PAINFUL PULPITIS

A Clinical and Microscopic Study

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THIS is a report of the findings made to date in a continuing study. Although toothache probably is the major basis for the existence of the dental profession, the pathoses underlying many different types of odontalgia have not been accurately diagnosed by most practitioners in the past. The subject has not been thoroughly taught in many schools. As a result, many teeth are extracted which could be saved by newer endodontic, periodontic, and restorative dentistry methods. Recently, 177 patients with toothache were examined at this school during a thirty-five-day period. The findings were analyzed and discussed previously.[1] One new patient in twelve reported with a toothache. Almost 50 per cent of these emergencies involved teeth with vital pulps, slightly more than 25 per cent involved teeth with necrotic pulps (and thus pain of periapical origin), 15 per cent were of gingival or periodontal origin, and about 3 per cent were due to other local factors, such as postextraction pain, maxillary sinusitis, and dental trauma to an opposing edentulous ridge.

The most common form of toothache is caused by pulpitis. Often it is difficult to locate the offending tooth, and it is impossible to ascertain the degree of inflammatory involvement without microscopic study. There is a continuing need for well-controlled studies of the clinical as compared with the microscopic aspects of pulpitis. Thirty-eight years ago, Raper[2] stated that the electric pulp tester was of great value in determining whether or not a pulp was vital but that it was not practicable to attempt to determine any particular pathologic state of a vital pulp with the electric pulp tester. This is still true. It has been pointed out that pulpal tissue seldom is able to destroy invading bacteria and that inflammation is likely to spread over the entire coronal portion and eventually into the root canal and periapical area. It is not known how long it takes for a microscopic inflammatory zone at the site of a tiny pulp exposure to spread and involve all of the pulp and eventually the periapical tissues.[3]

In 1929 Thoma[4] reported the first correlated clinical and microscopic study of pulp infection in fifteen teeth. Some of these teeth did not have pulpitis.

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This investigation was supported in part by Research Grant D600 from the United States Public Health Service.

In 1937 Stephan[5] reported findings in thirty-seven teeth, of which eleven were considered to have pulpitis. He also attempted to correlate clinical symptoms with histopathologic findings in the dental pulp and concluded that the acutely inflamed pulp causes spontaneous pain and generally results in a severely painful response which lasts longer than the normal reaction to applied heat and cold. Since this report was made twenty-three years ago, no similar study of permanent teeth has been reported in the literature of this country. A few attempts have been made by investigators abroad, but some of their findings may be questioned because of the inherent difficulties involved in the preparation of the dental pulp for accurate microscopic study.[6, 7] In this connection, in a recent letter Dr. Stephan says about his work:

> This [work] of course was done from 1932 to 1934. As you may have noticed, we were just beginning to recognize the importance of making an increased opening into the pulp chamber immediately after extraction of teeth in order to secure proper fixation of the pulp tissue; in fact, we learned this during the course of this study so that some of the first teeth studied were not treated, but later ones were, by drilling a hole into the pulp chamber or cracking in a vise before fixation.
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> It is interesting that I found no difference in response to pulp tests between normal pulps and pulps which were diagnosed microscopically as showing "atrophy" or "degeneration" as commonly described in the dental literature at that time.

Thus, for years, the terminology used to describe microscopic pulp findings has been confusing due to the use of such words as "reticular atrophy," "degeneration," "fibrosis," "vacuolization," "active and passive hyperemia," "open and closed pulpitis," and "acute, subacute, chronic, serous, purulent, ulcerated, and granulomatous pulpitis." In present-day analysis these terms seem to be related more to repetitious clinical mythology, to artefacts caused by the difficulties of preparation of pulp tissue for study, to misinterpretation than to facts.

MATERIAL AND METHODS

Clinical Investigation.—To standardize the clinical tests and findings, forms were developed which students used under an instructor's supervision in an effort to establish accurately the diagnosis of toothache on the basis of subjective and objective signs and symptoms. After the student had tentatively diagnosed a case, one of us repeated each of the clinical tests, first on a control tooth and then on the suspected tooth. Histologic sections were made of each of the twenty-six reported cases covered in this article. In order to standardize procedure, the following tests and materials were used:

Visual exploration.—Good light, a mouth mirror, and an explorer were used.

Pulp exposure.—A sharp explorer was used to determine whether or not the pulp was exposed. Exposure was said to exist if blood or pus was found by the explorer point, if the patient experienced sudden, severe, induced pain, or if the explorer was felt to "drop

into'' the pulp chamber. In some cases, it was necessary to excavate decay and debris in order to determine whether or not exposure was present. Occasionally the radiograph furnished suggestive evidence of exposure, but clinical inspection was always utilized.

Heat test.—A stick of temporary stopping was heated until it bent under its own weight; then it was placed against the tooth until the patient felt pain. Whenever possible, the applications of heat and cold and the electrical tester were applied to sound enamel overlying sound dentine during the tests. If pain resulted, the stimulus was immediately removed and the patient was asked to signal when the tooth again felt normal. Both severity and duration were noted and compared with reactions previously induced in control teeth in the same patient.

Cold test.—Two methods were used. The first involved the use of cotton pledgets sprayed with ethyl chloride and the second consisted of the direct application of ice. The pledget of cotton was sprayed until frosted and then immediately placed against the tooth. When ice was used, frozen water-filled anesthetic Carpules were employed. The ice was applied until the patient felt pain, at which time it was removed and the patient was asked to signal the return to normal. Both duration and severity were noted and compared with reactions induced in control teeth.

Percussion test.—The metal handle of a mouth mirror was used to tap each tooth in the quadrant or occasionally the half-mouth involved, and the patient was asked to signal when any tooth felt different or when pain was induced. On occasion, efforts were made to ''trick'' the patient in order to obtain the most objective results.

Electric pulp test.—One of two new electric unipolar testers was utilized.* If the tooth was vital, it was so noted and the number was recorded.

Radiographs.—A periapical radiograph was made in each case, and periapical involvement was noted if it existed.

All tests were of a comparative nature. Always the reactions of a normal tooth (or teeth) in the same person were compared with those of the abnormal tooth being tested. In no instance in this series was selective anesthesia necessary, although this technique has been useful occasionally in helping the clinician locate the offending tooth.

Preparations for Microscopic Investigation.—All involved teeth were tested clinically, removed at the same appointment, and placed in formalin solution. Before fixation, usually two chosen surfaces of the tooth were ground down under water until the pulp, running the length of the tooth, became faintly visible. In this way, fixation of the entire pulp was assured. If it was not possible to grind the involved tooth immediately, it was made certain that the

*Ritter Company, Inc., Rochester, New York; Burton Mfg. Company, Los Angeles, California.

pulp was exposed adequately to the preservative solution through a deep cavity, or else approximately one-third of the apical end of the root (or roots) was cut off.

After a fixation period of at least forty-eight hours, the tooth was decalcified in 10 per cent formic acid. Then the record of each case was reviewed, and the expected point of exposure was located by the microscopist. The decalcified tooth was trimmed in such a direction that the technician would make no mistake in orienting the specimen properly for cutting. The tooth then was infiltrated by and embedded in paraffin, and sections were cut with the microtome set at 8 microns. Semiserial sections were cut until the technician was reasonably sure, from inspection, that the point of exposure had been cut and passed. In some cases, complete serial sections were obtained. All sections were stained with hematoxylin and eosin, and they were evaluated by one person.

RESULTS

The findings in fifteen multirooted and eleven single-rooted teeth taken from thirteen female patients and thirteen male patients are presented in Table I. Of the twenty-six cases studied, more than half of the patients claimed severe symptoms. Eleven stated that the tooth was sensitive to heat, and all but six reported that the tooth was sensitive to cold. Two reported pain due to biting pressure. The average time elapsing between the start of the toothache and the time treatment was sought was thirty-one days. The range was from six hours to one year; however, half of the patients sought dental attention within the first week.

TABLE I. FINDINGS BASED ON TWENTY-SIX CASES OF PULPITIS

SUBJECTIVE SYMPTOMS		
Duration of Pain	*Reported Severity*	*Pain Induced by:*
Average—31 days	Mild—6	Heat alone—2
Range—6 hours to 1 year	Moderate—3	Cold alone—11
	Severe—17	Heat and cold—9
		Neither—4
		Biting pressure—2

OBJECTIVE TEST FINDINGS			
Percussion	*Thermal*	*Apical Radiolucency*	*Exposure Detected*
Negative—4	Heat alone—0	Suggestive—5	26
Positive—22	Cold alone—1	Negative—21	
Severe—1	Heat and cold—25		
Moderate—16			
Mild—5			

MICROSCOPIC FINDINGS

Number of sections per tooth—average, 58; range, 7 to 218
Estimate of coronal pulp inflammation—average, ⅔; range, ¼ to 4/4+

An analysis of the objective testing revealed a very consistent pattern. All teeth were sensitive to cold, and all but one were sensitive to heat. This particular tooth had had a calcium hydroxide and zinc oxide pulp-capping treatment five weeks prior to the date when emergency help was given, possibly influencing this result. All but four teeth were sensitive to percussion in varying

degrees of severity, and all teeth displayed a pulp exposure when examined with a sharp explorer. Only five displayed possible thickening of the apical periodontal membrane as revealed in the radiograph.

These findings indicate that pulpitis and actual pulp exposure, whether associated with deep caries, deep restorations, or other causes, go hand in hand. There was no correlation between the severity of pain and the extent of pulp involvement as studied. Subjective complaints of pain due to the intake of hot and cold foods or drinks are indicative of pulpitis, but they are not as reliable as careful, actual tests made by the clinician. No real difference in response to heat or cold was detected since, with one exception, all patients were sensitive to both as tested.

Fig. 1.—Lower left second molar from a 27-year-old man who had suffered intermittent dull pain for two to three weeks. All objective tests were positive, including minor periapical radiolucency. There is generalized inflammation throughout the coronal portion of the pulp, extending into the roots. This was the most extensive pulpitis encountered, and it does not correlate with the history of mild symptoms.

The microscopic findings indicate that all of these inflamed, vital pulps were involved most at the site of the exposure, with extension of the inflammatory zone varying from a tiny area of involvement of a pulp horn to an area extending into one or more root canals. No clinical test studied differentiated between such cases. (Compare the cases illustrated in Figs. 1 and 2.)

Serial sections of certain teeth revealed the pitfalls that are entailed in interpretation of only a few "representative" sections of pulps unless the specimen is carefully oriented so that the cut extends through both the exposure and the immediately underlying pulp tissue (Figs. 3 to 7).

Three teeth which were asymptomatic but considered likely to have pulpitis were tested and sectioned. (Fig. 8 illustrates such a case.) Thus, the subjective symptoms were negative. The results of the objective tests indicated pulpitis, and the microscopic findings supported this diagnosis. Why this pulpitis was not painful while others illustrated were painful is not clear.

Fig. 2.

Fig. 3.

Fig. 2.—Lower left second premolar from a 45-year-old woman who had experienced intermittent pain for two weeks and then "throbbing, pounding" pain. All objective tests were positive, except radiographic evidence of apical involvement. There is partial pulpitis of coronal pulp beneath exposure; 185 sections of this pulp were studied. (See Fig. 3.)

Fig. 3.—Same tooth as shown in Fig. 2. There is practically no evidence of inflammation in this section (compare with Fig. 2). This and subsequent figures illustrate the importance of cutting and studying sections directly through the exposure.

Fig. 4.—Lower left first molar from a 17-year-old boy who suffered continuous pain for two days (not severe at night). All objective test findings were positive, except for radiographic studies. No exposure or inflammation is seen. Vasodilatation ("hyperemia") is suggested. (Compare with Figs. 5 and 6.)

Fig. 5.—Same tooth as shown in Fig. 4. Note small focus of inflammatory cells in center of pulp, without apparent relationship to pulp exposure. In illustrations published in the past, such small zones of inflammation have been called "foci" and "early abscess" formation, presumably unrelated to pulp exposure. (Compare with Figs. 4 and 6.)

Fig. 6.—Same tooth as shown in Figs 4 and 5. Note small localized area of inflammation beneath pulp exposure; 204 sections of this tooth were studied before this small exposure was seen microscopically.

Nevertheless, if many more cases to be studied in this series support these early findings, it may be possible in the future to detect pulpitis in teeth which have presented no symptoms and thus avoid restoring such teeth only to have them later flare up with pain.

Fig. 7.—Upper left first molar from a 32-year-old man. Horizontal section through coronal portion of the pulp below exposure site at the base of a Class V cavity. Pulpitis involves approximately one-half of the pulp in this section. The straight line illustrates the fact that if a mesiodistal section were cut through this tooth along this plane only normal pulp tissue would be seen. The pulp exposure is not shown in this section.

Fig. 8.—Lower left second molar from a 19-year-old female patient. This nonpainful tooth had "hurt some in the past." Pulpitis was clinically suspected due to deep cavity. All objective tests were positive, except for radiographic studies. Approximately one-half of the coronal pulp in this section is inflamed. Why was this tooth without symptoms? Comparable findings have been seen in three such "control" teeth to date.

DISCUSSION

It is wise to listen carefully to the patient's description of subjective symptoms; however, his report should be used as a guide for further testing rather than as a total basis for diagnosis. Subjective symptoms are influenced by many things: the attitude of the patient toward the situation, his available time, hypersensitiveness, language barriers, and the manner in which the operator questions the patient.

The literature contains allusions to causative factors in toothache not borne out by this study, such as "gas" in the pulp chamber (which has not been demonstrated well), pulpstones (so common, yet never proved to cause pain), hypersensitive cementum (confused with sensitive dentine), pulp pain as a symptom of high blood pressure, premature contacts or minor excursive interferences ("high," recently placed restorations and manipulative tooth movements do make teeth tender, although there is no evidence to show that the pulp is adversely affected), and reputable restorative materials and tooth-cutting techniques judiciously used.

Some old diagnostic aids and ideas that no longer seem to be practical include such phrases as "cold hurts and heat relieves pulpitis," "heat hurts and cold relieves a necrotic pulp," "a necrotic pulp hurts more at night," "a lateral abscess hurts less than a periapical abscess," and "a throbbing pain denotes more serious pathosis than a dull pain."

Deciding by microscopic study whether one-fourth, one-half, or more of a pulp is involved proved to be presumptuous because of the size and three-dimensional nature of the pulps of single-rooted and multirooted teeth. Histopathologic serial sections of the tooth are in two dimensions, and the ability to analyze the third dimension depends upon the study of hundreds of sections of some teeth. A coronal pulp chamber may have a maximum dimension ranging up to 5 mm. Conservatively speaking, up to 500 microscopic sections could be made through this distance. The variation in inflammatory response in parts of the pulp in these sections is great, ranging from none to severe.

Such microscopic studies of the dental pulp must furnish adequate pulp fixation; there must be concientious searching on the part of the clinician, the laboratory technician, and the microscopist for pulp exposure; and many sections of the same tooth must be studied in order to rule out misinterpretation of the findings. It is our opinion that painful pulpitis and pulp exposure go hand in hand and that many of the so-called "degenerative" changes reported in the past are due to problems involving improper fixation, inaccurate orientation of the direction of cut for the sections, and misinterpretation of the findings seen in too few prepared sections. Needless to say, these general principles also apply to studies of pulp reactions to tooth-cutting techniques, filling materials, and pulp-capping materials.

In only seven teeth did the inflammation extend into root canals. In no instance was inflammation evident within the apical third of the root canal. Yet, twenty-two of these teeth were tender to percussion to some degree. Each of the five teeth which had suggestive apical radiolucency were tender to percussion; yet, many were tender without such radiographic findings. One can

only speculate regarding the reason why a tooth with one pulp horn inflamed should be tender to percussion. Could the mere shaking of the tooth cause pulpal pain by jarring the pulp and stimulating the nonmyelinated, nonproprioceptive pain fibers? If this were true, then periapical irritation of proprioceptive, myelinated nerves would not be necessary in order for a patient to have a localized response to a percussion test.

A word should be devoted to the "hyperemic pulp." From this and other studies,[3] it appears that this term is invalid, and published photomicrographs showing vessels filled with red blood cells *in the absence of inflammation* are based on sections cut from improperly oriented specimens and/or misinterpretation. No one has reported a study of a series of teeth with "reversible active hyperemia," for it is recognized clinically that these teeth usually recover and there is no need to extract them for microscopic study. It is our belief that if such a study were conducted, a mild, transient pulpitis localized to the pulpal ends of the freshly cut dentinal tubules would be seen. This inflammation would be evidence of *injury but not due to infection,* and hence reversible.

SUMMARY AND CONCLUSIONS

Because of the difficulties encountered in preparing for microscopic study the delicate pulp tissue enclosed in the extremely hard dental tissues, few studies have been made of relatively few teeth in an effort to correlate accurately the clinical symptoms of pulpitis with pulp histopathology.

We strongly believe that for many years misinterpretation of microscopic findings in these few studies has resulted in confusion. In an attempt to circumvent this problem, a carefully controlled study was initiated to analyze cases of painful pulpitis clinically, and after immediate extraction of these teeth microscopic studies were performed. The teeth were subjected to an electric vitality test, applications of controlled cold and heat, percussion, and exploratory probing. Radiographs then were made and periapical involvement was noted, if present. Normal-appearing control teeth were tested first in the same manner to enable us to judge better the patient's reaction to such stimuli. Laboratory preparation of the teeth for microscopic study was done with infinite care, and many sections of each tooth were studied. A carefully controlled method has been established that will be used in a continuing study of this problem.

On the basis of the first twenty-six cases, the following conclusions may be drawn at this time:

1. Teeth with painful pulpitis are vital, hypersensitive to applied heat and cold, and have pulp exposures that are usually detectable by a sharp explorer or excavator.

2. Such teeth usually, but not always, will exhibit sensitivity to the percussion test in varying degrees ranging from mild to severe.

3. Diagnosis by radiograph alone is nearly impossible in view of the fact that only five of the tested teeth displayed any thickening of the periodontal

membrane space in the periapical region. The radiograph is useful for suggesting possible pulp exposure due to a deep cavity or restoration, fracture, abrasion, erosion, or any other possible cause of exposure.

4. Invariably, the pulp inflammation is greatest immediately next to the pulp exposure. All cases showed "partial pulpitis" ranging from involvement of a pulp horn to complete involvement of the coronal pulp with extension of inflammation into the root canals.

5. If the practitioner will take pains to arrive at an accurate diagnosis of the pathosis causing "toothache," many more teeth will be saved from extraction by the judicious use of new and forthcoming therapeutic and restorative techniques.

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