JOURNAL OF ENDODONTICS
Printed in U.S.A.
VOL. 29, NO. 11, NOVEMBER 2003

Cognitive Dissonance in Endodontics†

Samuel Seltzer, D.D.S.,* and I. B. Bender, D.D.S.**

Cognitive dissonance is the existence of views, attitudes, or beliefs which are inconsistent or incompatible with one another but, nonetheless, are held simultaneously by the same person. In a penetrating article, Edwin G. Boring (1), Edgar Pierce Professor of Psychology Emeritus at Harvard University, has documented the existence of cognitive dissonance among scientists who, after all, he said, turn out to be human. One of the graphic examples offered is the persistence of smoking, despite evidence that it is hazardous to life itself. To rationalize, the smoker must change his cognition, thereby minimizing the scare about lung cancer, suppress or ignore the dissonance, or change his behavior by giving up smoking.

Is cognitive dissonance present in endodontics? It is our belief that, as scientific evidence accumulates, a greater and greater dissonance is emerging in both the theory and the practice of endodontics. At least, this is true for us, and we would like to share our dissonance with others in the scientific community who perhaps have similar but unexpressed views.

It has long been held that if the three basic principles—the so-called "endodontic triad"—are followed faithfully, the end result of endodontic treatment must be successful (2). These three "principles" are (a) thorough debridement of the root canal, (b) sterilization of the root canal, and (c) complete obturation of the root canal. Put down as a simple formula, it would be a + b + c = endodontic success.

In endodontic therapy, the a + b + c formula for success has been taught in most dental schools as the only sure way to achieve a lasting and permanent result. Deviations from this formula are almost certain to result in failure. So far, no dissonance. However, some dissonance begins to creep in when the dental student or general practitioner (or even the experienced endodontist, for that matter) follows the a + b + c formula and failure ensues (Fig. 1). Usually, the dissonance is resolved quickly by the rationalization that, somehow, there has been a break in adherence to the a + b + c formula. For example, maybe a small amount of necrotic tissue was left in the root canal and somehow escaped being removed, or perhaps the negative culture obtained was a false negative and there were some microorganisms lurking in hidden recesses just waiting for the opportunity to emerge and "vent their spleens" on the periapical tissues after treatment was completed. Perhaps the canal was not completely obturated and there were minute voids between the root filling and the dentinal wall, or perhaps the canal was overfilled and the filling material was irritating. The possibilities are numerous. Conversely, dissonance also arises when, through intention or neglect, the formula is not followed and success results anyway (Fig. 2). How can this be explained rationally?

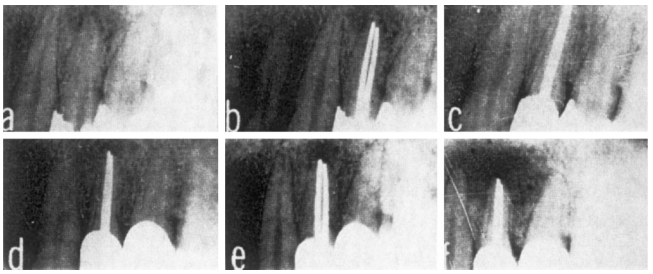
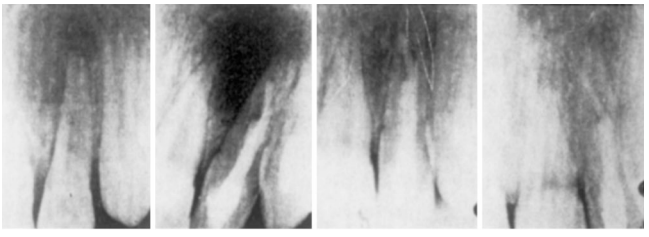


FIG 1. Roentgenograms showing failure of endodontic therapy despite thorough débridement, negative cultures, and complete obturation of the root canals. *a*, Pretreatment roentgenogram showing radiolucent area over upper right first premolar. *b*, At completion of endodontic therapy. *c*, At checkup 6 months later; note reduction of radiolucent area. *d*, At checkup 1 year later; area is reduced in size but persists. *e*, At checkup 3 years later; area is beginning to enlarge. *f*, At checkup 6 years later; area has increased tremendously, and patient has pain and swelling.

The cognitive dissonance rears its ugly head when we examine each part of the triad, as we shall now do.



A. *B.* *C.* *D.*

FIG 2. Successful endodontic therapy despite "poor" treatment. *A*, Preoperative roentgenogram showing large area of rarefaction around upper right lateral incisor. *B*, At completion of endodontic treatment; cultures were persistently positive and the root canal was obturated poorly. *C*, Roentgenogram made 1 year later shows that area of rarefaction has virtually disappeared. *D*, At checkup 6 years later; the periapical area is normal.

This investigation was supported in part by United States Public Health Service Research Grant DE 01930 from the National Institute of Dental Research, Bethesda, Md.

* Associate Professor of Oral Pathology.
** Associate Professor of Oral Medicine.
From the University of Pennsylvania School of Dental Medicine, Philadelphia, Pa.

† Reprinted from ORAL SURGERY, ORAL MEDICINE, ORAL PATHOLOGY, ORAL RADIOLOGY & ENDODONTICS, Vol 20:506-516 © 1965 with permission from Elsevier.

THOROUGH DEBRIDEMENT OF THE ROOT CANAL

Débridement of the root canal appears to be an essential part of endodontic therapy. Unless débridement is performed, there can be no cure. This seems to be reasonable, inasmuch as it has never been shown, as far as we are aware, that a periapical lesion resulting from an inflamed or necrotic pulp will regress or resorb and heal without treatment of the root canal or extraction of the tooth. In other words, a cure without treatment *cannot* and *does not* occur. But, how thorough must the débridement be, especially in teeth without periapical involvement? Is it possible that overzealous instrumentation and irrigation sometimes can cause more harm than good? There is some evidence that it can.

Strindberg (3), and Grahnén and Hannson (4) found that there were fewer failures in endodontically treated teeth that could *not* be reamed through the apex than in those which were reamed completely through the apex. They noticed this particularly in teeth with vital pulps.

In our current investigations of periapical tissue reactions to endodontic procedures, we have noted some bizarre reactions to pulp extirpation and canal instrumentation. The inflammatory reaction which invariably follows such procedures occasionally causes proliferation of cell rests of Mallasez in the vicinity of the root apex (Fig. 3). In time, cysts may form. Figs. 4 and 5 show stages of this epithelial proliferation. They illustrate the formation of periapical cysts following pulp extirpation, root canal reaming and filing of teeth in which the pulps were intact and uninflamed prior to treatment. "But how often does this happen?" the reader may ask. In our experiments we have observed cyst formation following endodontic procedures in a sufficient number of cases to believe that it can and does occur with some degree of frequency. However, we have not yet accumulated enough cases to establish significance. Despite these findings, the dissonance is not too great, since débridement is a prerequisite for wound repair anywhere in the human body. Complications can and do arise in the treatment of other bodily diseases. Why should endodontic treatment be different? Our main point here is that, in spite of faithful adherence to a basic principle, a failure can still result.

STERILITY OF THE ROOT CANAL

Eliminating infection from human tissue appears to be a reasonably sound goal. Why should this not also be the goal of good endodontic therapy? On the basis of reason and logic, therefore, obtaining one or two negative root canal cultures prior to completion of endodontic therapy has become one of the important "principles" of endodontic therapy. Who can argue with such a noble goal? In most previously quoted studies on comparative success and failure rates following endodontic therapy, the teeth with the negative root canal cultures always fared better than those with the positive cultures (5–11). There is no dissonance there, and that is as it should be. However, some dissonance begins to arise when it is noted that many of the same investigators never obtained less than an 80 to 85 per cent success rate, even with positive root canal cultures (Table 1). Even more outlandish is the paper of Lörinczy-Landgraf and Palóez (13), who reported the results of a one-visit endodontic procedure on 400 teeth in which the root canals were instrumented, irrigated with tap water, and filled. Cultures were not even taken! They obtained a 72 per cent success rate after 1 year, which increased to 79 per cent after 2 years (14). How can that be?

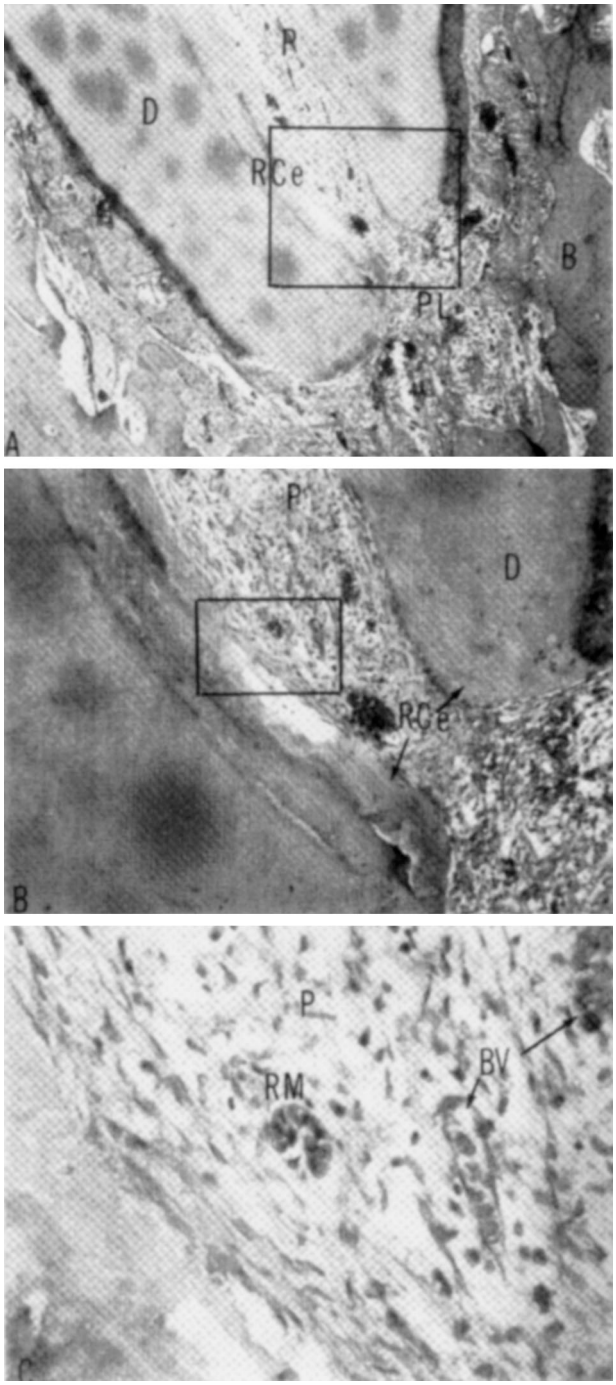


FIG 3. *A*, Root apex and surrounding tissues of an upper central incisor. *P*, Pulp; *D*, dentin; *RCe*, reparative cementum; *PL*, periodontal ligament; *B*, bone. (Magnification, ×54; reduced ¼.)
B, Higher magnification of region outlined by rectangle in *A*. *D*, Dentin; *P*, pulp; *RCe*, reparative cementum. (Magnification, ×135; reduced ¼.)
C, Higher magnification of region outlined by rectangle in *B*. *P*, Pulp; *BV*, blood vessels; *RM*, cell rest of Mallasez within the root canal, a rare occurrence. (Magnification, ×540; reduced ¼.)

Is it reasonable that such an important basic principle can be violated with impunity at least 80 per cent of the time?

An even greater dissonance occurs when the fallibilities of the culture technique are examined. The possibilities of obtaining false

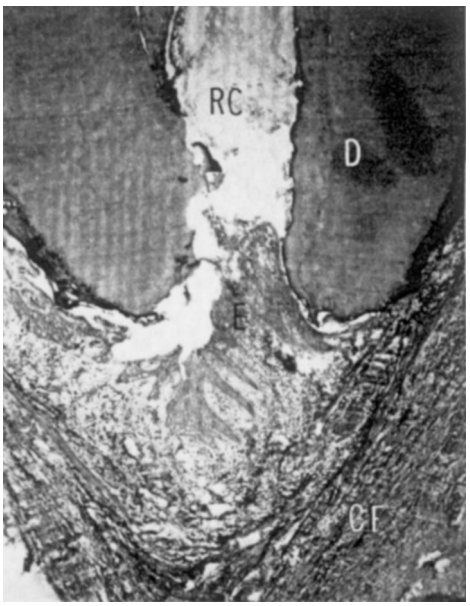


FIG 4. A small cyst has formed at the apex of an upper central incisor following endodontic therapy. *RC*, Root canal; *D*, dentin; *E*, epithelial proliferation; *CF*, collagen fibers. (Magnification, ×54; reduced ¼.)

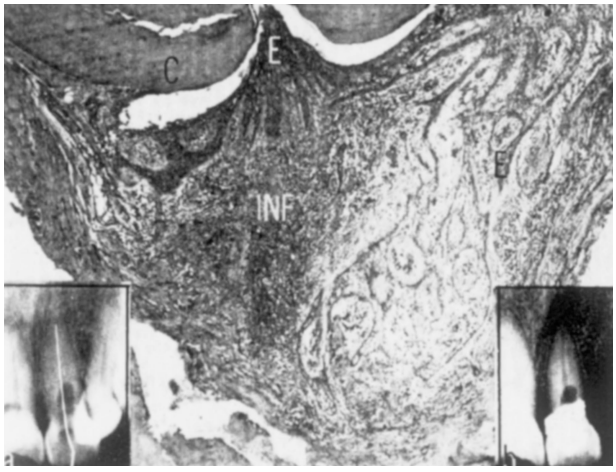


FIG 5. A cyst is forming following endodontic procedures on an upper right central incisor. Inset *a* shows diagnostic wire in root canal. Inset *b* shows area of rarefaction 6 months after instrumentation. *C*, cementum; *E*, epithelial proliferation; *INF*, inflammatory infiltrate. (Magnification, ×54; reduced ¼.)

negative cultures are so numerous that the credibility of a negative root canal culture is constantly in doubt. Being cognizant of this failing, some endodontists are willing to admit that the culture technique is a poor "tool" for determining the sterility of the root canal, but they argue that "a poor tool is better than no tool at all."

Can microorganisms actually be eliminated from an infected root canal? We seriously doubt that this achievement of sterility is possible. Histologic examinations of serial sections of the roots of many teeth have convinced us of the prevalence of multiple accessory and lateral canals (Fig. 6). We cannot conceive that these branches can be either débrided properly or sterilized, let alone cultured. All we can hope for is a reduction in the number of microorganisms in the main canal. Any success obtained from treatment of teeth with positive root-canal cultures can probably be ascribed to a reduction in the number of microorganisms, removal of most inflamed or necrotic tissue, and a favorable systemic background. This explanation appears to be reasonable, but how many microorganisms are permitted to remain alive in the root canal? And how is this number to be determined? Also, how does the practitioner determine the presence of a favorable systemic background? These are questions which we believe must yet be resolved, for they create cognitive dissonance within us.

COMPLETE OBTURATION OF THE ROOT CANAL

Complete obturation should yield a good result, assuming that the root canal is well débrided and sterilized. This conclusion is based on such dogmatic statements as the following: (1) Unless the canal is well filled, spaces between the root filling and the wall of the root canal may harbor microorganisms and/or tissue debris which will continue to act as a periapical irritant. (2) If voids are permitted to remain in the apical third of the root canal, tissue fluid or inflammatory exudate will stagnate and the breakdown products may then serve as an excellent culture medium for microorganisms. Both stagnated fluid and microorganisms are periapical tissue irritants. In either case, failure is sure to occur.

Ingle (2) found that 58.65 per cent of 104 endodontic failures were related to poorly filled root canals. We wondered what the percentage of poorly filled canals was in teeth in which endodontic treatment was successful. Our dissonance was augmented when we examined tissue sections of root-filled teeth and routinely found numerous voids between the filling material and the dentine, as well as accessory foramina containing uninflamed, inflamed, or necrotic tissue. These findings were in endodontically treated teeth that were both successful and unsuccessful. Furthermore, similar observations of unfilled accessory canals were made even in teeth with the most carefully filled main canals in which special efforts had been made to force the filling material into the foramina (Fig. 7).

As far as we can discover, the stagnation theory stems from observations made in 1931 by Rickert and Dixon (15) that inflammation persisted around implanted steel and platinum hypodermic needles in the skin of a rabbit. Macroscopic observation revealed irritation around the ends of the metal tubes but not in the median portions. They claimed: "This gave rather convincing evidence that the circulatory elements diffusing out of the openings of these tubes were not well tolerated by the vital tissues." On the basis of gross examination, they also observed: "Sterile implants of unfilled teeth [in rabbits] were not well tolerated in either skin or muscle." On the other hand, implants from extracted, sterilized, and root-filled teeth failed to show the gross irritation. The number of experiments performed was not documented; nor were there any histologic sections. This "hollow-tube effect" has been cited as definite evidence that complete obturation of the root canal is essential for periapical repair. As long as we accept this evidence, there is no dissonance. However, doubts become increasingly disturbing when in routine full-mouth roentgenograms one sees teeth, not needles, with partially filled root canals and complete absence of periapical pathosis (Fig. 8). Why does the "hollow-tube effect" not cause breakdown? There have also been some reports in the literature by a "naïve" practitioner who treated root canals until periapical regions of rarefaction disappeared before he filled the canals (16, 17). Whoever heard of such nonsense? How can the areas regress and disappear if the canals are not filled? Our cognitive dissonance assumed tremendous proportions when, in the course of our investigations, we discovered that the practitioner was not so naïve. We found, in roentgenograms and in histologic

TABLE 1. Success rates of endodontic treatment by various investigators

Investigator	No. of cases	Per cent with positive cultures	Per cent with negative cultures	Observation period (months)
Oliet (10)	98	55.2	83.8	6–12+
Frostell (11)	316	49.0	59.0	12
Frostell (11)	252	69.3	84.7	48–60
Rhein, Krasnow, and Gies (5)	492	85.5	93.9	24+
Buchbinder (7)	151	82.0*	92.0	20
Zeldow and Ingle (9)	56	83.3	92.9	24
Seltzer, Bender, and Turkenkopf (18)	2,335	81.8	84.4	6
Bender, Seltzer, and Turkenkopf (19)	706	82.2	81.9	24
Grahnén (12)	76	100.0†	68.4	48–60

* Not cultured—culture assumed positive.
† Only two teeth had prefilling positive cultures.

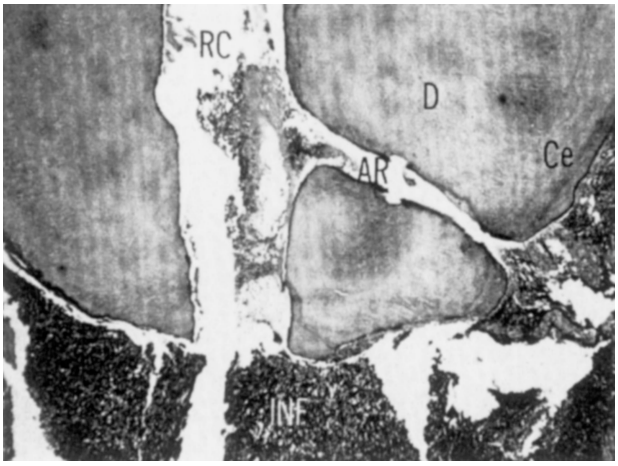


FIG 6. A large apical ramification (*AR*) communicates with the main root canal (*RC*). *D*, Dentin; *INF*, inflammatory infiltrate; *Ce*, cementum. (Magnification, ×54; reduced ¼.)

sections, that repair of periapical inflammatory lesions sometimes occurred following endodontic treatment in teeth without any root filling whatsoever (Fig. 9). We have noted similarly good results following vital pulp extirpation (Fig. 10). How can this repair be explained rationally when one part of the triad is completely missing? Again, we are victims of cognitive dissonance. Reason tells us that failure must ensue; yet the evidence before our very own eyes tells us the exact opposite. Why, then, should we fill a canal at all if we have evidence that repair occurs without a root-canal filling? Unfortunately, we have seen many other cases in which the canals were not filled and repair failed miserably.

How can we explain our failures rationally when we have faithfully followed an accepted formula for success? One way is to suppress or ignore the dissonance in the hope that perhaps it will go away. Is not this what many endodontists do? A few years ago a panel convened to discuss endodontic failures. They failed to come to grips with the issue simply because the panelists, all highly competent endodontic teachers, were unable to present any "real" endodontic failures. The cases that were presented were admittedly mismanaged or misdiagnosed cases or anomalies and not true endodontic failures. Is it possible that endodontists never see anything but successes with their treatments? We doubt it. We believe that the failures are subconsciously ignored to avoid dissonance. One of our foremost endodontists, who claimed 100 per cent success, finally admitted to us, after being badgered into it, that perhaps he was in error and that 99 per cent was the correct figure! Such success rates are unheard of in any other phases of medicine or dentistry. In our view, such distortion places

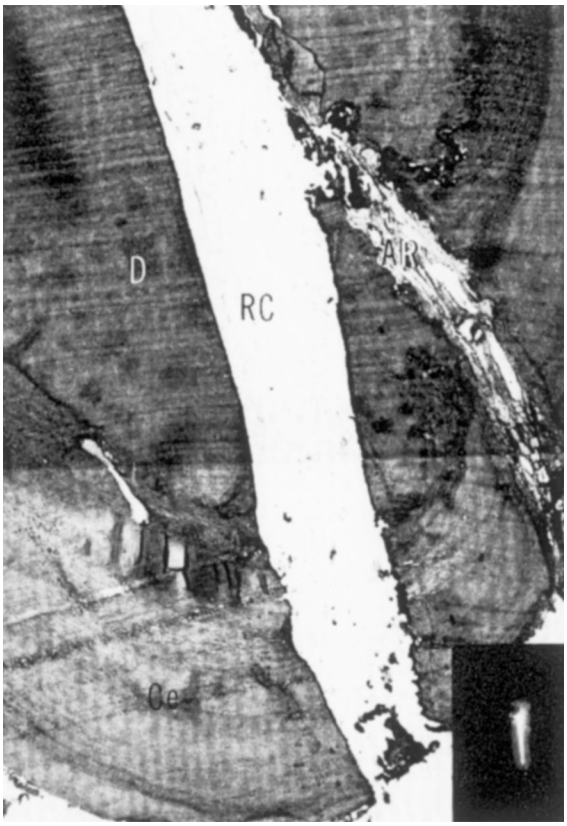
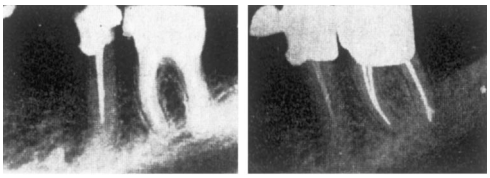


FIG 7. Palatal root of an upper molar. The root canal was instrumented and filling material was forced into the canal under pressure (inset). The main root canal (*RC*) was well-débrided and filled. An accessory ramification (*AR*) which contains pulp tissue is present. *D*, Dentin; *Ce*, cementum. (Magnification, ×54; reduced ¼.)

students and practitioners alike in a horrible, guilt-ridden position when they faithfully follow the dictates of their teachers and yet do not achieve perfect results. If the teachers can obtain them, why can't they? How inadequate they must feel! Is there justification for this feeling?

In studying our endodontic failures in the laboratory (we admit that we have some), we are frequently impressed with the inadequacy of our understanding of the reasons for those failures. Teeth with canals which have been débrided, sterilized, and filled in the accepted manner develop areas of rarefaction, or pre-existing areas of rarefaction get larger for no apparent reason. The case histories are frequently negative, and there are no known systemic conditions. Examination of the tissue sections reveals no special patterns. The periapical radiolu-



A. *B.*

FIG 8. Endodontic therapy on lower right second premolar and first molar. *A*, At completion of treatment the root canal of the second premolar is considerably underfilled. *B*, Five years later there is no periapical pathosis around the second premolar.

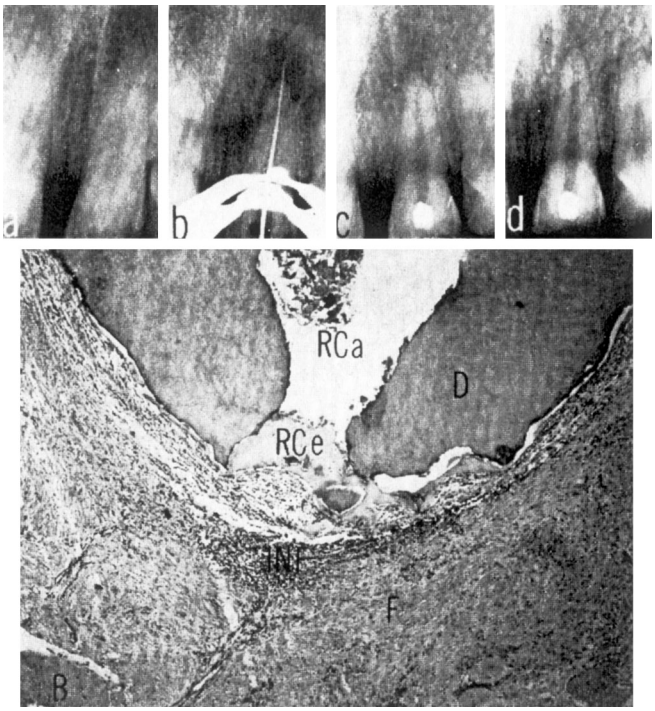


FIG 9. *A*, Endodontic treatment of an upper right central incisor with an area of rarefaction. *a*, Preoperative roentgenogram; *b*, diagnostic wire in root canal; *c*, 2 months following instrumentation, no root canal filling was placed; *d*, 4 months later, the periapical area is healing. *B*, The histologic section of the resected root apex reveals that reparative cementum (*RCe*) has been elaborated at the apex of the tooth. *RCa*, Root canal; *D*, dentin. A mild inflammatory infiltrate (*INF*) remains. *F*, Collagen fibers. (Magnification, ×54; reduced ¼.)

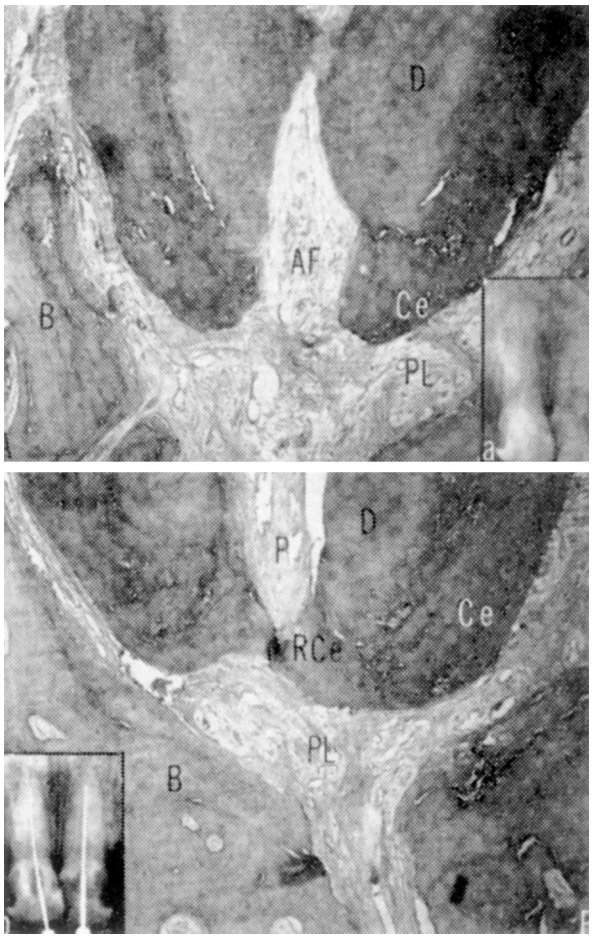


FIG 10. Two upper central incisors requiring endodontic therapy (inset *a*), have been instrumented (inset *b*), but the canals were not filled. Photomicrograph *A* of the left central incisor shows normal pulp tissue in the apical foramen (*AF*) and absence of inflammation of the periodontal ligament (*PL*). *D*, Dentin; *B*, bone; *Ce*, cementum. (Magnification, ×35; reduced ¼.)
Photomicrograph *B* of the right central incisor shows an intact pulp (*P*) and absence of inflammation of the periodontal ligament (*PL*). Reparative cementum (*RCe*) has been elaborated at the apex. *D*, Dentin; *Ce*, cementum; *B*, bone. (Magnification, ×54; reduced ¼.)

cencies represent mostly either granulomatous tissues or radicular cysts. One type of lesion does not occur more frequently than the other. Obviously, other factors—both local and/or systemic—are involved. But what are those factors? Periodontal disease? Traumatic occlusion? Leakage? The presence of undiscovered accessory canals? Psychologic factors? Systemic disease?

We trust that the questions raised here will not be misconstrued. We still believe that thorough débridement, reduction in number of microorganisms, and root-canal obturation are important in endodontic therapy. On the basis evidence thus far at hand, we think that this type of therapy usually, but not always, achieves results. Our only aim is to present our cognitive dissonance in some of the areas of endodontic theory and practice. Perhaps by so doing we may stimulate other endodontists to take a long, hard look at the a + b + c formula and realize that it has some dissonance. It is obvious, to us at least, that complacency with respect to endodontic therapy is out of order. There are too many unanswered questions. To get the answers, more and more research is needed and re-evaluations of previously accepted dicta are in order. Above all, we believe that it is time for the endodontic community (especially teachers) to become more realistic and to stop informing the dental profession that endodontic treatment is a sure-fire method which almost always succeeds. Endodontists themselves must be willing to stop ignoring the dissonance and admit that their treatments sometimes fail, for there can be no attempt at solution of a nonexistent problem.

References

1. Boring EG. Cognitive dissonance: Its use in science. Science 1964; 14:680.
2. Ingle JI. A standardized endodontic technique utilizing newly designed instruments and filling materials. Oral Surg, Oral Med & Oral Path 1961;14:83.
3. Strindberg LZ. The dependence of the results of pulp therapy on certain factors: An analytic study based on radiographic and clinical follow-up examinations. Acta Odont Scandinav 1956;14: suppl. 21.

4. Grahnén H, Hannson L. The prognosis of pulp and root canal therapy: A clinical and radiographic follow-up examination. Odont Revy 1961;12:146.
5. Rhein ML, Krasnow F, Gies WJ. A prolonged study of the electrolytic treatment of periapical infection: A preliminary report. Dental Cosmos 1926; 68:971.
6. Appleton JLT. A note on the clinical values of bacteriologically controlling the treatment of periapical infection. Dental Cosmos 1932;74:798.
7. Buchbinder M. A statistical comparison of cultured and non-cultured root canal cases. J D Res 1941;20:93.
8. Ingle JI, Zeldow BJ. An evaluation of mechanical instrumentation and the negative culture in endodontic therapy. J Am Dent A 1958;57:471.
9. Zeldow BJ, Ingle JI. Correlation of the positive culture to the prognosis of endodontically treated teeth: A clinical study. J Am Dent A 1963;66:9.
10. Oliet S. Evaluation of culturing in endodontic therapy. Oral Surg, Oral Med & Oral Path 1862;15:727.
11. Frostell G. Clinical significance of the root canal culture. *In* Grossman LI (editor): Transactions of the Third International Conference on Endodontics, Philadelphia, 1963, University of Pennsylvania, p. 112.
12. Grahnén H. The effect of instrumentation and flushing of nonvital teeth in endodontic therapy. II. A clinical and radiographic follow-up. Odont Revy 1963;14:361.
13. Lörinczy-Landgraf E, Palócz G. Kontrollergebaisse von in einer sitzung versorgten gangränzähnen. Deutsche Zahn Ztschr 1955;10:742.
14. Lörinczy-Landgraf E. Die biologische wertung des infizierten wurzelkanals: Eine Klinische Studie, Deutsche Zahn Ztschr 1957;12:438.
15. Richert UG, Dixon CM, Jr. The contolling of root surgery. Eighth Internat D Cong, Tr Supp 1931, Sec. IIIa, p. 15, 1931.
16. Dubrow H. Treatment of nonvital teeth with radiolucent periapical areas. Illinois D J 1958;27:675.
17. Dubrow H. A method of treating nonvital teeth with radiolucent periapical areas. New York State D J 1964;30:155.
18. Seltzer S, Bender IB, Turkenkopf S. Factors affecting successful repair after root canal therapy. J Am Dent A 1963;67:651.
19. Bender IB, Seltzer S, Turkenkopf S. To culture or not to culture? Oral Surg, Oral Med & Oral Path 1964;18:527.