or clotting factors (ie, hemophiliac patients)
Residents and staff of institutions for the mentally handicapped
Household and sexual contacts of persons with persistent hepatitis B antigen
Newborns of hepatitis B surface antigen carrier mother

Populations with high incidence of the disease
Alaskan natives
Indo-Chinese refugees
Haitian refugees
Native Pacific Islanders
Sub-Saharan Africans

Morticians and embalmers

Blood bank and plasma fractionation workers

Persons at increased risk of disease because of sexual practices

Prisoners

Users of illicit injectable drugs

International travelers

must be sterilized and stored in appropriate packages. Single-use items must be properly discarded.

2. **Semicritical items:** Instruments that touch mucous membranes but do not penetrate tissues. This includes amalgam condensers and saliva ejectors. These items should be sterilized; however, if this is not feasible, high-level disinfection or disposal is required.

3. **Noncritical items:** Those items that do not come in contact with oral mucosa but are touched by saliva- or blood-contaminated hands while treating patients. Such items include light switches, countertops, and drawer pulls on cabinets. These areas should be properly disinfected.

STERILIZATION

A recent Minnesota study suggests that approximately one of every five efforts at instrument sterilization in dental offices fails. Errors made by the sterilizer operator were found to be the major cause of failure.[212]

Four elements essential to ensuring proper sterilization are recommended:

1. High-quality sterilization equipment and maintenance
2. Correct operation of sterilization equipment
3. Comprehensive operator training
4. Weekly use of biologic indicators (*Bacillus subtilis* strips) to monitor sterilization effectiveness.[212]

The four methods of sterilization that are generally accepted in dentistry include **steam** under pressure, **chemical vapor, dry heat** sterilization, and **glutaraldehyde** solutions.[208,219] Ethylene oxide gas, ultraviolet light, microwave, and other forms of radiation are effective but have limited use in dentistry at present.[217,220] Glutaraldehyde solutions are reviewed with disinfectants because of difficulties of attaining sterilization using the medium.

Steam Under Pressure—Autoclaving

The commonly accepted criteria for autoclaving are 121°C (249°F) at 15 psi for 15 to 40 minutes. The time depends on the items to be autoclaved, the size of the load, and the type of container used. Included in this method is the "flash" sterilization technique, for which shorter times with higher temperatures are used. There is, however, a greater chance for sterilization error to occur. The disadvantages of autoclaving include rusting, corroding, and dulling of instruments, especially those composed of carbon steel. Instruments removed from the chamber are wet, which increases the turnaround time of sterilization. Certain plastics and rubber are also sensitive to heat and moisture and cannot be placed in the autoclave.

Chemical Vapor Sterilization—Chemclave

This method is based on the factors of heat, water, and chemical synergism. The chemicals include alcohol, acetone, ketones, and formaldehyde. The water content is below the 15% level, above which rust, corrosion, and dullness of metal occur. The composition of heat and chemicals is much kinder to metal surfaces than are other techniques. The temperature requirements are 132°C (270°F) for 20 minutes. The main advantages are the fast turnaround time and the protection of carbon steel instruments. The main disadvantage is the odor that is released when the chemicals are heated. This method has become a popular mode of sterilization in endodontic offices.

Dry Heat Sterilization

This technique of sterilization requires a temperature of 160°C (320°F) for 2 hours. The primary disadvantage of this technique is the long sterilization time. Initial cost of the dry heat method is lower than that of the two previously described. During the loading process, instruments must be separated to prevent the creation of air pockets (stratification) leading to ineffective sterilization. Some units also have problems of uneven heating.

Recently, a Rapid Heat-Transfer Sterilizer was introduced as the Cox sterilizer (Alfa Medical Equipment Co.; Hempstead, N.Y.). Operated at 190°C, it will, by rapid airflow, sterilize unpackaged instruments in 6 minutes and packaged instruments in 12 minutes.

Preparation for Sterilization

Instruments and equipment intended for sterilization or disinfection procedures must first be carefully prepared. This precleaning is essential to remove blood, saliva, tissue, and other debris that can interfere with the sterilization process. The instruments should be cleaned thoroughly by scrubbing with soap and water or a detergent solution, or with a mechanical device (ultrasonic cleaner). The use of a covered ultrasonic cleaner is an effective method of increasing the efficiency of cleaning and reducing the handling of sharp instruments.

When it is not possible to clean and process instruments immediately after their use, they may be held in a "holding solution" to prevent organic material from drying on them, making them difficult to clean. Water, a detergent solution, or an intermediate-level disinfectant may be used for this purpose.

Verification

In a study of sterilizers in endodontic offices, 15% of those tested failed to adequately sterilize items.[221] Dry heat sterilizers were the most likely to have failures, although the most common problem was human error. Inadequate exposure time, equipment overloading, improper wrapping, and poor internal circulation were cited as only some of the problems encountered. Few failures were caused by the equipment.

Several sterilization monitors are available, including process indicators, control indicators, and biologic monitors.[219] Process indicators (ink compound on tape or paper) determine that certain conditions have been met but do not indicate sterility. Control or certified indicators better show that sterilization parameters have been achieved but still do not conclusively indicate sterility. Biologic monitoring is the only dependable method to verify sterility. These monitors are composed of strips of paper with live, resistant spores, which should be killed if properly sterilized. Biologic monitoring should be done weekly and the results recorded and stored.

DISINFECTION

All items that can be sterilized should be sterilized. Disinfection is added to the methods for preventing cross-contamination for instances in which sterilization is not possible. Disinfection is a compromise over sterilization; however, it does contribute substantially to the reduction of microorganisms. A disinfectant is deemed acceptable for dentistry if the solution is registered with the US Environmental Protection Agency (EPA) or approved/accepted by the ADA. Instrument and surface disinfectants suitable for dentistry are listed in the sections that follow.[222,223] These compounds have been accepted by the ADA as liquid disinfectants.

Glutaraldehyde Preparations

A plethora of glutaraldehyde preparations exists today. Disinfection occurs in 10 to 30 minutes, and various types of preparations are capable of sterilization:

2% acidic—60°C for 1 hour
2% alkaline—at room temperature for 10 hours
2% alkaline with phenolic buffer—at room temperature for 6.75 hours
2% neutral—at room temperature for 10 hours

Glutaraldehydes are generally not recommended for sterilization because of the instability of the activated solutions, problems of dilution, and the inability to monitor sterilization. Some glutaraldehyde solutions are ADA approved as disinfectants and sterilizers if used according to manufacturers' instructions. The solutions are registered with the EPA as immersion disinfectants only. They can be used on operatory surfaces and act in 3 to 30 minutes, depending on the amount of debris and types of viruses present. Glutaraldehydes have disadvantages as surface disinfectants, however, such as vapor toxicity, hand and eye irritation, and expense. They are therefore not recommended. A monitor strip to test the solution potency is available and recommended, rather than depending on the number of days the solution is used. A 1986 survey indicated that 71% of the dental practices participating were using glutaraldehyde solutions in some formulation.[224]

Chlorine Dioxide

The chlorine dioxide compounds disinfect instruments and operatory surfaces in 1 to 3 minutes when used correctly. The solution requires no rinsing and leaves no residue after use. There are no special handling or disposal requirements. Solutions can sterilize items in 6 hours at room temperature. This substance has been reported to be nontoxic, nonirritating, and nonsensitizing. The disadvantages are the corrosion of easily oxidized metals and the need for fresh new solutions for each sterilization/disinfection process.

Sodium Hypochlorite (Household Bleach)

Sodium hypochlorite is more suitable for surface **disinfection** than for instrument sterilization because of its highly corrosive action on metals. Dilutions of 1:5 to 1:1 are generally recommended. On surfaces, sodium hypochlorite is virucidal, bactericidal, and tuberculocidal. Disinfection can occur in 3 to 30 minutes, depending on the amount of debris present. It is the least expensive of the surface disinfectants. The major disadvantage, as previously mentioned, is the corrosion factor. The solution also tends to be unstable and should be prepared daily. As a surface disinfectant, there is a strong, unpleasant odor. Plastic chair covers have a tendency to crack under prolonged use.

Iodophors

Iodophor is a broad-spectrum disinfectant that is effective against a host of pathogens, including HBV, *M. tuberculosis,* poliovirus, and herpes simplex virus. One of the inherent advantages of the compound is the slow release of elemental iodine to enhance the bactericidal activity. A surfactant carrier keeps the surface moist to protect the iodophor during this release, and the action may continue even after the surface appears dry. The most effective dilution for hard-surface iodophors is 1 part iodophor concentrate to 213 parts of soft or dis-

tilled water. **Hard water inactivates the iodophor**. Biocidal activity occurs within 30 minutes.

Iodophors also have a built-in color indicator. When the solution is fresh, an amber color is present. With age, the solution changes to light yellow, indicating the loss of the iodophor molecules. A mixture of iodophor with alcohol was thought to enhance the activity, but research evidence is insufficient to support this claim.[222] The iodophor compound is to be used solely as a disinfectant. The sporicidal capabilities of the substance have not been shown.

Alcohols

Alcohols are not accepted by the ADA for disinfection of surfaces or instruments.

Quarternary Ammonium Compounds

This group of compounds, including benzalkonium chloride, is no longer recommended for instrument or surface disinfection. All quarternary ammonium compounds have been disapproved by the ADA for use in dentistry.

DISINFECTION TECHNIQUES

The following disinfection techniques are recommended by the ADA and The Center For Disease Control:

1. **Immersion disinfection:** Solutions must be fresh and changed according to manufacturers' recommendations. All instruments must be cleaned by thorough scrubbing with soap and water or with a mechanical cleaner, such as an ultrasonic unit. Heavy-duty rubber gloves should be worn during instrument decontamination. Instruments must be dried before being placed in the disinfectant to prevent dilution.

2. **Surface disinfection:** Countertops and surfaces that have become contaminated with blood, saliva, and debris must be wiped and/or scrubbed to remove organic material after being sprayed with an appropriate surface disinfectant. Once cleaned, the surface is again sprayed and left moist for the recommended period. Surface decontamination is approximately 80% effective in bacterial control.[205]

3. **Decontamination of dental units:** Dental units have come under scrutiny in the environment of infection control. Check-valves have been recommended to prevent aspiration of infective materials into handpieces and water lines.[208] A major implication would be if a patient were infected with HBV, HIV, or tuberculosis and these organisms were aspirated into the unit and allowed to colonize. They could later be discharged into the mouths of subsequent patients. A comparison of units with and without check-valves showed significant decreases in the amount of bacteria in the dental units with check valves.[225] Microorganisms, however, were still found even in units using the check-valve. Eliminating the fluid retraction valve was the most effective way to prevent fluid retraction, but then water continued to drip from the units. The CDC recommends that handpieces be flushed for 20 to 30 seconds between patients and for several minutes at the beginning of each day to reduce any overnight bacterial accumulation in the units. They also recommend that sterile saline or sterile water be used as a coolant/irrigant for any surgical procedures.[226]

BARRIER TECHNIQUES

Three factors determine whether disease develops in the host after exposure: virulence of the disease agent, resistance of the host, and the quantity of the disease agent.[227] Barrier techniques in infection control address the quantity factor in disease prevention. This may encompass protection of the body surfaces, protection of the environmental surfaces, or blockage of bacteria from the source.

Gloves

Gloves provide the patient with protection from contamination of microorganisms on the practitioner's hands and protect dental health care workers from contamination by the patient's blood and saliva.[227] Small cuts and abrasions on the hands can serve as portals of entry into the body. Gloves can provide a barrier between open wounds and bacteria from blood and saliva. In one research study, traces of blood were found beneath the fingernails of 44% of ungloved general dentists.[228]

One of the main concerns about the use of gloves has been the worry about possible loss of tactile sense, especially in the practice of endodontics. In a study focusing on tactile sense, no significant differences were found among gloved versus ungloved clinicians.[229] In a time test of endodontic performance, no differences were found between gloved and ungloved hands.[230] The difficulty lies in the fact that many clinicians were trained when gloves were not used. Studies have shown that learning periods of 1 to 2 months are necessary to become accustomed to wearing gloves.[231] Proper fit is important for tactile control and comfort. Gloves vary in size between manufacturers and even within the same brand, depending on the type of glove (ie, examination versus surgical).

The reuse of gloves has been reviewed by many authors.[208,231,232] Gobetti and associates have stated that washing a gloved hand removed significant amounts of

bacteria.[233] If iodine scrub soap was used, the gloved hand would be free of bacteria. In a study of the evaluation of gloves, pinholes occurred randomly and were independent of the type or manufacturer.[231] Pinholes occurred in 1.7 to 9% of the gloves tested. Tear strength also varied. The investigators did not recommend reuse after conventional dental procedures because the clinician had no way to determine the integrity of the glove.

The ADA Council on Dental Therapeutics and the CDC recommend that gloves not be reused.[234] **Double gloves** may be indicated for patients with known infectious diseases, such as herpes, HBV, and HIV. Gloves that are known to have been contaminated with an infectious entity (ie, HBV, HIV) should be sterilized before being discarded.[234]

Hand Washing

Hands should be washed before gloves are placed and after gloves are removed because the integrity of the glove is not dependable.[208] Antimicrobial hand-washing solutions should be used.[209] If, during the course of treatment, a glove is torn, the glove should be removed and the hands washed and then regloved.

Face Masks

The face mask is an important barrier providing protection from inhalation of aerosols generated by high-speed handpieces and air-water syringes. The mask should remain dry to prevent transmission of organisms through moisture penetration. Masks may be composed of glass or synthetic fiber, paper, or gauze. The fiber-type mask is considered to be more efficient in filtering bacteria.[228] Masks should be worn by all treatment personnel and should be changed between patients because masks worn for prolonged periods may become a nidus of infection.

Eyeglasses

Protective eyewear is highly beneficial for dental care providers and for the patient. Herpes virus infection of the eye and infection from hepatitis B are possible consequences of viral contact with the eye.[227] Eyewear can prevent bacterial or viral contact with the eye by aerosol spray or droplet infection. Chin-length face shields are also effective in the prevention of splashing and splattering of blood and saliva; however, they do not provide protection from inhalation of aerosols.

Clothing

The general recommendations for clinic wear include reusable or disposable gowns and laboratory coats or uniforms with long sleeves. Head covers are also rec-ommended during procedures that result in splashing blood or other body fluids. Gowns should be changed at least daily. Laundering can be effectively accomplished with a high-temperature (60 to 70°C) wash cycle with normal bleach, followed by machine drying (100°C or more). According to the CDC reports, this method, along with dry cleaning and steam pressing, is effective in killing the AIDS virus.[234] Shoes should be changed at the office or kept out of reach of small children at home because they are in constant contact with saliva and blood splatter that settle on the floor.

Procedural Barriers

The rubber dam has been shown to be an effective barrier to reduce the number of organisms contained in aerosols.[227] The number of infectious particles can be reduced by 99%. The rubber dam prevents aerosolization of saliva and should be used whenever possible. **Operating fields**, isolated by a rubber dam, however, showed bacterial contamination in 53% of the cases after 1 hour.[235] When silicone and adhesives were used to further seal around the dam, bacterial leakage was reduced to 20%.

Although high-speed evacuation is not a true form of barrier control, it should be used whenever possible. Evacuation decreases the amount of particles that become airborne.[208]

Disposable impervious-backed paper, plastic, or aluminum wrap can be used to cover surfaces and operatory equipment.[208,227] This aids in the prevention of surface contamination from blood or saliva. Plastic is more resistant to water penetration and can be molded into any shape more easily than can paper. Specially designed covers are commercially available to protect light handles, chairs, and bracket and instrument tables. Ash et al. developed a technique wherein radiographic film can be wrapped and sealed with a plastic to prevent contamination with saliva.[236] After the film has been exposed, the wrap is opened and the film handed to someone who is not contaminated and therefore can then develop the saliva-free film. Another method is to open the contaminated film packet in the darkroom or developing box using disposable gloves. The films should be dropped out of the packets without touching the films. Drop the contaminated packets in a paper cup. After all packets have thus been opened, the discarded packets and the gloves can be removed before processing the films. A recent study has demonstrated that bacterial contamination on radiographic films can survive the processing, thus pointing out the importance of preventing cross-contamination for this dental procedure.[237]

SHARP INSTRUMENTS

Needles, endodontic files, scalpels, and other sharp instruments must be handled with care to prevent percutaneous injury. After anesthetics or other injectables have been administered, the needle should be kept in a "sterile" area either uncapped or recapped, using the "scoop technique" (holding the cap in a hemostat or using a manufactured cap holder).

After needles or scalpel blades have been used, they should be removed with a hemostat to prevent injury. All sharps should be placed into puncture-resistant receptacles, which are then disposed of according to local regulations.

IMMUNIZATION

Hepatitis B is a major health hazard for dental health care personnel. Because of this risk, the ADA Council on Dental Therapeutics and the CDC have recommended that all dental personnel involved in patient care receive the hepatitis B vaccine if they do not already have immunity as a result of previous exposure to the virus.[206,238] Two types of vaccines are currently available: a plasma-derived HB vaccine and a recombinant DNA HB vaccine. Both are considered safe and effective in producing immunity to HBV. To date, no serious side effects have been reported from recipients of either vaccine.

Vaccines play an important role in the infection control process, but many bloodborne pathogens exist for which there is presently no vaccine, including HIV and non-A/non-B hepatitis. Proper infection control procedures are therefore important to prevent transmission of any pathogen.

ENDODONTIC INSTRUMENTS AND MATERIALS

Glass bead sterilizers have been commonly used in endodontic offices. Sterilization of clean endodontic files can be achieved with glass beads at 218°C (424.4°F) for 15 seconds or with salt at the same temperature for 10 seconds.[239] It is important to note that there is a wide variability among units in achieving operating temperatures. Preheating times ranged from 15 minutes to 3.5 hours, according to a test of sterilizers.[240] Larger instruments and more porous materials should be immersed in sterilizers for a minimum of 20 seconds. If larger-size instruments are being reused, the handles are not sterilized and require alternate methods of sterilization between patients.

Gutta-percha points are sterile in the manufacturer's package. Contaminated points can be sterilized with 5.25% sodium hypochlorite.[241] Researchers have found that gutta-percha can be sterilized after exposure to gram-positive, gram-negative, and spore-forming microorganisms within 1 minute after immersion in undiluted sodium hypochlorite (Clorox). No mention was made of viral forms. No changes were noted in the dimensional stability or integrity of the points immersed for up to 5 minutes in sodium hypochlorite versus points that were placed in water.[241]

Immersion in polyvinylpyrrolidone-iodine for 6 minutes is an alternate method for the disinfection of gutta-percha.[242] The reliability of this method against tuberculosis bacilli and some spore forms is questionable, however.

OCCUPATIONAL HEALTH AND SAFETY ADMINISTRATION

The OSHA requires employers, including dentists, to provide a safe working environment for their employees. Endodontists must obey guidelines developed by their specific state administrations and information set forth by the CDC and the ADA.[243] According to Miyasaki and associates, informing, educating, and providing for one's employees are ways to minimize the chance of an OSHA inspection.[244] Practitioners should inform their employees of the risks of exposure to hazardous materials and bloodborne diseases, educate employees on the prevention of the spread of disease, and provide protective equipment. All infection control procedures should be documented. Lastly, the endodontist can consult with an OSHA consultant regarding current regulations.

Chemical hazards are another area of regulation by OSHA. Again, depending on the location of practice, the endodontist must be aware of state and local regulations. Even though the endodontic office has fewer hazardous substances than does a general practice, items such as mercury, formaldehyde, and nitrous oxide may often be found. Generally, a complete list of hazardous substances in the office must be kept on file. This should be updated as materials are added to the office. Material safety data sheets from manufacturers must be available to employees. This documentation includes handling and use precautions, emergency and first-aid procedures, and control measures. Practitioners must also have a hazard communication program to disperse information to their employees.

CONCLUSION

A checklist recommended by the ADA is printed as Figure 3-10.[209] Practitioners should attempt to adhere to these recommendations to protect their patients, staff, and themselves from the risk of cross-contamina-

Infection control for the dental office: A checklist

Immunization

- Health care workers should have appropriate immunizations such as that for hepatitis B virus.

Before patient treatment

- Obtain a thorough medical history.
- Disinfect prostheses and appliances received from the laboratory.
- Place disposable coverings to prevent contamination of surfaces, or disinfect surfaces after treatment.

During patient treatment

- Treat all patients as potentially infectious.
- Use protective attire and barrier techniques when contact with body fluids or mucous membranes is anticipated.
  —Wear gloves.
  —Wear mask.
  —Wear protective eyewear.
  —Wear uniforms, laboratory coats, or gowns.
- Open intraorally contaminated X-ray film packets in the dark room with disposable gloves without touching the films.
- Minimize formation of droplets, spatters, and aerosols.
- Use a rubber dam to isolate the tooth and field when appropriate.
- Use high-volume vacuum evacuation.
- Protect hands.
  —Wash hands before gloving and after gloves are removed.
  —Change gloves between each patient.
  —Discard gloves that are torn, cut, or punctured.
  —Avoid hand injuries.
- Avoid injury with sharp instruments and needles.
  —Handle sharp items carefully.
  —Do not bend or break disposable needles.
  —If needles are not recapped, place in separate field. If recapping is necessary, use a method that protects hands from injury such as a holder for the cap.
  —Place sharp items in appropriate containers.

After patient treatment

- Wear heavy-duty rubber gloves.
- Clean instruments thoroughly.
- Sterilize instruments.
  —Sterilize instruments that penetrate soft tissue or bone.
  —Sterilize, whenever possible, all instruments that come in contact with oral mucous membranes, body fluids, or those that have been contaminated with secretions of patients. Otherwise, use appropriate disinfection.
  —Monitor the sterilizer with biological monitors.
- Clean handpieces, dental units, and ultrasonic scalers.
  —Flush handpieces, dental units, ultrasonic scalers, and air/water syringes between patients.
  —Clean and sterilize air/water syringes and ultrasonic scalers if possible; otherwise, disinfect them.
  —Clean and sterilize handpieces if possible; otherwise, disinfect them.
- Handle sharp instruments with caution.
  —Place disposable needles, scalpels, and other sharp items intact into puncture-resistant containers before disposal.
- Decontaminate environmental surfaces
  —Wipe work surfaces with absorbent toweling to remove debris, and dispose of this toweling appropriately.
  —Disinfect with suitable chemical disinfectant.
  —Change protective coverings on light handles, x-ray unit head, and other items.
- Decontaminate supplies and materials.
  —Rinse and disinfect impressions, bite registrations, and appliances to be sent to the laboratory.
- Communicate infection control program to dental laboratory.
- Dispense a small amount of pumice in a disposable container for individual use on each case and discard any excess.
- Remove contaminated wastes appropriately.
  —Pour blood, suctioned fluids, and other liquid waste into drain connected to a sanitary sewer system.
  —Place solid waste contaminated with blood or saliva in sealed, sturdy impervious bags; dispose according to local government regulations.
- Remove gloves and wash hands.

Figure 3-10   Infection control for the dental office: a checklist. (Report, Council on Dental Materials, ADA.[209])[may be copied]

tion. Recommendations from federal, state, and local authorities can change frequently; therefore, one must remain constantly updated on current information.

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