A question that should be asked of any discipline or technique in dentistry is, “What degree of success should be expected?” Success, in turn, should be measured longitudinally in time—long-range success as opposed to short-term success. The beautiful resin restoration turning an ugly yellow in 1 year is not an unqualified success. By the same token, the denture “worn” in the bureau drawer is far from successful, nor is the well-fitting partial denture successful if it leads to clasp caries in 6 months. Moreover, the endodontically treated tooth with a large periradicular lesion that does not show signs of healing 2 years after treatment cannot be considered a success.

To answer the question, “How successful is endodontic therapy?” a study was undertaken at the University of Washington School of Dentistry to evaluate endodontically treated teeth to determine their rate of success. More important to the study, the rate of failure was also established, and the causes of failure were carefully examined. Analysis of the failures led to modifications in technique and treatment. Finally, the entire discipline of endodontic therapy was re-examined, and definitive improvements were made as a result. The improvements in treatment are reflected in the improvement in success, which increased to 94.45% from a former success rate of 91.10%, an improvement of 3.35 percentage points. In other words, nearly 95% of all endodontically treated teeth were successful.

There was also a hidden agenda to the Washington study—to prove ourselves to a profession that, at that time, was skeptical of root canal therapy. In light of today’s knowledge, the project had some design flaws and misinterpretations and was not that well controlled, even though each phase was subjected to statistical analysis. The null hypothesis was ignored in an effort to prove a point: root canal therapy could be successful if properly done. On the other hand, the figures of the Washington study compared favorably with other reports of success.1–13

A group at Temple University, for example, reported a 95.2% success rate at the end of 1 year with 458 canals filled by the gutta-percha-eucapercha method.5 They found that teeth that started with vital inflamed pulps had more success (98.2%) than teeth with nonvital pulps (93.1%). Contrary to other reports, however, they were far less successful with short-filled canals (71.1%) than with flush-filled or overfilled canals (100%). (Reports of 100% success must be questioned.)

South African researchers enjoyed a success rate similar to that of the Temple University group: 89% success at the end of 1 year.8 Also, as with the Temple group, they were successful 92% of the time in teeth filled to the apex and 91% of the time if the canals were overfilled. Filling short of the apex reduced their success rate to 82%.6

The poorest reported rate of success dealt with 845 Dutch military servicemen.7 After 17 years, 45% of the endodontically treated teeth had failed in nonaviators, whereas only 7% had failed in aviator patients. There is a simple explanation for this wide discrepancy in failure rate. The aviators were usually treated with gutta-percha or silver point fillings, whereas the non-aviators were more frequently treated by “therapy with special chemical compounds.” Furthermore, the aviators’ teeth were more frequently crowned.7

Hession, a highly respected endodontist in Australia, reported the highest rate of success: 98.7% of 151 teeth.8 Nelson reported lower rates in England: 81.9% of 299 teeth. With re-treatment, however, Nelson salvaged 11 of the treatment failures, raising the success rate to 85.6%.9 Kerekes and Tronstad, using the standardized technique recommended in this text, had a success rate similar to the Washington study,10 as did Sjögren and his associates from Sweden.11 Their remarkable study of 356 endodontic patients, re-examined 8 to 10 years later, reported a 96% success rate if the teeth had vital pulps prior to treatment. The success
rate dropped to 86% if the pulps were necrotic and the teeth had periradicular lesions and dropped still lower to 62% if the teeth had been re-treated. They concluded by stating that “teeth with pulp necrosis and periradicular lesions and those with periradicular lesions undergoing re-treatment constitute major therapeutic problems.” They surmised that bacteria in “sites inaccessible” might be the cause of increased failure.  

Worldwide, most controlled studies seem to agree that a lower success rate is associated with overfilled canals, teeth with preexisting periradicular lesions, and teeth not properly restored after root canal therapy. A Swedish group reported a high failure rate if canals were not totally obturated. Sjögren et al., quoted above, also noted a direct correlation between success and the point of termination of the root filling. As Figure 13-1 shows, teeth filled within 0 to 2 mm from the apex enjoyed a 94% success rate, which fell to 76% if the teeth were overfilled and fell further to 68% if they were filled more than 2 mm short.  

In reported re-treatment of initial endodontic failures, success figures have been unacceptably low. Only 50% of the overfilled teeth were acceptable (Figure 13-2). Similarly, a Japanese group reported a much higher failure rate if root fillings were overextended. Surprisingly, a Dutch group enjoyed as high a healing rate whether or not the canals were filled. The Dutch report was only a 2-year study. Unfilled canals had not been followed over a long period of time (such as 10 to 20 years). Most endodontically treated teeth should last as long as other teeth. Vire examined 116 root-filled teeth that were extracted because of failure and found that only 8.6% failed for endodontic reasons compared with 59.4% restorative failures and 32% periodontal failures.

**The Washington Study**

The figures above from the University of Washington are only a few from an exhaustive study encompassing many aspects of endodontic therapy. The modifications in treatment mentioned above were instituted following a pilot study of endodontic success and failure. Even with the limited number of patients in the pilot study, the causes of failure became apparent. Clinical techniques were then changed in an effort to overcome failure. Patients were recalled for follow-up at 6 months, 1 year, 2 years, and 5 years, and the recall radiographs were carefully evaluated for improvement or lack of improvement. Teeth included in the success group were those that demonstrated decided periradicular improvement (Figures 13-3 and 13-4) and those with continuing periradicular health (Figure 13-5). The failures were made up of those teeth that initially demonstrated periradicular damage and that had not improved (Figure 13-6), as well as those that had deteriorated since treatment (see Figure 13-6). As soon as a statistically significant group of cases built up in the file, the material was ready to be analyzed.  

The 2-year recall series was found to be ideal for this study because a statistically significant sample developed within this group. The 5-year recall sample was also analyzed but in understandably smaller, though significant, numbers. The study did not take into consideration any illnesses or systemic differences between patients.

**Two-Year Recall Analysis.** Of a total population of 3,678 patients who could have returned for 2 years, 1,229 actually did return, a recall rate of 33.41%—a statistically significant cohort. Before improvements in technique and case selection were instituted, there was
a 91.10% success rate—104 failures of 1,067 cases. After these improvements were instituted, the success rate rose to 94.45%—9 failures of 162 cases.

Five-Year Recall Analysis. From the beginning of the study, enough 5-year recalls had returned to make a statistically valid analysis of these cases. Of the 302 5-year recalls, 281 teeth were successful—a success rate of 93.05%; 21 teeth were considered failures—a failure rate of 6.95%. These figures compare favorably with the 2-year recall analysis.

Two-Year Recall Analysis by Age of Patient. Reference is frequently made to age as a criterion for

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**Figure 13-3** Special mount for radiographs taken of each case. Note that recall films are exposed at 6 months, 1 year, 2 years, and 5 years following therapy. Figures at the top of the card indicate that complete healing had occurred at 6 months and was maintained for 5 years.

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**Figure 13-4** A. "Before" radiograph, with large periradicular lesion. B. "Treatment" radiograph immediately following periradicular surgery. Also note the fractured root tip of a central incisor (arrow) that tests vital. C. Two-year postoperative radiograph of successfully treated case. Lack of total periradicular repair (moderate repair) is related to extensive surface of filling material over which cementum cannot grow. Also note fracture repair (arrow) of vital central incisor.
endodontic treatment. The youngest patient treated in the study was 2½ years old; the oldest patient was 92 years old. There was a fairly consistent rate of success and failure according to age as shown by statistical analysis ($\chi^2 = 2.72; p > .09$). Age, of course, has a great deal to do with the size and shape of the canal. It was found that older teeth, with more restricted canals, were more successfully obturated than very “young” teeth with large-diameter canals. It also became obvious that the size and shape of the lumen of the canal, as well as the direction of root curvature, play an important part in the successful completion of a root canal filling. Analysis of success, by individual tooth, will demonstrate these points.

Two-Year Recall Analysis by Individual Tooth. No significant difference in failure existed between any of the teeth in either arch ($\chi^2 = 2.45; p > .10$). Thus, no particular tooth can be considered a higher endodontic risk, although there was a wide discrepancy between the mandibular second premolar with a failure rate of 4.54% and the mandibular first premolar with a failure rate of 11.45%. Canal anatomy might account for the greater increase in failure in the first premolar. Pucci and Reig, in their monumental work Conductos Radiculares, pointed out the great anatomic differences
between the two mandibular premolars. While the mandibular second premolar has two canals and two foramina 11.5% of the time, the mandibular first premolar has branching canals, apical bifurcation, and trifurcation 26.5% of the time.

Others have found similar discrepancies between mandibular premolars, most recently Trope et al. at the University of Pennsylvania. These researchers confirmed one of Pucci and Reig’s findings almost exactly: 23.2% of the mandibular first premolars examined had two canals. They also found that 7.8% of second premolars had two canals, a figure differing from Pucci and Reig’s 11.5%. This discrepancy could well be explained on an ethnic basis: the researchers at Pennsylvania reporting on a US African American population and Pucci reporting on a Uruguayan population containing some aborigines.

The Pennsylvania study also confirmed the notion that African American patients more frequently have two canals in lower premolars than white patients. In the first premolars, 32.8% had two canals, compared with 13.7% of whites. In the second premolars, 7.8% of African Americans had two canals, compared with 2.8% of whites. Nearly 40% of the African Americans in the study had at least one premolar with two canals and 16% of the time presented two separate roots in mandibular first premolars.

Failures in the maxillary lateral incisor may also be explained by anatomic differences. Pucci and Reig showed extensive distal curvature of the maxillary lateral incisor root 49.2% of the time. Here again, poor judgment and preparation frequently prevent adequate instrumentation and obturation, with root perforation at the curvature a common occurrence (Figure 13-7). Increased failure rate in the maxillary lateral incisor is also related to continuing root resorption following treatment, a finding peculiar to these teeth.

The overall failure rate, mandible to maxilla, is striking but not statistically significant. Failure in the mandibular arch was encountered 6.65% of the time and 9.03% of the time in the maxilla.

Two-Year Recall Analysis: Nonsurgical versus Surgical Intervention. The degree of success achieved by surgical intervention in treated endodontic cases has long been a contentious point. The Washington study demonstrated that although nonsurgical treatment appears to be slightly more successful than surgical treatment, differences are not statistically significant ($\chi^2 = 3.44; p > .05$).

It is tempting to indict case selection as the basis for increased failure of surgical treatment. One of the indications for surgical intervention has been the grossly involved periradicular lesion, where a lower rate of repair might be expected. Although the 10 to 12 mm periradicular lesion appears to heal as readily, if not quite as rapidly, as the 2 mm lesion (Figure 13-8), this may not always be true. Others have shown that larger lesions, or cases with lesions versus those without, are less successful. Nelson reported that the failure rate was three times higher if a periradicular lesion existed before treatment. In another study, the failure rate was the same. The Dutch study, interestingly enough, found that “teeth with periradicular granulomas tend to heal less successfully than teeth showing cysts.” Similarly, Japanese researchers reported a wide discrepancy in success between treated teeth that had no periradicular rarefaction (88%) and those with a 5.0 mm or greater rarefaction (38.5%). For those teeth without periradicular lesions, Sjögren et al. reported a 96% success rate, but this rate dropped to 86% if periradicular lesions were present.

Two-Year Recall Analysis of Endodontic Failures. The final and most important portion of the Washington study dealt with 104 failure cases as a group. Careful analysis of these particular cases is most revealing.

Arrangement of Failures by Frequency of Occurrence. As Table 13-1 shows, the most striking finding is the 58.66% of the failures caused by incomplete obturation of the canal (Figure 13-9). This is a highly significant figure. This most common cause of failure is almost 50 percentage points ahead of the next greatest cause of failure, root perforation, which

Figure 13-7 Root tip biopsy from the curved apex of a maxillary lateral incisor. Severe inflammatory reaction surrounding the root end is related to perforation as well as failure to debride and obturate the curved portion of the canal. Reproduced with permission from Luebke RG, Glick DH, Ingle JI. Oral Surg 1964;18:97.
Inadequate cleaning and shaping and incomplete obturation. In other words, over two-thirds of all of the endodontic failures in the study were related to inadequate performance of two points of the endodontic triad, “canal instrumentation and canal obturation.” This alone would call for improved technique and attention to detail.

Categorical Arrangement of Causes of Failure. The 13 causes of endodontic failure may be arranged in three general categories of causes leading to failure: (1) apical percolation, (2) operative errors, and (3) errors in case selection (Table 13-2). Actually, a clear-cut delineation of the agents of failure is difficult; apical percolation into the canal accounts for almost all of these failures, as Table 13-2 shows.

Apical Percolation. Three of the causes of failure shown in Table 13-2 lead to apical percolation and subsequent diffusion stasis into the canal (Figures 13-10 and 13-11). Factor in the bacteria lurking in the region and these three causes together account for 63.47% of all of the endodontic failures in the study and demonstrate how vital careful therapy is to success. One must also consider the potential of microleakage under and around coronal restorations: bacteria penetrating from the crown to the periapex alongside poorly obturated canals.

Operative Errors. A category made up of errors in coronal cavity preparation and canal preparation accounts for almost 15% of the failures (root perforation, 9.61%; broken instrument, 0.96%; and canal grossly overfilled, 3.85%; total, 14.42% of all of the failures).
These operative errors are all related to inadequate coronal cavity preparation, improper use of endodontic instruments and filling materials, and lack of standardization of endodontic equipment and material as it arrives from the manufacturer. The latter problem has now been reduced through instrument standardization.

On the other hand, delicate root canal instruments must not be mistreated by the inexperienced, and one of the common complaints of the neophyte is “instrument breakage.” Also, improper use of the instruments causing root perforation and apex perforation accounted for 14 of the 104 failures in the study. Penetrating through the side of a curved root ultimately leads to incomplete instrumentation and incomplete obturation (Figure 13-12).

Opening wide the apical foramen during instrumentation illustrates that this is also a form of perforation and leads to gross overfilling or overextension. Healing is delayed and often incomplete around the grossly overfilled areas that may be caused by foreign-body reaction. Moreover, the perforated apical foramen has destroyed the apical “stop” and does not allow compaction during canal filling. Although the canal may appear overfilled, it is actually incompletely obturated, with resulting percolation and failure (Figure 13-13).

The paucity of failures in this study owing to broken instruments illustrates that this is not as desperate a situation as it is often considered to be. In analyzing the University of Washington caseload, Crump and Natkin found that failure following instrument fragmentation was the same as in other endodontic cases.20

Broken instruments, however, are not favored any more than overfilling and underfilling are favored, but, occasionally, both accidents may occur. Surgical treatment is recommended in operable teeth if the instrument is broken off in the apical one-third of the canal space.21

Table 13-2 Distribution of Failures of Treated Endodontic Cases: Two-Year Recall by Category of Cause of Failure

<table>
<thead>
<tr>
<th>Causes of Failure</th>
<th>Number of Failures</th>
<th>% Failures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical percolation—total</td>
<td>66</td>
<td>63.46</td>
</tr>
<tr>
<td>Incomplete obturation</td>
<td>61</td>
<td>58.66</td>
</tr>
<tr>
<td>Unfilled canal</td>
<td>3</td>
<td>2.88</td>
</tr>
<tr>
<td>Ag point inadvertently removed</td>
<td>2</td>
<td>1.92</td>
</tr>
<tr>
<td>Operative error—total</td>
<td>15</td>
<td>14.42</td>
</tr>
<tr>
<td>Root perforation</td>
<td>10</td>
<td>9.61</td>
</tr>
<tr>
<td>Canal grossly overfilled or overextended</td>
<td>4</td>
<td>3.85</td>
</tr>
<tr>
<td>Broken instrument</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>Errors in case selection—total</td>
<td>23</td>
<td>22.12</td>
</tr>
<tr>
<td>External root resorption</td>
<td>8</td>
<td>7.70</td>
</tr>
<tr>
<td>Coexistent periodontal-periradicular lesion</td>
<td>6</td>
<td>5.78</td>
</tr>
<tr>
<td>Developing apical cyst</td>
<td>3</td>
<td>2.88</td>
</tr>
<tr>
<td>Adjacent pulpless tooth</td>
<td>3</td>
<td>2.88</td>
</tr>
<tr>
<td>Accessory canal unfilled</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>Constant trauma</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>Perforation, nasal floor</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>Total failures</td>
<td>104</td>
<td>100.00</td>
</tr>
</tbody>
</table>

Distribution of 104 endodontic failures 2 years following therapy. Causes of failure may be categorized into three general groupings: apical percolation, which accounts for 63.46% of failures; operative errors, which account for 14.42% of failures; and errors in case selection, which account for 22.12% of the 104 failures.
Figure 13-10  A. Although the root canal filling appears to be overextended, the fuzzy appearance indicates a lack of density necessary for total obturation. Apical perforation destroyed the apical constriction necessary for compaction of the root canal filling. B. Root end biopsy of this failure case shows root canal cement and cellular debris rather than well-condensed gutta-percha filling. Constant percolation into the root canal space provides media for bacterial growth.

Figure 13-11  A. Constantly draining sinus tract opposite the mesiobuccal root of a maxillary first molar (arrow) was thought to be related to an advanced periodontal lesion. Total amputation of the mesiobuccal root is indicated. B. Amputated root reveals an error in diagnosis. The reamer is in an undetected second canal, whereas the arrow points to an obturated primary canal. Secondary canals in mesiobuccal roots of first permanent molars occur about 60% of the time.
Outcome of Endodontic Treatment and Retreatment

and is loose in the canal and cannot be removed or bypassed. Surgery may also be the approach if the canal is grossly overfilled with irretrievable gutta-percha and cement. Broken instruments may often be retrieved by “floating” out the piece with an ultrasonically powered instrument. Broken instruments, loose in the canal, have also been seen to “rust out.” Failures associated with underfilled canals can usually be remedied by retreatment rather than surgery (see Chapter 14).

All of these errors of operation may be prevented by careful cavity preparation and canal obturation. For example, if the operator is unsure of the instrument position in the canal, a radiograph should be taken. Also, more accuracy in fitting the primary or initial filling point will lead to less overfilling. In the final analysis, operative error is the simplest cause of failure to control and requires more patience, care, and understanding to overcome.

Errors in Case Selection. Errors in case selection are not as easily overcome as operative errors and may often be listed as the “bad breaks of the game” rather than errors in judgment. Who could predict, for example, that external root resorption would continue, an apical cyst would develop following treatment, an adjacent tooth would become pulpless, or an associated periodontal

Figure 13-12  Failure owing to root perforation accompanied by incomplete débridement and imperfect obturation. Note the break in the lamina dura opposite the perforation (arrow).

Figure 13-13  A, Although this incisor appears to be grossly overfilled, careful examination (arrow) reveals failure to totally obturate the canal. B, Tissue reaction to overfilling and underfilling in a single biopsy specimen from this failure case. Curved arrows direct attention to a mass of cement forced into periradicular tissue. Heavy black arrow (right) indicates noninflammatory tissue capsule that has developed as foreign-body reaction. Open arrow (bottom) points to a violent inflammatory reaction and an abscess related to bacterial products percolating from the unfilled canal.
lesion would lead to failure? These are all factors that led to some of the failures in the Washington study; factors that constitute 22.12% of the total failures. Some could well have been predicted at the time of therapy, but others were entirely unpredictable.

As Table 13-2 shows, there were also minor causes of failure: constant occlusal trauma from bruxism, perforation of the nasal floor, and unfilled accessory or lateral canals. The low incidence of failure associated with unfilled accessory canals came as a surprise considering the degree of emphasis placed by some on the absolute necessity of totally obliterating these lateral canals.

Returning to some of the greater causes of failure, one should note that all eight cases of continuing root resorption were in maxillary lateral incisors. So one should be wary in evaluating the future of these teeth if they exhibit extensive apical resorption prior to treatment. Most external apical root resorption stops in response to successful root canal treatment!

Endodontic failure associated with periodontal pockets is usually the fault of the dentist for not recognizing the pocket’s existence. One stumbles ahead with the endodontics before careful probing reveals the presence of an associated pocket and that concurrent periodontal/endodontal therapy will be necessary.

In some cases, concurrent treatment may not solve an unsolvable problem. Witness the destructive and irreparable nature of some dens invaginatus or radicular lingual grooves extending to the apex or the resorptive invasion from one endodontic lesion causing the necrosis of an adjoining tooth (Figure 13-15).

There are, of course, multiple causes of failure not revealed in the Washington study: retrofilling failures, root tip and foreign bodies left in surgical sites, root fenestration following surgery (Figure 13-16), cracked or split roots, or carious destruction unrelated to the root canal treatment (Figure 13-17). Ultimately, in all of these situations, bacteria are the final cause of failure. One must also recognize that one of the most frequent causes of failure of the treated pulpless tooth is fracture of the crown. The tooth must be carefully protected by an adequate restoration.

The Washington study has been faulted by some for being only a radiographic study. As Brynolf pointed out in her classic punch biopsy study of root-filled teeth in cadavers, histologic evaluation is a much more accurate method of determining if inflammation remains at the apex than is radiologic evidence. But her research was done on cadavers, proving the impracticality of punch biopsy on live patients. Green and Walton followed up on Brynolf’s approach, comparing histologic and radiographic findings on cadavers, and also found that 26% of the teeth with no radiolucencies showed chronic inflammation histologically. But that is not to say that these teeth were uncomfortable or contributing to the patient’s poor health. How success-

![Figure 13-14](image_url) Developmental defect, invagination, and radicular lingual groove, resulting in an unattached periodontal ligament tract to the apex. The defect at the cingulum probably communicated with the pulp. Endodontic and periodontic therapy were to no avail. Reproduced with permission from Simon JHS, Glick DH, Frank AL. Oral Surg 1971;37:823.)
ful is success? Better yet, what are the criteria of success? Comfort and function? Radiographic? Histologic? Since histologic evaluation is impractical, if not illegal, one would have to go with comfort and function and the radiographic findings.

There are well over 50 studies attempting to delineate how successful our treatment procedures are and what the prognosis is for a particular form of treatment. All in all, the studies that have been done suggest the following generalizations:

1. The more extensive and severe the endodontic pathosis, the poorer the prognosis. In other words, the highest percentage of success is with teeth with vital pulps and the worst prognosis is for those with large, long-standing periradicular lesions.
2. The more dental treatment that is done, the poorer the prognosis. In other words, good nonsurgical endodontic treatment has the best prognosis. The worst prognosis lies with teeth that have been re-treated nonsurgically and then re-treated surgically once or twice more.

MICROBES

To further elaborate on the role bacteria play in pulpal and periradicular disease, one must turn to the classic research by Kakehashi and his associates at the National Institute of Dental Research. They were able to show, in a gnotobiotic study, that microorganisms alone cause pulpal inflammation and necrosis as well as periradicular infection. It follows that inadequate removal of microbes (ie, bacteria, fungi, and viruses) from the canal leads to continued infection and inflammation and that all of the defects listed in the Washington study are ultimately reduced to bacterial invasion and/or colonization.

But what happens when everything is done right (ie, cleaning, shaping, and obturation), yet failure still occurs? Can bacteria still be present after adequate endodontic treatment?

There is strong evidence that bacteria may not be completely eliminated after thorough cleaning, shaping, and disinfection. Moreover, when obturation is postponed, bacteria may be able to recolonize in the canal. Furthermore, try as one might, no preparation

Figure 13-15  A, No radiolucency is apparent on a 4-year recall radiograph following root canal filling of a first premolar. Note the anatomic defect on the canine (arrow). B, Radiograph taken 6 years after the lesion was first noticed. Failed root canal filling in the premolar led to periradicular lesions, inflammatory external resorption of canine, and ultimately necrosis of the canine pulp. Reproduced with permission from Frank AL.

Figure 13-16  Severe bony and soft tissue dehiscence extending to the periapex of a traumatized incisor. Postoperative defects of this type may develop following surgery in an area of osseous fenestration. Reproduced with permission from Luebke RG, Glick DH, Ingle JI. Oral Surg 1964;18:97.
technique can totally eliminate the intracanal irritants, and a “critical amount” can sustain periradicular inflammation (Figure 13-18).³⁹,⁴⁰

In addition, as stated previously, the obturated canal may be recontaminated from coronal leakage.⁴¹ Indeed, gutta-percha root canal fillings do not resist salivary contamination.⁴²,⁴³ As Ray and Trope have been able to show, “long term prognosis of treatment seems to correlate directly with the quality of the coronal seal.”⁴⁴

Figure 13-17 Postoperative failure owing to extension of dental caries rather than endodontic therapy per se. Loss of these abutment teeth led to full denture in this case.

Figure 13-18 A, The radiograph shows a mandibular cuspid that appears well obturated with a well-fitting coronal restoration. B, A sinus tract is traced with a gutta-percha point to the apical lesion that has not responded to treatment. C, Surgical resection of the root end. D, High-power photomicrographs show bacteria containing plaque on the external root surface.
There are also times when an irritant, such as infected dentin chips, is packed at the apex or pushed through the apex, there to serve as a continuing irritant that overwhelms the body’s defense system. As stated before, the periapical tissue could become colonized by periodontal contamination, the virulence of the bacteria, or extrusion by overaggressive instrument action.

In most cases, the host’s immune system can overcome these antigens. On the other hand, some bacteria possess mechanisms to resist phagocytosis such as encapsulation or the production of proteases aimed against the immune system. Bacteria may also bury themselves in a thick matrix that acts as a sort of apical plaque (see Figure 13-18, D).

Many organisms can survive in periradicular lesions: Actinomyces, Peptostreptococcus, Propionibacterium, Prevotella and Porphyromonas, Staphylococcus, and Pseudomonas aeruginosa. Barnett, in fact, has stated that Pseudomonas refractory periradicular infection could be “cured” only by heavy doses of metronidazole (Flagyl) following the failure of re-treatment and apicoectomy.

FOREIGN BODIES

In spite of what has been stated above, “foreign-body giant cell reaction” can occur without the presence of bacteria, but no fulminating infection will develop without the bacteria.

A number of foreign bodies have been reported: lentil beans, other vegetables, popcorn kernels (Figure 13-19), paper points, cellulose, and a variety of unidentified materials. Human body defense cells are unable to digest cellulose or cotton pellets.

In addition, various lipids, cholesterols, and crystals have also been implicated as periradicular irritants. By their very presence, these intrinsic factors are capable of sustaining an apical lesion despite correct endodontic treatment.

Root canal sealers can also act as a foreign body and thus sustain a lesion, although, over time, extruded sealers (and gutta-percha) may be phagocytized by macrophages.

The cytotoxicity of freshly mixed and unset sealers is well documented. In the long term, however, obturation materials are far less irritating than microbes. It can be concluded that an overfill may cause a delay in healing but will not prevent it.

EPITHELIUM

The role of epithelium must also be taken into account when reviewing failure to heal periapically. If the resting cells of Malassez remain in the region, they may respond to the irritants and inflammation and proliferate into a cyst-like attempt to wall off the irritants.

It has been suggested that these latent epithelial cell rests could be activated by the epidermal growth factor present in saliva that contaminates canals left open for drainage. In the absence of treatment, or in the presence of persistent bacteria, epithelium continues to proliferate to become a bay (pocket) cyst and eventually a true cyst. The distinction between a bay (pocket) cyst and a true cyst is important from a clinical standpoint. Since root canal therapy can directly affect the lumen of the bay (pocket) cyst, the environmental change may bring resolution of the lesion.

There is a controversy over whether cysts heal after nonsurgical endodontic treatment. The true cyst is independent of the root canal system, so conventional therapy may have no effect on it. The prevalence of true cysts is less than 10%. Most practitioners now realize that true cysts, as well as some bay (pocket) cysts, probably do not heal with nonsurgical therapy. In spite of good cleaning, shaping, and filling, these lesions have to be surgically removed to effect healing.

The dangers delineated above emphasize the point initially stressed: if the case is carefully selected before treatment, if the canal is correctly instrumented and obturated, and if the crown is properly restored, the ultimate outcome should be successful.

Figure 13-19  Biopsy of what was thought to be a root tip reveals columnar-like cells (arrow) of the outer husk of a popcorn kernel that was trapped in a fresh surgical area and served as a severe irritant.
Measures to be Employed to Improve Success.

One may draw certain conclusions from this study and finally list a number of procedures to improve the rate of success of treated endodontic cases. These are the Ten Commandments of Endodontics:

1. **Use great care in case selection.** Be wary of the case that will be an obvious failure, but, at the same time, be daring within the limits of capability.

2. **Use greater care in treatment.** Do not hurry; maintain an organized approach. Be certain of instrument position and procedure before progressing.

3. **Establish adequate cavity preparation** of both the access cavity, which can be improved by modifications of the coronal preparation, and the radicular preparation, which can be improved by more thorough canal débridement—cleaning and shaping.

4. **Determine the exact length of tooth to the foramen** and be certain to operate only to the apical stop, about 0.5 to 1.0 mm from the external orifice of the foramen.

5. **Always use curved, sharp instruments in curved canals,** and especially remember to clean and reshape the curved instrument each time it is used. This applies to stainless steel instruments.

6. **Use great care in fitting the primary filling point.** One must be certain to obliterate the apical portion of the canal. Be more exacting in the total obturation of the entire root canal. **Always use a root canal sealer cement.**

7. **Use periradicular surgery only in those cases for which surgery is definitely indicated.**

8. **Always check the apical density of the completed root canal filling** of the patient undergoing periradicular surgical treatment, and this should be done by using a sharp right-angled explorer. If found wanting, the apical foramen is prepared and retrofilled.

9. **Properly restore each treated pulpless tooth to prevent coronal fracture and microleakage.**

10. **Practice endodontic techniques** until the procedures are as routine as the placement of an amalgam restoration or the extraction of a central incisor. Practice on extracted teeth mounted in acrylic blocks is especially recommended.

Careful attention to details in following the Ten Commandments of Endodontics will ensure a degree of success approaching 100%.
PROGNOSIS

All of this having been said, it should be possible to predict the outcome of endodontic treatment. With the benefit of education and experience and some plain luck, one should be able to choose the proper cases for endodontic treatment and reject those that will obviously fail. But it is not quite that simple.

The practicing dentist should not be cited for faulty judgment when even the experts tend to disagree on prognosis. A North Carolina group, for example, found considerable disparity in treatment choice between general practitioners and endodontists who were shown seven controversial cases. In another instance, Holland and his collaborators reviewed 17 prognosis studies dealing with success or failure of teeth with and without periradicular lesions. In the “with lesion” column, successful outcome ranged from a low of 31.8% to a high of 85%. On the other hand, there was general agreement among these 17 experts that a greater success rate may be enjoyed if there is no initial periradicular lesion present. This is not to say that teeth with periradicular lesions should be shunned. On the contrary, respectable success rates have been reported by many who have treated teeth with radiolucent lesions.

All in all, one must ultimately develop confidence in one’s own abilities. Being able to practice using a variety of techniques and not being “married” to a single approach in every case greatly enhances one’s capabilities. This is the basis of good prognosis, the result of skill, knowledge, and self-confidence. On the other hand, if a treated tooth does fail, it may be resurrected by skillful re-treatment!

SUCCESSFUL RE-TREATMENT

One must recognize that the patients in the Washington study were all controlled university clinic or specialty practice patients, as were the patients in many of the other success and failure reports, an important consideration in evaluating these outcomes.

In marked contrast, worldwide reports of endodontic success and failure in a cohort of general practice patients is an entirely different matter. To make matters worse, the periradicular lesions in these reports were associated with previously treated endodontic general practice cases and were much higher (24.5 to 46%) than the periradicular lesions in nontreated teeth (1.4 to 10%) (Table 13-3).

There is also a very disappointing finding in a general survey by Buckley and Spångberg at the University of Connecticut; they noted that periradicular lesions are found 5 to 10 times more often in endodontically treated teeth than for teeth without root fillings.

Mactou pointed out that “the quality of treatment plays a decisive role in the successful outcome of endodontic therapy” (Figure 13-21) (personal communication, April 2000).

Fortunately, treated root canals with inadequate results may be re-treated to improve the quality of the treatment and heal any apical lesion. In a 2-year follow-up study, Bergenholtz et al. also reported a 94% success rate for re-treatments done for one reason only, to improve root canal filling quality prior to prosthodontic therapy.

Friedman et al. claimed a 100% success rate in re-treatment of endodontic cases presenting without periradicular lesions, which suggests that if infection is not present, and re-treatment is performed by skilled clinicians, the success rate can be expected to be very high (Table 13-4). On the other hand, if periradicular lesions are present before re-treatment, the success rate can be expected to fall to around 70% (Table 13-5).

As many as 22 factors have been listed to affect the prognosis of endodontic re-treatment, the preoperative...
state of the tooth (as above) obviously being a key factor in outcome.\textsuperscript{106} Stabholz et al.\textsuperscript{107} and Friedman\textsuperscript{108} have noted that causes of endodontic failure fall into three categories: preoperative, intraoperative, and postoperative causes. Of these three, \textit{intraoperative}, that is, iatrogenic, causes are the most prevalent but the most easily controlled.\textsuperscript{109} For example, Nair has pointed out that refractory periradicular infections are found in acute symptomatic teeth.\textsuperscript{110} At the same time, others have noted that these bacteria may have been “planted” there as contaminants during previous endodontic treatment.\textsuperscript{110–116} As previously stated, infected dentin chips, extruded from overinstrumentation, may also be the “root” cause of refractory infections.\textsuperscript{103} Bergenholtz has stated that “…root filling material \textit{per se} was not the immediate cause of the unsuccessful cases, but that

Table 13-3  Cross-sectional Studies on the Presence of Periapical Periodontitis (PAP) in Nontreated and Endodontically Treated Teeth and Quality of Treatment

<table>
<thead>
<tr>
<th>Lead Author</th>
<th>Country</th>
<th>Teeth with PAP (%)</th>
<th>Endodontically Treated Teeth (%)</th>
<th>Endodontically Treated Teeth with PAP (%)</th>
<th>Technically Inadequate Treatment (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bergen, 1973</td>
<td>Sweden</td>
<td>6</td>
<td>12.7</td>
<td>31</td>
<td>—</td>
</tr>
<tr>
<td>Hansen, 1976</td>
<td>Norway</td>
<td>1.5</td>
<td>3.4</td>
<td>46</td>
<td>—</td>
</tr>
<tr>
<td>Petersson, 1986</td>
<td>Sweden</td>
<td>6.9</td>
<td>12.4</td>
<td>31</td>
<td>&gt; 60</td>
</tr>
<tr>
<td>Allard, 1986</td>
<td>Sweden</td>
<td>10</td>
<td>18</td>
<td>27</td>
<td>69</td>
</tr>
<tr>
<td>Eckerbom, 1987</td>
<td>Sweden</td>
<td>5.2</td>
<td>13</td>
<td>26.4</td>
<td>55</td>
</tr>
<tr>
<td>Eriksen, 1988</td>
<td>Norway</td>
<td>1.4</td>
<td>3.4</td>
<td>34</td>
<td>59</td>
</tr>
<tr>
<td>Odesjo, 1990</td>
<td>Sweden</td>
<td>2.9</td>
<td>8.6</td>
<td>24.5</td>
<td>70</td>
</tr>
<tr>
<td>Imfeld, 1991</td>
<td>Switzerland</td>
<td>8</td>
<td>20.3</td>
<td>31</td>
<td>64</td>
</tr>
<tr>
<td>De Cleen, 1993</td>
<td>Netherlands</td>
<td>6</td>
<td>2.3</td>
<td>38</td>
<td>50.6</td>
</tr>
<tr>
<td>Buckley, 1995</td>
<td>United States</td>
<td>4.1</td>
<td>5.5</td>
<td>31.3</td>
<td>58</td>
</tr>
<tr>
<td>Soikkonen, 1995</td>
<td>Finland</td>
<td>4</td>
<td>21</td>
<td>16</td>
<td>75</td>
</tr>
</tbody>
</table>

Figure 13-22  A, Failure case with apical and lateral periradicular lesions. B, Re-treatment eliminates bacterial infection through extensive recleaning, reshaping, and canal medication. Obturation by vertical compaction fills the lateral canal as well. C, Complete healing 1 year later. (Courtesy of Dr. Pierre Mactou.)
unsuccessful cases were caused...by a reinfection in the apical areas favored by overinstrumentation\(^{105}\) (Figure 13-23).

Mactou has laid out the important factors that must be adhered to if re-treatment is to be successful: first of all, complete re-cleaning and reshaping of the canals. This should be carried out in a step-down fashion: early coronal enlargement and canal body shaping prior to apical preparation (personal communication, April 2000).

Table 13-4  Nonsurgical Endodontic Re-treatment Outcome in Pulpless Teeth without Periapical Periodontitis

<table>
<thead>
<tr>
<th>Authors</th>
<th>Cases</th>
<th>Success (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strindberg, 1956</td>
<td>64</td>
<td>95</td>
</tr>
<tr>
<td>Grahnon and Hansson, 1961</td>
<td>323 (roots)</td>
<td>94</td>
</tr>
<tr>
<td>Engstrom et al., 1964</td>
<td>68</td>
<td>93</td>
</tr>
<tr>
<td>Bergenhotz et al., 1979</td>
<td>322 (roots)</td>
<td>94</td>
</tr>
<tr>
<td>Molven and Halse, 1988</td>
<td>76 (roots)</td>
<td>89</td>
</tr>
<tr>
<td>Allen et al., 1989</td>
<td>48</td>
<td>96</td>
</tr>
<tr>
<td>Sjögren et al., 1990</td>
<td>173 (roots)</td>
<td>98</td>
</tr>
<tr>
<td>Friedman et al., 1995</td>
<td>42</td>
<td>100</td>
</tr>
</tbody>
</table>

From Friedman.\(^{28}\)

Table 13-5  Nonsurgical Endodontic Re-treatment Outcome in Teeth with Periapical Periodontitis

<table>
<thead>
<tr>
<th>Authors</th>
<th>Cases</th>
<th>Success (%)</th>
<th>Uncertain (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strindberg, 1956</td>
<td>123</td>
<td>84</td>
<td>—</td>
</tr>
<tr>
<td>Grahnen and Hansson, 1961</td>
<td>118 (roots)</td>
<td>74</td>
<td>—</td>
</tr>
<tr>
<td>Engstrom et al., 1964</td>
<td>85</td>
<td>74</td>
<td>—</td>
</tr>
<tr>
<td>Bergenhotz et al., 1979</td>
<td>234 (roots)</td>
<td>48</td>
<td>30</td>
</tr>
<tr>
<td>Molven and Halse, 1988</td>
<td>98 (roots)</td>
<td>71</td>
<td>—</td>
</tr>
<tr>
<td>Sjögren et al., 1990</td>
<td>94 (roots)</td>
<td>62</td>
<td>—</td>
</tr>
<tr>
<td>Friedman et al., 1995</td>
<td>86</td>
<td>56</td>
<td>34</td>
</tr>
<tr>
<td>Sundqvist et al., 1998</td>
<td>50</td>
<td>74</td>
<td>—</td>
</tr>
</tbody>
</table>

From Friedman.\(^{28}\)

Figure 13-23  A. Failed endodontic treatment caused by overinstrumentation and failure to totally obturate the canal. Note the gross overextension of sealer and that the final 6 mm of the canal are sealer only. B. Re-treatment by complete recleaning, reshaping, and canal medication before final obturation. C. Twelve-year recall shows total healing. Note recurrent decay at the distal margin of the crown (arrow) that can lead to microleakage and potential failure. (Courtesy of Dr. Pierre Mactou.)
The canal should be cleaned segment by segment through a coronal reservoir of 2.5% of sodium hypochlorite. Working length at the radiographic terminus is established late in treatment, when the remainder of the canal has already been cleaned and shaped. Maintenance of apical potency and constant recapitulation with fine files avoids canal blockage with dentin debris. It also allows the sodium hypochlorite to reach this area. Great care must be exercised when removing the previous filling material, particularly when solvents such as chloroform are used.

It is very difficult to remove all of the sealer and gutta-percha from the canal walls. Wilcox et al. contended that the last remnants of sealer can be removed only by vigorous reinstrumentation and reshaping of the canal \(^{117,118}\) but cautioned that overenlargement may result. \(^{119}\) It would appear that sealer removal is most important since bacteria can easily “hide” under previous sealer.

**CANAL MEDICATION**

Re-treatment cases are notorious for continued failure! In all probability this is caused by the failure to remove or kill the refractory bacteria responsible for the lesions in the first place. One is warned that these cases are challenging, and this is probably not the occasion for **one-appointment therapy**. Continuing failure is undoubtedly owing to remaining bacteria, and their elimination calls for extra effort. New data regarding failed root canals indicate that these microflora\(^ {120-122}\) differ from those of untreated necrotic teeth.

Enterococci, for example, are quite prevalent in previously root-filled teeth and are quite difficult to eliminate. *Enterococcus faecalis* is the most frequent strain and, as a monoinfection, is found in 33% of the cases. \(^ {123}\) Enteroccoci are quite resistant to calcium hydroxide, so Safau et al. have recommended the addition of iodine potassium iodide in these cases. \(^ {124}\) Heling and Chandler have recommended a mixture of 3% hydrogen peroxide and 1.8% chlorhexidine as an alternative against *E. faecalis*. \(^ {125}\) *Staphylococcus aureus* and *Pseudomonos* are also notorious refractory contaminants and may require a prescription of metronidazole and amoxicillin to rid the periapex of these bacteria. \(^ {126-128}\)

**OBTURATION AND RESTORATION**

It goes without saying that total obturation is the **sine qua non** of successful re-treatment.

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**Figure 13-24**  A. Pretreatment radiograph reveals failed endodontic treatment and a periradicular lesion. B, Re-treatment by total re-cleaning, reshaping, and canal medication. Final obturation by vertical compaction fills the lateral canal as well. C, Six-year follow-up. (Courtesy of Dr. Pierre Mactou.)
For this, the reader is referred to chapter 11. Furthermore, since many failures are believed to be related to microleakage from around coronal restorations, it is imperative that the re-treated tooth be properly restored, at once, not weeks later (Figure 13-24). Molven and Halse have said it best: “Success depends on the elimination of root canal infection present when treatment starts, and the prevention of both contamination during treatment and re-infection later. So success rates reflect the standard of the cleaning, shaping and filling of root canals”

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