ENDODONTICS

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FOREWORD

In view of recent advances in the field of endodontics, both in concept and technique, the earlier course, Endodontics, NavPers 10782, has been revised and enlarged. The purpose has been to give more assistance to the dental officer in incorporating endodontic therapy to greater advantage in his everyday practice. The course is designed to assist the dental officer at the isolated duty station, as well as the dental officer at the large clinic where endodontics may be a major part of his practice. While it is true that the large clinic can equip and maintain a dental operating room especially for endodontic treatment and function very efficiently, the fact that a dental officer is stationed at a small clinic or at sea does not preclude endodontics from becoming a most rewarding part of his practice.

All of the principal phases of endodontics—etiology, diagnosis, and treatment—are encompassed in this course. Emphasis is given to the histological aspects of pulpal and periapical disease in the hope that an understanding of these factors will enable the dental officer to use reasoning power—in place of blind cookbook methods—in diagnosis and treatment.

In chapter 2, the development, histological character, and resolution of pathological conditions of the pulp and periapical tissues are described. Diagnosis of these conditions and the evaluation of cases for treatment are dealt with in chapter 3. In the remainder of the text nonsurgical and surgical endodontic treatment and certain auxiliary procedures such as sterilization, culturing of root-canal samples, and bleaching of pulpless teeth are discussed.

In studying this text the dental officer should bear in mind that space does not permit complete discussion of the various phases of endodontics, and therefore he should seek further knowledge in current textbooks and periodicals. The articles on endodontics appearing in Oral Surgery, Oral Medicine & Oral Pathology are especially recommended because they have been reviewed and approved for publication by a member of the American Association of Endodontists.

This text was prepared for the Bureau of Medicine and Surgery, under the direction of the Commanding Officer, U. S. Naval Dental School, by Captain John F. Bucher, Dental Corps, U. S. Navy, Head of the Operative Dentistry Department and Head of the Endodontics Division, assisted by Mrs. Elizabeth W. Graeff, Technical Publications Writer-Editor.

Grateful appreciation is expressed to the Armed Forces Institute of Pathology, to Captain R. A. Colby, DC, USN (ret.), and to Dr. H. R. Stanley of the National Institute of Dental Research, for their contribution of many of the transparencies that accompany the text. The cooperation and assistance of many members of the staff of the U. S. Naval Dental School in giving guidance, in providing current information relative to their specialties, and in supplying transparencies illustrating the subject matter is likewise gratefully acknowledged.

The student is encouraged to submit any comments and suggestions he may have regarding the text and course materials.

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CHAPTER 1

INTRODUCTION

Endodontics is the branch of dentistry that deals with the etiology, diagnosis, and treatment of pulpal and periapical disease. The goals of endodontic treatment are (1) to relieve pain; (2) to preserve the pulp, if possible; and (3) to undertake all procedures required to restore a pulpally or periapically involved tooth to a condition of good health in its supporting structures.

Not many years ago endodontic therapy was approached by the average practitioner with hesitation and pronounced reservations; but as a result of the clear vision, the strong faith, and the sustained educational efforts of a core of dedicated men, the dental profession is awakening to the opportunities afforded by endodontics.

Progress in endodontics is being built upon a foundation of dental research and the application of scientific principles. Educational opportunities are increasing rapidly. The development of improved techniques has provided additional stimulus for the use of endodontic treatment.

Today, endodontic therapy is becoming one of the most versatile and practical dental procedures. Dentists are gaining confidence in endodontics and in its widening possibilities in their everyday practice. Patients are becoming aware that pulp-involved teeth need not be extracted but can, in most cases, be maintained in the mouth. Teeth with involved pulps or periapical lesions are now, as a matter of course, carefully evaluated for endodontic treatment.

Nevertheless, questions arise in the minds of some men: "Are we placing too much faith in the endodontically treated tooth?" "When the pulp is removed and endodontic treatment is completed, is the pulpless tooth a 'dead' tooth?" The dental officer is familiar with the terms "dead tooth," "nonvital tooth," and "devital tooth." These terms convey a very unpleasant meaning to the patient, and they are unfair!

One should not think of the pulp as the very fountainhead of the tooth's vitality. The pulp is a highly important organ, of course, and every effort should be made to maintain it, but it is not the sine qua non of the tooth's existence. The periodontally sound, endodontically treated pulpless tooth is not a foreign object but a part of the body. There is still a ligamentous attachment between cementum and alveolar bone; there is still a nutritional exchange between tooth and body through the rich network of periodontal vessels that literally sheathes the root. There is still a metabolic response of cementum to physiologic drift or orthodontic movement.

On the other hand, when the periodontal support is threatened by advancing periodontal disease, the tooth is in very serious trouble. When periodontal support is gone, the tooth is indeed "dead" (regardless of the condition of the pulp).

With a well-filled canal, a restored crown, an intact periodontal attachment, and the resolution of any periapical lesion, the treated pulpless tooth is a healthy, functioning member of the dentition. Therefore, in discussion of these teeth with patients and medical officers, the dental officer should stamp out the expressions "dead tooth" or "nonvital" tooth. In their place the following terms are suggested:

<u>Pulp-Involved Tooth:</u> A tooth in which the pulp is involved by inflammatory or retrograde changes, or in which the pulp has undergone necrosis.

<u>Pulpless Tooth:</u> A tooth in which the pulp spaces have been properly treated and filled. With complete postoperative healing, it is reasonable to refer to this tooth as a "healthy pulpless tooth."

Endodontic therapy is, of course, concerned with the saving of individual teeth, but a highly significant part of its mission is to maintain the integrity of the dental arch. Through prevention of the needless loss of teeth from the arch, the role of endodontics in preventive dentistry becomes evident. If the first interruption in the natural dentition can be prevented, many of the untoward results of that interruption (tooth drifting, dental caries, periodontal involvement) may be prevented. In addition, by sound endodontic procedures, a strategic pulp-involved tooth may be saved from extraction with possible disfiguring loss of bone.

Preservation of a key abutment tooth may make possible the retention of a fixed prosthetic appliance in the arch and preclude the need for a removable partial denture; similarly, preservation of a strategic abutment may make the difference between a removable partial denture and a full denture. Thus, endodontics can aid and abet good health rather than undermine it.[1]

REFERENCE

1. Rudolph, C. E., Jr. Arch continuity through endodontics. Military Med. 122:385-389 June 1958.

CHAPTER 2
PULPAL AND PERIAPICAL PATHOSIS

The material in this chapter pertains principally to pathological conditions of the dental pulp and the periapical tissues. Basically, most of the changes that take place in pulpal and periapical tissues are those of inflammation. The pulp responds to irritants coming from the external environment by inflammation. The periapical tissues respond to irritants stemming from the root canal by the formation of inflammatory lesions.

In order that this chapter may be thoroughly understood, the essentials of inflammation will be reviewed briefly.

INFLAMMATION

Whenever tissue cells are injured or killed, a series of local defensive reactions occurs at the site of injury. These reactions constitute inflammation. The irritant causing injury or death may be physical, chemical, or bacterial. Regardless of the type of irritant, the tissue changes in inflammation are essentially the same. The invasion of the irritant and the inflammatory response in the tissues may be interpreted as a battle between an attacking injurious force and the defensive capability of the tissues.

The objectives of inflammation are (1) to bring to the area certain phagocytic cells that engulf and digest bacteria, dead cells, or other debris; (2) to bring antibodies to the site; (3) to neutralize and dilute the irritant (by edema); (4) to limit the spread of inflammation (by fibrin formation, or walling off with granulation tissue); and (5) to initiate repair.[1] Thus, it should be realized, inflammation is a purposeful protective response.

Inflammation may be divided into two very broad types: acute and chronic. These types are not entirely separate and distinct: areas of blending and transition exist. The appearance of one type or the other depends on the relative strength of irritant and host.

Cells of Inflammation

Several types of cells are characteristically associated with the process of inflammation. These cells, and the part they play in the process, can be described as follows:

1. Polymorphonuclear neutrophil leukocytes (neutrophils) (SLIDE 1) are the predominant cells of acute inflammation, the first cells to arrive in an injured area. They ingest bacteria and liberate proteolytic enzymes that liquefy, or cause the dissolution of (lyse), bacteria, fibrin, and cellular debris.
2. Macrophages (SLIDE 2) comprise the second line of defense. These large mononuclear cells, which originate in the tissues as well as in the blood, act as scavengers, ingesting certain bacteria and tissue debris. Together, the neutrophils and the macrophages clean up the injured area and prepare it for angioblastic and fibroblastic proliferation that ultimately results in complete repair.
3. Lymphocytes (SLIDE 3) enter the area from the blood, and in some instances from nearby lymph tissue. They are believed to carry antibodies.
4. Plasma cells (SLIDE 4) are believed to produce and carry antibodies. Together with lymphocytes, plasma cells provide a picture characteristic of chronic inflammation.
5. Eosinophils, probably all from the blood, are slightly phagocytic and are numerous in inflammations of an allergic nature, in parasitism, and in certain longstanding chronic processes.
6. Basophils are believed to be a source of heparin, which prevents the formation of massive clots.

Acute Inflammation

If the irritant is overwhelming in relation to the local defensive capability of the tissues, the response is acute inflammation. It is a rapid, sharp emergency response. In the injured area a momentary vasoconstriction is followed by vasodilation (hyperemia). Soon the current in the dilated vessels slows progressively, and the cellular elements, particularly the neutrophils, move from the axial stream to the vessel walls (margination). It is believed that injured tissues release chemical substances which, among other actions, increase the permeability of capillaries and attract neutrophils to the injured site. The space between endothelial cells of the capillary walls opens to several times the normal width. This allows the neutrophils to squeeze through the capillary walls by ameboid movement (diapedesis) and move by chemical attraction in increasing numbers to the injured site (chemotaxis). Here the neutrophils phagocytize bacteria, dead cells, or other debris.

Crystalloids and proteins (albumin, globulin, and fibrinogen) also escape from the bloodstream. Fibrinogen is converted into fibrin, which plugs the lymphatics and thereby helps prevent the spread of the injurious agent. Fibrin also serves as a network upon which the neutrophils move toward the injured cells. Escaping gamma globulins are altered to form antibodies, which assist in tissue defense.

Along with the cells and plasma proteins, six or seven times the normal amount of fluid escapes into the tissues and is retained there because of an altered pressure gradient between the tissues and the venous capillaries. This edematous fluid (or exudate) serves to neutralize and dilute the irritant.

Thus, microscopically, acute inflammation is characterized primarily by pronounced edema and large numbers of polymorphonuclear neutrophils (SLIDE 5). There is no time for the development of connective tissue barriers.

On a clinical level, acute inflammation is characterized by redness and heat (because of hyperemia), swelling (because of edema), and pain (because of pressure on, and irritation of, local nerve endings by the products of the inflammatory process).

When the irritant is eliminated, the inflammatory process begins to resolve. The edematous fluid is drained by lymphatics and veins, and dead cells and debris are phagocytized. The tissue may gradually return to normal; or if the irritant is merely subdued or reduced in intensity, the acute phase may proceed to a subacute or chronic phase.

Acute inflammation may also take a special form: an <u>acute abscess</u>. This lesion forms when an inflammatory exudate containing many polymorphonuclear neutrophils is able to confine an irritant of great intensity that has killed numerous cells. The great numbers of neutrophils liberate a large amount of a proteolytic enzyme that liquefies the necrotic cells, forming a semiliquid material: pus. The wall of an acute abscess is composed only of inflammatory cells, whereas fibrous tissue is found in the outer wall of a chronic abscess.

Subacute Inflammation

This is considered a transitional phase between the acute and chronic stages. Acute exudative changes are still present, but chronic proliferative changes have begun.

Chronic Inflammation

Chronic inflammation is a low-grade, prolonged response, characterized by a continuing impulse to repair. It occurs when the irritant is mild and long lasting and the resistance of the host is good—in other words, when irritant and host are evenly matched. Whereas acute inflammation lasts from days to 2 or 3 weeks, chronic inflammation extends over a period of months or years. It is a deliberate response in which all defensive forces may be brought into action.

Microscopically, the defensive phase of chronic inflammation is characterized by the presence of exudate and defensive cells: lymphocytes, plasma cells, and macrophages (SLIDE 6). Even as the irritant is being diluted, phagocytized, and as far as possible eliminated, the repair phase is beginning. Young connective tissue cells (fibroblasts) and endothelial cells start to multiply. Delicate connective tissue fibrils and budding new vascular channels proliferate into and permeate the exudate, producing a highly vascularized, reddish granular mass termed <u>granulation tissue</u> (SLIDE 7).

Since the chronic inflammatory exudate undergoes so many subtle changes in its response to an irritant, it is difficult to decide at what point granulation tissue begins and ends. Oral pathologists offer a wide latitude in interpretation. In order to avoid misunderstanding, then, the term "granulation tissue" will be used here to embrace the entire chronic response—from the chronic exudate in the primarily defensive phase to the proliferating connective tissue and vascular channels in the predominantly reparative phase.

It is granulation tissue that proliferates into a wound clot to facilitate complete repair. In a periapical lesion granulation tissue represents the valiant effort of local tissues to lay a foundation for complete repair in the face of continuing irritation. If the source of irritation is removed by sound endodontic therapy, granulation tissue is the steppingstone to complete repair—hopefully, osteogenesis.

Relationship of Inflammation to Infection

The reader may possibly be confused about the relationship between inflammation and infection. He may wonder, for instance, how an infected periapical abscess can be termed "inflammation."

When pathogenic microorganisms invade and multiply in a tissue, the resultant defensive reaction is basically inflammation. Infectious lesions are inflammatory lesions, and all the fundamental principles governing the body's reaction to injury strictly apply to them. Any special features of infection depend upon certain peculiarities of the microorganisms themselves: (1) Unlike purely chemical or physical irritants, bacteria can multiply within the body, thus affording a prolonged, continuing stimulus for inflammatory reaction; (2) bacterial infection may spread in a manner unlike that of nonliving injurious agents. This wider extension occurs through the action of toxins elaborated by the microorganisms—toxins that produce edema, dissolve fibrin, or dissolve the ground substance that normally holds the cells together.

Resolution of Inflammation

Inflammation eventually ends in one of two ways: repair or necrosis.

Repair is an all-inclusive term that embraces (1) regeneration, or the replacement of damaged tissue with cells similar to those destroyed, for example, osteogenesis; and (2) those processes wherein the damaged tissues is replaced by dissimilar cells, for example, replacement of bone with dense collagenous "scar" tissue.

Necrosis is simply death of tissue. There are several types, including (1) coagulation (dry) necrosis and (2) liquefactive (moist) necrosis. In coagulation necrosis, which is commonly produced by cutting off the blood supply, the decomposed tissue appears as a dried-up, brownish mass. In liquefactive necrosis, commonly produced by proteolytic enzymes, the dead tissue becomes softened and eventually liquefies. (In abscesses tissue debris and cells are subject to liquefactive necrosis.)

Gangrene is merely necrosis of tissue to which there is usually added an invasion by saprophytic bacteria (those that live on dead or decaying organic matter). Gas gangrene, characterized by a foul odor, occurs when invading saprophytes are of the gas-forming group, for example, the Welch bacillus.[2]

Inflammation is a fascinating study in itself. For a detailed discussion of it the reader is referred to the general pathology textbooks by Boyd[3] or Anderson,[4] as well as Menkin's Dynamics of Inflammation.[5]

THE NORMAL DENTAL PULP

Before the dentist can diagnose inflammation or degeneration of the dental pulp, he must be familiar with the structure and function of the normal pulp.

The reader may wonder how a knowledge of the pulp's histological structure and function can possibly be significant in his everyday practice of endodontics. Without this knowledge, however, and without a knowledge of what is transpiring histologically inside the diseased pulp, the behavior of the pulp as evidenced by clinical signs and symptoms is a mystery. The dentist is reduced to guessing and to practicing cookbook endodontics.

The endodontist should be as keenly interested in the living pulp as in the handling of the tooth after the pulp is lost. In essence, the objective of endodontic treatment is to maintain each tooth in a healthy condition in its supporting structures. The endodontist would clearly fail his patient and himself if, through ignorance or laziness, he failed to assess properly the true condition of the pulp and sacrificed it unnecessarily. After all, preventive endodontics is an integral part of dental practice.

The dental pulp is a highly vascular and innervated mass of connective tissue enclosed in and supported by the dentin. The outer surface of the pulp roughly follows the outline of the outer surface of the dentin. Like connective tissue in any part of the body, the pulp consists of a matrix or ground substance in which are suspended fibers, cells, blood vessels, and nerves.

Ground Substance

The ground substance of the pulp is more dense, more highly polymerized, than that of loose connective tissue, giving the pulp a gelatinous consistency.[6] Pulp fibers are of two types: reticular and collagenous. With age, the balance shifts toward the collagenous type.

Cellular Elements

The cells of the pulp are of extreme importance to its integrity and defense. Of these the most characteristic and significant cells are the odontoblasts. These highly specialized connective tissue cells, in palisade form (SLIDE 8), comprise the outer layer of the pulp adjacent to the dentin. A long protoplasmic process extending from the cell body of the odontoblast is maintained within the dentinal tubule that it helped to form. During periods of active dentin formation this principal protoplasmic extension of the odontoblast increases in length as the odontoblast recedes toward the center of the tooth. This may be likened to a cable-laying procedure. Sicher[7] has calculated that three-quarters of the cell's protoplasm lies within the dentinal tubule. Thus, it is evident that the odontoblasts stand as important relay stations, connecting dentin and the external environment with the pulp (SLIDE 9). This function will be explored more fully later.

In addition to the odontoblasts the normal pulp contains fibroblasts; undifferentiated mesenchymal cells (reserve cells, playing a dominant role in repair); and macrophages, polymorphonuclear neutrophils, and lymphocytes (all concerned with defense).

Blood Vessels

The pulp is an extremely vascular tissue. A pulpal artery (or arteries) enters the apical foramen, branching as it passes occlusally and finally forming a rich arborization of capillaries near the surface of the pulp among the odontoblasts. From these capillaries the blood is collected into venules, then into veins, which leave the pulp through the apical foramen. Vessels of the pulp are unusually thin walled and delicate and for this reason are susceptible to pressure changes incident to inflammation.

A significant point is that odontoblasts comprise nutritional way stations between the vascular system and the dentin. From the rich capillary network over the odontoblastic cell bodies, fluid is transported to the dentin through the protoplasmic extensions; fluid interchange may thus occur between dentin and pulp. The significance of this to the endodontist is that when the pulp is lost the odontoblastic extensions are lost and the dentin becomes appreciably more dry. However, studies[8,9] have shown that the dentin may also receive fluid via the cementum and enamel, thus preventing dehydration in the periodontally healthy pulpless tooth.

Lymph Drainage

Investigators have demonstrated a rudimentary lymphatic drainage of the pulp. Drainage is believed to occur via perivascular spaces or sheaths, into the submandibular (submaxillary) lymph nodes.

Nerve Supply and Function

The nerve supply of the dental pulp is abundant. Thick nerve bundles enter the apical foramen with blood vessels. Nerves of the pulp are of two types: (1) Small nonmyelinated fibers from the sympathetic nervous system follow the arborization of the arteries and arterioles, regulating their blood flow; (2) myelinated sensory fibers branch progressively and follow a course toward the odontoblasts. The sensory fibers lose their myelin sheath as they form a rich plexus in a cell-free zone just beneath the odontoblasts. From here, fibrils form a network around and terminate between the odontoblasts.[10] These are the fibers that provide an early warning system against external irritation or injury.

The pulp is connected with the external environment not through nerve fibers but through the odontoblastic extensions in the dentinal tubules. Investigators believe that injury to these protoplasmic extensions causes the release of a chemical substance in the odontoblastic cell body in the pulp. This danger signal is picked up by the interodontoblastic network of sensory fibers and relayed to the brain.[11]

The sensory nerve fibers in the pulp respond to danger in another way. Because of the gelatinous consistency of the pulp tissue and because of the confinement of this tissue in an unyielding dentin chamber, the highly sensitive sensory fibers are bound to be affected by an increase in intrapulpal pressure. This increased pressure can be brought about by edema incident to inflammation.

Regardless of whether the irritant causing inflammation is bacterial, chemical, or physical, the sensory nerves of the pulp relay these messages to the brain as pain. The nerves of the pulp provide no sense of touch or localization. Pain originating from a pulp can seem to originate from almost any point on one side of the face.[12] Pulpal pain cannot be localized until the inflammatory process reaches the apical periodontal ligament space, where proprioceptive fibers relay the correct localization to the brain. This is of great diagnostic significance to the dentist.

PULPAL PATHOSIS

Pulpal pathosis, or pulp disease, will be discussed under two headings: "Pulpitis" (inflammation of the pulp); and "Retrograde Alterations of the Pulp."

PULPITIS

When an irritant attacks a tooth, the pulp responds in the same manner as does the connective tissue elsewhere in the body; that is, by inflammation. However, there are certain restrictions imposed upon this inflammatory response in the pulp. The fact that pulp tissue is encased within the unyielding walls of dentin limits to some extent the normal swelling of tissue that occurs in the edematous phases of inflammation. The fact that the blood vessels nourishing the pulp tissue must enter the tooth through small apical foramina appreciably limits the development of a collateral blood supply to the inflamed part.

What are the irritants that cause inflammation of the pulp? These irritants may be bacterial, chemical, or physical. Bacterial invasion through dentinal tubules of carious lesions is believed to be one of the prime causes of pulpitis. Tooth fracture may expose the pulp directly to bacteria in the oral fluids. Chemical irritants include bacterial toxins, acids from various cements, and highly irritating medicaments used in cavity disinfection. Physical irritants such as those applied in operative procedures—pressures, severe thermal change, placement of extensive, poorly insulated restorations—may cause inflammation. Traumatic blows or partial evulsion may sever the apical vessels and cause starvation of pulp tissue with resultant direct necrosis of the entire pulp.

Generally speaking, when the irritant is overwhelming, the result is an exudative response—acute pulpitis. This may resolve when the irritant is subdued by the pulp's defenses. When the irritant is mild and long lasting, the result is a low-grade, proliferative response—chronic pulpitis. This may persist for a long period of time. Inflammation may be partial (involving a portion of the pulp), or total (involving the entire pulp). The pulp may go from acute inflammation to chronic inflammation and back to acute inflammation, depending on circumstances. Various phases of acute and chronic inflammation may be mixed, with one predominating. The outcome may be repair, but in most cases total breakdown and necrosis of the pulp ensues.

In this chapter pulpitis will be discussed under the headings of "Reversible Pulpitis" (an early, potentially reparative acute or chronic response); "Acute Pulpitis (Irreversible)" and "Chronic Pulpitis (Irreversible)"; and the possible end point, "Total Necrosis of the Pulp." Later, excerpts from an important clinicopathological study will be included to illustrate the significant change in concepts that is taking place in the study of the dynamics of pulpal inflammation.

Reversible Pulpitis

In response to external stimulation the pulp is able to protect itself by deposition of sclerotic or secondary dentin; but if the stimulation increases to the point of irritation, the pulp's response is inflammation. In response to moderate irritants, such as injudicious operative procedures, transmission of excessive thermal changes through metallic restorations, or toxins and bacteria in approaching caries, incipient inflammatory changes begin to take place in the pulp. These changes usually occur in the portion of the pulp subjacent to the dentinal tubules involved by the irritant.

The pulp reacts to deep-seated caries by dilation of vessels and the presence of chronic inflammatory cells, indicating that the pulp is being subjected to a low-grade irritant. This condition will resolve after the caries is removed and a proper restoration is placed. The pulp reacts to operative procedures (cavity preparation, chemical disinfection of the cavity, manipulative pressures) by a localized acute inflammation (SLIDE 10). Local blood vessels dilate; fluid leaks out and distends the surrounding connective tissue; polymorphonuclear neutrophils migrate to the region.

These teeth may be sensitive for s short period. Their response to external stimuli (cold, hot, or sweet food or drink) is exaggerated as compared with the response of an uninflamed tooth. Characteristically, the pain is of short duration, disappearing soon after the stimulus is removed. This pain response serves as a warning signal.

"Hyperemia" is the term that has been applied to the condition just described. Actually, hyperemia means an increased content of blood in a part with dilatation of blood vessels. Until recently it was generally accepted that the warning clinical symptoms were due to pressure induced by engorgement and vasodilation of the vessels in the pulp, in the absence of inflammation. Recent research[13] has shown that wherever dilated vessels are seen, serial sectioning reveals further evidence of inflammation. Thus, hyperemia is a part of inflammation and is not a discrete clinical condition. The characteristic sensitivity, or warning response, of the pulp is not hyperemia but incipient inflammation.

If the irritant is removed or discontinued and if the local defense mechanism is adequate, the inflammatory process may be resolved. The tissue may not return to a "normal" or uninflamed state but to an asymptomatic, clinically quiet, depleted state. If the foregoing conditions are not met, the inflammation proceeds in an irreversible direction.

Acute Pulpitis (Irreversible)

Acute pulpitis may arise as an incipient, potentially reversible reaction in a previously uninflamed pulp—for instance, in response to operative procedures. On the other hand, if the irritant is especially destructive or virulent, the acute response is irreversible. (This is especially true if the acute response is superimposed on pulp tissue that has endured previous inflammatory episodes). The acute inflammation soon spreads to other parts of the pulp. If the pulp is closed—or if, perhaps, a carious exposure is very small and there is little opportunity for drainage—there is marked pressure from the exudate and the entire pulp tissue undergoes rapid breakdown. Numerous abscesses form (SLIDE 11), and eventually the pulp undergoes liquefaction and necrosis.

The patient with advanced acute pulpitis usually experiences severe pain and desires immediate attention. It is this clinical picture that the dent l officer associates with acute pulpitis.

Chronic Pulpitis (Irreversible)

Chronic pulpitis occurs in response to a low-grade irritant, such as slowly advancing caries. If the irritant is removed, some measure of repair may occur. Without invasion of an overwhelming irritant, the pulp may remain chronically inflamed and asymptomatic for years.

In most cases the chronically inflamed pulp is doomed. If a carious pulp exposure occurs, incoming microorganisms set off an acute exacerbation. If the exposure is wide and drainage can occur, the acute reaction may subside and granulation tissue may be able to wall off the denuded area of the pulp for awhile, (SLIDE 12).

In any event, areas of necrosis eventually appear in the chronically inflamed pulp. The pulp is inevitably involved in its entirety by the chronic inflammatory process—and total necrosis is the long-term result.

Chronic hyperplastic pulpitis may be classified as a rather uncommon development in chronic pulpitis. When a widely exposed pulp exists in a young tooth with an incompletely formed apex and a liberal blood supply, the chronically inflamed pulp may actually succeed in its attempt to survive. In a valiant attempt at repair, granulation tissue grows exuberantly out of the carious cavity (SLIDE 13), forming an easily recognizable pulp polyp.

Total Necrosis of the Pulp

Necrosis is simply death of pulp tissue. It is the end point of pulpal inflammation. Thrombosis of vessels and failure of nutrition as a result of the inflammatory process cause the pulp tissue to die. First, portions of the inflamed pulp are involved; then total necrosis ensues. The pulp spaces are filled with an amorphous material (SLIDE 14).

Necrosis may occur directly, without inflammation. If a tooth is subjected to a hard blow or partial evulsion and apical vessels are crushed or severed, the pulp loses its source of nutrition. Starvation or strangulation of the entire pulp occurs rather rapidly, and total necrosis ensues.

Gangrene of the pulp should not be considered a special form of pulpal pathosis. Gangrene refers to a secondary infiltration of a necrotic pulp by saprophytic organisms and does not change the necrotic state. If the organisms are of the gas-forming group, the necrotic pulp is characterized by a disagreeable, foul odor.

Recent Research on Pulpal Inflammation

After the foregoing brief review of the broad categories of pulpal inflammation, it is considered highly important to include in this chapter excerpts from a clinicopathological research study conducted by Seltzer, Bender, and Ziontz.[13] These men investigated the histopathological status of pulps of 166 serially sectioned teeth and correlated these findings with previously recorded clinical signs and symptoms manifested in these same teeth. Their research brought to light many significant facets in the story of pulpal inflammation. It is strongly recommended that the entire article be read.

The authors found that the pulps they examined in their study could be divided into the following overlapping categories, on a histological basis:

1. Intact, uninflamed pulp
2. Atrophic pulp (the aged or traumatized pulp, still in the "normal range")
3. Intact pulp with scattered inflammatory cells (transitional stage)
4. Acute partial pulpitis (occurring in previously uninflamed pulp)*
5. Chronic partial pulpitis
   a. Without partial necrosis

- -

   b. With partial necrosis

6. Chronic total pulpitis (Acute exacerbation of chronic total pulpitis)
7. Total necrosis

In the article referred to, Seltzer et al.[13] did not discuss the possible outcome of the various histopathological conditions of the pulp. Subsequent personal communication with the authors has indicated, however, that in their opinion all the observed categories up to and including chronic partial pulpitis without necrosis may resolve, either spontaneously or following palliative treatment. On the other hand, chronic partial pulpitis with necrosis, chronic total pulpitis (always observed with areas of necrosis), and acute exacerbations of chronic total pulpitis are considered by the authors to be beyond hope of resolution; that is, these conditions may be termed "irreversible pulpitis." (The broken line indicates the line of demarcation between the conditions that may resolve and those that will not resolve.)

In tracing the development of pulpal inflammation Seltzer et al. found that pulpitis develops differently under the influence of caries than as a result of operative procedures. The following paragraphs describe the difference:

"Development of Pulpitis From Dental Caries.—The persistence of dental caries for weeks, months, or years provides a continuous stimulus for an inflammatory response to occur within the dental pulp. The pulp protects itself adequately in several ways. It reacts to the process of dental caries by the formation of sclerotic dentine in the primary dentinal tubules and also by the elaboration of reparative dentine under the region of the involved dentinal tubules. In effect, the pulp volume is reduced with the elaboration of reparative dentine, and the aging process of the pulp is accelerated. The formation of reparative dentine increases the collagenous portion of the pulp and decreases its cellular content. This, in effect, reduces the ability of the pulp to defend itself against further irritants. The amount of reparative dentine elaborated tends to keep pace with the amount of dentine removed by the oncoming process of dental caries. When the caries progresses at a more rapid rate than the reparative dentine is elaborated, the blood vessels of the pulp dilate and scattered cells of the chronic inflammatory series (predominantly macrophages and lymphocytes) become evident in the pulp tissue. They appear in small numbers at first, but gradually, as the decay involves the reparative dentine, the numbers increase and the exudate becomes much more proliferative. In terms of the numbers of inflammatory cells present, the response is mild inasmuch as the pulp is being only mildly irritated by the products elaborated from the carious process. As the decay comes closer to the pulp, more and more macrophages and lymphocytes are found scattered throughout the pulp, especially subjacent to the region of the involved dentinal tubules. Thus, the pulp is already chronically inflamed when it is almost exposed. Should the decay remain untreated,

*Acute pulpitis alone was not detected in this study, since none of the teeth was removed immediately following operative procedures. It has, however, been noted under experimental conditions by Stanley.[14]

with further decalcification of the dentinal tubules, a frank exposure occurs. The pulp reacts at the site of exposure with an infiltration of acute inflammatory cells (that is, polymorphonuclear leukocytes), and the chronic pulpitis then becomes acute. A small abscess develops within the coronal portion of the pulp under the region of the exposure. Here dead or dying polymorphonuclear leukocytes, together with dead tissue, are present. . . . The remainder of the pulp may be uninflamed, or, if the exposure has been present for a long period of time, the pulp may have been converted to granulation tissue. Once the pulp has become exposed by dental caries, the lesion may be called ulcerative pulpitis . . . , inasmuch as the surface covering of the pulp (the dentine) has been removed, leaving the pulp exposed to the oral fluids . . . . [Ulcerative pulpitis] usually requires endodontic treatment. Generally, the prognosis for capping an exposed, chronically inflamed pulp is poor. Recovery is possible but unlikely. Instead, the entire pulp may become acutely inflamed or total necrosis may ensue. If left untreated, the pulpitis may become acute as a result of food packing or pressure, or chronic pulpitis may gradually involve the entire pulp and consequently the apical tissues as well."[13]

"Development of Pulpitis from Operative Procedures. — Following operative procedures, such as cavity or crown preparation, pulp-capping or pulpotomy, restorations, etc., an acute partial pulpitis develops within the dental pulp. This may be mild or severe in intensity, and the involvement of the pulp may be partial or total. This may be superimposed over a 'transitional stage' or 'chronic pulpitis' which was already there as a result of dental caries, or it may occur within a previously uninflamed pulp. When restorations are placed in a tooth, this acute pulpitis may be converted to chronic pulpitis with eventual recovery, or the acute partial pulpitis may rapidly develop into an acute total pulpitis with accompanying involvement of the apical periodontal tissues. In the latter instance, an acute apical periodontitis occurs.

"As a rule, the acute partial pulpitis following operative procedures subsides and a chronic partial pulpitis ensues with eventual resolution. . . . following operative procedures, pulps may remain chronically inflamed for months or even years. . . . The persistence of chronic pulpitis for long periods of time may explain why pulps which remain symptomless following operative procedures may eventually exhibit painful symptoms. . . .

"Chronic pulpitis which has persisted following operative procedures may become acute when another operative procedure is carried out. This might explain pain episodes following minor traumatic procedures on previously treated teeth.

. . . .

"Chronic pulp inflammation under restorations may be partial, involving only part or all of the coronal portion of the pulp, or the inflammation may eventually involve the entire pulp (chronic total pulpitis). There is no actual separation between chronic total pulpitis and apical pericementitis. The apical tissues become involved once the entire pulp becomes inflamed.

. . . .

"Following mechanical exposure, an acute inflammation occurs in the pulp at the site of the exposure, but the remainder of the pulp is usually unaffected. The prognosis for healing following mechanical exposures of the pulp is much better than that following carious exposures. The reason is that the pulpitis which develops after mechanical exposure is usually not complicated by previous inflammation and infection. When treated, the acute inflammation is converted to chronic inflammation, and repair may then occur. Repair depends upon the amount of tissue destruction, the amount of hemorrhage, the patient's age (and, hence, the blood supply of the tissue), the resistance of the host, and other factors which influence the ability of the pulp connective tissue to repair itself in the face of injury."[13]

RETROGRADE ALTERATIONS OF THE PULP

Although the retrograde changes discussed here are considered to be noninflammatory, they are closely associated with and coexistent with inflammation of the pulp.

Calcifications

Pulp tissue has a tendency to form discrete foci and diffuse areas of calcification. Pulp stones (denticles) (SLIDE 15) are present in the pulp chambers of almost all teeth, although very few are visible roentgenographically. Most consist of amorphous, atubular calcified material; a few are composed of true dentin. Although they may be present in young, unerupted teeth, their size and number increase with age. Referred or obscure pulpal pain is sometimes attributed to pulp stones; however, the extirpation of pulps that have large pulp stones rarely eliminates such pain.

There is a tendency for calcium to be deposited upon atrophic or necrotic pulp cells. This deposit forms a nucleus for further adtion of calcium salts. Diffuse calcifications (SLIDE 16) are found in increased amounts in the chambers and canals of chronically inflamed pulps; that is, pulps that have been subjected to caries, operative procedures, and advanced periodontal involvement.

Internal Resorption

Internal resorption is a retrograde alteration of the pulp that causes resorption of the hard structures of the tooth. In most cases no clear cause can be determined, although a history of trauma is sometimes found. The pulp undergoes a change to a highly vascularized tissue closely resembling granulation tissue. Fibroblasts are converted to cells that resorb dentin from the pulpal walls. If the resorptive process begins in the pulp chamber, the vascular tissue may reach the enamel, where it is visible as a pink spot. If the process begins in the root canal, the resorption spreads laterally (SLIDE 17) and may perforate through the cementum to the periodontal ligament.

Although internal resorption may cease spontaneously, a program of "watchful waiting" is not justified; in all likelihood the process will proceed until penetration or fracture of the tooth occurs. Thorough endodontic treatment, if undertaken before perforation, will halt the resorption and save the tooth.

Pulp Aging

With increasing age, retrograde changes occur in the pulp; and this process is accelerated by external insults. In teeth that have been subjected to abrasion, attrition, erosion, extensive caries, or extensive operative procedures, the pulp undergoes atrophic changes; that is, diminution in the number of cells, increase in the amount of collagenous fibers, and profuse dystrophic calcification. The volume of the pulp is reduced by the formation of reparative dentin under the region of the involved dentinal tubules. The dentin sometimes obliterates almost the entire coronal portion of the pulp. Similarly, the lumina of root canals are narrowed. The presence of periodontal lesions also induces pulp aging. Pulps of periodontally involved teeth (without caries or restorations) show many regions of atrophy, necrosis, and calcification; and narrowing of the root canals is also evident.[13]

What does this mean to the clinician? Teeth of older patients and teeth in which pulp aging has been induced will have poor recuperative powers against further operative insults; prognosis for recovery from pulpitis is poor. When endodontic treatment is begun, the dental officer should expect to encounter constricted pulp chambers and canals. His instrumentation will be hampered by diffuse calcifications.

PERIAPICAL PATHOSIS

If diseases of the dental pulp do not resolve themselves, or if the root canals of pulp-involved teeth are not treated and completely sealed, trouble proceeds in an apical direction. Periapical pathosis is the body's response to irritants passing from the root canal(s) of a tooth into the periapical tissues. The periapical region becomes a battleground in which the defensive forces of the body attempt to defend themselves against bacterial warfare, chemical warfare, and traumatic warfare waged by irritants invading from the root canal. The site of this struggle—the battleground itself—is a periapical lesion.

In discussing this battle let us first examine the offensive forces. What are the injurious forces in the root canals that invade the periapical tissues and initiate the inflammatory response?

1. Bacterial irritants. Microorganisms that have broken down the defense mechanism of the pulp form a region of infection in the pulp. From this source, bacterial toxins and a few bacteria pass into the periapical tissues to produce continual lysis of the tissues.

2. Chemical irritants

a. Necrotic pulp debris in an untreated or inadequately filled canal breaks down into protein degradation products that gradually seep through the apical foramen. These irritating products provoke a continual low-grade inflammatory response in the periapical tissues.

b. Drugs used as root-canal medicaments may produce mild irritation in the periapical tissues, or they may cause severe inflammation. The amount of irritation produced is directly proportional to the nature of the drug, the amount used, the length of time it remains in the canal, and the size of the apical foramen.

3. Traumatic irritants

a. During endodontic treatment, repeated overextension of root-canal instruments and paper points traumatizes periapical tissues.

b. When a root canal is overfilled with a gutta-percha point, or more particularly a silver point, the extruded material, arcing with the masticatory movement of the tooth, stabs and tears periapical tissue.

c. With a sharp blow to the crown, the tooth itself acts as a battering ram, crushing tissues in the apical periodontal ligament space. If the foraminal vessels are severed, pulpal starvation necrosis occurs with consequent chemical irritation of the periapical tissues.

Opposing the offensive forces in the periapical region is the local defense mechanism—the tissue changes that constitute the inflammatory response. Compared with the situation in the pulp—in which the defense mechanism is seriously hampered by lack of space and lack of collateral circulation—in the periapical spaces the defensive capability is considerably greater.

The character of the inflammatory response in the periapical tissues depends on the strength of the attacking forces. Broadly speaking, if the attacking forces are highly toxic and have an overwhelming superiority over the local defensive capability of the tissues, the reaction is acute. (In a few instances highly invasive microorganisms may even penetrate the local defense mechanism and spread to adjacent tissues.) If, on the other hand, the invasive agent is mild and long lasting and the opposing forces are almost evenly matched, the inflammatory reaction will be chronic. Within the periapical region a stalemate develops that may continue for months or years. It should be made clear that this battle is seldom purely acute or purely chronic. The two types of reaction usually coexist, but one type may predominate over the other at any given moment depending upon which of the opposing forces is stronger. If treatment is begun and the canal contents are forced by overzealous instrumentation into the periapical region, a quiet, symptomless chronic lesion may quickly undergo an acute exacerbation.

It is important to emphasize that the focus of irritation, or the focus of infection, is located within the pulp cavity. The periapical lesion merely represents the body's response to the viable and nonviable irritants from the pulp cavity.

Classification

To lend order to a discussion of the development of periapical lesions, some type of classification is necessary. Several classifications are in existence, each excellent in its own right. However, all methods of classification cannot be used in this text: a reasonable choice must be made. Periapical lesions will be discussed under the following headings:

1. Acute periapical inflammation
   a. Acute apical periodontitis
   b. Acute periapical abscess
2. Subacute periapical inflammation ("Subacute" or "chronic" abscess)
3. Chronic periapical inflammation (Periapical granuloma)
4. Periapical cyst (Radicular cyst, periapical periodontal cyst, apical cyst)

It is important to realize that periapical lesions do not represent individual and distinct entities, but rather that there is a subtle transformation from one type of lesion into another type in most cases. Furthermore, some lesions are reversible.[15]

ACUTE PERIAPICAL INFLAMMATION

Acute periapical inflammation is a rapidly developing response elicited by irritants of overwhelming toxicity or virulence stemming from the root canal. The acute response may develop directly in vital periodontal tissue, or it may arise as an acute flareup, or exacerbation, in an already existing chronic inflammatory lesion. In order to trace the development of acute periapical lesions, the initial acute response will be described.

Acute Apical Periodontitis

This term refers to acute inflammation in the apical periodontal ligament tissues and immediately adjacent alveolar bone. Acute inflammation does not usually stay long in this limited space.

What happens if highly virulent microorganisms or their toxins move (or are forced) apically through the apical foramen? A rapid response occurs in the soft tissues of the confined periodontal ligament space, with rapid accumulation of exudate. Since there is no time for resorption of bone, the hydraulic pressure of the exudate actually raises the tooth in its socket. This is visible roentgenographically by a widening of the periodontal ligament space and an intact lamina dura (SLIDE 18) (in roentgenograms the cribriform plate, or alveolar bone proper, appears dense and thus is frequently called the lamina dura). The tooth feels elongated and is sensitive to percussion and to masticatory pressures because the nerves in the periodontal ligament are irritated by the inflammatory process.

In mobilizing emergency defensive measures against the overwhelming attack, the local defensive forces bring in thousands of polymorphonuclear neutrophils, many of which are killed in the increasingly acid environment. Near the apical foramen a nucleus of pus is formed, surrounded by newly arrived neutrophils.

In the presence of this overwhelming irritant there is no time for a fully organized defense—for a connective tissue walling off of the irritant, as in chronic inflammation. As more tissue is destroyed and fluid accumulates and more defensive cells are killed, pus builds up and, demanding escape, pushes through the numerous spaces in the cribriform plate and into the marrow spaces of the surrounding bone.

As the inflammation spreads beyond the periodontal ligament spaces into bone, the reaction is frequently labeled "acute alveolar abscess," or "acute periapical abscess."

Acute Periapical Abscess

Within the bone, still under pressure, the purulent exudate moves slowly through the cancellous spaces, seeking a path of least resistance. (Actually, a localized osteomyelitis exists. In rare instances when the virulence of the microorganisms is high and the resistance of the patient is low, a widespread osteomyelitis might occur.)

In a few days the pus reaches and perforates either the buccolabial or the lingual cortical plate of bone. As the pus emerges, it meets considerable resistance from the tough periosteum. Under pressure, the purulent exudate spreads along the bone surface, separating the periosteum—together with the overlying mucous membrane—from the bone. This action increases the pain to such an intensity that the patient usually seeks help. Opening the root canal (or reopening it if treatment has already begun) will allow drainage and relieve the pain almost immediately in most cases. Sometimes the pus is too viscous to flow through a narrow apical foramen. When this becomes apparent, the inflammatory exudate may be induced by warm saline rinses to "point" on the overlying mucous membrane. Drainage of pus, with consequent release of pressure, may then be obtained by incision or spontaneous rupture of the mucosa, and pain is alleviated (SLIDE 19).

The course and point of discharge of a periapical abscess varies; however, the following guidelines may be of value. Pus follows the line of least resistance and, in general, takes the shortest path to the surface. It is somewhat affected by gravity. In the maxilla, root apices are closer to the buccolabial cortical plate, and discharge usually occurs on that surface (SLIDE 20, A). However, abscesses associated with maxillary lateral incisors (SLIDE 20, B) and palatal roots of maxillary molars may discharge palatally (SLIDE 20, C). Abscesses associated with palatal molar roots may discharge into the antrum (SLIDE 20, D). In the mandible, discharge usually takes place in the vestibule along the buccolabial cortical plate (SLIDE 21, A), but in abscesses associated with lower molars the lingual cortical plate may be pierced (SLIDE 21, B). Pus may burrow along the side of a periodontally involved tooth and discharge at the gingival line (SLIDE 21, C), but a healthy periodontal lingament is usually too dense and firm to allow passage.

Under certain circumstances an acute periapical abscess takes an especially violent course. It is believed that in these cases highly virulent and invasive microorganisms are certainly the attacking agents. Some microorganisms spread rapidly and diffusely and, by producing increased permeability of capillaries, cause marked serous inflammation and swelling of the soft tissues ("cellulitis"), which tends to spread widely through tissue spaces and along fascial planes of the face (SLIDE 22). This condition is frequently accompanied by signs of general toxemia.

Treatment of these acute conditions will be discussed later. In current practice, the severely acute conditions are seldom encountered because patients seek help sooner than they used to and because the supportive action of antibiotics is available.

SUBACUTE PERIAPICAL INFLAMMATION ("SUBACUTE" OR "CHRONIC" ABSCESS)

After the distressing symptoms of the acute periapical abscess have subsided and comfort is restored, some patients do not follow through with recommended endodontic therapy. In this event the abscess may waiver between periods of quiescence and episodes of discharge for a period of time. The periapical abscess under these conditions may be termed "subacute."

With the release of pus and reduction of pressure, the tide of battle is changed in favor of the tissues, and attempts at repair begin. Viewing the abscess region—or space just outside the apical foramen—under the microscope, we would see a core of pus surrounded by a primary wall of defense (densely packed neutrophils). Around this we would see a zone of cellular activity (plasma cells, lymphocytes, macrophages, young fibroblasts, and newly formed capillaries) constituting granulation tissue. The gradual flow of irritant from the root canal attacks the wall of neutrophils and continually produces pus, while the new capillaries continually bring in a fresh supply of neutrophils to maintain a balance of power.

The pus escapes through the drainage tract formed in the acute stage. If pus formation continues to be active, the tract may remain open; but if pus is formed slowly, the tract may be closed temporarily at the surface by proliferation of epithelium. When a mass of pus has accumulated, the resultant pressure forces it along the drainage tract and a vesicle ("parulis") (SLIDE 23) is formed in the mucosa. The vesicle ruptures and disappears until the next exacerbation.

In an attempt to establish a state of equilibrium with the irritant, granulation tissue proliferates. As it does so, bundles of collagen become condensed on the periphery to separate the granulation tissue from the bone.

Microscopically, quite a picture of chronicity is presented: a wall of granulation tissue with a collagenous capsule, surrounding a periapical core of smoldering suppuration (SLIDE 24). The lesion is quiescent clinically. For these reasons many investigators choose to term the lesion at this stage a "chronic periapical abscess" (or "chronic alveolar abscess").

Because of the persistent nature of the irritant stemming from the root canal (usually pyogenic microorganisms) the defensive and reparative capabilities of the granulation tissue are seldom sufficient to resolve the periapical core of tissue disintegration and suppuration. It is a standoff struggle for supremacy. There is very little difference between a granuloma that is attempting to contain irritants within the pulp of a tooth and a chronic abscess that is attempting to contain irritants within a tooth and within an area of varying size around the apex of the tooth.[16]

It is important to point out that whereas the chronic abscess may represent a "quieting down" or resting stage of an acute or subacute periapical abscess, as just shown, it arises more often as a slow but steadily progressive disintegration, with suppuration, of a periapical granuloma. This may happen when the tissue's defenses are not able to cope with a new influx of pyogenic microorganisms from the canal or in a few instances from the bloodstream. Sometimes a drainage tract may be slowly formed without disturbing symptoms.

What eventually happens to these tracts, or pathways, that permit drainage of periapical abscesses? Since the tract is lined with chronic inflammatory tissue that becomes strongly reparative as soon as the irritant is reduced or removed, it is not difficult to understand that when endodontic treatment is instituted the inflammatory drainage tract (fistula) closes and heals quickly, often before treatment is completed. Very infrequently, in longstanding untreated cases, oral epithelium may proliferate into and line the tract. In this event the tract may remain patent, maintaining the periapical lesion in communication with the oral cavity. The treatment of choice in this case is surgical curettage of the lesion accompanied by careful surgical excision of the epitheliated tract.

CHRONIC PERIAPICAL INFLAMMATION (PERIAPICAL GRANULOMA)

Chronic periapical inflammation is a slow, well-organized response of the periapical tissues to a mild irritant. It is a transformation of the apical periodontal ligament and adjacent bone into a defensive and reparative tissue—granulation tissue. This chronic response is initiated by mild irritants emanating from the root canal.

In review, what are these irritants? Bacterial colonies of low virulence, living in the culture medium provided by a necrotic pulp and protected from bodily defenses, exert a continual toxic effect upon the cells of the periapical tissue. Necrotic tissue in untreated or grossly underfilled canals furnishes a continual supply of protein degradation products which, in turn, produce continual lysis of periapical tissue.

Chronic periapical inflammation does not just suddenly develop. In many cases it is an extension of chronic inflammation in the root canal. In any event, when the toxins from the canal reach the vital, vascular connective tissue of the periodontal ligament space, these tissues respond with an organized exudative and proliferative inflammatory reaction. As a chronic exudate slowly builds up in the tissues of that narrow space between unyielding bone and cementum, it is clear that room must be gained. Therefore, vital apical connective tissue is stimulated to form special giant cells, or osteoclasts, whose function it is to resorb bone. These osteoclasts gradually resorb the dense bone lining the alveolus and may proceed into cancellous bone. (It should not seem strange that similar giant cells are busily engaged in resorbing the cementum and in some instances the dentin of the root apex.)

Bone continues to be resorbed slowly until the inflammatory exudate can dilute, phagocytize, and in some way reach a state of equilibrum with, the invading toxins. However, even as the lesion is expanding, this chronic inflammatory exudate—this granulation tissue—is laying the groundwork for repair: new connective tissue fibrils and budding vascular channels permeate the space.

It is essential to point out that from the very beginning, as the granulation tissue expanded in the periodontal ligament space, it pushed and compressed vital connective tissue ahead of it, forming a dense collagenous capsule between itself and the bone. This capsule is continuous with the periodontal ligament. Thus, this radiolucent lesion constitutes a wall of granulation and collagenous tissue that has been built around the apex of a tooth in an effort to contain the invading irritants within the canal (SLIDE 25).

The lesion just described is referred to in most professional literature as a "periapical granuloma" or a "dental granuloma." The term "granuloma," although criticized by some authorities, will be used here because of its widespread identification with the lesion.

The periapical granuloma represents an uneasy stalemate between the persistent irritant and the strong tendency for healing by the host tissues. The balance could be tipped either way.

If a powerful irritant, such as highly virulent microorganisms or a highly irritating drug, were forced from the root canal into the granuloma, an acute flareup, or exacerbation, would occur. Under these conditions the chronic inflammatory exudate would change to an acute type characterized by polymorphonuclear neutrophils and eventually by pus. An inflammatory tract, or "blowoff valve," might be created. Without treatment the lesion would in all probability quiet down to a standoff state—the chronic alveolar abscess.

If, on the other hand, the irritant were largely subdued, the granulation tissue would become predominantly reparative—filled almost solidly with plump fibroblasts, young connective tissue, budding new vascular channels. (This would account for its clinical appearance on an extracted root apex: a reddish sac, granular in texture.)

If the irritant were completely eliminated by competent nonsurgical endodontic therapy and the local reparative mechanism were adequate, the lesion would go on to complete healing—hopefully, osteogenesis—unless a cyst were forming in the granuloma.

PERIAPICAL CYST (RADICULAR CYST, PERIAPICAL PERIODONTAL CYST, APICAL CYST)

At some time during its existence at the apex of a pulpless tooth, the mass of chronic inflammatory tissue known as a "periapical granuloma" may give rise to a periapical cyst. When a cyst does occur, it is a true cyst—a closed epithelium-lined space containing a fluid or a semifluid. Even though it is a cyst within a granuloma, the histological diagnosis becomes "periapical cyst."

There are several theories concerning the method of formation of a periapical cyst, but only the most widely accepted one will be discussed. Understanding the method of formation of a cyst is of great benefit in understanding the rationale of treatment.

One fact regarding granulomas that has not been mentioned is the presence of epithelium. Serial sectioning has revealed the presence of nests, cords, or sheets of epithelium in all granulomas observed microscopically. This epithelium originates in nearly all cases from remnants of Hertwig's sheath (debris of Malassez), found in the periodontal ligament (SLIDE 26). In some instances, however, this epithelium may arise from (1) respiratory epithelium of the maxillary sinus when the periapical lesion has perforated the sinus wall, (2) oral epithelium proliferating inward through an inflammatory tract, or (3) oral epithelium proliferating apically from a periodontal pocket.[17]

Stimulated by the underlying inflammatory process, the epithelium proliferates from one or several foci and forms a large mass of cells. Since the epithelium has no blood vessels of its own, its blood supply must come from the surrounding connective tissue. Because the central cells in the epithelial mass are farthest from the blood supply, they degenerate and form a small cavity, which is filled with tissue fluid and cell debris and lined with epithelium (SLIDE 27). This is the beginning of the periapical cyst. Since cell debris is composed of protein, the intracystic pressure becomes greater than the pressure in the surrounding granulation tissue. Tissue fluid is therefore drawn into the cystic cavity by osmosis. The slight hydraulic pressure produced within the cyst is exerted against the granulation tissue-connective tissue wall. This outward pressure stimulates osteoclastic destruction of bone (with a resultant increase in the size of the lesion) (SLIDE 28).

If the periapical cyst remains untreated, it imbibes more fluid and slowly increases in size at the expense of the surrounding bone. Cysts vary considerably in size, ranging from a few millimeters in diameter to a space involving two or more teeth. Quite rarely, these cysts involve almost an entire quadrant of jaw.

The treatment of cysts consists in thorough surgical curettage of the entire cystic sac. When a very large cyst involves neighboring structures, the cyst should be reduced in size before surgery is performed. It thus becomes important to point out that if an opening through the cyst wall to the oral cavity were to be created and maintained, generation of internal pressure would be impossible. The cyst would collapse and begin decreasing in size. This aspect will be discussed in the chapter on surgical endodontics.

HEALING OF PERIAPICAL LESIONS FOLLOWING ENDODONTIC THERAPY

In review, an acute periapical inflammatory lesion becomes subacute following proper emergency treatment. With further reduction of the irritant, the lesion may become chronic. The histological picture of a subacute periapical lesion (subacute abscess) is that of a wall of chronic inflammatory exudate—granulation tissue—trying to close in on a sometimes rebellious purulent core at the apical foramen. The picture of chronic periapical inflammation (granuloma) is that of an encapsulated, predominantly reparative wall of granulation tissue with evidence of a slight area of necrosis at the apical foramen.

Two facts regarding granulation tissue should be recalled: (1) This is first of all a defensive tissue, or exudate, containing cells with antibacterial and antitoxic properties; (2) it is next a tissue of repair, featuring proliferation of new connective tissue fibers and vascular channels.

For many years competent endodontists advocated root resection and periapical curettage as the best means of removing <u>all</u> of these inflammatory periapical lesions—considered to be dangerous pathoses. Although these men were sure that infected canals could be sterilized and completely sealed, they did not know whether the periapical tissue might still harbor microorganisms and hence could represent a source of residual infection.[18]

In 1950 a study was undertaken by Hedman[19] to determine whether microorganisms still remained in the periapical tissues after the canal had been rendered sterile. By a very ingenious method Hedman inserted cannulas through the root canals of selected teeth and took samples of periapical tissue for culturing. His investigation showed that microorganisms could not be recovered from the periapical tissues after the sterility of the canal had been established.

This investigation, corroborated by Shindell,[20] demonstrated that the main focus of infecting microorganisms is in the root canal and that once infection has been eliminated from within the canal the defensive cells of granulation tissue are able to dispose of any residual organisms in the periapical area. It has become apparent to endodontists that resection and curettage remove the very tissue which nature has laid down as a defensive and reparative tissue. By removal of the granulation tissue, the return of the area to normal bone would be considerably retarded.

It has gradually become common practice in cases in which a periapical radiolucency is associated with pulpal involvement to proceed with endodontic therapy and postpone surgical resection or curettage in the hope that the periapical lesion will eventually be replaced by new bone formation. The fact that this can and does occur has been demonstrated by literally hundreds of clinicians and researchers.

When all irritants (bacterial or otherwise) have been removed from the canal and the canal has been thoroughly filled, the balance of power is shifted strongly in favor of the local tissues. With the irritant gone, the defensive forces are soon disbanded and the reparative forces take over; that is, the inflammatory cells in the granulation tissue gradually recede, fibroblastic activity increases, and vascularity diminishes. In the fibrous mass tiny spicules of osteoid tissue appear, surrounded by bone-forming cells, or osteoblasts. The spicules enlarge and fill the entire fibrous defect in a haphazard, crisscrossing pattern. Soon calcification of the osteoid tissue begins to occur. Roentgenographic evidence of healing lags far behind actual osteogenesis because there is insufficient calcium in new bone to stop the roentgen rays. Over a period of several months, differing with each patient's capabilities for repair, osteogenesis will be complete. Roentgenographically, there will be no sign of the original lesion.[21]

Most lesions will heal readily without further treatment following competent, nonsurgical root-canal therapy, but a small percentage stubbornly refuse to heal.

Why does osteogenesis fail to take place? There are four main reasons:

1. Failure or inadequacy of the local reparative power of the tissue.

2. Persistence of a source of irritation which is not detectable by roentgenographic observation and which the tissues are not able to overcome. Examples would be a hidden, extruded silver point or unfilled accessory canals packed with necrotic debris.

3. Filling in of the bony defect by healthy, dense, fibrous connective tissue ("scar" tissue). In these few cases (about 2 percent of all lesions) repair does occur—not by regeneration of bone but by replacement of bone with a less specialized tissue: connective tissue. These lesions are radiolucent and are not clinically distinguishable from unhealed periapical lesions.

Penick[22] has observed that healing by scar tissue has occurred where there is a history of a longstanding lesion that has destroyed the overlying plate of bone and the periosteum. He has suggested that these predisposing factors may permit an ingrowth of fibrous tissue from the overlying mucosa during the healing process.

4. Transformation of the lesion to a cyst. In a periapical lesion in which there is a central cavity completely lined with patent epithelium, bony healing cannot occur. The only prognosis is maintenance of the status quo or slow expansion until surgical intervention is accomplished.

For years authorities have suspected that some cysts may heal spontaneously, without surgery. Recent investigation[23] has strongly pointed toward the possibility that many cysts may be resolved (without specific intent) following nonsurgical endodontic therapy. It is possible that as a result of overinstrumentation or the formation of an acute inflammation within the cyst a portion of the epithelial lining may be destroyed. Granulation tissue then may proliferate into the cystic cavity and, following complete elimination of the irritant by root-canal therapy, the lesion may resolve. Further research is in progress.

This concept may account for the unplanned, spontaneous healing of some cysts. However, the treatment of choice for all periapical radiolucent lesions that persist for a reasonable period (6 to 12 months depending on the original size of the lesion) following nonsurgical endodontic therapy is surgical curettage.

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CHAPTER 3
DIAGNOSTIC PROCEDURE IN ENDODONTICS

The diagnostic approach to pulpal and periapical pathosis cannot be hit and miss but must be organized. A great number of patients who come for treatment with pulpal or periapical problems are experiencing some degree of pain, but even under emergency conditions it is highly important that the diagnostic procedure be systematic.

The heart of the diagnostic procedure is the examination of the patient. This examination covers the medical and dental history, the subjective symptoms, clinical observations, clinical tests, and the roentgenographic examination.

The purpose of the examination is to provide signs, symptoms—clues—that will allow the dental officer to interpret what changes (if any) are transpiring in the pulp and the periapical tissues of suspected teeth. By means of examination (plus cerebration) the dental officer should be able to:

1. Locate the involved tooth.
2. Determine the extent of pulpal involvement.

    a. First, it must be decided whether pulpal inflammation is reversible (the inflammation may resolve) or irreversible (inflammation has proceeded too far to be resolved, and endodontic treatment of some type must be instituted).

    b. If inflammation is judged to be irreversible, the examination should further indicate the nature and severity of the pathosis as an aid in choosing the direction of endodontic treatment.

3. Determine the existence and status of periapical inflammation stemming from the pulp.

The consequences of diagnostic error can be quite serious to the patient. For example, if the dental officer correctly interprets the symptoms of early, reversible pulpitis, he can remove all possible sources of irritation; if caries is the irritant, he can remove it and apply a sedative dressing that will in most instances eliminate the pain and preserve the health of the pulp tissue. If, on the other hand, he interprets the symptoms incorrectly, he may remove the pulp or extract the tooth unnecessarily.

EXAMINATION

The examination should be conducted with a definite routine. To accomplish this, the dental officer will find it helpful to use a chart as a guide. He should remember that no one test is infallible and that he must correlate the results of several tests to arrive at the diagnosis. The essential information is discussed in the following paragraphs.

Medical and Dental History

The medical history should include a record of sensitivities to, or reactions to, any drugs or antibiotics; the presence of cardiovascular disease (especially valvular disease with a history of rheumatic fever); blood dyscrasias; hormonal diseases, and so forth. It should include a record of current or recent medication and past exposure to therapeutic doses of radiation. Knowledge of these systemic factors will modify subsequent local treatment.

The dental history should include a history of trauma and of dental procedures. History of trauma to a local area, even years ago, would indicate a possibility of pulp deaths in that area. A history of dental procedures, such as pain following a recent pulp capping or placement of an extensive restoration, would focus attention on a particular tooth.

Subjective Symptoms

What the patient has to say regarding his symptoms is important and deserves attention. Many of his presenting symptoms are centered around pain. Every dentist is aware of the wide range in reaction to pain among individuals. A mild stimulus in a highly neurotic or apprehensive patient may cause what seems to him to be excruciating pain; whereas a much stronger stimulus in another individual may cause pain that is scarcely noticed. The state of health plays an important role in the reaction to pain.

The task of the dental officer is to interpret these symptoms. There is no foolproof method of relating clinical symptoms to the exact histopathological status of the pulp. Over past years each of the traditional phases of pulpal inflammation has become matched with a certain set of clinical signs and symptoms—for instance: Heat aggravates and cold may relieve acute suppurative pulpitis; a throbbing pain denotes more advanced pulpitis than a sharp, intermittent pain; a hyperemic pulp requires less current than a control tooth to elicit a response, and so on. Using this widely accepted method and entering the pulp, the clinician has often been chagrined to find that his diagnosis was wrong.

In recent years investigators have attempted to re-evaluate previous data and have stirred up doubts regarding their validity. Larger samplings of teeth have been used; the study of serial sections has been employed to great advantage. Mitchell and Tarplee[1] provided evidence that evaluation of the type or degree of pulpitis by the response to either heat or cold is misleading, since in their study most patients with any type of pulpitis exhibited increased sensitivity to both heat and cold.

Based on their study comparing clinical signs and symptoms with the histological status of 166 teeth, Seltzer, Bender, and Ziontz[2] have provided certain new guideposts for the diagnosis of pulp-involved teeth. Their observations are summarized in part as follows:

1. Previous history of pain is an important diagnostic means of establishing the presence of destructive pulpal pathosis.

2. Severity of pain is only partially related to severity of the inflammatory response. Severity of pain appears to be related to the patient's previous experiences, his ethnic background, whether or not drainage has been established, and other factors. (In their study the most severe pain occurred when the diagnosis was chronic partial pulpitis with partial necrosis.)

3. There is no significant correlation between the character of pain and the histological diagnosis, although the authors observed that there appears to be a trend toward an increased awareness of the various types of pain (sharp, dull, throbbing, localized, diffuse, intermittent, and continuous) in the categories of chronic partial pulpitis and chronic total pulpitis with necrosis.

4. Pain responses to heat or cold are not pathognomonic for specific types of pulpal inflammation. Inflamed pulps may react with pain to heat or cold, or both.

Quite apart from the observations of Seltzer et al, it should be brought to the reader's attention that the onset and duration of pain may be significant. If pain is caused by a stimulus (such as hot, cold, or sweet food or liquid) and ceases when the stimulus is removed, this may be a warning signal that the pulp is in an early stage of inflammation. Search should be made for a causative factor, such as a deep metallic restoration with an inadequate base. If this factor is removed, the pulp may recover. If, however, a stimulus triggers pain that continues for hours after the stimulus is removed, or if pain occurs without apparent provocation and persists for long periods, this behavior is indicative of advanced, irreversible inflammation of the pulp.

Clinical Observations

Visual examination should not be confined to the oral cavity but should take in the patient as a whole. Any questionable signs or symptoms should be investigated thoroughly and discussed with the medical officer.

Within the oral cavity the following observations are significant:

1. The presence of a deep carious lesion or recurrent caries beneath a restoration points to the possibility of a carious exposure. Deep caries or questionable, cariously involved restorations should be examined carefully. If a pulp exposure can be found, the likelihood is great that this is the involved pulp and is the source of the pain. If a frank exposure can be found beneath deep caries or an extensive restoration, the prognosis of the pulp is poor because preexisting inflammation has depleted the pulp's recuperative powers.[2] Endodontic therapy is indicated.

2. Discoloration of the tooth is often a manifestation of pulp death, caused by the penetration of dentin by breakdown products of the necrotic pulp. However, the dental officer should be aware of the fact that discoloration may also be present in a vital tooth. A young tooth with open root ends may survive a hemorrhagic inflammatory reaction after a blow, and the pulp may recover even though the

dentin has been discolored by pigment from the breakdown of red blood cells.

3. Observation of a draining or quiescent discharge point of an inflammatory tract ("fistula") immediately suggests pulpal-periapical involvement. Actually, either a periapical or lateral periodontal abscess may be the causative agent. Placement of a thin, flexible, sterile wire into the tract will often reveal, by roentgenography (SLIDE 29), the source of inflammation. The pulp may be found innocent.

Clinical Tests

Application of Heat or Cold.—The application of extremes of cold (ice cone or ethyl chloride spray) or heat (hot ball burnisher or heated gutta-percha) will assist in locating an inflamed pulp. The concept that reaction to cold alone indicates reversible pulpitis ("hyperemia") and that reaction to heat alone indicates advanced pulpitis is not dependable. There is no magic in the difference between heat and cold: they are both irritants. Actually, an inflamed pulp will react to heat or cold, or both. A totally necrotic pulp will not respond to heat or cold. The idea that heat expands the "gas" in gangrenous pulps to produce pressure pain has been largely discounted.

Percussion.—Pain on percussion immediately suggests pulpal-periapical inflammation. Actually, pain on percussion should immediately suggest inflammation of the periodontal ligament, which may stem from traumatic occlusion, or from periodontal or pulpal disease. When there is pain on percussion, it is wise to look first for an occlusal prematurity, which might be caused by slight drifting of the tooth or a recently placed "high" restoration. Exploring the gingivae and probing for possible periodontal pockets may lead to the source of pain. If occlusal prematurity or lateral periodontal involvement is not discovered, the pulp is likely to be the culprit. When pulpal inflammation reaches the periapex, the pulp is considered to be irreversibly involved.

Electric Pulp Tester.—The electric pulp tester is a useful diagnostic aid, if its limitations are known. This instrument, by sending an electric current through the pulp, indicates with fair accuracy the presence or absence of living nerve tissue in the pulp. Patients may respond to the electric pulp tester at various readings on the scale, but these readings do not accurately correspond to degrees of involvement of the pulp. The concept that a lower reading than that for a normal control tooth means "hyperemia" or early acute pulpal inflammation—or that a higher reading means advanced pulpal inflammation—is not borne out by histological evidence.[2] A major shortcoming of this test is that the selected control tooth is not always normal but may be chronically inflamed.

The electric pulp tester is most accurate in diagnosing a necrotic pulp by its lack of response. However, the dental officer should be aware that a response is sometimes elicited from a pulp that has undergone liquefaction necrosis. In other instances, though no response to the tester is detected, vital nerve tissue may still be present in the apical portion of the root canal of an anterior tooth, or in one of the canals of a posterior tooth. Thus, caution should be observed when entering any canal without anesthesia.

When the electric pulp tester is used, it is important that the dental officer not contribute to the patient's apprehension by using such words as electricity, current, pain, or shock. The patient should merely be asked to raise his hand when he feels a warm or tingling sensation in the tooth. The procedure for using the pulp tester is as follows: The tooth is isolated under cotton rolls; the crown is air dried; and the electrode, coated with an electrolyte such as toothpaste to ensure contact, is placed on sound tooth structure about 3 to 4 mm. from the gingiva. The current is increased slowly until the patient signals a response.

Test Cavity.—Application of a bur to the dentino-enamel junction will reveal whether a dental pulp is vital or not. When the electric pulp tester cannot be used effectively because of the presence of extensive restorations, or when the results of other pulp tests are extremely doubtful, the test cavity can be used as a last resort.

Anesthetic Test.—When a pulp-involved, painful tooth cannot be located with certainty by other methods, the anesthetic test is used. This test is based on the fact that pain, even when referred, does not cross the midline of the face. Usually the patient can tell whether pain is present in the upper or lower jaw. By means of systematic infiltration or conduction, teeth are anesthetized (moving in an anterior direction in the maxilla and in a posterior direction in the mandible) until the pain ceases and the involved tooth is located. If the pain continues, it evidently does not stem from the teeth—provided, of course, that the anesthetic has been administered properly.

Trial Treatment.--Diagnostic tests do not always pinpoint the trouble but may narrow the field of suspicion to one or two teeth. On the other hand, the involved tooth may be located but the extent of involvement may be uncertain. Where such uncertainty exists, the dental officer is justified in undertaking trial treatment. The procedure is as follows: Under the rubber dam, caries and questionable restorations are removed. If a definite exposure is found beneath deep caries or deep restorations, the prognosis for the pulp is poor, as previously explained, and endodontic treatment should be decided upon. If no exposure is found, a treatment restoration (zinc oxide and eugenol cement) is placed for a trial period of several days or weeks. With a cooperative, understanding patient this waiting period may give time for emergence of new symptoms. If the toothache disappears and the suspected tooth (or teeth) remains vital for a period of approximately 6 weeks, the prognosis is favorable and a permanent restoration may be placed.

Roentgenographic Interpretation of Pulpal Pathosis

The roentgenogram, when properly exposed and processed, can be a valuable aid in diagnosing pulpal (as well as periapical) inflammation. However, the dental officer must learn to interpret what he sees. The roentgenogram will not directly show pulpal inflammation, infection, or degeneration; it will show factors that predispose to pulpal involvement. In examining the crown and root, one should look for unusually deep cavities or restorations, root fracture, and internal or external resorption.

In examining the periapical region, especially if a definite radiolucent lesion is not present, one may still detect evidence of advanced pulpal disease. If the periodontal ligament space is widened (SLIDE 30), this points to extension of pulpal inflammation into the apical periodontal space. If the continuity of the lamina dura is interrupted around the root apex (SLIDE 31), this is a strong sign that the pulpal-periodontal tissue inflammation is extending into and resorbing bone.

Roentgenographic Interpretation of Periapical Pathosis

With careful, knowledgeable interpretation, roentgenograms of good quality can be an effective means of diagnosing periapical lesions.

In the interpretation of periapical radiolucencies, several factors must be kept firmly in mind. First, the osteolytic process moves in advance of and is more extensive than the radiolucent image of bone destruction on the roentgenogram. In their investigation of artificial lesions made in mandibles from human cadavers, Bender and Seltzer[3] showed that intrabony lesions often may not be detected roentgenographically until there is perforation or considerable resorption of the overlying cortical bone. They demonstrated that lesions in cancellous bone cannot be detected roentgenographically. Thus, extensive disease of bone may be present even when there is no evidence of it on roentgenograms.

Second, it must be kept in mind that the type of periapical lesion cannot be established through the roentgenogram. Many investigators[4-7] have demonstrated clearly that the dental granuloma, periapical cyst, or periapical abscess cannot be differentiated on the basis of roentgenographic evidence alone. Kronfeld warned: "It cannot be overemphasized that roentgenograms do not show infections, granulomas, or cysts, but merely differences in the relative density of the bone shadow."[8]

Lastly, the dental officer must realize that all periapical radiolucencies do not stem from pulpal pathoses. Radiolucent areas, normal or abnormal, not related to pulpal pathosis may appear superimposed over the root apices. To avoid mistakes in diagnosis it is necessary to have a full knowledge of the most common radiolucent areas around the apices, whether they represent normal anatomical areas or radiolucent areas due to other diseases.

Anatomical areas that may be superimposed over root apices are the nasopalatine foramen, the maxillary sinus, the mental foramen, or prominent nutrient canals. Pathological lesions that might be mistaken for sequelae of pulpitis are a median maxillary cyst (SLIDE 32), a globulo-maxillary cyst (SLIDE 33), a hemorrhagic traumatic bone cyst (SLIDE 34), a lateral periodontal cyst (SLIDE 35), a neoplasm

(SLIDE 36), or a cementoma (especially in its early stage of development) (SLIDE 37).

In order to establish a differential diagnosis and to eliminate the possibility of confusing any anatomical or pathological areas with periapical pathosis, several procedures may be employed:

1. Take several roentgenograms at various angles. Anatomical areas will shift from the apex with the change in angulation (SLIDE 38). A radiolucent area due to pulpal pathosis will not move (SLIDE 39).

2. Study the roentgenograms carefully. The presence of an unbroken lamina dura is a strong indication that no periapical pathosis is present.

3. Take electric pulp tests. If a response is elicited, this may establish the vitality of the tooth and prevent error in recognition of lesions (such as a cementoma) which bear a strong roentgenographic resemblance to a granuloma or a cyst. If no response is elicited, the lesion may be presumed to be a sequela of pulpal pathosis.

In summary, the diagnosis of pulp disease and its sequelae is made on the basis of accumulative evidence. This evidence is obtained by means of a history, a visual examination, roentgenograms, the electric pulp tester, percussion, and by any other means that might be helpful. A diagnosis should not be made on the basis of evidence obtained by means of only one diagnostic aid.

DIAGNOSTIC SUMMARIES

DIAGNOSTIC SUMMARY OF PULPAL PATHOSIS

Reversible Pulpitis

The clinical category of "Reversible Pulpitis" includes those conditions of the pulp that are capable of resolving--not necessarily returning to a completely "normal" state, but possibly to an asymptomatic, atrophic state. Acute pulpitis arising in a previously inflamed pulp as a result of operative procedures is in most cases reversible. Similarly, a chronic partial pulpitis (without partial necrosis) may, to some extent, undergo repair.

Symptoms

Reversible pulp changes may occur on a subclinical, asymptomatic level. Gradual repair of inflamed tissue may occur without knowledge of the patient. Sometimes, however, the pressures incident to this localized inflammation—distention of tissues by exudate--cause the pulp to respond in an exaggerated or hyperreactive manner to various stimuli. The pulp may react with transient pain to cold, hot, or sweet food or drink. If the pain is of momentary duration and disappears when the stimulus is removed, and if the pain is not spontaneous but occurs only in response to a stimulus, this is an indication (not a guarantee) that early inflammatory changes are occurring in the pulp and may be reversible.

Clinical Observations

Any one of several situations may be visible—for example, a recently placed restoration or a moderate carious lesion (no exposure). Translucency of the crown is not impaired.

Clinical Tests

Thermal: Exaggerated response to extremes of temperature (heat or cold) of short duration.

Electric pulp test: Inconclusive; may require less current than "control" tooth to elicit response (if control tooth is indeed normal).

Percussion: No discomfort (unless other factors, not related to the pulp, are involved.)

Roentgenographic Appearance

A deep, poorly insulated metallic restoration or recurrent caries beneath a restoration may be visible roentgenographically. There is no periapical change.

Clinically Acute Pulpitis (Irreversible)

On a histopathological level, cases of clinically acute irreversible pulpitis consist for the most part of acute exacerbations of chronic inflammation. The prognosis for recovery is very poor.

Symptoms

Patients will describe the pain in various ways, but in general the pain is more severe than with reversible pulpitis. Pain may occur in response to thermal stimuli (hot or cold) or may occur without any apparent provocation. Pain may persist for hours or days, intermittently or continuously. In a large percentage of cases the patient will give a history of previous episodes of pain.

Clinical Observations

Any one or a combination of situations may be present, such as a new restoration or a crown; an inadequately insulated restoration; an extensive carious lesion, possibly with exposure of the pulp; recurrent caries beneath a restoration.

Clinical Tests

Thermal: Abnormal response to heat or cold.

Electric pulp test: Not entirely conclusive, but may respond with less current than "control" tooth.

Percussion: Negative, except when acute reaction reaches the apical periodontal tissues.

Roentgenographic Appearance

The roentgenographic appearance is negative. (In advanced stages, when inflammation reaches the apical periodontal tissues, a thickening of the periodontal ligament space is evident.)

Clinically Chronic Pulpitis (Irreversible)

The category of "Clinically Chronic Pulpitis (Irreversible)" includes chronic inflammation of the pulp that has progressed beyond the early, potentially reversible stage.

Symptoms

Pain is often absent. Serious involvement of the pulp may be present with no serious symptoms. Where an exudate cannot secure drainage, as when food particles pack into an exposed carious lesion, a dull, low-grade pain may be experienced, which may become severe if the cause is not removed, since an acute reaction may set in.

Clinical Observations

Any one of several situations may be visible, such as an extensive restoration or a crown; recurrent caries beneath a restoration or a crown; an extensive carious lesion with possible exposure of the pulp. The exposure in the absence of acute symptoms is diagnostic. A history of previous episodes of pain is often given.

Clinical Tests

Thermal: Not diagnostic; response may be either normal or abnormal.

Electric pulp test: Not diagnostic; more current may be required to elicit a response than with normal tooth as nerve tissue degenerates over a long period of time.

Percussion: Negative.

Roentgenographic Appearance

The roentgenographic appearance is negative, except that there may be a carious exposure. In longstanding cases, a thickening of the apical periodontal ligament may be evident.

Chronic Hyperplastic Pulpitis

Hyperplastic pulpitis might be classed as an uncommon subdivision of chronic pulpitis, occurring almost exclusively in the younger age groups and involving mostly deciduous molars and first permanent molars with large open carious lesions. Aided by an excellent blood supply, chronically inflamed pulp grows exuberantly out of the pulp chamber and fills the cavity with pink polypoid tissue that is easily diagnosed (SLIDE 40). The polyp, frequently covered with transplanted oral epithelium, may be differentiated from hypertrophic gingival tissue by careful probing to establish whether the mass stems from the pulp or the gingiva. Because granulation tissue contains few nerves, the polyp is relatively insensitive. Although pulpotomy might be accomplished in some instances, the treatment of choice is pulpectomy.

Total Necrosis of the Pulp

Necrosis of the pulp may occur directly and swiftly as the result of a blow that severs the blood supply. In most cases it occurs gradually as the end point of inflammation of the pulp.

As such, it may be only a transitional step to inflammation of the apical periodontal tissues.

Symptoms

Pain symptoms are usually absent because nerve tissue has been destroyed; however, the patient often gives a history of previous episodes of pain. A necrotic pulp may not be discovered until inflammation of apical periodontal tissues causes tenderness or a full-blown acute reaction.

Clinical Observations

The crown has lost its translucency. There may be fracture of the incisal or occlusal surface of the involved tooth and adjacent teeth.

Clinical Tests

Thermal: No response.

Electric pulp test: Usually, there is no response. Following a traumatic injury where pulp tests are negative and necrosis is suspected, it is wise to delay endodontic therapy (provided no other conclusive signs or tests are evident) for at least 3 weeks. A stunned pulp may not respond to electric pulp tests or thermal tests for several weeks but may recover later.

Percussion: Negative response. (Sometimes there is tenderness because it is difficult to draw a line between total necrosis and apical periodontitis.)

Roentgenographic Appearance

The roentgenographic appearance is negative; or the periodontal ligament space may appear thickened.

DIAGNOSTIC SUMMARY OF PERIAPICAL LESIONS

Acute Apical Periodontitis

When strong irritants (virulent microorganisms, highly irritating drugs) invade from the root canal, their first point of attack is in the tissues of the apical periodontal ligament, where they produce an acute response.

As a point of differential diagnosis it is well to remember that occlusal trauma may also produce inflammation of the apical periodontal tissues, and lateral periodontal involvement may produce a similar acute response.

Symptoms

There is tenderness to occlusal pressure and masticatory movement, and a feeling of elongation of the tooth.

Clinical Observations

The presence of occlusal wear facets or malocclusion may indicate that tenderness is due to occlusal trauma. The presence of deep periodontal pockets points to lateral periodontal inflammation. In these latter cases the pulp must not be blamed.

Clinical Tests

Percussion: Painful response.

Electric pulp test: No response when tooth is pulpless; near normal response if the cause is not due to involvement of the pulp.

Roentgenographic Appearance

There is no change in the roentgenographic appearance, or perhaps slight widening of the apical periodontal ligament space. The lamina dura is intact. (Early bone changes cannot be visualized.)

Acute Periapical Abscess

As the acute inflammatory response, under increasing pressure, moves outward from the confines of the periodontal ligament spaces into surrounding bone, the inflammatory response becomes known as an acute periapical abscess.

Symptoms

As the pus travels, under pressure, through the cancellous bone and penetrates the buccolabial or lingual cortical plate of bone, intermittent to continuous throbbing pain is felt. When the purulent exudate builds up behind the tough periosteum and creates a subperiosteal abscess, excruciating pain is often experienced.

In severe cases, signs of toxemia are manifested: chills, fever of 99 F. to 103 F., or higher; malaise; and a feeling of nervousness and apprehension.

Clinical Observations

As the acute reaction reaches its advanced stages, some mucosal swelling and possibly a soft fluctuant subperiosteal mass may be palpated near the apical area of the involved root. A parulis (popularly called "gum boil") may be observed on the buccolabial or lingual mucosa; possibly discharge of pus may be noted in the final acute stages. Mild to severe swelling of the soft tissues of the face is evident in some cases. Local lymphadenopathy may be found.

Clinical Tests

Percussion: As mounting pressure forces exudate through the bone, it also forces exudate occlusally and incisally along the lateral periodontal spaces and the tooth becomes extremely sore to pressure in any direction.
Vitality tests: All negative.

Roentgenographic Appearance

Slight widening of the apical periodontal ligament space may be noted. It is important to realize that this acute abscess may be superimposed on a previously existing chronic lesion, in which case a periapical radiolucency of varying size will be observed.

"Subacute" or "Chronic"
Periapical Abscess

In the quieting down phase between severely acute periapical inflammation and chronic inflammation the reaction becomes "subacute" (possessing characteristics of each). This is usually a short-lived phase, and chronic inflammatory tissue soon begins to enclose the suppurative area in an effort to confine it and overcome it, but the attempt is never completely successful. Thus, the "chronic" periapical abscess represents a wall of granulation tissue with a collagenous connective tissue capsule which surrounds an area of tissue disintegration or suppuration at the apex of a tooth. The reparative process is held at bay by the slow flow of pyogenic irritants from the canal. (If the onslaught of pyogenic microorganisms were reduced in intensity and the area of suppuration were to be overcome, the lesion would be a granuloma.)

Symptoms

Pain is absent in most cases.

Clinical Observations

At times there may be evidence of a draining inflammatory tract ("fistula") on the mucosa near the apex of an involved tooth. A vesicle filled with pus may form, discharge, and disappear.

Clinical Tests

Pulp vitality tests: Negative.
Percussion: Usually no response.

Roentgenographic Appearance

A diffuse area of rarefaction is visible around the apex. This is indistinguishable from any other periapical lesion.

Periapical Granuloma

This is a chronic, low-grade inflammatory lesion—a wall of granulation tissue in a collagenous connective tissue capsule, surrounding the apex of a tooth and limiting the spread of irritants from the canal. It is a clinically quiet lesion (unless an acute exacerbation is induced). The first indication of its presence is often found when examining full-mouth roentgenograms.

Symptoms

Pain is absent.

Clinical Observations

The crown has lost its translucency. Mobility is negative unless a large amount of periapical bone has been destroyed.

Clinical Tests

Vitality tests: Negative.
Percussion: Negative.

Roentgenographic Appearance

The lesion appears as a diffuse or circumscribed area of rarefaction around the root apex (SLIDE 41), varying in size from a marked thickening of the apical periodontal ligament space to a lesion 10 mm. or more in diameter. It is indistinguishable roentgenographically from other periapical lesions.

Periapical Cyst (Periapical Periodontal Cyst, Radicular Cyst)

The periapical cyst is a sequela of the formation of a periapical granuloma. It is a slow-growing, epithelium-lined sac containing a fluid or semi-fluid. Details have been discussed earlier.

Symptoms

Pain is usually absent.

Clinical Observations

Crown shows loss of translucency. Mobility is negative unless loss of bone involves a large part of the root. Palpation of overlying mucosa may reveal enlargement of contour or crepitus. All clinical vitality tests are negative.

Roentgenographic Appearance

The periapical cyst appears as a periapical radiolucency which may have either a ragged (SLIDE 42) or smooth border and may or may not exhibit the peripheral thin, opaque line representing reactive bone formation (SLIDE 43). Because of its expanding character the apical cyst may grow larger than the granuloma, but this is not a reliable diagnostic criterion. It is not possible to differentiate an apical cyst from a granuloma or other periapical lesion by roentgenographic means alone.

Diagnosis of a Periapical Cyst During Treatment.—A large number of investigators have reported 90 to 95 percent success using conservative (nonsurgical) endodontic therapy. Possibly some cysts may spontaneously heal following conservative therapy, but this cannot be relied upon. The only certain way of treating a cyst is by surgical curettement of the entire epithelium-lined sac. It would be of immense help to the endodontist if he could diagnose or identify a periapical cyst, possibly even during treatment, and select this lesion for surgical excision.

Patterson, Shafer, and Healy[9] in their excellent clinical and histological study of the periapical lesions of 501 patients conclude, in part, that the endodontist should suspect existence of a cyst if (1) the root canal continues to exude a serous fluid, (2) it is impossible to obtain a negative culture test, or (3) the lesion emits a foul odor. The authors point out that it seems unreasonable that the periapical cyst could be sterile, not only because its fluid contents are an excellent culture medium for microorganisms, but also because these microorganisms are separated and protected from the phagocytic activities of the neutrophils and macrophages in the connective tissue wall by the limiting band of epithelium. Furthermore, if the cyst is sterile, and this would be rare, the excessive moisture in the root canal should indicate to the endodontist the strong possibility of the existence of a cyst. Apical surgery could then be resorted to after disinfection and root canal filling.[10]

EVALUATION OF CASES FOR ENDODONTIC TREATMENT (CASE SELECTION)

As previously stated, the first major step in diagnosis is to locate the pulp-involved tooth; the second step is to determine to what extent the tooth is involved, both pulpally and periapically. In other words, the dental officer must answer the question, "Does the tooth need endodontic treatment?" Once the need for endodontic treatment has been definitely established, the dental officer must evaluate the tooth, and the patient, and decide whether endodontic treatment can indeed be carried out for this patient or whether the tooth must be extracted.

It is difficult to lay down hard and fast rules for determining whether a tooth can be treated endodontically. Each case must be considered individually, and many factors must be appraised. Not all teeth are amenable to treatment. Further, variables introduced by both patient and dental officer can markedly influence the success of treatment. For example, the skilled dental officer might be able to cope satisfactorily with certain mechanical complications that the less proficient or less experienced officer might find insurmountable. The one with the least experience should be the most discriminating in selecting or rejecting cases for treatment.

In selecting those cases in which endodontic treatment—either nonsurgical or surgical—will have a strong chance of success, the dental officer should answer these four questions:

1. Can the tooth be treated?
2. Is the tooth worth treating?
3. Does the patient's health warrant endodontic treatment?
4. Does the patient desire the treatment?

Answering these questions will complete the diagnosis, determine the manner of treatment, and indicate the prognosis.

1. Can the tooth be treated? The answer to the question of whether the tooth can be treated depends upon the skill and experience of the dental officer and the anatomical and biological situation of the tooth. The dental officer must evaluate the risks involved. In general, if any one or a combination of the following situations exists, endodontic treatment is not indicated:

a. The crown of the tooth is so badly broken down that the tooth cannot be isolated and a sterile field maintained.

b. The roots are too curved or malformed to be treated nonsurgically, and it is not anatomically or medically feasible to treat them surgically.

c. One of the following types of fracture is present:

(1) Fracture parallel with the long axis of the tooth.

(2) Fracture of the cervical two thirds of the root followed by death of the pulp. (If the pulp remains vital, healing may occur without treatment other than stabilization. If the fracture is in the apical third of the root, the canal may be filled and the apical portion removed surgically.)

d. The tooth has multiple, irregular external resorptive processes.

e. There is advanced internal resorption, and possibly perforation, in the cervical two thirds of the root.

f. An inflammatory tract communicates between a periapical lesion and the gingival sulcus of the tooth. (The prognosis is poor, but competent combined periodontic-endodontic therapy may result in success.)

g. There is extensive periodontal inflammation. (This condition should first be brought under control.)

h. As a result of advanced periodontal disease, the bone support is inadequate. (If over half of the root length is unsupported by bone or if marked mobility exists, treatment is contraindicated.)

2. Is the tooth worth treating? The dental officer must consider the strategic value of the involved tooth. Can the successfully treated tooth serve the patient better functionally than an artificial replacement? Is the tooth essential for anchorage of either a fixed or a removable partial denture? Would loss of this tooth necessitate going from a fixed to a removable partial denture, or from a partial to a complete denture? With regard to esthetics, would the treated tooth, with possible bleaching, look better than a prosthetic replacement? Is the tooth malaligned or has it extensive carious involvement, and could the situation be corrected by placement of a post crown? In essence, the patient's future welfare must always be considered rather than merely present expediency.

3. Does the patient's health warrant endodontic treatment? Many uncontrolled systemic disorders, active infectious diseases, or debilitating conditions will inhibit periapical healing either partially or completely. If the dental officer suspects that the patient has uncontrolled syphilis, tuberculosis, diabetes, or anemia, for example, he should consult the medical officer before initiating endodontic treatment.

Surgical endodontic treatment is hazardous for patients in certain stages of pregnancy, for those with certain blood dycrasias, for those receiving supportive medication such as anticoagulants, and for those who have previously undergone intensive radiation treatment of the head and neck. Nonsurgical endodontic treatment is often a necessity for these patients, and this fact emphasizes the value of this type of treatment.

Age is not necessarily a consideration in deciding upon endodontic treatment, though one should expect slower repair of periapical tissues in older patients.

4. Does the patient desire endodontic treatment? Every patient confronted with the dilemma that a pulp-involved tooth poses deserves an explanation of the rationale of endodontic therapy. He should be told that the endodontically treated tooth is not a dead tooth but a healthy pulpless tooth and that there are great advantages to be gained by retaining the tooth and preserving an intact dentition. For mutual planning purposes, the total time involved and

the time intervals of treatment should also be made known to the patient. Given a careful explanation, most patients will choose endodontic therapy, and a valuable dental and educational service will have been rendered.[11]

It is hoped that the reader has not gained the impression from the preceding discussion that diagnosis and evaluation of cases for treatment (case selection) are complicated and time-consuming procedures. Quite the contrary; within a short time the dental officer can develop enough skill to carry out these procedures in 10 to 15 minutes—a very small investment of time indeed when the preservation of the tooth and perhaps the integrity of the dental arch are at stake.

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CHAPTER 4
ASEPTIC AND MICROBIOLOGICAL PROCEDURES IN ENDODONTICS

A climate of asepsis is absolutely essential in the practice of endodontics. (Asepsis requires the constant prevention of access of microorganisms to the vicinity of the pulp spaces.) The destruction of microorganisms that may already be present in the root canal is also essential. Failure to observe these principles may result in eventual loss of the tooth being treated as well as in bacterial contamination of the patient's bloodstream.

In this chapter a discussion of certain aseptic and microbiological procedures and considerations is presented: Acceptable methods of instrument sterilization or disinfection are described; the importance of eliminating microorganisms that may gain access to the root canal is stressed; and microbiological techniques for ensuring that root canals have been properly disinfected are discussed.

STERILIZATION AND DISINFECTION OF INSTRUMENTS AND SUPPLIES

Sterilization or disinfection of all instruments and other articles used in endodontic treatment must be carried out with scrupulous care. The best available methods, and indications for using them, are discussed in this section.

First of all, the two terms should be clearly distinguished. Sterilization is a process by which all microorganisms are killed. Disinfection is a process by which most microorganisms are rendered incapable of causing infection.[1] Spores of spore-forming organisms are not killed by disinfecting procedures; and neither are Mycobacterium tuberculosis and the viruses—for example, those causing the viral forms of hepatitis. For instruments that will enter the root canal, disinfection is not acceptable. All such instruments must be sterilized. Some ordinarily nonpathogenic organisms that might be carried into the canal on disinfected instruments could acquire virulence when tissues are traumatized or the patient's resistance to infection is lowered.

STERILIZING METHODS

The Autoclave

Steam under pressure should not be used for sterilizing small endodontic instruments because they are certain to rust. Items used for apical surgery—rubber gloves, gauze squares, surgical linen, and instruments—should be assembled in packets and autoclaved. It is the live steam that destroys the microorganisms, and not the temperature or the pressure. Therefore it is necessary first to ensure free access of steam to all parts of instruments and packets, and then to sterilize at 120° C. (250°F.) for at least 20 minutes. Hypodermic syringes should be autoclaved; and presterilized disposable needles should be used to prevent the spread of viral diseases.

The Dry Heat Oven

The dry heat oven is excellent for sterilizing small endodontic instruments because they do not rust. To guarantee sterilization, the oven must be heated to a temperature of 160° C. (320°F.), and this temperature must be maintained for at least 1 hour. The temperature is critical because solder on root-canal files and reamers will melt at a temperature of approximately 170° C. (340° F.). An excellent oven for sterilizing dental instruments is now on the supply table (FSC 6530-962-9965, Sterilizer, Surgical Instrument, Dry heat type, electrically heated. Cost, approximately $75.).

Reamers, files, broaches, burs, paper points, and cotton pellets should be placed in a compartmented metal box for sterilization in the oven. A large selection of such boxes is available, on an open purchase basis. These boxes make excellent storage kits, and all items are easily accessible for use (SLIDE 44). To lessen the possibility of air contamination, the box should be resterilized at least once a week, and sterile items should be removed from the box with sterile, flamed forceps.

All the other items to be used during endodontic treatment can also be assembled in a compact kit. Each kit should include all items that may be needed during one nonsurgical endodontic treatment: rubber-dam frame, mirrors, explorer, cotton pliers, spoon excavators, extra gauze squares, irrigation syringes, hemostats, scissors, plastic instruments, cement spatulas—even a small tile mixing slab (SLIDE 45). A pair of instrument forceps should be on top (SLIDE 46). This kit can be sterilized in either the autoclave or the dry heat oven.

The Open Flame

Holding instruments in an open flame until they are cherry red is a limited method of sterilization. The instrument must be heated thoroughly so that microorganisms are burned off, as is customary in sterilizing inoculating wires in microbiology laboratories. Because the high temperature alters the temper and physical properties of instruments, it is particularly important that reamers, files, and broaches not be flamed; loss of temper could result in breakage within the root canal. Flaming is most useful for sterilizing cotton pliers (which have been sterilized but may have become contaminated upon exposure to air) immediately before they are used to remove small instruments from the sterilized instrument box.

DISINFECTING METHODS

The Molten Metal, Glass Bead, or Salt Sterilizer

The heated-well bracket-table sterilizer is widely used in the practice of endodontics, but it has shortcomings that should be discussed. There is doubt concerning the effectiveness of this device—doubt concerning its ability to kill spore-formers on small instruments in a reasonable length of time.[2, 3] Therefore it will be discussed under "Disinfecting Methods."

Concerning the heat-transfer materials used in the sterilizer, there are individual difficulties. Molten metal has been largely discarded because of the accumulation of dross (metallic oxide) on the surfaces of instruments and materials and the danger of carrying bits of metal into the root canal. Of course, there is the constant danger of upsetting the sterilizer and burning the patient or the dental officer. Glass beads (less than 1 mm. in diameter) are cleaner to use; however, there is danger of carrying beads into the root canal. Grossman[4] warned that there can be a variation of 30° C. between the upper and lower levels of the beads. Table salt is inexpensive, is easy to obtain, and can be dissolved if accidentally carried into root canals. On the debit side, however, recent reports indicate that there is a marked temperature gradient from the walls of the crucible to the center of the heated mass. With salt, and also with glass beads, gradients of as much as 140° C. have been reported. In one study in which fine lava beads were used, a gradient of 125° C. was noted until the sterilizer had heated for 20 minutes.[5]

In view of these variabilities, a suitable thermometer must be employed regularly to ensure that the temperature reaches 218° C. (425° F.) in that part of the sterilizing medium into which the root-canal instruments will be introduced. Care must then be taken to plunge root-canal instruments deep enough into the mass. An exposure time of at least 10 seconds should be employed to ensure an adequate margin of safety for most root-canal instruments.

If the heated-well sterilizer is employed correctly, the time required to heat the material in the sterilizer and then to sterilize every instrument, paper point, and cotton pellet makes endodontic treatment a tedious operation. The dentist might better conserve his time by using articles unquestionably sterilized previously by other methods.

The bead or salt sterilizer might be used as an auxiliary device; for instance, in cases of accidental contamination of small instruments or other articles when time does not permit their resterilization by hot air. This sterilizer can be used for silver points that become contaminated during handling and shaping; submersion for at least 10 seconds (preferably longer) is suggested. Instruments of larger bulk, such as scissors and forceps, cannot be reliably sterilized in the bead or salt sterilizer.

Boiling Water

Boiling water sterilizers are not considered reliable. This method is not suitable for sterilization of the endodontic armamentarium.

Disinfectant Solutions

Certain endodontic materials, such as gutta-percha points or temporary stopping, cannot be sterilized by moist or dry heat. By necessity, then, one must rely on chemical solutions for their disinfection. The Council on Dental Therapeutics of the American Dental Association stipulates that acceptable chemical disinfectants must be effective in killing vegetative forms of pathogens, except possibly Mycobacterium tuberculosis, within 5 minutes. However, it recommends a minimum period of exposure of 15 to 30 minutes in order to provide a further margin of safety.[6]

The disinfectant solution of choice is 70-percent ethyl alcohol. Sufficient gutta-percha points for several days' use should be immersed in the alcohol for 15 to 30 minutes, then dried with sterile gauze and stored in a dry sterile glass container with lid. Extended immersion of the gutta-percha points will cause them to become excessively soft. A fresh solution of alcohol should be obtained for use each time. Alcohol volatilizes more rapidly than water, and within 1 week the strength of the solution is reduced to approximately 60 percent.

STERILIZATION OR DISINFECTION OF DENTAL HANDPIECES

Dental handpieces can be sterilized or disinfected in many ways. When a method is being selected, consideration must be given to the intended use of the instrument and to the peculiarities of each type of handpiece with respect to the care required by finely machined metal or plastic parts. To avoid damage, the recommendations of the manufacturer must be followed.

For apical surgery, sterilization of the handpiece is mandatory. Advanced speed stainless steel handpieces with no plastic components are available for surgery. With the use of a suitable lubricant such as silicone oil, these handpieces can be autoclaved without damage.

For conventional endodontic or operative procedures, sterilization of the handpiece is highly desirable, but the handpiece must be selected on the basis of its performance. Autoclave sterilization is the method of choice for those handpieces that can be subjected to the necessary heat and moisture. Most standard speed handpieces may be systematically cleaned, lubricated, and autoclaved. Manufacturers are directing strong efforts toward production of advanced speed handpieces with Teflon or all-metal components that can withstand autoclaving. The advanced speed handpiece that will not stand autoclaving is a weak link in the chain of sterilization or disinfection in the dental office. For these handpieces, cleanliness should be maintained by the best available procedure; namely, cleansing by wiping with a generous quantity of 70-percent alcohol after each use.

ROOT-CANAL CULTURES

Bacteria and other microorganisms gain access to the pulp or the pulp cavity through the tubules of cut or carious dentin, or directly through exposure of the pulp. Far less frequently they gain access along the periodontal ligament in periodontal disease, or through the bloodstream during bacteremia or septicemia. Secondary invaders (contaminants) may be introduced into the root canal during endodontic treatment by faulty or careless technique. From the pulp or the root canal, microorganisms may move or be forced into the periapical tissues.

It is important to realize that following death of the pulp, microorganisms in the root canal live in a cozy environment conducive to their growth and inaccessible to the body's defense system. In the periapical tissues of healthy individuals, on the other hand, the microorganisms are exposed to the full range of defensive activity and are sooner or later destroyed—depending upon the relative strength of the invaders and the defense mechanism.

Hedman's investigation[7] gave considerable support to this concept. He demonstrated that once the source of bacteria in the root canal is eliminated the remaining bacteria in the periapical tissues do not long survive. This leads us to the point that it is the community of microorganisms within the root canal—the potential source of trouble or focus of infection—that must be dealt with. Healing of periapical tissues and restoration of the treated tooth to a state of health will not take place until all sources of irritation within the canal have been eliminated. All pulpal breakdown products must be removed; all microorganisms must be eliminated or reduced to negligible numbers. It is the clear-cut objective of chemomechanical preparation of the root canal to accomplish this.

Obviously, there must be some way of determining whether microorganisms have been eliminated or reduced to negligible numbers before the root canal is sealed. The only practical, reliable way of doing this is by the culture method. Clinical observations are not reliable because the presence of infection cannot be determined by these means. (The term "infection" is taken to mean not only the presence of pathogenic microorganisms but the reaction of the tissues to these organisms.) Infection cannot be detected by odor, by the presence or absence of pain, or by clinical examination of the tissues. Although pus may be observed, it is not necessarily a manifestation of infection; it is an indication of a foreign body reaction, and that foreign body may be a chemotoxic drug or a mechanical irritant rather than a bacterial irritant. It is also impossible to determine the presence of infection by roentgenographic examination.

It should be pointed out that with the simple culturing methods ordinarily available to the dental officer only the presence or absence of microorganisms is determined. The organisms are not identified and there is no way of knowing whether they are potentially harmful or not. That is why it is necessary to eliminate all microorganisms or reduce them to negligible numbers.

Microorganisms Found in the Root Canal

Although the microorganisms found in the root canal are not usually identified in culturing, the dental officer should have a general knowledge of them. No single microorganism produces pulpal or periapical disease. Various types are found, but some are consistently predominant.

Streptococci are the organisms most commonly isolated from root canals. In various studies [8-11] investigators have found that 50 to 82 percent of organisms isolated from root canals are streptococci in pure and mixed culture. Streptococci are also the most prevalent organisms in the oral cavity, and the same types prevail in both the oral cavity and the root canals. Although the types of streptococci isolated from root canals are not usually considered pathogenic, they may set up low-grade infections when natural resistance is lowered or tissues are traumatized.[12]

Staphylococci rank second in frequency of appearance in root canals of pulpless teeth. Depending upon the study, staphylococci have been found to be present in 7 to 25 percent of canals examined.[8-11] Of the staphylococci isolated, only a small percentage are Staphylococcus aureus, the pathogenic type.

Lactobacilli are isolated from exposed root canals. Since they are not pathogenic in soft tissues, it is speculated that they are contaminants from saliva or carious dentin.

Various other microorganisms are found in root canals in small percentages: yeasts, actinomycetes, gram-negative bacilli, gram-negative cocci, and many others. For a more complete discussion of the microorganisms found in the root canal, the reader is referred to two texts[13, 14] and two highly significant research studies[15, 16] in addition to the studies already referenced.

Culture Media

While it is true that no one culture medium is suitable for growth of all organisms that may be found in the root canal, still it is not practical for the practicing dentist to use a number of media to check the sterility of his root canals. He must use a medium that will be capable of growing the greatest variety of microorganisms. Among the numerous requirements for an adequate culture medium, the following should be specially noted:[17]

1. It should supply varied oxygen tension for support of aerobes, strict anaerobes, and facultative anaerobes (which can grow equally well in the presence or absence of molecular oxygen). The addition of 0.1-percent agar to the culture medium permits varying levels of oxygen tension: aerobes may grow at the top of the medium, and anaerobes deeper.
2. It should have the correct pH. A pH of 7.4, that of body fluids, is needed to permit growth of pathogenic organisms.
3. It should be fluid, since microorganisms multiply more readily in a fluid medium than in a solid medium.
4. It should contain enriched body fluids—blood, serum, or ascitic fluid—for the growth of many fastidious pathogenic microorganisms. Ascitic fluid, 5.0 percent, is recommended; this fluid is prepared by commercial laboratories from serous fluids removed aseptically from human peritoneal cavities.[18]
5. It should have sufficient depth and volume (about 8 to 10 cc. per tube) to cover the paper points, to allow for varying oxygen tension, and to neutralize traces of drugs carried over on the points.

6. It should contain neutralizers to prevent inhibition of bacterial growth by a drug that is carried over on the paper point. Penicillin may be effectively neutralized by use of penicillinase in the culture broth (available commercially as Penase Ascites Medium). Unfortunately, no practical methods of neutralizing the other anitbiotics are available, and therefore false negative cultures may be obtained with the use of these drugs.

Competent investigators[19-21] have compared various media for use by the clinical endodontist. Two media, recommended here, may be purchased commercially or prepared by trained medical laboratory technicians:

1. Glucose ascites medium is an excellent preparation that facilitates the rapid growth of streptococci and staphylococci. It is prepared by adding 0.2-percent glucose (to allow better growth of such organisms as streptococci, lactobacilli, and yeasts) and 0.1-percent agar to brain-heart or beef infusion broth and then adjusting the pH of the mixture to 7.4. About 10 ml. of the mixture is poured into each of several test tubes, and the tubes are capped with metal screw caps and autoclaved. After the tubes have been autoclaved, 0.5 ml. (5 percent by volume) of sterile ascitic fluid is added to each tube under strict aseptic conditions. The tubes are then incubated for 48 hours to ascertain whether or not they are contaminated; and if no growth is observed, they are stored in a cool place until needed.

2. Trypticase soy broth is another excellent all-purpose medium for use in endodontics. It can be purchased ready for use, or it can be prepared from the anhydrous mixture, available in 1-pound lots, as follows: First, the powder is rehydrated by suspending 30 gm. of it in 1 liter of distilled water. Next, 1/2 to 1 gm. of agar is added. Then 10 ml. of the suspension is poured into each of several test tubes, and the tubes are capped and autoclaved. Following autoclaving, 0.5 ml. of ascitic fluid may be added to each tube before incubating and storing.

The Incubator

Culture tubes should be placed in an incubator at 98.6° F. for a minimum of 48 hours. An incubator is essential for endodontic practice and is obtainable from laboratory or dental supply dealers. Small ones are relatively inexpensive.

On a do-it-yourself basis, any container with a capacity of about 1 cubic foot in which a temperature of 98° F. (plus or minus a degree) can be maintained will serve as a satisfactory incubator. The box can be constructed of sheet acrylic resin or wood. A controlled source of heat can be provided by connecting an inexpensive porcelain lamp socket (with a 60- to 100-watt bulb) in series with a chicken-brooder thermostat, and plugging the circuit into a 110-volt outlet. The cost of this homemade incubator is about $5. (SLIDE 47). Even a discarded insulated food container can be converted into an incubator with a minimum of effort and expense.[22]

Interpretation of Cultures

Any change in the appearance of the medium, such as turbidity or general cloudiness, or the presence of sediment or surface scum, indicates bacterial growth, and the result is called a positive culture (SLIDE 48). Although growth on the surface may be due to airborne contamination, it is better to take another specimen for culturing than to make this assumption. If no signs of growth are observed at the end of the incubation period; that is, if the culture medium is still clear, the result is called a negative culture (SLIDE 49).

A positive culture may indicate either infection or contamination. The presence of contaminant organisms as a result of faulty endodontic technique causes needless waste of time. Because it is nearly impossible to differentiate infective microorganisms from contaminants in the positive culture, medication must continue until negative cultures are obtained. Contaminant organisms enter the pulp spaces on unsterile instruments or via saliva through leaks in the tooth crown or between-treatment seal. Crown patency should be frequently checked and a rigid aseptic technique followed.

Absence of growth in the culture tube may mean that the canal is sterile. However, false negative cultures may occur for several reasons:

1. Microorganisms may be present in such small numbers that they cannot initiate growth in the medium.

2. The culture medium may not be versatile enough to support growth of certain fastidious organisms present in the canal.[15, 16]

3. Facultative anaerobes, lying dormant in the canal under anaerobic conditions, may not-

reproduce when the canal is opened and a sample is taken on the first appointment. After a few days of increased oxygen tension in the canal the next culture will show positive growth.

4. The samples on the paper points may be inadequate if the points are not placed to the full working distance.

5. The growth of organisms may be inhibited if an agent used in root-canal medication is transferred to the culture tube on the paper point. This is true if penicillinase is not used to neutralize penicillin or if other antibiotics are used in the canal.

The reader may be disturbed by the fact that negative cultures do not always guarantee that the canal is sterile. However, if a thoroughly aseptic technique is followed and all extraneous reasons for false negative cultures are eliminated, the only other possibility is that small numbers of microorganisms remain in the canal. If these few organisms are able to invade the periapical area, the local defense mechanism will, in a very high percentage of cases, be able to overcome them. If, on the other hand, the canal is filled when the cultures are positive, that is, in the presence of demonstrable numbers of microorganisms, the population of organisms is presumed to be greater and the chances that the defense mechanism will overcome them are reduced.

In recent years there has been a spirited controversy among endodontists regarding the justification for culturing. Many competent investigators[23-25] have undertaken clinical studies and presented evidence to show that their percentage of success was significantly higher for teeth filled when cultures were negative than for those filled when cultures were positive. Success was based on clinical and roentgenographic evidence of repair. It would seem that safe practice demands that all practitioners—but particularly those with limited endodontic experience—take the extra few minutes of time required to take samples for culturing and provide themselves with an additional check on their technique.

On the basis of available evidence, it seems reasonable to conclude that (1) a positive culture definitely indicates that a root canal is not ready to be filled, and (2) two successive negative cultures obtained at least 48 hours apart indicate beyond a reasonable doubt that a canal is ready to be filled. The obtaining of two negative cultures does not eliminate the risk of failure in endodontic treatment; it reduces the risk. Cultures are an aid—a tool—in endodontic therapy, not a gold-plated guarantee of success.

Technique for Taking Culture Samples

The first sample for culturing should be taken on the initial appointment, after access has been obtained, the pulp has been removed by gross debridement, and the working distance of the canal or canals has been determined. No irrigating solution should be used before the culture is taken. The technique, which is simple and requires only a few minutes, is as follows:

A sterile paper point is placed into the open root canal—care being taken to place the point to the limit of, but not beyond, the working distance. (The apical third of the canal is more likely to contain organisms than the rest of the canal. Small canals may have to be enlarged with instruments before paper points can be inserted the entire length of the canal.) The paper point is allowed to remain in the canal for at least 1 minute. The cap of the culture tube is removed, and the lips of the tube are flamed. The point is carried swiftly and directly to the tube in sterile forceps (SLIDE 50), the tube is flamed, and the cap is replaced lightly.

The procedure is repeated with another point from the same canal if it is a single-rooted tooth, or a point from each canal if it is a multirooted tooth. The inoculated culture tubes are placed in an incubator for at least 48 hours.

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CHAPTER 5
NONSURGICAL ENDODONTIC TREATMENT: CHEMOMECHANICAL PREPARATION OF THE ROOT CANAL FOR FILLING

In this chapter and the one that follows it, nonsurgical endodontic treatment is discussed. Clinical preparations for treatment and chemomechanical preparation of the root canal to receive a filling, as well as the management of of acute cases, are included in this chapter. Filling materials and techniques are dealt with in chapter 6.

Throughout the discussion of endodontic treatment, principles as well as technical details are emphasized. The dental officer should recognize that there are several acceptable methods of treatment and should understand the advantages and disadvantages of each. He should also know which methods are best in particular circumstances. It is better to develop a sound endodontic philosophy and treat each case on the basis of individual requirements than to search for one ideal technique based on the assumption that all cases will fall into a similar pattern for treatment.

The reader is advised to supplement this discussion by referring to textbooks such as those by Sommer, Ostrander, and Crowley;[1] Healey;[2] Grossman;[3] and Coolidge and Kesel.[4] If the reader is stationed at a small activity or at sea, he should read the 6820 series of BuMed Instructions for information on the procurement of textbooks and periodicals.

PRINCIPLES AND OBJECTIVES OF CHEMOMECHANICAL PREPARATION

Chemomechanical preparation of root canals implies the use of chemical agents and mechanical methods in preparing the canals to receive a proper filling. One all-important ingredient must be added to this procedure: "TLC"—Tender Loving Care. The root canal leads straight to the periapical tissues, and the dental officer must protect those tissues with the same zeal with which he would protect an intact pulp when performing operative procedures. Ultimate success depends upon maintaining the periapical tissues in good health—or in restoring them to health if inflammation has already developed. The tissues must not be stabbed, crushed, or inoculated with bacteria through careless instrumentation; nor must they be injured with strong drugs.

The objectives of chemomechanical root-canal preparation are as follows:

1. To remove all irritants from the canal; this includes:

   a. Removal of all organic pulp tissue and debris from the canal.

   b. Elimination of microorganisms in the canal, if present.

2. To enlarge, taper, and smooth the canal in order to create a definite preparation to receive the desired filling material.

These objectives are attained by a series of steps that consist essentially of instrumentation and irrigation, usually followed by medication. Another necessary procedure is the taking of a sample from the root canal for culturing.

Chemomechanical preparation of the root canal for filling requires at least two appointments—more if two successive negative cultures are not obtained routinely. Treatment can usually begin on the first appointment. Summaries of procedures carried out on the first and second appointments and a suggested appointment schedule are given later in this chapter.

CLINICAL PREPARATIONS FOR TREATMENT

PREPARATION OF PATIENT

Once the diagnosis is made and the type of treatment is determined, the patient can be prepared for treatment.

Premedication

If the patient has been made to feel at ease and the endodontic procedure has been fully explained to him, premedication is usually not necessary. Occasionally a patient may be quite apprehensive; in this case premedication suitable for the patient's needs should be given.

Obtaining Anesthesia

For extirpation of a vital pulp, profound local anesthesia is required. Removal of a vital pulp is more likely to cause pain than operative or even surgical procedures. In general, the same injections are used for extirpation of a vital pulp as would be employed for extraction of the tooth. When the tooth gives absolutely no response to pulp tests, anesthesia is not needed. However, the dental officer should observe caution in entering the canals of these teeth. In single-rooted teeth the coronal pulp may be necrotic, but vital nerve tissue may be present in the apical portion of the root canal; in molars one or two canals may contain vital tissue. Nerve tissue is the last to lose function in a dying pulp.

In cases of acute periapical inflammation where edema of the periodontal ligament spaces has caused the tooth to be extremely sore to the touch, it may be possible to gain access to the pulp chamber without anesthesia by the use of an advanced speed handpiece with its attendant minimal vibration. When anesthesia is absolutely needed, block injections should be employed. The reasons for not infiltrating into an inflammatory area are (1) to avoid increasing the fluid pressure in the periapical area, (2) to avoid increasing the local chemical toxicity in this area, and (3) to avoid the local ischemic effect of the vasoconstricting agent, thus reducing the possibility of local infection as the direct result of poor circulation and consequent lowered tissue resistance in this area. Furthermore, better blood supply means quicker healing and less postoperative pain.

Establishing the Integrity of the Crown

During endodontic treatment only two openings into the canal may exist: the access opening and the apical foramen. Leaky restorations or carious areas provide sources of contamination that cannot be tolerated. Faulty restorations must be removed, and carious areas must be cleaned out and temporarily replaced with cement. If the crown is badly broken down by caries and if proximal surfaces are involved, the crown may be reinforced with a carefully contoured copper or stainless steel band cemented to place, free from occlusion (SLIDE 51). An aluminum crown form may also be used. The restored crown must be capable of being reopened and sealed several times during the course of therapy without extraneous leakage.

Establishing an Aseptic Field of Operation

If the reader has not been using the rubber dam in recent years, endodontics will give him a chance to become reacquainted with it—for in endodontics the rubber dam is an absolute necessity and will remain as such until someone can find a better way to isolate a tooth and maintain an aseptic field of operation.

In endodontics only the tooth to be treated need be isolated. The procedure is simple and usually takes less than a minute, as follows: The tooth is cleansed of all debris; the proper clamp is selected; a single hole is punched in the rubber dam; the dam is positioned; and the clamp and rubber dam holder are applied. Some dental officers prefer a single clamp (SLIDES 52, 53); others prefer an additional clamp or two placed over the dam in order to widen the the corridor of access to the tooth (SLIDES 54, 55). The type of rubber dam holder is not important, but many dental officers find Young's frame (as shown) most convenient. The frame should be positioned so that the rubber dam acts as a barrier between the patient's nostrils and the field of operation.

Once the tooth has been isolated, an aseptic field of operation must be established. (It would be impossible to establish a sterile field unless rigid surgical operating room conditions prevailed, but much can be done to minimize the amount of contamination introduced during operative procedures.) Starting with the tooth and working outward at least 1 inch on all sides, the area should be swabbed several times with an effective antiseptic.

One of the most effective antiseptics for this purpose is 7-percent tincture of iodine, although it has the disadvantage of staining silicate and acrylic restorations. Where such restorations are present and are not to be replaced after the canal has been filled, untinted nitromersol tincture, N. F. (Tincture of Metaphen), or surgical soap may be substituted. The outline of the swabbed area on the rubber dam serves as a reminder that all instruments entering the area must be sterile.

The rubber dam not only makes it possible to establish an aseptic field of operation but also protects the patient from irrigating solutions and from accidental swallowing of broaches or files should he cough or sneeze.

OBTAINING ACCESS TO THE ROOT CANAL

Success in endodontics depends largely upon proper preparation of the root canal to receive an adequate filling. A root canal cannot be competently instrumented and prepared unless the coronal approach to that canal is properly made. Thus, proper access is indeed a cornerstone of success in endodontics.

Gaining access to the root canal does not consist simply in providing a large opening into the canal; in fact, a large opening is usually contraindicated. The objectives in gaining access are (1) to uncover the roof of the pulp chamber in such a way that all pulp horns and crevices hiding carious tooth structure and pulp tissue are completely eliminated; (2) to preserve all anatomical landmarks of the pulp chamber, particularly the floor of the chamber; and (3) to provide a straight path of entrance into the root canal.

It is very important to study the roentgenogram of the tooth. This shows the size and shape of the pulp chamber and the curvature and direction of the root canals. It is essential to visualize these pulp spaces in three dimensions within the tooth.

Access to the root canal(s) of four representative teeth will be described:

Anterior Teeth

The enamel is penetrated in the exact center of the lingual surface, with a fissure bur or diamond stone directed perpendicularly to the enamel surface (to avoid slippage over the cingulum). As soon as the bur encounters dentin, it is moved without stopping to a direction parallel with the long axis of the tooth (SLIDE 56). Enough enamel is removed lingual to the incisal edge to permit a round bur to slide into the chamber. The chamber is uncovered from the inside outward; and the lingual shoulder is removed (SLIDE 57) with a round bur. This shoulder must be removed or it will direct root-canal instruments labially. The final product should be a smooth funnel into the root canal with all the walls visible and no residual pulp tissue in the chamber (SLIDE 58). The opening is triangular in the youngster, to encompass pulp horns, and oval in the adult (SLIDE 59). Access to the canal through the proximal surface is totally unacceptable (SLIDE 60); it leads to ditched pulp chambers and broken instruments (SLIDE 61).

Maxillary Bicuspid (Two Roots)

From the center of the occlusal surface in the central groove, a bur is directed apically. Special care should be taken that the bur is correctly lined up with the inclination of the crown. As soon as the bur breaks through into the pulp chamber, the chamber is carefully uncovered, from the inside outward. The access outline will be like the traditional Class I cavity outline turned 90°; that is, ovoid in shape buccolingually, narrow mesiodistally, reflecting the shape of the pulp chamber. The limits of the margins are established by inserting small files into the canals and making sure that the access opening is wide enough not to impede free use of the files. If the roots are widespread, there is plenty of room for files to enter (SLIDE 62). If the canals converge, the preparation may need to be widened (SLIDE 63), but it is a rare occasion when the cusp heights need to be involved.

(In the <u>mandibular bicuspid</u> the crown tilts lingually. Caution must therefore be observed in gaining access. The occlusal opening is started perpendicularly to the crown. As soon as enamel is penetrated, the direction is changed to one parallel with the long axis of the tooth (SLIDE 64).)

Maxillary First Molar

The access opening on the occlusal surface of the maxillary molars should be roughly triangular, with the apex of the triangle toward the lingual surface. This is a projection of the shape of the floor of the chamber onto the occlusal surface (SLIDE 65).

The initial entrance is made through the mesial pit, with the bur directed lingually toward the large opening of the easily located lingual canal. Next, the roof of the chamber and the overhanging dentin are carefully removed, with the dental officer working by touch from the inside outward. From the conservative access opening, the walls of the preparation should actually flare toward the canal orifices, providing a guide for insertion of instruments (SLIDE 66). The angle at which a file emerges from the lingual canal is shown in SLIDE 67; from the distobuccal canal in SLIDE 68; and from the mesiobuccal canal in SLIDE 69. It should also be noted that the entire preparation can be kept in the mesial half of the tooth.

Mandibular First Molar

The access opening on the occlusal surface of mandibular molars should be triangular, with the apex of the triangle toward the distal surface, reflecting the "molar triangle" anatomy (SLIDE 70). The preparation should be completely within the mesial half of the tooth.

The initial entrance is made just mesial to the central pit, the bur being inclined distally (SLIDE 71). This will direct it into the large orifice of the distal canal. One of the commonest mistakes, especially in the molars of older patients where the pulp chamber is shallow, is to overextend the bur and perforate the floor of the chamber. If a contra-angle bur of normal length is used, the hub of the bur will be flush with the occlusal surface of the tooth when the bur reaches the floor of the chamber (SLIDE 72). Use of a surgical length bur, in inexperienced hands, may lead to perforation (SLIDE 73).

With a round bur, the roof of the pulp chamber is removed and the overhanging dentin toward the mesial wall is cut away (SLIDE 74). Careful placement of small-sized files into the canals helps govern the size of the access opening. No overhanging tooth structure should bind a file or reamer during instrumentation. The angle at which the instruments enter the distal canal is shown in SLIDE 75; the mesiobuccal canal in SLIDE 76; and the mesiolingual canal in SLIDE 77.

If intelligent care is employed in gaining access to the root canal, the chance of success in the subsequent steps of canal instrumentation and filling will be greatly enhanced.

INSTRUMENTATION AND IRRIGATION OF THE CANAL

After proper access to the root canal has been obtained, instrumentation and irrigation comprise the principal means of preparing the canal for filling. While the role of instrumentation is of paramount importance, the part that irrigation plays should not be underestimated. Frequent irrigation of the canal during instrumentation prevents clogging of the canal and removes pulpal debris and dentinal filings that otherwise might be forced through the foramen.

In reviewing the objectives of chemomechanical preparation of the root canals, one phase deserves amplification: elimination of the focus of irritants in the root canal. Instrumentation and irrigation of the canal accomplish the following results:

1. All organic pulp tissue and debris are removed. This tissue would provide sustenance for microorganisms if it were present; and, in the form of toxic protein degradation products, would provide a continuing source of irritation to periapical tissues.
2. The irregular, cobblestone-like lining of the root canal is planed to hard, smooth dentin, thereby eliminating many possible hiding places for microorganisms and exposing the dentinal tubules to irrigant action.
3. The flushing action of the irrigant removes many microorganisms.

It is not surprising that in a clinical study Auerbach[5] demonstrated that with careful mechanical cleansing and irrigation of the canal alone, without use of further medication, a large proportion of the teeth thus treated yielded growth-free cultures. Stewart,[7] in a modified study, found similar results.

INSTRUMENTS

The instrument setup for the instrumentation and irrigation procedure is simple (SLIDE 78). The metal small-instrument box may be on the bracket table (or on a nearby auxiliary table). Sterile syringes for irrigation may be placed on sterile gauze pads. Commonly used hand instruments (such as a mirror, double-ended spoon excavator, and plastic instrument) are placed in a folded towel to minimize air contamination. A pair of cotton pliers (for removal of files, reamers, pellets, and other small items from the instrument box) may be placed in a well-type holder for easy access to the flame. An aspirator tip composed of a large-bore needle may be positioned in a bracket for ready access.

Three basic types of instruments are used to remove the contents of the root canal and prepare it for the reception of the filling material. They are the barbed broach, the root-canal file, and the root-canal reamer.

Barbed Broach

The barbed broach is an inherently weak instrument and has limited use. It is manufactured by notching a soft steel wire and then elevating the cuts to produce a series of barbs along the shaft (SLIDE 79, A). This instrument is used to remove intact masses of pulp tissue, paper points, and gross debris. The barbed broach should never be forced into a canal: it would almost certainly break upon withdrawal. In proper use the broach is inserted in the canal until the walls are first contacted. It is then withdrawn slightly, rotated slowly so as to entangle the barbs in the contents of the canal, and withdrawn completely from the canal.[7]

Root-Canal File

The root-canal file is an important and versatile instrument for the endodontist. It is made by planing the surfaces of a carbon or stainless steel wire to form a pyramidal shaft and then twisting the shaft so that the resultant spiraling of the edges produces a series of cutting flutes (SLIDE 79, B). Files cut on withdrawal. They are used in removing the contents of the canal and in filing or rasping the canal walls. The root-canal file is used by inserting it in the canal to the desired depth and then giving it one quarter turn clockwise. The flutes engage the dentinal wall and cut it as the file is withdrawn. The dentin-laden file should be cleaned by twisting it on a sterile towel or cotton roll before it is reinserted in the canal.[7] The file should never be locked in a canal and turned more than 180 degrees: excessive torque will weaken or break the instrument.

Root-Canal Reamer

A root-canal reamer is similar in design to a file, but in manufacture the steel shaft is not turned so tightly. Consequently, the spiral is looser, and there are fewer flutes per blade length (SLIDE 79, C), with resultant increased flexibility. Reamers cut on the insertion stroke. They are most effective when a rotary shaving action upon the canal wall is required, as in the final finish of a preparation, and when a boring action upon the canal's contents is needed.[7] Reamers can be used to remove old gutta-percha fillings from canals that are to be re-treated. A reamer is inserted in the canal to the desired depth, rotated one-half to one full turn clockwise, and then withdrawn.

Standardization of Instruments

Until recently, standardization in the manufacture of root-canal instruments was unknown. Before the late 1950's there was no uniform system of measurement, no uniform progression in size and taper. Instruments of the same number varied greatly in diameter and taper even with one manufacturer, and more among different manufacturers. Moreover, there was only accidental agreement in size and taper between instruments and filling materials.

A strong demand for more accurate tolerances in the manufacture of root-canal instruments and materials was made by the profession. In response, from the Second International Conference on Endodontics (Philadelphia, 1958) came the designs for standardized instruments and silver points.[8] These designs and their nomenclature were endorsed by the American Association of Endodontists in 1962.

Figure 1 represents the shaft of a standardized root-canal file, while table 1 shows how the standardized instruments correspond to earlier instruments. The size of a standardized instrument is indicated by the size of its shaft at the beginning of its spiral (D_1). Each instrument has a spiraled shaft 16 mm. in length, which tapers 0.30 mm. The diameter at the beginning of the spiral (D_1) is 0.30 mm. less than the diameter at the end of the spiral (D_2).

Figure 1.—Shaft of standardized root-canal file.

Table 1. --Numbering System and Diameter of Root-Canal Instruments.

Conventional numbers (closest)	Standardized numbers	Diameter of files and reamers (mm.)	
		D_1	D_2
0	10	0.10	0.40
1	15	0.15	0.45
2	20	0.20	0.50
3	25	0.25	0.55
4	30	0.30	0.60
--	35	0.35	0.65
5	40	0.40	0.70
--	45	0.45	0.75
6	50	0.50	0.80
--	55	0.55	0.85
7	60	0.60	0.90
8	70	0.70	1.00
9	80	0.80	1.10
10	90	0.90	1.20
11	100	1.00	1.30
12	120	1.20	1.50
--	140	1.40	1.70

With standardized instruments, uniformity of size increment and taper has been achieved. A standardized file makes the same bore as a standardized reamer of the same number. Moreover, a standardized silver point can be adjusted to fit a prepared canal with only minor adjustment in length.

With these standards as a basis, rigid specifications for the diameter, taper, and physical characteristics of root-canal instruments and filling materials are being set up by the Council on Dental Research of the American Dental Association.

IRRIGATING SOLUTIONS

The most generally accepted irrigant is sodium hypochlorite solution, N. F. This 5-percent solution, although mildly caustic to viable connective tissue, has the following advantages when used liberally throughout instrumentation:

1. It dissolves necrotic pulp and other organic debris.
2. It acts as a sluicing agent, floating out debris.
3. It lubricates root-canal instruments, improving their efficiency and lessening the hazard of breakage.
4. It acts as a mild antiseptic.
5. It acts as a bleaching agent, lightening the color of dentin.

Some dental officers prefer to irrigate alternately with sodium hypochlorite solution and 3-percent hydrogen peroxide solution, U. S. P. This procedure has one advantage in that the two solutions act as catalysts for each other, and there is marked effervescence of nascent oxygen and chlorine, which literally boils organic debris out of the canal. Following irrigation, the hydrogen peroxide must be thoroughly washed from the canal with the sodium hypochlorite solution, and the canal must be well dried. If this precaution is not observed, the continued liberation of oxygen after sealing may result in tissue emboli and severe apical periodontitis. Because of this possible disadvantage, it is suggested that the use of hydrogen peroxide with sodium hypochlorite solution be limited to wide canals.

In view of the mildly irritating effect of sodium hypochlorite and in order to treat normal and inflamed periapical tissue as gently as possible, many endodontists prefer to use a very bland irrigant, such as a sterile isotonic solution of sodium chloride. This is a satisfactory, nonirritating irrigant.

Chelating Agents

Another chemical agent has been placed in the dentist's armamentarium to aid him in enlarging the diameter of the root canal: the chelating agent. Chelating agents have an affinity for calcium ions and act as decalcifying agents. They can also be used as irrigants. Nygaard

Östby[9] investigated the use of solutions of disodium ethylene-diamine tetra-acetate (EDTA) for chelation of constricted canals. He recommended the use of a 15-percent solution of the salt, with the addition of a quaternary ammonium compound (Cetavlon) to increase the solution's antiseptic value. (This chelating solution may be purchased under the trade name of EDTAC.)

Introduced into the root canal EDTA (or EDTAC) substitutes sodium ions for calcium ions in the calcified pulpal remnants and dentinal walls of the constricted canal. Obviously, a completely obstructed root canal cannot be made accessible by any dentin-dissolving agent; but if there is a minute lumen left, the chelating agent will seep into it. The resultant softened structure is easier to slide through and remove with instruments.

Von der Fehr and Nygaard Östby[10] found that EDTAC produces significant demineralization in 5 minutes and is self-limiting in a short time. Patterson[11] found that slight chelating action may go on for as long as 5 days, until all of the available EDTAC has formed a stable bond with calcium of the dentin. In any event, the most advantageous time for using the chelate is during the period of instrumentation; there is no significant advantage (or danger) in sealing the agent in the canal between treatments. The chelating agent should never be forced under pressure into the canal, and should be carefully flushed from the canal following use.

Patterson,[11] in a clinical study of 200 patients, employed EDTA as an irrigant and chelating agent in facilitating root-canal preparation. He found that 10-percent EDTA produced no deleterious effects upon the apical periodontal tissues.

INSTRUMENTATION AND IRRIGATION PROCEDURE

The first step, after access to the root canal has been obtained, is to remove the vital pulp or diseased pulpal debris with a fine barbed broach. A sample for culturing is then taken (as described in chapter 4). This should be done before the canal is irrigated. The next step is to determine the "working distance" of the tooth.

Determining the Working Distance

The working distance is the distance from the incisal edge or occlusal surface to the dentinocemental junction. The dentinocemental junction is an anatomical constriction, approximately 0.5 to 1 mm. from the apex, formed by an invagination of cementum into the apical foramen.

With a good diagnostic roentgenogram being used as a guide in judging the approximate working distance, a small-sized file is inserted in the canal, and the apical constriction is located by probing gently with the file's tip. By means of a new roentgenogram the position of the tip of the file is then determined. The position of the file is adjusted, with the aid of a new roentgenogram if necessary, until the tip of the file is within 0.5 mm. to 1 mm. of the apex. The file is then grasped and removed with a hemostat that has been positioned at the incisal edge of the tooth. The distance from the hemostat (or the incisal edge) to the tip of the file is the true working distance. This distance must be recorded so that all instruments used in the canal will reach the full working distance but will not exceed it. The instruments can be fitted with mechanical or rubber stops to prevent their reaching too far (SLIDE 80).

Determining the location of the files is sometimes difficult in roentgenograms of posterior teeth. When canals are superimposed, as, for example, in maxillary first bicuspids or the mesial root of mandibular molars, the dental officer cannot tell which file is in the buccal canal and which is in the lingual (SLIDE 81). A suggested solution is to direct the cone of the X-ray tubehead from a mesial or distal angle to separate the roots and, in addition, to differentiate the diagnostic instruments by placing a reamer in the buccal canal and a file in the lingual canal (SLIDE 82), by using files of different sizes, or by blunting the point of one file and not that of the other. Careful scrutiny of the roentgenogram will reveal the difference.

There are good reasons for terminating the canal preparation at the dentinocemental junction or a level 0.5 mm. to 1 mm. from the apex. If instrumentation is carefully confined within the canal, delicate, possibly inflamed periapical tissue will not be repeatedly lacerated. The natural dentinocemental constriction (where it can be found) affords a solid stop for seating of the filling material and minimizes the possibility of overextension of the filling into periapical spaces.

After the working distance of each canal has been determined and recorded, the canal should be carefully irrigated.

Irrigating the Canal

Irrigating solution may be carried into the canal with a 2-cc. glass (Luer) syringe (SLIDE 83). Care should be taken that the needle does not bind in the canal, or the solution will be forced through the apical foramen. The solution may be gently aspirated with a 14- to 17-gauge needle that has been curved, blunted, and placed in the hub of a saliva ejector connected to an aspirator (SLIDE 84).

Enlarging the Canal

Enlarging the root canal should not be a difficult procedure. A point to remember is that this procedure cannot be hurried. Except for final finishing or in instances where a boring action upon canal contents is required, enlarging the canal is usually accomplished with files. The following guidelines for the procedure are suggested:

1. Begin with the largest file that can easily be inserted the full working distance.
2. Progress in sequence to larger sizes; do not skip sizes.
3. Irrigate thoroughly before changing to a larger file.
4. Introduce instruments slowly with a slight rotary back and forth action so as not to push the contents of the canal apically.
5. Insert each file precisely to the working distance, rotate one quarter turn clockwise, and file on withdrawal.
6. In instrumenting curved canals, make a slight bend in the apical 2 mm. of the file so that it can be gently teased around the curve (SLIDE 85).
7. Continue filing until the canal is smooth and polished throughout its working distance.

The canals of anterior teeth and of bicuspids with a single canal, the lingual canal of upper molars, and the distal canal of lower molars should usually be enlarged to at least the size of a standardized No. 50 (conventional No. 6) file. Other canals should be enlarged anywhere from the size of a No. 25 (No. 3) file to that of a No. 50 (No. 6) file, depending upon the amount of curvature and the size of the canal. In general, a canal that cannot be enlarged to at least the size of a No. 25 (No. 3) file cannot be medicated and filled adequately, and success in treatment is doubtful.

Instrumentation of root canals must be carried out thoroughly. If it is not, subsequent procedures will be carried out under a severe handicap. Root-canal medication is far more effective in a canal that has been adequately prepared. Moreover, a canal that is properly prepared is one that can be filled with the greatest ease and assurance.

ROOT-CANAL MEDICATION

Although instrumentation and irrigation succeed in removing many microorganisms from the root canal, between-treatment, intra-canal medication is of considerable value. It is well to place a medicament in the canal routinely at the end of each appointment until two successive negative cultures have been obtained and the canal can be filled.

MEDICAMENTS

The ideal medicament for endodontic use would be:

1. Rapidly effective in destroying all organisms found within the root canal.
2. Capable of neutralizing or eliminating any toxic products present within the root canal.
3. Nonirritating to healthy tissue and incapable of producing toxic or allergic reactions.
4. Incapable of interfering with the accuracy of culturing results.
5. Capable of penetrating deep within the dentinal tubules.
6. Effective in the presence of organic debris.
7. Incapable of staining tooth structure.
8. Chemically stable, even with long periods of storage.
9. Easy to apply, odorless, and tasteless.
10. Inexpensive and readily available.

No root-canal medicament now available meets all of the requirements listed. One of those that comes closest to the ideal should be chosen.

Nonspecific Drugs

Camphorated parachlorophenol, N. F., (CMCP) comes rather close to the ideal It is a combination of 3 parts of crystalline parachlorophenol, with 7 parts of gum camphor as a vehicle. Camphorated parachlorophenol is effective, penetrates dentin well, and is well tolerated by the tissues.[12] It does not interfere with the accuracy of culturing results after being sealed in the root canal for 48 hours or more. It is inexpensive and remains chemically stable for years.

Metacresyl acetate (Cresatin) has a low surface tension and penetrates deeply. It is not so effective a germicide or fungicide as CMCP but is noted because it causes little damage to tissue. It is effective against pathogens commonly found in the root canal.

A mixture of parachlorophenol (25 gm.), metacresyl acetate (25 gm.), and camphor (50 gm.), called XP-7,[13] is judged to be an excellent germicide and fungicide. It can be made on prescription by a pharmacist.

A zinc iodide-iodine solution is particularly useful when exudates persist in the root canal. In the concentration recommended[14] (zinc iodide 15.0 gm.; iodine crystals 0.6 gm.; distilled water 50.0 ml.) iodine is not significantly irritating or discoloring. Zinc iodide is an active astringent and is quite effective in drying up exudates. It is employed here as an intracanal dressing, not as an electrolyte.

Antibiotic Preparations

Almost every antibiotic used in medicine has been tried as a root-canal antiseptic dressing. Various combinations have been tested under laboratory and clinical conditions in a sincere attempt to produce the most effective bactericide and fungicide possible, while minimizing toxicity.

Grossman[15] introduced the polyantibiotic approach to the treatment of root-canal and periapical infections. His original formula (PBSC) was as follows:

Potassium penicillin G	1,000,000 units
Bacitracin	10,000 units
Streptomycin sulfate	1.0 gm.
Caprylate sodium	1.0 gm.
DC 200 silicone fluid	3 cc.

Later, the fungicidal agent sodium caprylate was replaced by the less irritating nystatin (10,000 units). Many other antibiotic preparations[13] are in active clinical or experimental use today.

Polyantibiotic preparations have stood the test of clinical use and are undeniably effective root-canal medicaments. They do, however, have certain shortcomings that should be mentioned:

1. Their use may result in the development of resistant forms of microorganisms, which renders the antibiotic ineffectual for possible future systemic use.

2. The patient may become sensitized to the antibiotic, and as a result may have an allergic response to it in the future. (Caution should be observed when the patient has any history of allergies.)

3. Polyantibiotic pastes tend to invalidate the results of culturing: trace amounts of antibiotics carried to the culture medium inactivate bacteria in the medium and give false negative results for as long as a week. There is one exception: trace amounts of penicillin can be neutralized by the addition of penicillinase to the medium. Methods of neutralizing residual amounts of other antibiotics are impractical for general use.

The use of a penicillin-camphorated parachlorophenol mixture has been advocated for highly infected root canals.[16] This is prepared immediately before use by placing a 50,000-unit tablet of soluble penicillin on a sterile glass slab, adding a drop of CMCP, and then spatulating. This produces a smooth, creamy paste that can easily be introduced into the root canal on a file or reamer. Excellent clinical results have been obtained with this mixture. It is not recommended for routine use but is of value when repeated positive cultures have been obtained with CMCP alone. When penicillinase is used in the culture medium, this penicillin-CMCP mixture does not interfere with the accuracy of the results.

In the sphere of honest controversy surrounding the relative merits of various medicaments, no single preparation seems to have outstanding advantages. Because of the built-in disadvantages of polyantibiotic preparations mentioned in preceding paragraphs, the use of the nonspecific drug CMCP is generally preferred.

Unacceptable Medicaments

Formocresol, widely used since 1900, is an excellent germicide but is extremely irritating to tissue; hence, it is contraindicated for endodontic use.

Beechwood creosote is an effective root-canal medicament and has enjoyed wide usage for years. Because of its moderately severe irritant effects and its disagreeable pungent odor, it is passing out of favor.

Phenol is a potent germicide but is highly caustic to tissue and should not be used for root-canal medication.

Sulfuric acid and other strong acids produce marked tissue damage and should never be used in a root canal. They were formerly used in selected cases as an aid in penetrating occluded canals.

Eugenol is a poor germicide and, contrary to popular belief, is somewhat irritating to tissue.

Electromedication

The principle of electromedication (electrosterilization, ionic medication) has been applied in endodontics to carry an antiseptic (usually an iodide-iodine solution) into the dentin and periapical tissues by means of a direct current. For details concerning the procedure the reader is referred to standard endodontic texts.[1-4]

In recent years the popularity of electromedication has diminished greatly, owing to the amount of chair time it consumes and the fact that results obtained with electromedication are similar to those obtained with the much less time-consuming use of the better root-canal medicaments.[17] Nevertheless, it would be wrong to condemn electromedication when so many dentists have used it successfully for so many years.

PROCEDURE

After the canal has been thoroughly instrumented and irrigated, it is dried with absorbent points. The medicament is then carefully wiped on the canal walls by means of an absorbent point saturated (but not dripping) with the medicament. Most effective medicaments are at best mildly to moderately irritating to connective tissues; thus it is essential to keep the drug within the canal and away from periapical tissue. For this reason the absorbent point is not left within the canal between appointments. The medicated absorbent point that fills the canal and touches the canal walls would expand when tissue fluids enter the canal. This expansion would cause fluid pressure within the canal, resulting in increased inflammation and tenderness in the periapical region. In addition, the expanded point might become wedged in the canal and tend to disintegrate upon later removal.

If the medicament is in paste or cream form, it is carried into the root canal by means of a syringe or a metal instrument, depending on its viscosity.

A small pledget of cotton containing a small amount of medicament is placed in the pulp chamber to act as a reservoir and prevent pieces of outer seal from falling into the canal when the seal is removed.

Placing the Double Seal

In order to prevent the medicament from escaping from the tooth, a double seal is employed (SLIDE 86). The inner seal, which is placed on top of the pledget of cotton in the pulp chamber, consists of gutta-percha. This layer of gutta-percha is carefully luted or sealed to the walls with a hot instrument and trimmed to make room for the outer seal. The outer seal should consist of zinc oxide and eugenol cement, or a zinc oxide and polyvinyl preparation (Cavit). Zinc oxyphosphate cement should not be used because it leaks consistently. The recommended cements produce a more effective seal, as shown by Parris and Kapsimalis.[18]

This double-seal technique is essential for preventing medicaments from escaping into the oral cavity, and also for preventing saliva, with its contained bacteria, from gaining entrance to the root canal.

SUMMARY OF PROCEDURES FOR CHEMOMECHANICAL PREPARATION OF THE ROOT CANAL

In the absence of acute symptoms, chemomechanical preparation of the root canal for filling can be accomplished in accordance with the following schedule:

First Appointment

1. Repair leaks in crown of tooth.
2. Anesthetize tooth, if necessary.
3. Isolate tooth by applying rubber dam.
4. Disinfect operating area.
5. Obtain access to root canal.
6. Grossly debride root canal with barbed broach.
7. Take sample for bacteriological culturing.
8. Establish working distance.
9. Irrigate canal thoroughly.
10. Enlarge and smooth canal with files or reamers, or both.

11. Flush and dry canal.
12. Dress canal with medicament of choice.
13. Place double seal.

Second Appointment

A minimum of 2 days should elapse between visits in order to allow time for cultures to develop and the medicament to take effect. (A period longer than 5 to 7 days is contraindicated—tissue fluids may accumulate in the canal.) The procedure on the second visit, regardless of whether the culture reading is positive or negative, is as follows:

1. Isolate tooth by applying rubber dam.
2. Disinfect operating area.
3. Remove cement portion of double seal with bur.
4. Repeat disinfection of opening to level of inner seal. (This is necessary because of percolation around margins.)
5. Remove gutta-percha portion of double seal and cotton pellet.
6. Debride root canal with broach and aspirate debris.
7. Take sample for bacteriological culturing.
8. Irrigate and aspirate canal.
9. Using largest file previously employed in canal, scrape all walls of canal throughout entire working distance. (Exudate from periapex frequently diffuses into canal and coagulates to encrust canal walls. Unless crust is removed, it decreases medicament's effectiveness.)
10. Irrigate and aspirate several times.
11. Dry canal with paper points.
12. Dress canal with medicament of choice.
13. Place medicated cotton pellet.
14. Place double seal.

The procedure outlined for the second visit should be repeated until the following requirements are met:

1. Two successive negative cultures have been obtained, each culture having been incubated for a minimum of 48 hours (preferably 72 hours).
2. The tooth is comfortable (no signs of acute periodontitis).
3. The canal is dry—with little or no exudate entering it.
4. The orifice to any previously existing discharge tract is closed and healed.

When these conditions are met after the second (or third or fourth appointment, if necessary), the root canal is ready to be filled.

MANAGEMENT OF ACUTE CASES

Up to this point, discussion has been centered on teeth that are asymptomatic, or relatively so, when the patient first presents himself for treatment and remain asymptomatic during treatment.

Sometimes the patient arrives in the dental office with acute, distressing symptoms. Acute cases require special consideration. First of all, treatment will differ depending upon whether the difficulty stems from the pulp alone or whether the periapical tissues are involved.

CLINICALLY ACUTE PULPITIS

Cases of clinically acute pulpitis are not of the incipient, histologically acute type but consist, in most instances, of acute exacerbations of a chronic pulpitis. The overriding symptom is pain created by exudate that is confined, or dammed up, in the pulp chamber and has no path of release. This pain is often difficult to localize to any one tooth, but intelligent observation of all diagnostic factors helps pinpoint the involved tooth.

Treatment

Effective local anesthesia is needed. The preliminary procedures of isolating the tooth under aseptic conditions and obtaining access to the root canal are carried out as previously outlined. Once the canal has been entered, the procedure is altered. The pulp is extirpated and the canal is carefully filled with irrigating solution and aspirated. Irrigation may be repeated several times to remove gross debris. Minimal instrumentation is undertaken: the canal is not enlarged fully on the first visit. Extreme care must be taken not to push debris, bacteria, instruments, or drugs through the foramen. A mild medicament such as metacresyl acetate (Cresatin) may be sealed in the canal, to remain until the second appointment; or the canal may be sealed without medication. The tooth should be checked to see that it is not in supraocclusion. On the second appointment, in 2 or 3 days, normal endodontic treatment procedure is followed.

ACUTE PERIAPICAL ABSCESS

Earlier, it was pointed out that movement of any powerful irritant (bacterial, chemical, or mechanical) through the apical foramen would precipitate an acute response. However, when acute symptoms are present initially, before any root-canal procedures have been started, the invading agent is usually bacterial. When bacteria gain access to the periapical spaces, an acute response will result if the microorganisms are extremely virulent or the local defense mechanism is inadequate (this may be related to lowered general resistance due to unfavorable systemic factors, including nutritional or metabolic disturbances).

Initially, when the acute response is limited to the apical periodontal ligament space, slight to extreme tenderness to percussion is felt. The tooth gradually becomes highly sensitive to any type of pressure. As the inflammatory exudate moves through the bone and seeks to penetrate the periosteum, an intense, throbbing pain is experienced. Finally, at the height of the infective process, characteristic local signs, and systemic symptoms such as fever, chills, and malaise, are present. The roentgenogram may show no demonstrable periapical change, or perhaps a thickening of the apical periodontal ligament space or a preexisting periapical radiolucency.

Treatment

Effective treatment of an acute periapical abscess depends upon the stage or intensity of the acute reaction when the patient enters the dental office.

Early Stage.—(This tooth has never been treated; tenderness or possibly severe pain are present but no appreciable mucosal swelling or symptoms of toxemia.)

The patient may be given sedatives for pain. Drainage by way of the root canal is an all-important objective. The tooth is first isolated under aseptic conditions as described earlier. In establishing drainage it is necessary to penetrate the enamel of an extremely sensitive tooth. This should be carried out with a minimum of vibration, preferably with advanced speed equipment such as an air-turbine handpiece. Infiltration anesthesia is contraindicated; block anesthesia is permissible but not always necessary.

The canal should be carefully cleared of pulpal remnants with a broach and gently flushed with sodium hypochlorite solution or sterile isotonic solution of sodium chloride. Extreme care should be exercised that no debris is pushed or flushed through the apex. The next step is to make certain that the root canal is open wide enough to facilitate drainage. Because the apical foramen may have become plugged with thick exudate or tissue debris, this is the one time it is necessary to instrument carefully 1 mm. to 2 mm. beyond the working distance so that the foramen is open. The canal need be instrumented only to clear gross debris; it should not be fully instrumented and prepared at this visit. The canal is then left open for a few days to drain freely. A small pledget of cotton may be placed in the pulp chamber to prevent clogging of the canal with food debris. Warm saline rinses are recommended. A very important step is to take the tooth slightly out of occlusion.

Advanced Stage.—The patient who presents himself for treatment of an acute periapical abscess in the advanced stage is a sick patient. He has experienced excruciating pain and loss of sleep; he manifests mucosal or facial swelling, or both, and systemic symptoms of toxemia.

Drainage is still a prime objective. In this case the suppurative process has continued along the path of least resistance and has culminated in a subperiosteal abscess. If this fluctuant lesion can be palpated beneath the mucosa, it must be incised surgically to release the contained purulent material. Since this incision is extremely painful to an already exhausted patient, one of the following measures may be taken to relieve the pain: (1) Administer a local anesthetic by block injection; (2) carefully inject an anesthetic into the mucosa around the periphery of the raised area; (3) apply ethyl chloride spray to the area; or (4) apply a topical anesthetic.

The incision should be made down to the bone. Following this, a hemostat should be inserted into the incision and the beaks spread. A rubber-dam I wick may be inserted into the incision, one bar of the I being allowed to extend out into the oral cavity. This should be checked daily to be sure that drainage can continue.

Although establishment of drainage is of prime importance, the dental officer must provide assistance to the natural defense mechanisms that are combating the severe infection. This supportive treatment consists of several aids:

First, systemic treatment with antibiotics should be initiated. The patient's history sheet should reveal possible allergies or sensitivity to antibiotics. The safest and most effective procedure is to obtain a sample of the exudate (if possible from the root canal) and, with the aid of local medical laboratory facilities, determine the specific antibiotic that would be effective against the microorganism involved. It is approved practice, however, to proceed with an antibiotic of choice while waiting for test results. Phenoxymethyl penicillin, U. S. P. (Pen-Vee, V-Cillin), taken orally, 250 mg. every 6 hours, is an effective antibiotic. For penicillin-resistant organisms, erythromycin, U. S. P., taken orally, 250 mg. every 6 hours, is also effective. The usual course of antibiotic therapy extends for 5 days or until 24 hours after the patient's temperature has returned to normal.

In order that the reader may become thoroughly familiar with the value, the limitations, and the dangers of antibiotics, it is recommended that he take advantage of excellent descriptions regarding their use in endodontic practice to be found in current endodontic texts.

For further supportive treatment in controlling the acute infection, it is essential to prescribe (1) analgesic and sedative agents for relief of pain; (2) a mild cathartic to assist in elimination; (3) a diet of light, nourishing foods; and (4) adequate rest and sleep.

A rather rapid decrease in the acute symptoms follows the initiation of systemic drug therapy and the establishment of drainage. When the acute symptoms have disappeared, usually within 3 days, routine treatment of the canal should be started. Thorough root-canal therapy results in the elimination of the infection in the canal, and this makes it possible for the local defense mechanism to return the affected tissues to a state of health.

Acute Exacerbations Between Appointments

When between-appointment flareups occur in the periapical tissues of teeth under treatment, the causes point directly to carelessness or lack of experience on the part of the dental officer. These acute exacerbations occur more frequently following the first appointment than at any other time. Common causes are overinstrumentation; forcing of toxic protein degradation products through the foramen; forcing of microorganisms and their toxins into the periapical space, with attendant inoculation and infection of the area; and sealing in of highly irritating medicaments, possibly in excessive amounts.

Usually these between-treatment exacerbations are painful enough to bring the patient to the dental office but are not of the intensity and magnitude just described. Treatment should include the following steps:

1. Open the canal under aseptic conditions.
2. Irrigate the canal gently and thoroughly.
3. If exudate is present, allow it to drain completely. Dry the canal.
4. Seal in glycerite of iodine with a double seal. (This is a special-purpose medicament that is effective in reducing edema in periodontal ligament spaces. The iodine has a disinfecting effect, but glycerine is the active ingredient. Glycerine, being hydroscopic, pulls water from the edematous periodontal spaces into the canal and slowly reduces the discomfort brought on by the congestion.)
5. Make sure the tooth is very slightly out of occlusion.

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CHAPTER 6

NONSURGICAL ENDODONTIC TREATMENT: FILLING THE ROOT CANAL

The objective in filling the root canal is to obliterate the pulp space within the canal throughout the working distance. If space remains in the canal in communication with the apex, tissue exudate can accumulate in the void. The decomposition products from the accumulated exudate can have an irritating effect on the periapical tissues and may delay or prevent healing. Also, at the time of transient bacteremias, microorganisms may make their way to this accumulated fluid and find it a suitable medium in which to localize and proliferate. Their toxins could further irritate the periapical tissues. [1]

APICAL EXTENT OF FILLING MATERIAL

There is a difference of opinion regarding the ideal apical extent of the filling material. Some authorities think that it should extend beyond the apex (overfilling the canal); some think that it should be flush with the apex; and others believe that it should end at the dentinocemental junction (underfilling the canal a distance of 0.5 to 1 mm.)

An overfilled canal does not necessarily ensure a well-condensed filling and should be avoided because the protruding filling material is always an irritant. In the normal masticatory movement of the tooth in its alveolus, protruding material would continually lacerate the periapical tissues and prevent normal healing. It is true that many overfillings seem to be tolerated, but the reparative power of the tissues cannot be determined in advance—so why take the chance?

Filling a root canal exactly to the apical foramen is extremely difficult to accomplish. The reasons are these: The apical opening of the root canal is usually not at the tip end, or apex, of the root. In the fully formed tooth the canal usually curves and makes its exit on a lateral surface. Therefore, if one side of the filling point is flush with the foramen, the other side is protruding (SLIDE 87). If the canal foramen opens above the apex on the buccal or lingual surface of the root, the canal may be grossly overfilled but appear to be filled flush with the apex in a roentgenographic view. (SLIDE 88 shows a diagrammatic view of this possible situation, and SLIDES 89 and 90 show extracted teeth from actual clinical cases in which this error was made.)

A further potential fault in filling exactly to (or flush with) the apex should be pointed out. Root resorption occurs in varying degrees where there is inflammation of the periodontal ligament and destruction of large areas of periapical tissue. When a root canal is filled exactly to the apical foramen in a tooth in which resorption is likely to continue during the healing process, it is possible that such a tooth ultimately may be overfilled[2] (SLIDE 91).

Siskin[2] has capably described the preferred apical extent of the filling material:

"In the procedure of choice, the root canal should routinely be obturated just short of the shortest length of the canal. The canal should be filled so that the proliferating periodontal membrane will at first project into the unfilled portion and then ultimately lay down secondary cementum to produce the final seal of the canal. When the space between all aspects of the actual apical orifice and the apical extent of the obturator is from 0.5 to 1 mm., there will be a free physical and chemical interchange between the fluids in the unfilled portion of the canal and the fluids around the apical end of the root, and consequently no stagnation."

FILLING MATERIALS

The ideal filling material for root canals would be:

1. Nonirritating to periapical tissues.
2. Impervious to tissue fluids.
3. Adaptable and adhesive.
4. Radiopaque.
5. Nonvolatile and nonshrinking.
6. Bacteriostatic.

7. Able to penetrate lateral or accessory canals.
8. Easy to sterilize.
9. Nonstaining.
10. Easy to prepare, manipulate, and place.
11. Easy to remove from the pulp chamber (and from the root canal if necessary).
12. Stable.

No single material now available fulfills all these requirements. However, a combination of gutta-percha points and sealer, or of silver points and sealer, comes close. Just how close depends on the dental officer's skill.

Gutta-percha Points

Gutta-percha is a latex exudate, similar to rubber in chemical composition. It is supplied in hand-rolled points varying in length, taper, and diameter. Some manufacturers make the points conform as closely as possible to sizes of files and reamers. Gutta-percha is valuable as a root-canal filling material because it is pliable and may be compressed against the walls of the root canal. It will not discolor the tooth or irritate periapical tissues (if not overextended). Also, gutta-percha is opaque to roentgen rays; and, if necessary, it can easily be removed from the canal.

Silver Points

Silver points are cones of pure metallic silver, manufactured to correspond to the sizes of files and reamers. They are particularly useful for filling long, fine root canals.

Silver points have some advantage over gutta-percha points: (1) They are flexible enough to be inserted or threaded into fine, curved canals, yet rigid enough to be seated with firm pressure with less likelihood of buckling; (2) they can be removed from the canal, adjusted, and reinserted as many times as deemed advisable without fear of breakage or distortion; and (3) previous to each insertion they can be sterilized in the heated-well sterilizer, provided they are immersed for a sufficient length of time.

In the use of silver points, careful preparation of the root canal becomes a matter of paramount importance. The apical third, in particular, must be prepared so that the point used to fill it can fit snugly against the walls. All irregularities must be filed away from the walls, and the entire canal must be made as nearly conical as possible. Advantage can be taken of the fact that in almost all cases the apical third of all root canals is round in cross section. The silver point that corresponds most closely in size and taper to the size and taper of the apical third of the canal must then be selected. This point will ordinarily correspond in number to the file used in the preparation of this portion of the canal.

Root-Canal Sealer

The requirements of the ideal root-canal sealer closely parallel those of the ideal root-canal filling material. In addition, the sealer must have a long enough setting time for the dental officer to adjust the position of the gutta-percha or silver point.

Zinc oxide and eugenol are the basic ingredients of the most widely used sealers. When mixed, these ingredients form a creamy paste that sets in a desirable length of time and, once set, can be dissolved with chloroform or dimethylbenzene (Xylene), if necessary.

A commercial product based on Rickert's formula (Kerr's Root Canal Sealer) incorporates precipitated silver, resin, and thymol iodide in the zinc oxide powder, and canada balsam in the eugenol. The mixture comes close to being the ideal sealer and has been used successfully for years. It has one disadvantage, however: The silver in the mixture tends to stain tooth structure. Therefore, extreme care should be taken that all sealer is removed from the pulp chamber.

Grossman advocates a sealer that contains bismuth subcarbonate and barium sulfate instead of silver compounds. It does not stain tooth structure and has excellent handling properties. The formula for Grossman's sealer[3] is as follows:

<u>Powder</u>	
Zinc oxide, reagent or C. P.	20.0 gm.
Staybelite resin	13.0 gm.
Barium sulfate	7.5 gm.
Bismuth subnitrate	7.5 gm.
Sodium borate, anhydrous	2.0 gm.
<u>Liquid</u>	
Eugenol	

Plastic Root-Canal Filling Materials

At the present time a significant amount of attention is being directed toward the development of new filling materials. Plastic root-canal points of acrylic, polyethylene, polypropylene, nylon, copolyvinyl, and the epoxy resins have been investigated. Of these, the polypropylene and epoxy resin points warrant further experimental trial. [3] Resins and silicone rubber are being considered as cementing media. From these investigations it is hoped that a superior root-canal filling material will emerge.

Unacceptable Materials

In view of both the objective of filling root canals and the requirements of the ideal filling material, it is apparent that some materials, and techniques of handling them, are unacceptable. Only two examples will be discussed here:

1. Overfilling the canal with an absorbable paste is advocated by some dentists on the assumption that the body will absorb all excess material. However, there is no basis for assuming that only the material extruded through the apical foramen will be absorbed. The paste in the root canal may also be absorbed, and the seal will be lost.
2. The use of gutta-percha in conjunction with a solvent, such as chloroform or eucalyptol, has been advocated as an easy method of filling root canals and their ramifications (SLIDE 92). Most authorities agree, however, that any filling technique involving the use of a solvent has a major disadvantage: in time, the solvent evaporates and the filling shrinks; and when the filling shrinks, the seal is lost.

FILLING TECHNIQUES

GUTTA-PERCHA POINT LATERAL CONDENSATION TECHNIQUE

The gutta-percha point lateral condensation technique is an effective method of filling the broad canals of anterior teeth and single-rooted posterior teeth. It is particularly useful for filling an ovoid or ribbon-shaped canal. Because the filling is placed under heavy pressure, this method should be reserved for canals with a definite apical taper, or constriction. Otherwise, the filling material may be extruded through the apical foramen.

A trial point is selected which, when inserted to the correct working distance, fits snugly in the apical 3 or 4 mm. of the canal. Great care should be given to selection and fitting of this trial point. Repeated roentgenograms may be necessary, and the trial point may have to be snipped at the tapered end several times to ensure the best possible fit.

After the trial point has been selected and adjusted, it is cut even with the incisal edge or with the tip of a facial cusp so that its length is equal to the working distance. The trial point is then removed from the canal and laid aside on a piece of sterile gauze. While the dental officer dries the canal with paper points, the technician prepares a creamy mix of sealer. The trial point is lightly coated on its apical half with sealer and inserted to the predetermined distance. Next, a No. 3 spreader is inserted alongside the trial point under moderate pressure. The spreader is then withdrawn with a rotary motion, and a gutta-percha point slightly smaller than the spreader is quickly inserted in its place (SLIDE 93). This procedure is repeated, with the pressure gradually being increased, until the spreader will no longer penetrate beyond the cervical line; it may take 20 to 30 small points to fill a large canal completely (SLIDE 94).

A final roentgenogram is made; and if the filling appears satisfactory, the excess gutta-percha and sealer are removed from the pulp chamber (up to the level of the gingival attachment). A cotton pellet saturated with dimethylbenzene (Xylene) or chloroform is helpful in cleaning out the pulp chamber, as either of these solvents dissolves both gutta-percha and sealer.

Once the pulp chamber has been completely cleaned, the opening is sealed temporarily by placing a cotton pellet in the pulp chamber and closing the opening with cement. This temporary seal can be removed easily in the highly unlikely event that the periapical region becomes inflamed and drainage via the root canal becomes necessary. If the tooth remains asymptomatic for 2 to 3 weeks, a permanent restoration may be placed.

Before the restoration is placed, particular care should be exercised in cleaning the pulp chamber. The portion to be filled, particularly the incisal portion, should be thoroughly debrided and again washed with dimethylbenzene or chloroform to prevent discoloration.

SECTIONAL GUTTA-PERCHA POINT TECHNIQUE

In the sectional technique, sections of gutta-percha points are carried into the root canal on the tip of a warmed plugger and then condensed until the canal is filled in all dimensions (SLIDE 95). Each section of gutta-percha may be warmed to facilitate condensation, or sealer may be used to ensure a complete seal. The use of solvents, however, is contraindicated.

Assuming that the canal has been adequately prepared, filling it successfully by the sectional method depends primarily on the proper selection and placement of the apical section of gutta-percha. If this section fits tightly in the apical third of the canal, subsequent sections can be condensed against it with considerable pressure. If the first section does not fit tightly, it may be forced through the apical foramen when pressure is applied.

The problem in using the sectional technique is to select a section of gutta-percha point with the same dimensions as the apical third of the canal. This can be done by selecting a section with the same dimensions as the end of the largest file used in preparing the canal. After the apical section has been chosen, a plugger must be selected (and sometimes adapted by bending) for carrying the section to the dentinocemental junction. (Having determined the working distance previously, the dental officer can control the depth of insertion.) If resistance is felt at the proper depth, the plugger is rotated slightly and withdrawn. A roentgenogram is then made to check the position of the apical section; any necessary adjustments are made; and the rest of the canal is filled.

SINGLE SILVER POINT TECHNIQUE

In canals that are thin and threadlike; that is, buccal canals in maxillary molars, buccal and lingual canals in maxillary bicuspids, mesial canals in mandibular molars, and occasionally a canal in a mandibular incisor, it is possible to adapt a silver point to the entire length of the canal.

The first step in the single silver point technique for posterior teeth is choosing the point that most nearly fits the canal, particularly the apical third. The choice should be made with the aid of roentgenograms (SLIDE 96).

When the points are being fitted to the root canals of the tooth, each point should be removed from its canal by grasping it with a hemostat horizontally, using a certain cusp (or combination of cusps) as an occlusal guide marker (SLIDE 97). Each point should remain in its own labeled hemostat to avoid confusion concerning which point fits in which canal. Each time a point is inserted in its canal, it should be resterilized by immersion in the heated-well sterilizer for at least 20 seconds. An alternate method is flaming, but there is danger of melting the tip or altering the characteristics of the metal.

After the points have been fitted, their tips are smoothed to remove any roughness, and each point is cemented firmly to position. Baseplate gutta-percha is then packed into the flaring canal orifices, and into the floor of the chamber to a depth of 2 to 3 mm. This makes later removal of the points possible, if necessary. The remainder of the pulp chamber is filled with cement, care being taken to pack it securely around the protruding silver points.

After the cement has hardened, both cement and points are cut down to the desired level with a No. 37 bur (SLIDE 98). In mandibular anterior teeth this method of cutting down silver points is impractical because there is not enough room for an instrument to be placed alongside the silver point to pack the gutta-percha or cement, or to cut down the point. A practical method is to notch the silver point at the gingival level before the point is sealed in position. This is done by carefully measuring the length of the clinical crown and transferring this length to the point, which is then notched with a disk (SLIDE 99). After the silver point has been positioned, carefully rocking it back and forth a few times will break it off at the correct level.

COMBINATION SILVER POINT AND GUTTA-PERCHA POINT TECHNIQUE

This technique is used in canals with a wide taper, where the silver point may fit snugly in the apical third but there is space lateral to the point in the occlusal two thirds. The technique is particularly useful for filling the ovoid or ribbon-shaped canal of a single-rooted bicuspid or the distal root of a mandibular molar. It could also be employed in the wide lingual canal of a maxillary molar.

The silver point is positioned with sealer in the usual manner. Adaptation of the silver point to the canal should be perfect in the apical third. In the occlusal two thirds of the canal the space lateral to the silver point is filled with as many fine gutta-percha points as possible, with a No. 3 spreader being used in the method described for the gutta-percha point lateral condensation technique. (Of course, the gutta-percha point lateral condensation technique could be used in this type of canal as an elective alternate method.)

NOTCHED SILVER POINT TECHNIQUE PRIOR TO DOWEL RESTORATION

When the crown of a pulpless tooth has been fractured, or when the crown is comparatively weak and fracture might occur later, it is wise to take this fact into account in choosing the canal filling—for perhaps immediately or years hence a dowel restoration will be made for the tooth. When the entire root canal, from apex to cervical level, has been filled with gutta-percha or a silver point, it is usually impossible to remove the cervical portion of the filling without disturbing the seal established in the apical portion. Loss of this seal generally leads to failure of the root-canal filling and generation of apical pathosis. The technique to be described is designed to minimize this danger through use of a two-layer root-canal filling, which is virtually immune to such disturbance.

The canal is enlarged, if possible, to at least the size of a No. 50 (conventional No. 6) instrument (a reamer should be used for the final part of the procedure). It is advisable to create the so-called Washington Monument effect in the preparation (SLIDE 100). This consists of terminating the preparation close to the dentinocemental junction, approximately 0.5 to 1 mm. from the true apex, thus providing a V-shaped backstop that greatly facilitates fitting the silver point.

A silver point of the same size as the largest reamer used is tapered and smoothed to fit the shape of the canal in the apical third; that is, when the point has been inserted to the correct working distance, the point should bind tightly in the apical 5 to 7 mm. (SLIDE 101). If a small, straight hemostat is clamped onto the silver point perpendicular to the point's long axis and touching the incisal edge of the tooth, a positive reference for depth of insertion is obtained. The point is then removed from the canal and notched on each side to a depth of one-third of the diameter by means of a thin diamond wheel or disk (SLIDE 102). The level of the cuts is determined by the desired depth of the dowel, but, on the average, they should be about 5 to 7 mm. from the dentiocemental junction.

After the canal has been dried, the silver point is coated from apex to notch with sealer, and firmly seated to the depth of the preparation. The incisal portion of the point is twisted off by means of apical pressure and 360-degree rotation until the break occurs (SLIDE 103).

If the dowel preparation is to be placed within a short time, the remainder of the canal may be filled with a dry absorbent point and double sealed. If a dowel preparation is planned for the future—whenever needed—the remainder of the canal may be filled with gutta-percha points or warm zinc oxide and eugenol paste and topped off with the restoration of choice. This cervical portion of the filling can easily be removed without disturbing the apical seal.

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CHAPTER 7

SURGICAL ENDODONTIC TREATMENT

The primary purpose of endodontic treatment is to eliminate all irritants from the root canal and completely obliterate the spaces where irritants might accumulate. In this way, the defensive and reparative elements in the periapical tissues are allowed to accomplish healing.

In some cases, because of the anatomical configuration of the root or because of mechanical difficulties, the dental officer cannot navigate the canal and cannot completely fill the root-canal spaces from the coronal approach. In these cases a surgical approach is indicated. In other cases a periapical lesion fails to show roentgenographic and clinical evidence of repair within a reasonable time, in spite of the fact that the tooth with which it is associated has been given careful and thorough root-canal treatment. In these cases, fortunately in the minority, surgery is definitely the treatment of choice.

The surgical procedures to be described in this chapter include (1) periapical curettage, (2) root resection, (3) resection with root-end seal, and (4) the reduction of large periapical radiolucencies prior to curettage.

<u>Periapical curettage</u> involves the removal of all soft tissue in the lesion surrounding the apex of a pulpless tooth without disturbing the root itself. Maintenance of the integrity of the root is desired, where possible, because a longer root means better support and an intact cementum predisposes to better healing. Curettage is indicated in the following situations:

1. A broken root-canal instrument has been forced through the apical foramen into the periapical spaces.

2. A root canal is grossly overfilled. (Protruding filling material, especially a silver point, constantly irritates the periapical tissues. In most cases the protruding material can be severed and smoothed off to establish a seal at the apex.)

3. Conservative (nonsurgical) endodontic treatment has been thoroughly and capably completed, but an existent periapical lesion has either failed to show evidence of repair or has expanded in size. It is important to refer back to the section on periapical pathosis for possible reasons for failure of osteogenesis to occur. Two pertinent reasons stated there are (1) persistence of a source of irritation not detectable by roentgenographic means and (2) presence of a cyst.

With regard to the first reason for failure of osteogenesis to occur, when surgical access is obtained for curettage, it is absolutely essential to examine the apical seal thoroughly. If examination reveals a faulty seal or an apical condition that might provide a source of irritation, the safest procedure is thorough curettage plus resection with amalgam seal. With regard to the second reason, if the root canal of a tooth associated with a periapical radiolucency continues to exude a serous fluid in spite of all efforts to stop it, or if it is impossible to obtain a negative culture, the presence of a cyst must be strongly suspected. In these latter cases, if surgery is mechanically and medically feasible, curettage (with careful evaluation of the apex) is indicated.

<u>Root resection</u> involves the cutting off of some portion of the root end and includes curettage when a lesion is present. The root canal must first be completely filled to the level of obstruction or difficulty; then the root end is removed back to the level of sound filling material. Root resection is indicated in the following situations:

1. A tooth with a sharp apical curvature cannot be adequately instrumented and sealed to the dentinocemental junction.

2. A broken instrument has wedged in the apical third of the root canal.

3. An instrument has perforated the root in the apical third.

4. A root is fractured in its apical third, and the pulp is nonvital. (If the pulp retains vitality, healing will occur without endodontic intervention.)

5. During curettage, a root tip denies access to all parts of a lesion.

Sometimes, access through the crown of a tooth is impractical or impossible; thus conventional treatment, including the all-important sealing of the apex, is impossible. In these cases surgical access is employed. Resection with root-end seal is a modification of the standard root resection procedure involving placement of a filling material in a root-end preparation to secure a positive seal. Resection with root-end seal is indicated in the following situations:

1. Excessive deposits of calcium or secondary dentin make it impossible to carry out instrumentation of the canal.
2. Periapical pathosis has developed around a tooth after a post crown has been placed.
3. Periapical pathosis has developed because of a faulty root-canal filling which cannot be removed.
4. A broken instrument is wedged in the incisal two thirds of the canal.
5. Nonsurgical endodontic treatment might lead to fracture of a restoration (a porcelain jacket crown, for example), or to fracture of a fragile dentin stump under a restoration.
6. An immature anterior tooth with a wide-open or funnel-shaped foramen cannot be adequately sealed from the coronal approach. (In this case the canal must first be filled as completely as possible from the coronal approach.)

CONTRAINDICATIONS FOR PERIAPICAL SURGERY

Periapical surgery is contraindicated in the following circumstances:

1. Roentgenograms indicate that the root-canal filling is faulty. (Re-treatment by nonsurgical means is the procedure of choice.)
2. The roots are in proximity to the nasal cavity, the maxillary sinus, the mandibular canal, the mental foramen, or other anatomical space that might be endangered by surgery. (If apical surgery can be done at all in these circumstances, it must be accomplished by a dental officer well qualified and experienced in surgical procedure.)
3. The region of periapical radiolucency is so extensive that the apices of adjacent vital teeth are involved. (In this situation it may be advisable to reduce the lesion by drainage before performing surgery.)
4. The remaining root will be so short after resection, or there is so severe a periodontal bone loss, that inadequate periodontal retention is anticipated.[1]
5. There is an untreated oral infection or acute periodontal involvement.
6. There is an uncontrolled systemic condition, such as a chronic debilitating disease or some form of blood dyscrasia.

PRESURGICAL CONSIDERATIONS

Examination

The examination, of course, is conducted during the diagnostic phase, which was discussed earlier. If the preliminary examination and history show that the patient is not in good health or that he is undergoing any type of medical treatment—therapeutic radiation, for example—the dental officer should most certainly consult the medical officer. Any oral infection or acute periodontal involvement should be brought under control before endodontic treatment is begun.

Presurgical Root-Canal Treatment

Sound root-canal therapy is an absolute prerequisite to surgical curettage or root resection. This calls for aseptic technique at every step of the procedure. It requires thorough instrumentation and irrigation. It requires elimination of all microorganisms from the canal just as in any sound endodontic therapy, and the only reliable means of determining sterility of the canal is by means of cultures. The canal must be filled completely as far as is mechanically possible. Unless the causes of periapical involvement have been removed and the canal completely sealed, bone repair will not follow curettage or resection. Surgery is an adjunct to proper therapy, not a patch-up or cure for poor root-canal treatment.

Control of Apprehension; Premedication

An often neglected yet most important consideration in any surgical procedure is control of apprehension in the patient. It is of prime importance that the dental officer show a little human understanding. His demeanor should be such that the patient is confident of being in good hands. Above all, the patient must be made to feel that the dental officer regards him as a sensitive human being and not simply as an interesting case. In addition, it is often advisable to use premedication to allay apprehension.

Supportive medication may also be necessary in some cases. In particular, patients with valvular heart involvements or a history of rheumatic fever or other febrile diseases that predispose to valvular damage should have prophylactic supportive therapy before any surgical procedure. This is recommended because of the possibility that during surgery microorganisms may be forced into the bloodstream, become attached to the damaged valves, and cause the onset of subacute bacterial endocarditis. Penicillin is the antibiotic of choice; but if the patient has a history of previous reaction to penicillin, erythromycin or a broad-spectrum antibiotic should be employed. For current recommendations regarding prophylactic antibiotic dosages, the reader is referred to the latest edition of Accepted Dental Remedies.[2] Any drug administered should be specific for the needs of the individual patient.

Obtaining Anesthesia

Obtaining anesthesia should not present a problem; the methods commonly used in exodontia are applicable to apical surgery. The local anesthetic solution should be injected slowly, an aspirating syringe being employed to prevent intravascular injection. Minimal adequate amounts of anesthetic should be used to ensure control of pain. When apical surgery is contemplated on an upper incisor, care must be taken to secure adequate palatal anesthesia. This is provided by injecting a few drops of anesthetic into the anterior palatine foramen, care being taken not to pass the needle through the sensitive papilla. Pain can be minimized by inserting the needle into the tissue at the side of the papilla and then directing it toward the foramen.

Maintaining Asepsis

Although it is not possible to reach the level of asepsis of a hospital operating room, much can be done in surgical endodontic procedures to minimize the amount of contamination within the surgical field:

1. The patient should be draped with sterile towels.
2. The exposed part of the patient's face should be cleansed with surgical soap.
3. An effort should be made to control salivary contamination. (In most instances, placing gauze squares in the buccal vestibule is sufficient, though administration of an antisialagogue may be advisable.)
4. The operative area should be painted with an antiseptic such as tincture of metaphen.
5. The dental officer and his assistant should wear masks, sterile rubber gloves, and sterile chest drapes.
6. All items entering the surgical field must be sterile.

SURGICAL METHOD

In this section surgical method will be discussed in general terms: principles rather than instrumentation will be stressed. Each dental officer prefers to select his own instruments for apical surgery, and rightly so, for each knows best which instruments work well in his hands.

The surgical approach for curettage, resection, and resection with root-end seal is identical up to the point of resection of the root. Thus, the paragraphs that immediately follow apply to all of these procedures.

OBTAINING ACCESS TO THE ROOT APEX

Making the Incision

There are two main types of incision for gaining access to the root apex: the semilunar incision and the full-flap incision. The governing factors in determining which to use are the adequacy of the blood supply to the tissue of the flap and the need to make (and close) the incision over a sound table of bone. Most dental officers prefer the semilunar incision (SLIDE 104).

The semilunar incision should extend far enough on either side of the tooth to be treated—about the distance of two teeth—so that reflection of the flap will provide proper access to the periapical region. If the labial frenum is encountered, one should cut around it (if at all possible) to avoid the formation of excessive scar tissue in this area. The vertical placement of the incision is also important. If it is placed too near the mucobuccal fold, relatively large vessels will be severed and unnecessary hemorrhage will result. If, on the other hand, it is placed too near the gingival crest, loss of blood supply to the gingival tissues may result. Ideally, the incision should be started and ended about 2 mm. from the mucogingival line and should be kept at least 4 mm. away from the gingival crest. This design will afford little hemorrhage into the operative site. It will provide adequate blood supply to all tissues and will eliminate tension when the incision is sutured.

Where roentgenographic examination or palpation show that the lesion extends to the middle portion of the root, so that there is not a sufficient table of bone, the full-flap incision must be used (SLIDE 105). In this incision tissue is reflected apically from the gingival level. Care must be taken that the vertical incision of the full flap does not terminate on thin mucosa overlying a root prominence.

In either type it is important that the incision be carried all the way to the bone, and that the periosteum be carefully elevated and retracted along with the tissue flap. If the periosteum is not elevated and retracted, it will be damaged when bone is removed to expose the root apex. If the periosteal elevator is used carelessly, the periosteum will be pushed under the tissue flap. In either case healing will be delayed.

A retractor or a periosteal elevator may be used to retract the tissue flap. Because of the enormous mechanical advantage of these instruments, the bone being used as a fulcrum, they must be used with great care. The soft tissues, particularly the lips, should be handled as gently as possible to prevent postoperative swelling and possibly a hematoma.

Locating the Lesion

In most instances, locating the periapical lesion after the flap has been elevated is no problem, for the cortical bone has usually been perforated by the vascular granulation tissue (SLIDE 106). If the cortical bone over the lesion is still intact, it is usually very thin, and the lesion can be outlined by punching through the bone several times with a sharp explorer. The overlying bone can then be removed easily with hand instruments.

When the overlying bone is very dense or very thick and there is no sign of the lesion, the relative position of the root may be visualized by observing the contour of the overlying bone. The level of the apex can be pinpointed by using sterilized dividers preset to the length of the tooth (SLIDE 107). When the apex has been located, the next procedure depends upon the speed of the surgical handpiece available.

With a standard speed handpiece the root can be outlined by inserting a No. 6 bur at the apex and then inserting the same bur, mesially and distally, at the junction of the middle and apical thirds of the root.[3] A fissure bur can then be used to join the openings made with the round bur, and the bone fragment can be elevated with hand instruments.

With use of an advanced speed handpiece in surgical procedures the bone overlying the root can literally be "wiped" away with a round bur. Great care should be exercised. Whenever a bur is used to cut bone, the operating field must be copiously flooded with sterile isotonic solution of sodium chloride to dissipate heat, to prevent the bur from becoming clogged, and to facilitate clear vision.

Once the dental officer has opened a bony window overlying the root apex and thus has gained access to the apical region (SLIDE 108), he is ready to carry out the procedure he has chosen—either periapical curettage, root resection, or root resection with root-end seal.

PERIAPICAL CURETTAGE

Where simple curettage is indicated without the need of removing root structure, it is accomplished by judicious use of surgical curettes. If the root tip denies access to all parts of the lesion, the dental officer should consider the possibility of enlarging the bony window rather than removing the root tip to gain access. Bone will fill in, but root structure is lost forever.

During curettement, great care should be exercised to ensure that neighboring anatomical spaces such as the nasal cavity, the maxillary sinus, or the mandibular canal are not entered. Care should also be taken not to destroy adjacent bone. For example, if palatal cortical bone is needlessly lost, normal bony healing may not occur, and an "operative defect" (healing by dense, collagenous connective tissue) will result (SLIDE 109). Still another reason for caution is that curetting too far laterally could destroy the blood supply to adjacent teeth.

Where possible, a tissue specimen (as large and intact as possible) should be placed in 10-percent formalin and sent to the oral pathologist.

ROOT RESECTION

In this procedure mechanical difficulties have dictated that a portion of the root apex be removed. It is very important that the root tip be in clear view. There must be no guesswork at this point, or confusion because of excessive bleeding. Unless the dental officer is sure of the location of the root tip, he may cut across the periapical bone beyond the root tip and perforate the wall of an adjacent space (SLIDE 110); he may cut across the root at a level closer to the cervix than desired (SLIDE 111); he may not cut the root tip completely off, but leave a sharp spicule of root to irritate the surrounding bone (SLIDE 112); or he may cut too far laterally and cut into an adjacent sound tooth, possibly far enough to devitalize its pulp (SLIDE 113). Copious irrigation and proper aspiration make it easier to distinguish between bone and tooth structure by their slight difference in density and color.

Regarding the level of resection, here are a few points to remember. It is not necessary to resect the root at the lowest level of a maxillary periapical lesion (or the highest level of a mandibular lesion). This would result in needless loss of root length (SLIDE 114). The cut should be made at a level where the broken instrument or the sharp apical curvature—in other words, the reason for resection—can be eliminated (SLIDE 115). If better access to hard-to-reach tissue is needed (SLIDE 116), a logical approach is to enlarge the access window by conservative removal of bone. (In the case of maxillary anteriors this could be accomplished at the superior aspect (SLIDE 117).)

In any event, it is important to remove no more of the root than is absolutely necessary (SLIDE 118). If too much root structure is removed (SLIDE 119), the tooth may not be able to withstand normal stress; healing will not occur; and the tooth will be lost.

The root tip should be cut off with a fine fissure bur, if possible in an advanced speed handpiece. Further smoothing and rounding of the edges can be done with a fine file or a finishing bur to leave a nonirritating surface. The surgical wound should be flushed, dried, and thoroughly examined. The apical face of the filling material should be probed to make sure the apical seal is tight. If the material is gutta-percha, it may be sealed to the canal walls with a hot plastic instrument. When the operative area is clean, closure is routine.

RESECTION WITH ROOT-END SEAL

Resection with root-end seal is the procedure used when an effective apical seal cannot be obtained by way of the conventional coronal approach. A number of materials, including amalgam, gutta-percha, silver points, zinc oxide-eugenol cement, and zinc oxyphosphate cement, have been employed for the purpose. Because of its superior sealing qualities, amalgam is preferred.

The procedure for resection with root-end seal is basically the same as that for standard resection up until the point when the root is resected. In the resection with root-end seal method, the apex is cut off at a 45-degree angle, with the apical surface slanting toward the dental officer performing the operation (SLIDE 120). This affords a clearer view of the apical surface of the root. A sharp explorer is used to locate the foramen or the point of the closed foramen. A small round bur is used to enlarge the foramen (or create an artificial foramen) to a depth of about 3 mm. With an inverted cone bur a retention undercut is formed in the enlarged foramen (SLIDE 121). When an immature tooth with a funnel-shaped or flared-out foramen is being treated, the sharp terminal margin of the apex is smoothed and the apical portion of the canal is cleaned and prepared to receive a filling.

In either case, after the foramen has been prepared to receive the amalgam, the field is irrigated with sterile isotonic solution of sodium chloride, aspirated, and dried with gauze sponges (SLIDE 122). The root tip should then be carefully isolated with gauze sponges to prevent moisture from contaminating the amalgam and to prevent amalgam fillings from falling into the bony defect.

Amalgam is inserted in the foramen with a serrated plugger. Only very small quantities should be inserted at a time, and meticulous care must be taken not to spill excess particles outside the limits of the canal. Excess amalgam is easily embedded in the walls of the bony cavity around the apex and presents a very disturbing roentgenographic appearance (SLIDE 123). However, no untoward reactions have been reported from such embedded particles: amalgam is apparently well tolerated by the tissues.[1] After the amalgam has been thoroughly condensed, the surface is smoothed with a plastic instrument. All bits of amalgam should be removed from the area. The isolating sponges (SLIDE 124) should now be removed and the wound irrigated with saline solution (SLIDE 125). Light curettage of the wound will stimulate bleeding and ensure an adequate blood clot.

Although the root-end seal technique was originally developed as a specialized means of sealing those blocked canals that could not be adequately instrumented and filled from the coronal approach, use of the technique has broadened. Attainment of a positive apical seal is an all-important goal in endodontic therapy, and the apical placement of amalgam does indeed provide such a seal. It is not suggested that apical surgery be undertaken purely to place this seal. However, in those cases where root resection is indicated, there is an increasing trend for endodontists to forego the usual root resection technique of smoothing off the apical face of the existent filling material. In order to ensure that much-desired positive seal of the apex, they proceed to sever the root at a 45-degree angle and place an amalgam or other type of restoration.

Nicholls[4] has devised an ingenious alteration of the root-end seal technique whereby a restoration may be placed in the facial surface of the apical portion of the root.

CLOSING THE INCISION

In all three of the procedures just described—periapical curettage, root resection, and resection with root-end seal--closure of the surgical site is the same. A sufficient number of sutures is used to return the edges to their natural relationship. Any fine suture material is acceptable. The sutures should be placed a little closer together than usual, since eating and talking place a strain on the incision. At this point the patient should be allowed to inspect the incision with a hand mirror. Otherwise, as soon as he leaves the office, he might go to the nearest mirror to have a good look—and tear the sutures out.

POSTOPERATIVE RECOVERY

The amount of postoperative swelling and discomfort is usually proportional to the amount of trauma inflicted during the operation. Careful regard for asepsis, the use of normal saline as a coolant, careful retraction of the flap and periosteum, judicious use of the curet--all help to minimize postoperative discomfort. In this connection, escharotic drugs, such as silver nitrate or phenol, have no place in apical surgery. Even if these drugs are confined to the amputated root end, they may delay or prevent the deposition of cementum over the denuded root surface.

Apical surgery is best scheduled the first thing in the morning. In this way the patient can recuperate in the recovery room or the sick bay under the care of a dental technician or a hospital corpsman and can be checked from time to time by the dental officer. This procedure has several advantages: (1) The patient remains under supervision until the effects of the premedicant have worn off; (2) the technician or the corpsman can supervise the application of an ice bag to the operative site (on 10 minutes, then off 10 minutes, for the first 3 hours), which helps prevent swelling; and (3) aspirin or codeine can be administered when needed.

When the patient is well enough to be dismissed, he is given final instructions (preferably in writing) and appointments, usually for 2 days later (for postoperative check) and for 5 days later (for removal of the sutures).

Postoperative complications are infrequent. Some edema and ecchymosis may be expected, but these conditions do not usually cause too much discomfort. Antibiotic therapy usually is unnecessary unless the patient has a history of rheumatic heart disease, or a significant rise in temperature is noted during recovery.

Follow-up roentgenograms should be made to determine the extent of repair of the bony defect, the periodontium, and the cementum. Healing time varies considerably with individuals. In some instances evidence of repair may be noted in 2 months (SLIDE 126), and complete repair may be evident in 5 to 7 months (SLIDE 127). In others, evidence of repair may not be apparent for 6 months, and complete repair may take more than a year.

If healing fails to occur after apical surgery, one or a combination of the following conditions probably is the cause:

1. Cystic epithelium has regrown (because of inadequate curettage).
2. There has been excessive loss of bone (because of overzealous curettage).
3. The periosteum was damaged during surgery (by careless elevation and retraction of the flap).
4. The tooth is mobile (because of excessive removal of root structure).
5. The root canal was inadequately filled.
6. A residual infection is present.
7. Contamination has entered the tissues from a pulp-involved adjacent tooth.
8. The general health of the patient is poor.

THE ONE-SITTING TECHNIQUE

In recent years the use of a one-sitting technique for endodontic treatment has been much discussed. Such treatment consists in debriding, enlarging, disinfecting, and filling the canal and then performing a root resection or periapical curettage, or both, all at one sitting. (The canal is disinfected by means of a single application of a very strong drug such as phenol or phenolsulfonic acid or, in one technique, a single application of electromedication.)

The major advantage of the one-sitting technique is the brevity of treatment time. This technique has been used, with varying degrees of success, at many military installations especially during wartime, when circumstances have demanded a maximum economy of time. Under ordinary circumstances, however, this approach to endodontics is not only unnecessary but also disadvantageous. The fact that no drug used in a canal during one appointment can ensure destruction of all microorganisms is reason enough to limit the technique to cases of great expediency. For example, a patient requiring treatment may be available for only a day or two and is being sent to an area where dental treatment is not readily available.

Burnett and Scherp[5] have stated: "This [one-sitting] procedure is perhaps as immediately successful as the longer, older method of treatment but it has been observed that infection is more likely to recur at some future time in the periapical areas of root canals treated by the one operation technique than it is about the roots of teeth filled after prolonged and more palliative types of treatment."

REDUCTION OF LARGE PERIAPICAL RADIOLUCENCY PRIOR TO CURETTAGE

Infrequently, a very large periapical radiolucency is encountered that encroaches on the apices of adjacent teeth (SLIDE 128) or on neighboring structures such as the maxillary sinus or the mandibular canal. Because of the continually expanding behavior of this lesion, it may tentatively be regarded as a cyst. Surgical removal of this cyst might endanger the vitality of the adjacent teeth because of interruption of their blood supply during curettage. A reasonable solution to this problem has been to drain the cyst surgically, causing a reduction in its size, and then, later, to proceed with curettage of the lesion and endodontic treatment of the tooth. The principle is this: If the cystic contents are drained, the osmotic pressure no longer exists. Thus, there can be no further cause for fluid to be drawn into the cavity and no further cause for expansion of the lesion. In fact, the opposite occurs: over a period of months the cystic space reduces in size as new bone is laid down on the periphery.

In order to accomplish this reduction in size of the cyst, various drainage procedures have been recommended. One highly effective method has been suggested by Patterson.[6] In his method constant drainage of the cavity is maintained by insertion of a nonirritating polyethylene tube into a surgically created window in the cavity, the tube being held in position by means of a labial acrylic splint.

In Patterson's technique an impression is taken and a cast is made of the arch involved. A mark is made on the cast at a point corresponding to a point on the mucosa where the drainage exit is desired (SLIDE 129). (If an inflammatory drainage tract already exists, this may be the chosen point.) Into this point on the cast a hole 4 mm. in diameter and approximately 1 inch deep is drilled with a round bur. A length of polyethylene tubing 3 mm. in diameter is inserted in this hole with its exterior end protruding slightly from the canal (SLIDE 130). A labial splint made of a denture acrylic material is processed to, and around, the protruding end of the tubing and is polished well (SLIDE 131).

At this stage, a surgical opening about 4 mm. in diameter is made into the lesion, and the fluid contents are aspirated. The acrylic splint is positioned in the mouth, and the polyethylene tube is inserted in the holes previously made in the splint and into the lesion. The length of the tube is then adjusted so that the tube terminates in the approximate center of the lesion (SLIDE 132). The patient should be instructed in cleansing and wearing of the appliance. He should be strongly impressed with the need for periodic visits for reduction of the tube length as the lesion closes in from the periphery. Osteogenesis will occur gradually. When it has proceeded far enough inward from the periphery that surgical curettage of the cyst membrane, if present, can be accomplished safely, this and endodontic treatment of the tooth (or teeth) involved (SLIDE 133) will make possible the complete filling in of bone (SLIDE 134).

With the appliance devised by Patterson adequate drainage is maintained, excessive food debris is kept from the bone cavity, and periodic examination of the healing lesion is possible.

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CHAPTER 8
BLEACHING PULPLESS TEETH

After the root-canal filling has been sealed into the canal, the crown may be bleached if the tooth is discolored. Surgically treated teeth may also require bleaching.

Before the methods of bleaching pulpless teeth are discussed, it would be well to consider the causes of discoloration. The usual causes are:

1. Failure to remove pulp tissue from the pulp chamber, especially the pulp horns.
2. Failure to remove blood or exudate from the pulp chamber.
3. Failure to remove grossly stained dentin from the walls of the pulp chamber.
4. Failure to replace defective restorations.
5. Failure to limit the root-canal filling to the clinical root of the tooth.
6. Failure to remove all gutta-percha or sealer from the pulp chamber.
7. Failure to use nonstaining drugs for medication.
8. Failure to select the proper restorative material for filling the pulp chamber.
9. Intrinsic staining of the crown due to hemorrhage resulting from traumatic injury to the pulp.
10. Intrinsic staining due to the chemical products of pulp degeneration and necrosis.

It is evident from this list that discoloration can often be prevented by a scrupulous regard for esthetics, from the opening of the pulp chamber to the final placement of the restorative material. If a tooth is not stained before treatment or re-treatment, changes in color or in translucency can be minimized by avoiding the pitfalls listed in items 1 through 8. If, on the other hand, a previously untreated tooth is already discolored because of intrinsic staining, or a previously treated tooth is discolored for any reason, a remarkable improvement can usually be obtained by bleaching the crown.

BLEACHING SOLUTIONS

In the past, many different agents have been used for bleaching pulpless teeth, but only one has been successful enough to endure: hydrogen peroxide. A 30-percent aqueous solution (sold commercially as Superoxol) is recommended for use. Another solution, of 25 percent hydrogen peroxide in 75 percent ether (Pyrozone), has been used but is not recommended because it has a short shelf life and, more importantly, because its ether component is flammable and potentially explosive.

Thirty percent hydrogen peroxide must be handled with extreme caution. It should be refrigerated, not only to prolong its potency but also to prevent gas pressure from building up inside the bottle. When the bottle is being uncapped, the opening should be pointed away from the face, and the hands should be protected with a towel. It is best to pour a small amount of the solution into a dappen dish, then to recap the bottle and replace it in the refrigerator. If by any chance the bleaching solution comes in contact with the skin, the area should be rinsed with water immediately. In fact, it is a good practice to rinse the hands routinely immediately after handling the solution. Another excellent precaution is to wear rubber gloves during the bleaching procedure.

BLEACHING PROCEDURE

Although the rubber dam is very effective in keeping saliva from an isolated tooth, extra precautions are necessary to prevent the hydrogen peroxide solution from leaking at the junction of the dam and the tooth during the bleaching procedure. The hydrogen peroxide solution, because it lacks viscosity and is effervescent, will penetrate a minute opening that saliva will not enter.

The following precautions must be taken to protect the patient from the caustic effects of the hydrogen peroxide solution:

1. Make sure the tooth to be isolated is free of all calculus, stain, and debris.
2. Coat the surrounding gingival tissue and mucous membrane with cocoa butter.
3. Avoid punching a jagged hole in the rubber dam.

4. Ligate the dam in position if necessary.
5. Apply cavity varnish to the margin of the dam to ensure a better seal.
6. Use a rubber dam napkin to protect the patient's face.
7. Use extra towels, as needed, to protect the patient's clothing.
8. Do not leave the patient unattended during the bleaching procedure.

After the rubber dam has been securely positioned to the satisfaction of the dental officer, the pulp chamber should be thoroughly cleaned of all debris, grossly stained dentin, gutta-percha, and sealer. This is best accomplished with a No. 4 or a No. 6 bur. The same bur is then used to remove any caries or defective restorations. Next, the dental officer must make sure that the root-canal filling is sealed into the canal and does not extend incisally beyond the level of the gingival attachment. If roentgenographic or visual examination of the root-canal filling reveals an unsatisfactory seal, the tooth should be re-treated before it is bleached.

The importance of cleaning the pulp chamber thoroughly cannot be overemphasized. This does not mean, however, that sound dentin should be promiscuously sacrificed to eliminate discoloration. On the contrary, dentin should be removed as judiciously as possible. If removing grossly stained dentin and defective restorations would leave the crown too weak to withstand normal stress, it would be better to construct a jacket crown. Under no circumstances should the patient be left with a badly discolored tooth.

Experience has shown that it is advisable to discuss with the patient the possible results of bleaching before the procedure is started. In this regard it is better to promise little and leave the patient pleasantly surprised than to promise a great deal and leave him disappointed. Moreover, before any work is attempted on a tooth in which the crown has very little dentin support or is subject to heavy stress, it is advisable to tell the patient that it may be necessary to use a post crown after bleaching.

The bleaching procedure itself is simple. After the pulp chamber has been cleaned, it is loosely filled with cotton. The cotton is then saturated with 30-percent hydrogen peroxide solution, which is carried into the opening with a curved glass medicine dropper. It is recommended that the solution be "pumped" into the dentin with an orangewood stick or a gutta-percha plunger. This may be done several times during the bleaching procedure and serves to ensure penetration of the solution into the dentinal tubules. A few strands of cotton impregnated with the hydrogen peroxide solution should also be placed on the facial surface of the tooth to aid in bleaching stained enamel.

The bleaching action will be accelerated if some source of light or heat is supplied to activate the bleaching solution and hasten the liberation of oxygen. One of the simplest and most convenient sources of heat for this purpose is a No. 2 photographic floodlamp, mounted in a metal reflector and attached to the light arm of the dental unit by a spring clamp. The lamp should be mounted about 18 inches away from the tooth during bleaching (SLIDE 135). Certain additional precautions must be taken to protect the patient:

1. Shield the patient's eyes with a towel.
2. Cover any metal object (such as the rubber dam clamp) in proximity to the tissue with moist gauze to prevent overheating (SLIDE 136).
3. Turn the lamp off from time to time to reduce the patient's discomfort.
4. Do not leave the patient unattended.

For maximum effectiveness, the hydrogen peroxide solution should be aspirated from the pulp chamber and fresh solution added every 10 minutes. After 30 or 40 minutes, the treatment is ended by aspirating the hydrogen peroxide solution, removing the cotton, and flooding the pulp chamber with 5-percent sodium hypochlorite solution to neutralize any remaining hydrogen peroxide.

When the tooth is severely stained—and only then—the hydrogen peroxide solution may be left in the pulp chamber under a seal of cotton and zinc oxide and eugenol cement to continue the bleaching process for 3 to 5 days. When the cement is inserted into the opening, a piece of rubber dam should be held over the setting cement until it hardens; otherwise, oxygen bubbles coming up through the cement may cause a faulty seal.

Pain sometimes follows the bleaching procedure. Apparently the solution penetrates the dentin and the cementum and causes a reaction in the periodontal tissues. The pain can be relieved almost immediately by removing the seal, flooding the pulp chamber with sodium hypochlorite solution, and resealing with cotton and cement.

A satisfactory result can usually be produced with one or two bleaching treatments. A few cases of discoloration do not seem to respond well at all. In even the most stubborn case (SLIDE 137), however, considerable improvement is usually noted (SLIDE 138).

Often the tooth being bleached will show dramatic results after the first 24 hours—even to the point of being lighter than the adjacent teeth (SLIDE 139 and 140). In the next few days, however, the tooth will usually darken somewhat to a more normal shade or become slightly darker than normal. The patient should be warned about this darkening or he may become discouraged.

ALTERNATE PROCEDURE

Nutting and Poe[1] have devised another method of bleaching pulpless teeth that does not require so long an appointment time as the method just described but reportedly achieves comparable results. It is recommended as an alternate bleaching method. All preparatory steps are the same, but the saturated cotton and the lamp are not used. Instead, the pulp chamber is filled with a bleaching paste. (This is prepared by adding a sufficient quantity of finely powdered sodium perborate to two drops of 30-percent hydrogen peroxide solution on a glass slab to make a paste.) The pulp chamber is then sealed and the patient is dismissed. If in 3 to 4 days the tooth shade is not satisfactory, the procedure is repeated. Usually no more than two treatments are necessary.

When, in either technique, the desired shade has been achieved, the coronal portion of the tooth is again cleaned thoroughly and swabbed out with chloroform or dimethylbenzene (Xylene) and dried with air.

When the final restoration is placed in an anterior tooth, care must be taken to choose a silicate of a shade that will come closest to restoring the natural translucency of the tooth. The shade chosen will depend on the degree of success obtained with the bleaching procedure, the amount of dentin left in the crown, and the type of material used.

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