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DIAGNOSTIC CRITERIA FOR THE TREATMENT OF CARIES-INDUCED PULPITI--ETC(U)
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Teeth with deep carious lesions approximating the dental pulp present a treatment dilemma for endodontists and restorative clinicians; should therapy be directed toward conservation of the pulp or should the diseased pulp be extirpated in a lengthier root canal filling procedure of established reliability? Naidorf (1) asserts that choice of treatment is usually based on the clinical experience and expertise of the dentist, together with his prior conditioning and unintentional biases formulated from previous successes or failures. The general dentist may enjoy a preponderance of long-term clinical successes with varied conservative pulp therapy techniques, but the endodontist sees only the failures. The dichotomy exists among pulp biologists as well as clinicians. Baume (2) states unreservedly that, "the living pulp remains the best root canal filling". Seltzer and Bender (3) advocate maintenance of pulp vitality if there is no evidence of severe pathosis, even though a chronic pulp inflammation may persist. In disagreement, Langeland, et al. (4) contend that lack of symptoms is not a success criterion and continued chronic pulpitis after vital therapy should be considered failure. A summarization of participant commentary in the 1971 Workshop on the Biologic Basis of Modern Endodontic Practice by Nygaard-Östby, Schilder and Zeldow (5) relates the view that rationale for indirect capping procedures is undermined by the difficulty in predicting the extent of pulpal damage.

Several authors (6,7,8,9) provide clinical and histologic evidence that the inflamed dental pulp can heal with conservative treatment. Three clinical studies (10,11,12) describe the radiographic resolution of periapical periodontal ligament space thickening after direct and indirect pulp-capping therapy. However, two publications (8,9) confirm the common reservation of endodontists that dystrophic changes of calcification and resorption may subsequently occur in the pulp spaces.

There appears to be universal agreement (1,3,5,9,13-16) that a new clinical classification of pulp disease is required, based on the reversibility or treatability of the condition. Past attempts (14-25) to correlate clinical findings with complicated histopathologic classifications of pulp disease have resulted in limited success. Most of these investigators conclude that no single clinical criterion can be relied upon to predict the existing status of the dental pulp. Baume (14) states, "... it is absolutely hazardous to establish a histopathologic diagnosis by clinical means. Signs and symptoms, however, give a valid lead for choosing the appropriate treatment". Seltzer and Bender (3) maintain that the dentist is generally unable to make an accurate pathologic diagnosis of the state of the pulp by clinical means. They recommend that a probable categorization be made according to seven histopathologic classifications. These histologic states are then divided into potentially treatable or nontreatable categories according to the presence or absence of pulp necrosis. They reason that it is easier to determine clinically if a pulp is necrotic than to ascertain the extent of pulposis or pulpitis. As a result, the diagnosis is considered, "... a prediction, based on clinical judgement," or, "... an educated guess as to whether or not the pulp can be treated and saved." Other researchers (2,15,26,27) concur that pulp necrosis must be considered irreversible and that it appears under deep caries simultaneously with bacterial invasion of the pulp. However, the severity of chronic pulpitis which a dental pulp can withstand and remain amenable to healing procedures has not been determined.

This study was an attempt to correlate commonly-used clinical examination findings from history, tests and radiographs with the reversibility (treatability) of pulp disease beneath deep caries. In an effort to refine some of the factors which influence clinical judgement, the significance and predictive value of each clinical variable for indicating reversible and irreversible pulp disease was calculated.

MATERIALS AND METHODS

The material consisted of 176 human permanent teeth with untreated carious lesions, ranging from superficial enamel-confined lesions to dental pulp perforation by caries. The specimens were selected from teeth designated for extraction by impartial examiners at the Naval Regional Dental Center, Great Lakes, Illinois. Most of the teeth were condemned as unmanageable during treatment-planning for complete and partial prostheses. Only a few teeth had caused recent symptoms severe enough to induce the patient to seek treatment. All of the volunteer donors were male naval recruits, ranging in age from 17 to 26 years (Median = 19). The samples included 18 anterior teeth, 36 bicuspids and 122 molars, of which 10 were third molars. Teeth with sinus tracts, swelling, extensive periodontitis involving furcations, mobility >1 mm or insufficient tooth structure for practical restoration with retentive pins were not selected for study.

Clinical data from history, examination, and tests were obtained immediately prior to extraction in most cases, but no longer than two weeks before extraction. The study was blind, with clinical information collected by three calibrated examiners at Great Lakes, Illinois and histologic evaluation carried out at the University of Connecticut.

I. Clinical Evaluation

A. Pain History. Each patient was asked if the subject tooth had ever caused pain, under any circumstances. If the response was "YES", detailed information was elicited and categorized as follows:

1. Duration of pain; <1 minute, >1 minute or >1 hour.

2. Pain intensity; mild, moderate or severe. Pain which was slightly annoying, but did not interfere with daily activities was designated "mild." "Moderate" pain was that which interfered with daily activities to the extent that the patient would attempt a home remedy. Pain which prevented sleep, caused interruption of normal activities or prompted the seeking of professional aid was termed "severe".

3. Type of pain; throbbing, sustained or sharp (twinge).

4. Frequency of pain episodes; $\leq$1/month, 1/week, 1/day or >1/day.

5. Interval since last pain episode; >30 days, 8 to 30 days, 1 to 7 days or <1 day.

6. Stimuli which provoked pain; hot, cold, sweets, pressure, meals, spontaneous or other.

7. Remedies which relieved pain; hot rinse, cold rinse, brushing, topical medicament, non-narcotic analgesic, narcotic analgesic, or other.

B. Tests. All responses to thermal, electrical and percussive tests were compared with responses elicited from control teeth of the same individual. The contralateral tooth in the same arch was used as the preferred control. If it was missing or had extensive caries or restorations, one or more intact alternative teeth of the same type (molar, bicuspid) were selected for control comparison. Responses elicited from test teeth were recorded as hyper-, hypo- or normo-responsive when compared to control responses. A lack of response in both test teeth and control teeth was also recorded. A special notation was made of any painful response which was sustained after removal of the stimulus.

1. Electric pulp test (EPT). A Model No. 205MB Burton Vitalometer* was used with a small amount of toothpaste applied to the concavity of the probe tip to act as a conductive medium. The tested teeth were isolated with cotton rolls and air-dried. The probe tip was placed at the cervical third of the facial or lingual crown surfaces, with care to avoid gingival contact. In several cases, where most of the crown was missing, testing was carried out directly on carious or eburnated dentin. A response from the test tooth at a deviation of three or more units on the Vitalometer scale from that recorded for control tooth response was evaluated as hyper- or hypo-responsive.

2. Cold test. Ice sticks were prepared by freezing wood-handled cotton swabs and water in molds made from discarded plastic protective sheaths of hypodermic needles. After isolating the test tooth with cotton rolls and air-drying, the tip of the ice stick was placed on the facial or lingual tooth surfaces. Melted run-off was carefully absorbed with gauze or cotton rolls to avoid gingival or adjacent tooth stimulation. The application interval was timed until the patient indicated sensation. A difference in intervals of ten seconds between test and control teeth responses was required to be graded as hyper- or hypo-sensitive. A lack of response was recorded if the patient did not experience sensation from the ice after a 30 second interval.

3. Heat test. A heating device was constructed from a pencil-shaped 15 watt soldering instrument† with 1/16" spade-end tip. Temperature was varied with a rheostat.† Electrical hazard protection was provided by an 1/8 Amp. (0.125 mAmp) fuse in the rheostat and a ground fault interrupter§ placed between the rheostat and the wall outlet. The device was tested for

*Burton Division, Cavitron Corp., Van Nuys, CA 91406.
†Antex Precision Miniature Soldering Iron, M. M. Newman Corp., Marblehead, MA 09145.
†Dremel Model 219, Dremel Mfg. Division, Emerson Electric Co., Racine, WI 53406.
§Circuit Guard, GEP-115, Harvey Hubbell Inc., Bridgeport, CT 06602.

electrical faults by the Medical Repair Unit, Naval Regional Medical Center, Great Lakes, Illinois and its use approved by the Protection of Human Subjects Committee of the same medical center. A ventilated protective sheath was constructed of metal tubing and acrylic so that only 15 mm of the probe tip was exposed. A temperature of 80°C at the probe was continuously monitored by means of a digital thermometer,// with its thermocouple affixed at a point 15 mm from the probe tip. A 2 mm ball of temporary stopping** was used as a contact medium between the probe tip and the dry, isolated tooth. Again, the tip was placed at the gingival third of the crown, with care to avoid soft tissue contact.

4. Percussion test. A mirror handle was used for repeated strikes of each tooth in a direction as parallel to the long axis of the tooth as access would allow. Hypersensitivity was recorded only if the patient noted pain, rather than proprioceptive or auditory differences from adjacent and contralateral controls.

C. Radiographs. At least one periapical film was exposed for each tooth. All radiographs were evaluated by the three clinical investigators at Great Lakes, Illinois. A Realist Microform Reader# was used for simultaneous viewing at 7.5X magnification. If the root apices were not visible, the case was eliminated from radiographic evaluation.

1. Thickening of the periodontal ligament space (PDL). A bulge in the radiolucent PDL shawdow which is noticeably wider (not darker) at the root apices than at the lateral surfaces of the roots.

2. Loss of lamina dura. A visible break in the continuity of laminar alveolar bone at the root apices which cannot be considered to be caused by poor contrast, angulation or normal anatomy. A tooth was evaluated by this criterion only if it did not demonstrate definite thickening of the PDL.

3. Sclerosis (condensing osteitis). A marked thickening of the lamina dura or halo-like sclerosing of trabecular bone surrounding the root apices. The condition appears more radiopaque than neighboring hard structures, cannot be identified as a natural anatomic feature and is not seen about apparently non-diseased adjacent roots.

4. Depth of caries. Classification was made into one of four groups: caries involving <1/4 of dentin thickness, 1/4 to 3/4 of dentin thickness, >3/4 of dentin thickness, and apparent radiographic exposure. In cases where the caries shadow overlapped that of the pulp spaces, no designation of exposure was made if the pulp outline was well delineated and had no breaks in density which would indicate communication with the caries. Where consensus was lacking on the presence or absence of a radiographic feature, the case was eliminated from consideration only with respect to that criterion.

//Model 5650 Digital Thermometer, Markson Science Inc., Del Mar, CA 92014.
**Hygienic Dental Mfg. Co., Akron, OH 44310.
#ADA Products, Inc., Milwaukee, WI 53217.

II. Histologic Classification.

After extraction, each specimen was trimmed with diamond-blade cuts on opposite sides of the tooth, parallel with its long axis. The root tips and any attached soft tissue remained intact. This trimming provided good penetration of the fixative, 10% neutral formalin, and orientation of the teeth for histologic sectioning. The specimens were shipped in fixative to the University of Connecticut for histologic processing and evaluation. Five-micron serial sections were stained alternately with hematoxylin and eosin, Masson's trichrome, or Brown-Brenn techniques. All microscopic classification was by one individual (KL), without access to the clinical data for each specimen. The specimens were divided into three groups according to the following criteria:

A. Irreversible pulp disease. Either partial or total necrosis of the dental pulp is present. At least one area of the coronal pulp has undergone liquefaction or coagulation, surrounded by masses of live and dead polymorphonuclear leukocytes (PMNs). Peripherally, concentrations of lymphocytes, plasma cells and macrophages form a dense halo around these central zones of abscess (Fig. 1).

B. Pulp disease of questionable reversibility. Heavy concentrations of chronic inflammatory cells, macrophages and a few PMNs can be seen beneath the affected dentin tubules. These cells almost obliterate the usual pulp morphology, but liquefaction or coagulation necrosis cannot be found in any of the serial sections. The inflammatory process is usually confined to the coronal pulp and bacterial cells have advanced to the point of near-exposure. Odontoblasts beneath the affected tubuli are very sparse, with no palisading. The coronal blood vessels are engorged, with great amounts of perivascular brown pigment which is birefringent in polarized light (Fig. 2).

C. Reversible pulp disease. This group includes specimens with intact uninflamed pulps and atrophic pulps. The atrophic pulp appears less cellular than the young, healthy pulp with fewer fibroblasts but greater amounts of collagen bundles. The odontoblastic layer may be reduced and flattened. Islands of calcification may be seen throughout the coronal and root pulp, with thick layers of irritation dentin reducing the volume of the pulp spaces.

Also included are specimens with evidence of moderate chronic inflammation confined to the coronal pulp (Fig. 3). Lymphocytes and plasma cells are seen gathered in moderate concentrations beneath the deepest areas of caries penetration, but do not obscure the normal architecture. Some dilation of vessels can be seen, with small amounts of perivascular brown pigment. This moderate chronic inflammation is similar to that described as "transitional stage" by Seltzer and Bender (3).

Specimens with minimal inflammatory or atrophic changes in the pulp are also included in the reversible category.

In addition to classifying each tooth according to healing potential, the depth of bacterial penetration was measured microscopically. Massler's (15) distinction between infected dentin containing bacteria

and demineralized, but bacteria-free affected dentin was used to reference measurements. The dentin tubuli were designated unaffected only when bacterial cells and demineralization were absent. All measurements were made in a direction parallel with the course of the dentin tubules. The extent of calcific apposition at the pulpodentinal junction was estimated relatively as none, slight, moderate or heavy.

RESULTS

Five specimens were eliminated from study because insufficient tissue had been recovered at extraction to make an accurate microscopic evaluation of the dental pulp. Of the remaining 171 teeth, 105 were placed in the reversible category, 50 were judged irreversible and 16 had questionable healing potential.

The frequency with which a history, test, or radiographic response occurred was tabulated for each of the three histologic categories. Occurrence, non-occurrence and intermediate categories were paired against each other to test significance by means of Chi Square analysis. The distribution of frequencies and p-values for each set of clinical variables are presented in Tables 1-4. The ability of each variable to predict reversible or irreversible disease was calculated only if the probability was 5% or less than the distribution of frequencies was due to chance. The predictive value (28) of a response was determined from the percentage of truly classified cases which manifest a characteristic out of all cases which manifest the characteristic. No subject reported the use of narcotic analgesics. Predictive values were not calculated for topical and non-narcotic therapy due to the low frequency of use.

Mobility less than 1 mm was noted in six teeth. Four of these cases were categorized reversible and two were irreversible. There were four teeth with which the patients experienced prolonged pain to thermal, electrical or percussion testing (pain persisted after the stimulus was removed). In one case, prolonged pain occurred with both EPT and cold; in another with both EPT and percussion. In the third tooth, prolonged pain was caused only by cold and in the fourth, only by heat. All of these teeth were categorized histologically as irreversible pulpitis.

The clinical usefulness of these responses as diagnostic criteria can best be visualized if they are ranked according to predictive value. The rankings of the most useful evaluations for predicting reversible pulp disease are presented in Table 5 and rankings for irreversible disease in Table 6.

The depth of bacterial penetration in each tooth was compared with the histologic assessment of pulp disease reversibility. Both primary and irregular dentin (29) were included in the measurement from the deepest bacterial penetration to the pulp. The results for all specimens are presented in Table 7. All 47 cases with complete bacterial penetration to the pulp were graded irreversible or questionable. There were six cases of necrosis in which total penetration was not confirmed histologically (12%). Eighty-five percent of the teeth with bacterial invasion extending to any depth short of the pulpodentinal junction were placed in the reversible disease category. Of the cases having bacteria-to-pulp distances of 0.5 mm/or more, 89% were judged reversible.

As seen in Table 8, where the extent of actual bacterial penetration is compared to the radiographic estimate of caries depth, 44% of the teeth judged to have carious exposures on the x-ray films did not have confirmed bacterial invasion to the pulp. There was a tendency for estimates of deepest caries advance to be greater than the measured bacterial penetration. At microscopic examination, demineralization commonly preceded the advance of bacteria in well-defined zones. However, in several cases, isolated groups of dentin tubules contained "pioneer" bacterial cells which had penetrated much deeper than detectable morphologic or staining changes indicative of dentin demineralization.

All teeth in the irreversible and questionable categories had moderate to heavy amounts of irregular dentin. Table 9 illustrates that 77% of the cases in the reversible category also had moderate or heavy amounts of irregular dentin.

DISCUSSION

When assessing the relevance of this study's results to clinical diagnostic use, it must be emphasized that the predictive values calculated for the tests and symptoms are applicable only to the sample evaluated and to other populations of similarly selected teeth. The results should not be generalized for use in all endodontic diagnosis, but must be restricted to treatment planning in deeply carious teeth. Cases with minimal caries involvement were included for comparison. However, only 20 of 171 cases had caries advancement through less than three-fourths the dentin thickness and any pulpitis present in these teeth was judged reversible.

The remaining 151 teeth had caries involvement through three-fourths or more of the dentin thickness at the deepest penetration. These are the teeth which pose the greatest problems in treatment planning. Some of the predictive values for reversibility of pulpitis under deep caries might be improved if the cases with minimal caries were removed from the study. However, since teeth with shallow caries comprised less than 12% of the total, their inclusion should have had negligible influence on the value rankings of the clinical variables.

Distribution of response frequencies was not statistically significant from histories for type of pain, frequency of pain episodes or for the interval since the last occurrence of pain in a tooth. The pain-relief effects of brushing, rinsing, topical medication or systemic analgesic therapy were also non-contributory in diagnosing pulp disease status. But, absence of pain or a pain duration of less than one minute were good predictors of reversible pulpitis. A pain duration history of one hour or more was a valuable indicator of irreversible disease. Pain of a severity sufficient to interrupt daily activities or sleep also indicated irreversibility, while a history of mild pain or no pain signaled treatability.

A history of responses to some of the stimuli which induce dental pain gave additional diagnostic evidence. Past sensitivities to hot or cold stimuli were not significant criteria, but historical absences of pain at meals or from pressure were good indicators of reversibility.

Spontaneous pain, when it occurred, was a valuable predictor of necrosis, but only 5 of 50 patients with teeth in the irreversible category reported pain without apparent stimulus. (Thirteen patients having teeth with pulp necrosis claimed that they had never experienced dental discomfort.) The distribution of responses for sweets sensitivity was significant, but the predictive values were relatively low to be of clinical use.

The unreliability of thermal and electrical pulp testing has been documented in many previous investigations. Results in Table 3 would tend to support those findings because of the many control teeth which failed to respond to these stimuli (responses of subject teeth were not recorded unless same-patient controls responded). Considering control response variability, the many types of electric test instruments in use, and the unique heat tester used in the study, it is probably best not to emphasize the nuances of hyper- and hypo-responses to these tests when ranking the clinical applicability of the variables for pulp diagnosis. The simplest evaluation, response/no response, reveals that EPT, hot and cold tests all are useful predictors of reversible pulpitis when a response is elicited. Lack of response to these tests is an indication of irreversible pulp disease. Instances of prolonged pain which persisted after the withdrawal of thermal, electric, and percussion stimuli occurred only in irreversible cases.

Absence of pain with percussion is a fair predictor of reversibility, but conversely, pain from percussion is not an indication of pulp necrosis. Equal numbers of reversible and irreversible cases experienced percussion pain, although the percentage was much higher in non-treatable cases. With the absence of severe periodontal pathosis, it can be reasoned that inflammation of the periapical tissues may result from caries toxins and the cellular breakdown products of pulpitis before necrosis can be observed in the pulp. This fact was borne out in histologic specimens which had portions of periapical soft tissue attached to the root apices. As Mitchell and Tarplee (21) observed, aggregations of chronic inflammatory cells could be seen in the periapical tissues of a few teeth whose pulps had partial chronic pulpitis without necrosis. More often, however, specimens were noted which had a well-circumscribed area of necrosis in the coronal pulp, an uninflamed root pulp, and granulomatous tissue at the root apex.

Thoma (17), in his evaluation of roentgenograms for pulp diagnosis, concluded that, "The secondary changes in the periapical tissue are evidence, almost per se, of pulp infection." In the present study, ten cases without pulp infection or necrosis were judged to have periapical PDL thickening on radiographs before the histologic assessments were made (two of these teeth had caused a pain response when percussed). Nevertheless, only four teeth with irreversible changes did not have evidence of PDL thickening, so that lack of radiographic PDL enlargement has a high predictive value (89.9%) for treatable pulpitis. Evidence of PDL enlargement is not an absolute indicator of irreversible pulp disease, but is a useful criterion, with a predictive value of 69.7%.

Loss of alveolar lamina dura is generally considered to be an early radiographic sign of periapical inflammation. These changes were evaluated only if PDL enlargement was not evident. Just 2 of 97 cases without PDL enlargement were judged to have early bone changes so that determinations of periapical lamina dura loss does not appear to be a useful procedure.

Condensing osteitis, or sclerosis of the bone surrounding the root apices, when observed, was the best predictor of irreversible disease. However, only 31% of irreversible disease radiographs had evidence of this phenomenon, so that a lack of sclerosis has a more limited applicability as an indicator of treatable disease.

Percentages of response frequencies for the questionable group tended to match those of the irreversible group in histories of presence or absence of pain, pain duration, pain severity and sensitivity to pressure. Conversely, questionable group response frequencies were similar to reversible group percentages in electric, hot and cold testing. For history of spontaneous pain, periapical PDL enlargement and sclerosis, questionable group column percentages from Chi Square analysis were intermediate between those of the reversible and irreversible group. A majority of patients with questionable category teeth complained of pain to cold stimuli and pain while eating regular meals. Most of the Chi Square analyses confirmed that the questionable cases are truly an intermediate group and should be considered to have minimal potential for healing.

Future multivariate analysis of the data may disclose that pairs or triplets of criteria from histories, tests and radiographs have better predictive values for diagnosing the reversibility of pulp disease. For instance, by simple manual matching and grouping of just a few variables, the potential of more sophisticated analysis was demonstrated; dual lack of responses to EPT and cold tests, when controls responded to both, was 94.1% predictive of irreversible pulp disease. However, there were only 17 cases in which these circumstances occurred. A coupling of pain duration of over one hour with enlargement of the periapical PDL space resulted in a predictive value of 85.7% for irreversible changes. (N=14, with two cases evaluated as questionable histologically, having severe inflammation.)

Upon re-examination of individual cases, it was noted that much of the longer duration pain reported by subjects having teeth in the reversible group was due to sweets. Further, relief was almost immediate upon rinsing or brushing. A short, sharp twinge due to cold was the most common type of pain reported for teeth in the reversible category. These data corroborate Massler's (15) description of treatable, "dentinal pain", but his characterization of "throbbing" pulpal pain was verified in only 24% of the irreversible category patients with pain histories.

In the histologic evaluation of tooth specimens, pin-point necrosis usually appeared simultaneously with bacterial penetration to the pulp. However, the extent of bacterial penetration cannot be estimated with any degree of accuracy from clinical radiographs. The development of a non-toxic disclosant which will differentiate between infected and uninfected dentin during cavity excavation is indicated.

Eighty of the 104 reversible cases in which calcific apposition could be evaluated had moderate to heavy amounts of irregular dentin. Twenty of the reversible category teeth were minimally penetrated by caries and most of these were listed among the teeth with none or slight irregular dentin. Thus, it can be concluded that almost all teeth with caries invasion through three-fourths or more of the dentin thickness have moderate to heavy amounts of irregular dentin -- regardless of the pulp's health. It is quite possible

that most of the irregular dentin observed in histologic specimens which have previously undergone pulp conservation therapy was deposited in response to the caries, not the treatment. Nevertheless, in pulp diagnosis and treatment planning, the clinician must first determine the present condition of the dental pulp. Secondly, he must consider the cummulative effects of the iatrogenic trauma (4,30) he will impose during restorative procedures on the pulp's healing potential. Finally, the dental pulp's ability to survive the continued sources of irritation during function of the tooth in its proposed role must be evaluated.

The choice of conservative pulp therapy or root canal filling for deeply carious teeth remains a matter of clinical judgement. To refine that judgement, this study has evaluated the reliability of common clinical findings in diagnosing the presenting health status of the dental pulp. Most histologic investigations of pulp treatment techniques and materials have been conducted on the intact, uninflamed dental pulps of animals, with little relevance to therapy for the diseased, exhausted pulps encountered clinically. If the findings with high predictive values are used as criteria for case selection in future clinical studies of pulp conservation, the effects of restorative and functional trauma on atrophic, diseased, but "treatable" dental pulps can be elaborated.

SUMMARY AND CONCLUSIONS

The reliability of commonly-used clinical diagnostic findings for predicting the reversiblity of pulp disease under deep caries was investigated in this study. Results of clinical histories, tests and radiographs were compared with the treatability, or potential for healing, of pulpitis in 171 teeth. None of these teeth had swelling, sinus tracts or other clinically obvious signs of necrotic pulps. The histologic evaluation for the reversibility of pulp disease was made according to the presence or absence of pulp necrosis. The following conclusions were drawn:

1. The clinical criteria of: radiographic periapical bone sclerosis; enlargement of the periapical periodontal ligament (PDL) space; a history of pain duration longer than one hour; sponteneous pain; tooth sensitivity to pressure; severe pain intensity and lack of response to thermal and electrical testing (when controls respond) -- are good indicators of irreversible pulp disease.

2. The clinical criteria of: uniform thickness of radiographic PDL space; absence of sclerosis; a history of no pain, mild or moderate pain of a duration less than one minute; absence of sensitivity to pressure or at meals; lack of pain to percussion and responses to thermal and electrical testing -- are good indicators of treatable pulp conditions.

3. Prolonged pain after removal of thermal, electrical or percussive stimuli is indicative of irreversible pulp disease.

4. Short, sharp twinges of pain from cold stimuli are common in reversible pulpitis cases.

5. Patients with treatable pulpitis may experience prolonged pain from sweets which is relieved upon rinsing or brushing.

6. No irreversible pulp disease was observed in teeth where caries involved less than three-fourths the dentin thickness.

7. Focal pulp necrosis occurred simultaneously with bacterial penetration to the pulp.

8. Pulp necrosis often occurs in the absence of pain symptoms.

9. The periapical tissues may have chronic inflammation before there is observable necrosis of the pulp.

10. Radiographic periapical PDL space enlargement does not always indicate pulp necrosis.

11. Bacterial penetration may precede demineralization of the dentin tubules. An accurate estimation of bacterial penetration cannot be made from radiographs.

12. Nearly all teeth with deep caries have moderate to heavy deposits of irregular dentin.

The use of these clinical criteria in case selection of treatable teeth for future pulp conservation studies would allow more accurate evaluation of the effects of techniques and materials on diseased, but non-necrotic dental pulps.

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Table 1. Frequency distribution and predictive values of pain history variables for diagnosing reversible and irreversible pulpitis

Pain History	Reversible Pulpitis (REV)	Questionable Potential	Irreversible Pulpitis (IRREV)	p<	Predictive Value REV(%)	Predictive Value IRREV(%)
A. None	56	4	13		76.7	
Pain	48	12	37	.005		38.1
B. Duration						
None or <1 Min.	85	9	26		70.8	
>1 Min.	19	7	24	.005		48.0
None or <1 Hr.	102	14	37		66.7	
>1 Hr.	2	2	13	.005		76.5
C. Intensity						
None or Mild	79	6	23		73.1	
Moderate or Severe	25	10	27	.005		43.5
None, Mild or Moderate	101	10	35		69.2	
Severe	3	6	15	.005		62.5
D. Type						
Throbbing	5	3	9	N.S.*		
Sustained	15	4	14	(in any pairing)		
Sharp (Twinge)	29	5	14			
E. Frequency						
≤1/Month	12	2	17			
1/Week	19	1	7			
1/Day	4	1	3			
>1/Day	12	8	9	N.S.		
F. Last Episode						
>30 Days	10	2	14			
8-30 Days	12	1	9			
1-7 Days	12	2	4			
<1 Day	14	7	10	N.S.		

*Not significant.

Table 2. Frequency distribution and predictive values of pain stimuli and remedies for diagnosing reversible and irreversible pulpitis

	Reversible Pulpitis (REV)	Questionable Potential	Irreversible Pulpitis (IRREV)	p<	Predictive Value REV(%)	Predictive Value IRREV(%)
A. Stimuli						
1. Hot						
Not Sensitive	98	13	45			
Sensitive	6	2	4	N.S.*		
2. Cold						
Not Sensitive	67	6	24			
Sensitive	37	10	24	N.S.		
3. Sweets						
Not Sensitive	73	5	34		65.2	
Sensitive	31	11	16	.005		27.6
4. Pressure						
Not Sensitive	101	11	33		69.7	
Sensitive	3	5	14	.005		63.6
5. Meals						
Not Sensitive	95	7	36		68.8	
Sensitive	9	9	14	.005		43.8
6. Spontaneous						
No	103	15	45		63.2	
Yes	1	1	5	.05		71.4
B. Remedies						
1. Brush, Rinse						
No Effect	80	9	36			
Relieves	20	6	8	N.S.		
2. Topical Medicaments						
No Effect	0	0	1			
Relieves	0	2	6			
Not Used	104	13	43	Not Done		
3. Non-Narcotic Analgesics						
No Effect	0	1	6			
Relieves	6	1	5			
Not Used	98	13	39	Not Done		

*Not significant.

Table 3. Frequency distribution and predictive values of clinical tests for diagnosing reversible and irreversible pulpitis

	Reversible Pulpitis (REV)	Questionable Potential	Irreversible Pulpitis (IRREV)	p<	Predictive Value REV(%)	Predictive Value IRREV(%)
A. Electric Pulp Test						
Hyper-responsive	26	3	8		70.3	
Normo-responsive	38	9	7		70.4	
Hypo-responsive	13	4	7		54.2	
No Response	12	0	24	.005		66.7
Response	77	16	22		67.0	
No Response	12	0	24	.005		66.7
B. Cold Test						
Hyper-responsive	40	3	11		74.1	
Normo-responsive	34	8	7		69.4	
Hypo-responsive	11	4	9		45.8	
No Response	6	1	20	.005		74.1
Response	85	15	27		66.9	
No Response	6	1	20	.005		74.1
C. Heat Test						
Hyper-responsive	39	4	13		69.6	
Normo-responsive	40	9	3		76.9	
Hypo-responsive	3	0	6			66.7
No Response	5	0	13	.005		72.2
Response	82	13	22		70.1	
No Response	5	0	13	.005		72.2
D. Percussion						
Normo-responsive	87	9	32		68.0	
Hyper-responsive	18	7	18	.010		41.7

Table 4. Frequency distribution and predictive values of radiographic findings for reversible and irreversible pulpitis

	Reversible Pulpitis (REV)	Questionable Potential	Irreversible Pulpitis (IRREV)	p<	Predictive Value REV(%)	Predictive Value IRREV(%)
A. Periodontal Ligament Space						
No Thickening	89	6	4		89.9	
Thickening	10	10	46	.005		69.7
B. Lamina Dura						
No Loss	88	5	4			
Loss	1	1	0	Not Done		
C. Sclerosis						
Absent	102	13	33		68.9	
Present	2	2	15	.005		78.9
D. Depth of Caries						
<3/4 Dentin	20	0	0		100.0	
<3/4 Dentin	85	16	50	.005		33.1

Table 5. Most valuable clinical predictors of reversible pulpitis

Criterion	N*	Predictive Value
Radiograph - caries depth <3/4 dentin thickness	20	100
Radiograph - no PDL space enlargment	89	89.9
History - no pain	56	76.7
History - no pain or mild intensity	79	73.1
History - no pain, or duration <1 min.	85	70.8
Test - responds to heat (control responds also)	82	70.1
History - not sensitive to pressure	101	69.7
History - no pain, or mild to moderate intensity	101	69.2
Radiograph - no sclerosis	102	68.9
History - not sensitive at meals	95	68.8
Test - normo-responsive to percussion	87	68.0
Test - responds to EPT (control responds also)	77	67.0
Test - responds to cold (control responds also)	85	66.9

*Reversible cases only.

Table 6. Most valuable clinical predictors of irreversible disease

Criterion	N*	Predictive Value (%)
Radiograph - sclerosis present	15	78.9
History - pain duration >1 hr.	13	76.5
Test - no response to cold (control responds)	20	74.1
Test - no response to heat (control responds)	13	72.2
History - spontaneous pain	5	71.4
Radiograph - enlargement of PDL space	46	69.7
Test - no response to EPT (control responds)	24	66.7
History - sensitivity to pressure	14	63.6
History - severe pain intensity	15	62.5

*Irreversible cases only.

Table 7. Bacterial penetration in relation to the severity of pulp disease

Measured Minimum Uninfected Dentin Thickness (mm)	Histologic Classification (Teeth)		
	Reversible	Questionable	Irreversible
0	0	4	43
<0.5	10	3	3
0.5 to 1.0	16	4	0
>1.0	78	5	3

Table 8. Bacterial penetration in relation to the radiographic estimate of caries depth

Maximum Thickness of Dentin Infected	Radiographic Estimate of Affected Dentin Thickness (Teeth)			
	Apparent Exposure	>3/4	1/4 to 3/4	<1/4
All (exposure)	33	14	0	0
>3/4	17	22	1	0
1/4 to 3/4	8	50	5	3
<1/4	1	4	2	9

Table 9. Quantities of irregular dentin according to histologic classifications

Histologic Evaluation of Irritation Dentin	Histologic Classification (Teeth)		
	Reversible	Questionable	Irreversible
None	6	0	0
Slight	18	0	0
Moderate	36	1	9
Heavy	44	15	40

Fig 1 -- Irreversible pulp disease. A zone of focal necrosis, surrounded by acute and chronic inflammatory cells, is located beneath a pulp horn exposure. (H&E, orig mag X53).

Fig 2 -- Pulp disease of questionable reversibility. The odontoblastic layer is absent and a heavy infiltrate of chronic inflammatory cells obscures normal pulp architecture. No necrotic areas are seen. (H&E, orig mag X198).

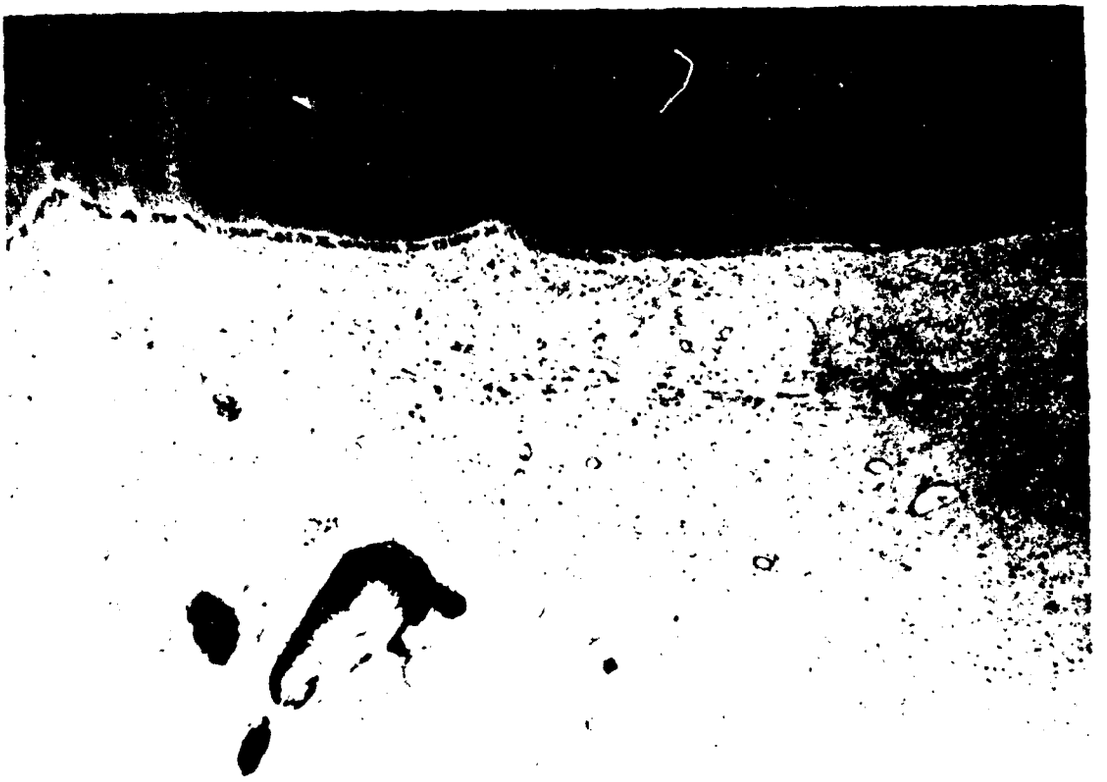

Fig 3 -- Reversible pulp disease. Although the pulp is atrophic, with calcifications and few cells, the degree of chronic inflammatory response is slight to moderate. (H&E, orig mag x80).

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Clinical findings from histories, tests and radiographs were compared with the histologic assessments of pulp disease in 171 teeth. Eighty-eight percent of the samples had deep caries. No dental pulp necrosis was observed in teeth having caries penetration through less than three-fourths the dentin thickness. Clinical criteria for selection of conservative pulp treatment or pulpectomy were compiled from clinical responses with the highest predictive values of reversibility of dental pulp disease, as judged from the histologic assessments.

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