Correlation between Clinical and Histologic Pulp Diagnoses

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Abstract

Introduction: Clinicians routinely face conditions in which they have to decide whether the dental pulp can be saved or not. This study evaluated how reliable the clinical diagnosis of normal pulp/reversible pulpitis (savable pulp) or irreversible pulpitis (nonsavable pulp) is when compared with the histologic diagnosis. **Methods:** The study material consisted of 95 teeth collected consecutively in a general practice over a 5-year period and extracted for reasons not related to this study. Based on clinical criteria, teeth were categorized as having normal pulps, reversible pulpitis, or irreversible pulpitis. The former 2 were grouped together because they represent similar conditions in terms of prognosis. Teeth were processed for histologic and histobacteriologic analyses, and pulps were categorized as healthy, reversibly inflamed, or irreversibly inflamed according to defined criteria. The number of matching clinical/histologic diagnosis was recorded. **Results:** The clinical diagnosis of normal pulp/reversible pulpitis matched the histologic diagnosis in 57 of 59 (96.6%) teeth. Correspondence of the clinical and histologic diagnosis of irreversible pulpitis occurred in 27 of 32 (84.4%) cases. Infection advancing to the pulp tissue was a common finding in teeth with irreversible pulpitis but was never observed in normal/reversibly inflamed pulps. **Conclusions:** Findings using defined criteria for clinical and histologic classification of pulp conditions revealed a good agreement, especially for cases with no disease or reversible disease. This means that the classification of pulp conditions as normal pulps, reversible pulpitis, and irreversible pulpitis has high chances of guiding the correct therapy in the large majority of cases. However, there is still a need for refined and improved means for reliable pulp diagnosis. *(J Endod 2014;40:1932–1939)*

Key Words

Clinical diagnosis, histologic, irreversible pulpitis, reversible pulpitis

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Pulpitis consists of pulp inflammation in response to irritants of microbial, chemical, or physical (mechanical and thermal) origin. Early attempts to classify pulpitis for clinical diagnosis were based on the type and severity of inflammation (eg, acute serous pulpitis, acute suppurative pulpitis, and chronic ulcerative pulpitis) (1), but studies showed that this classification is not accurate and has poor correlation with the histologic conditions of the pulp tissue (2–5).

The most currently used and generally accepted classification of pulpitis is based on the prognosis of treatment (6, 7). Accordingly, pulpitis is deemed as reversible when the pulp can predictably return to normal conditions after the removal of the irritant stimulus. In teeth with irreversible pulpitis, pulp conditions have little chance to be reverted to normal only by the removal of the irritants; most cases require partial or total excision of the affected pulp tissue. Knowledge of the pulp response to advancing caries lesions permits one to assume that pulp exposure by caries is a good indicator of irreversible inflammation. As caries destroys dentin and approaches the pulp, the inflammatory response becomes intense and increased in magnitude (8). However, inflammation does not usually become severe to the point of being considered irreversible until the caries lesion reaches the point of near exposure or the pulp is frankly exposed. In these conditions, the pulp tissue enters in direct contact with bacteria from the caries biofilm and almost invariably undergoes severe inflammation followed by necrosis and then infection (9). These processes occur by tissue compartments and gradually migrate in the apical direction (10, 11).

In many clinical situations, it is not possible for the clinician to ascertain whether the pulp is exposed, especially in the presence of large restorations or when the therapeutic decision is for stepwise excavation and indirect pulp capping. Thus, diagnosis is mostly based on other features including depth of the caries or restoration as evidenced by radiographs and clinical examination; presence, duration, type, and severity of symptoms; and pulp response to sensibility tests. It remains a matter of debate whether or not the diagnosis based on these features matches the pulp histologic diagnosis.

For instance, symptoms have been widely accepted as indicators of the inflammatory status of the pulp. It is by and large accepted that the presence of relatively mild symptoms relates to reversible pulpitis, whereas more severe symptoms are associated with irreversible pulpitis (12). However, a systematic review (13) concluded that there is insufficient evidence to determine whether the presence, nature, and duration of symptoms offer accurate information about the extent of pulp inflammation.

Clinicians often have to decide whether the pulp can be saved or not. Consequently, it is important to determine how reliable the clinical diagnosis is in distinguishing cases with savable pulps from those with nonsavable pulps. The purpose of this study was to correlate the clinical diagnosis of normal pulps, reversible pulpitis, and irreversible pulpitis with the histologic diagnosis in a series of teeth extracted consecutively in a dental practice over a certain period of time.

Materials and Methods

The material for this study consisted of 95 human teeth collected consecutively in a general practice operated by a single dentist over a period of 5 years. These were obtained from 79 patients (50 women and 29 men) aged 18–75 years (mean = 37.9 years). The teeth were extracted for prosthetic, periodontal, or orthodontic reasons; treatment plan needs; or the patient's desire of not having the tooth treated. The study was approved by the institutional review board, and all patients gave consent for histologic analysis. From this pool, 58 teeth had untreated caries lesions, 33 teeth had amalgam or composite restorations, and 4 teeth were intact. The teeth were clinically

categorized as having "normal pulp," "reversible pulpitis," or "irreversible pulpitis" on the basis of the following clinical criteria.

Normal Pulps/Reversible Pulpitis

Teeth with the clinical diagnosis of normal pulp or reversible pulpitis were grouped together because they represent clinical conditions in which the pulp can be usually maintained. Teeth included in this group had no history of spontaneous pain and presented only mild sensitivity to cold or sweet stimuli. Pulp sensibility tests yielded responses within normal limits or slightly exaggerated (thermal tests). Pain elicited with the application of the stimulus ceased within a few seconds or immediately upon removal of the stimulus. All teeth were negative to percussion and palpation. Radiographs showed normal periradicular conditions.

A total of 59 teeth were included in this group. Of these, 35 were unrestored teeth with caries lesions of varying extent, which were arbitrarily classified as shallow, medium, or deep. Shallow caries were those penetrating the enamel and involving less than one quarter of dentin thickness, medium caries were those involving approximately one quarter to three quarters of dentin thickness, and deep caries were those involving more than three quarters of dentin thickness (Supplemental Table S1) (14). The other 24 teeth exhibited amalgam or composite restorations (Supplemental Table S1 is available online at www.jendodon.com).

Irreversible Pulpitis

This group consisted of teeth with a clinical diagnosis of irreversible pulpitis. These teeth were associated with severe pain that prompted the patient to seek professional aid or teeth with a history of repeated pain episodes, and often led to self-medication with analgesics. All patients reported that pain was either provoked (by temperature changes, posture changes, or chewing) or spontaneous and had become continuous. In some cases, the patient woke up in the night or pain caused the interruption of normal activities. Pain was defined as throbbing, dull, or sharp. In all cases, it was graded as severe. All patients had difficulty locating the precise tooth that was the source of the pain, and in some instances the patient confused the maxillary and mandibular arches. Sometimes the patient declared that pain was radiating to the ear, the temporal or orbital regions, or the neck. Sensibility tests (heat and cold) gave exaggerated responses. Pain did not cease upon removal of the causative stimulus. The percussion test was negative or slightly positive for some cases. A periapical radiograph was taken for each tooth, and no evident periradicular changes were present, except for widening of the periodontal ligament space in a few cases. A total of 32 teeth were included in this group; 23 of these were teeth with untreated deep caries lesions, whereas 9 were teeth exhibiting amalgam or composite restorations of varying extents (Supplemental Table S2). All cases with a clear indication of pulp necrosis (no response to sensibility tests) associated or not with clinical and radiographic evidence of apical periodontitis were excluded from the study (Supplemental Table S2 is available online at www.jendodon.com).

Control Group

The control group consisted of 4 caries-free maxillary molars whose pulp responded normally to all tests (Supplemental Table S3) (Supplemental Table S3 is available online at www.jendodon.com).

Histologic Processing

Immediately after extraction, the following approaches were undertaken in order to allow proper fixation of the pulp tissue and correct orientation of the specimen in the paraffin block. The teeth were ground under magnification with high-speed diamond burs under water spray on a mesiodistal or buccolingual plane until 1 or 2 pulp horns were encountered. In some teeth, the roots were separated 2–3 mm apically to the root canal orifices before grinding the crowns. Subsequently, they were immersed in a 10% neutral buffered formalin solution for at least 48 hours. Demineralization was performed in an aqueous solution consisting of a mixture of 22.5% (v/v) formic acid and 10% (w/v) sodium citrate for 3–4 weeks with the end point being determined radiographically. All specimens were then washed in running tap water for 48 hours, dehydrated in ascending grades of ethanol, cleared in xylene, infiltrated, and embedded in paraffin (melting point 56°C) according to standard procedures. With the microtome set at 4–5 μm/L, meticulous longitudinal serial sections were taken until the pulp was exhausted. This implied that 500–600 sections were cut for molar teeth. Every fifth section was stained with hematoxylin-eosin for screening purposes and the assessment of inflammation. These sections were used to locate the areas with the most severe inflammatory reaction. Based on this initial evaluation, all slides adjacent to the location with the most severe reaction were stained. In addition, a modified Brown and Brenn technique for staining bacteria (15, 16) was used for selected slides. Coverslips were then placed on the slides, which were examined under a light microscope.

The worst histologic condition observed was recorded for each pulp. Slides were examined by 2 evaluators (D.R. and S.L.). Evaluations were performed separately, and whenever disagreement occurred, it was resolved by joint discussion.

Histologic Criteria

The teeth were histologically classified into 3 categories according to a slight modification of the criteria proposed by Anderson et al (14).

1. Reversible pulp disease: this group included specimens with uninflamed pulps and atrophic pulps. The atrophic pulp appears less cellular than the young healthy pulp with fewer fibroblasts but a greater amount of collagen bundles. The odontoblastic layer may be reduced and flattened. Islands of calcification may be seen throughout the pulp tissue, with thick layers of tertiary dentin reducing the volume of the pulp space. Specimens with evidence of moderate chronic inflammation confined to the coronal pulp were also included. In these cases, lymphocytes and plasma cells are seen gathered in moderate concentrations beneath the deepest areas of caries penetration but do not obscure the normal architecture. Areas of coagulation or liquefaction necrosis, as well as bacteria, are absent.
2. Irreversible pulp disease: either partial or total necrosis of the coronal pulp is present. At least 1 area, even if it is very small, of the pulp tissue has undergone liquefaction or coagulation surrounded by masses of live and dead polymorphonuclear neutrophils (PMNs). Peripherally, concentrations of chronic inflammatory cells (lymphocytes, plasma cells, and macrophages) form a dense halo around these central zones of abscess. Bacterial aggregations/biofilms are observed colonizing the necrotic pulp tissue or the adjacent dentin walls. The presence/absence of a direct communication between the caries cavity and the pulp chamber (ie, perforation) was recorded.
3. Healthy pulp: pulp with no changes in the dentin/predentin/odontoblast complex is present. Dentinal tubules running parallel to each other through dentin and predentin with no reduction in numbers are also observed. There is no reduction of the odontoblast layer or the odontoblast cell size. Tertiary dentin and other calcifications

are absent. No inflammatory cell accumulations, dilated vessels, or bacteria are present.

Results

Normal Pulp/Reversible Pulpitis

The clinical diagnosis of normal pulp or reversible pulpitis matched the histologic diagnosis of reversibility of the pulp inflammatory state for 57 (96.6%) of the 59 teeth (Supplemental Table S1). For 2 teeth, despite the absence of clinical symptoms and a normal response to pulp sensibility tests, the histologic diagnosis was irreversible pulp inflammation. One of them (case #17, Supplemental Table S1) exhibited a large mesial composite restoration performed 3 years before, and histologic sections revealed an abscess in the pulp chamber surrounded by an area of chronic inflammation tapering off at some distance and uninflamed tissue at the root canal orifices. Bacterial staining revealed that the tubules of the thin layer of residual dentin (0.2 mm) were heavily colonized by bacteria, and bacterial aggregations were present in the central part of the pulp abscess cavity. The other tooth was a mandibular third molar of a 20-year-old woman (case #48, Supplemental Table S1) with a deep occlusal caries lesion (Fig. 1*A*). Only a slight sensitivity to cold stimuli was reported, and the pulp responded to tests within normal limits. Histologic sections revealed a microabscess limited to the mesial pulp horn area, and a large amount of tertiary dentin layering the pulp chamber roof (Fig. 1*B* and *C*). The abscess was characterized by necrotic tissue partially washed away during histologic processing, colonized by bacteria, and surrounded by a dense accumulation of PMNs (Fig. 1*D*). At a short distance from the abscess, the remainder of the pulp tissue showed normal histologic characteristics (Fig. 1*B* and *C*). No perforation (pulp exposure) could be observed in any of the histologic serial sections of teeth from this group (Supplemental Table S1 is available online at www.jendodon.com).

In all the untreated carious teeth with correspondence of the clinical and histologic diagnosis of reversible pulpitis, pathologic changes of varying severity were present, such as a mild to moderate accumulation of chronic inflammatory cells beneath the tubules affected by caries (Fig. 2*A–F*). Another common feature was the presence of tertiary dentin and a reduction of the odontoblast layer (Fig. 2*C–E*). In some areas of teeth with medium and deep caries, no odontoblasts could be seen, having been replaced by flattened cells resembling fibroblasts (Fig. 2*D* and *E*).

In the pulps of the restored teeth with correspondence of the clinical and histologic diagnosis of normal pulp/reversible pulpitis, the presence of varying amounts of tertiary dentin with the reduction of the odontoblast layer in the areas subjacent to the cavities was a common finding. The pulps in general exhibited an atrophic aspect with less cells and an abundance of collagen fibers in the absence of inflammation. Some pulps showed mild to moderate

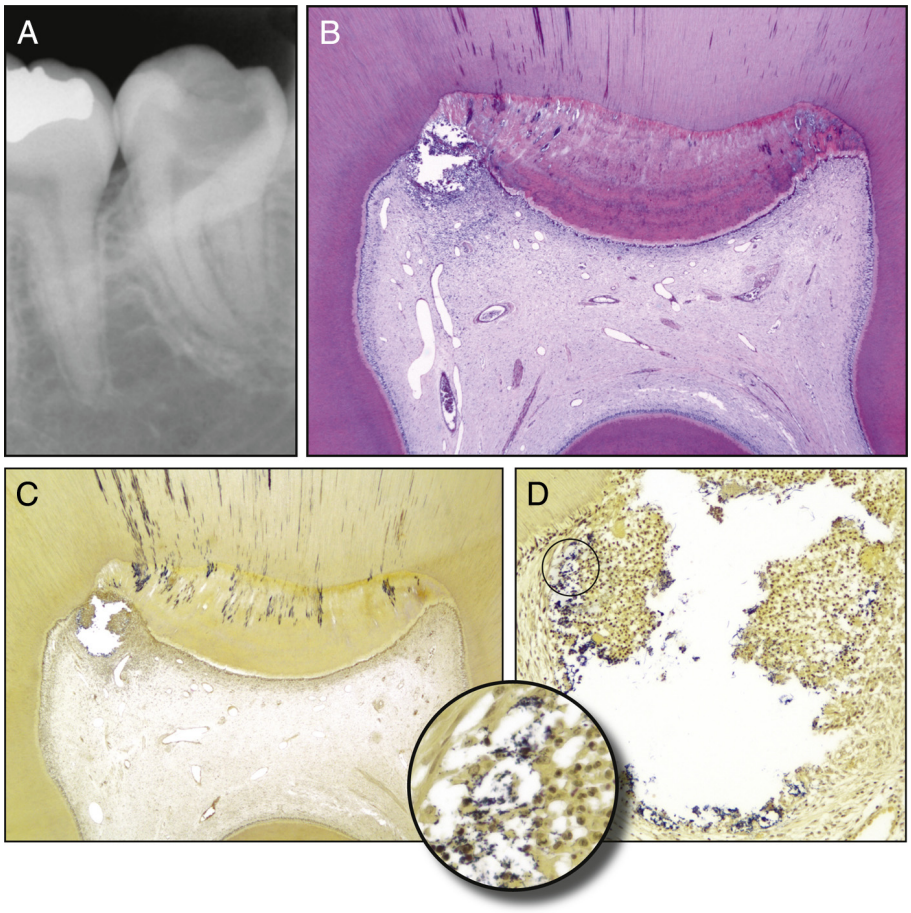

Figure 1. Clinical reversibility not matching histologic reversibility (case #48, Table S1). (*A*) A radiograph of tooth #17 with deep occlusal caries. The clinical diagnosis was reversible pulp inflammation. (*B*) A limited area of necrosis is present in the mesial pulp horn. A large amount of tertiary dentin on the roof can be seen (hematoxylin-eosin, original magnification ×16). (*C*) A section close to that in *B* (Taylor's modified Brown and Brenn, original magnification ×16). (*D*) A detailed view of the microabscess. Bacteria surrounded by PMNs on the right and by fibroblasts on the left (original magnification ×100, inset ×400).

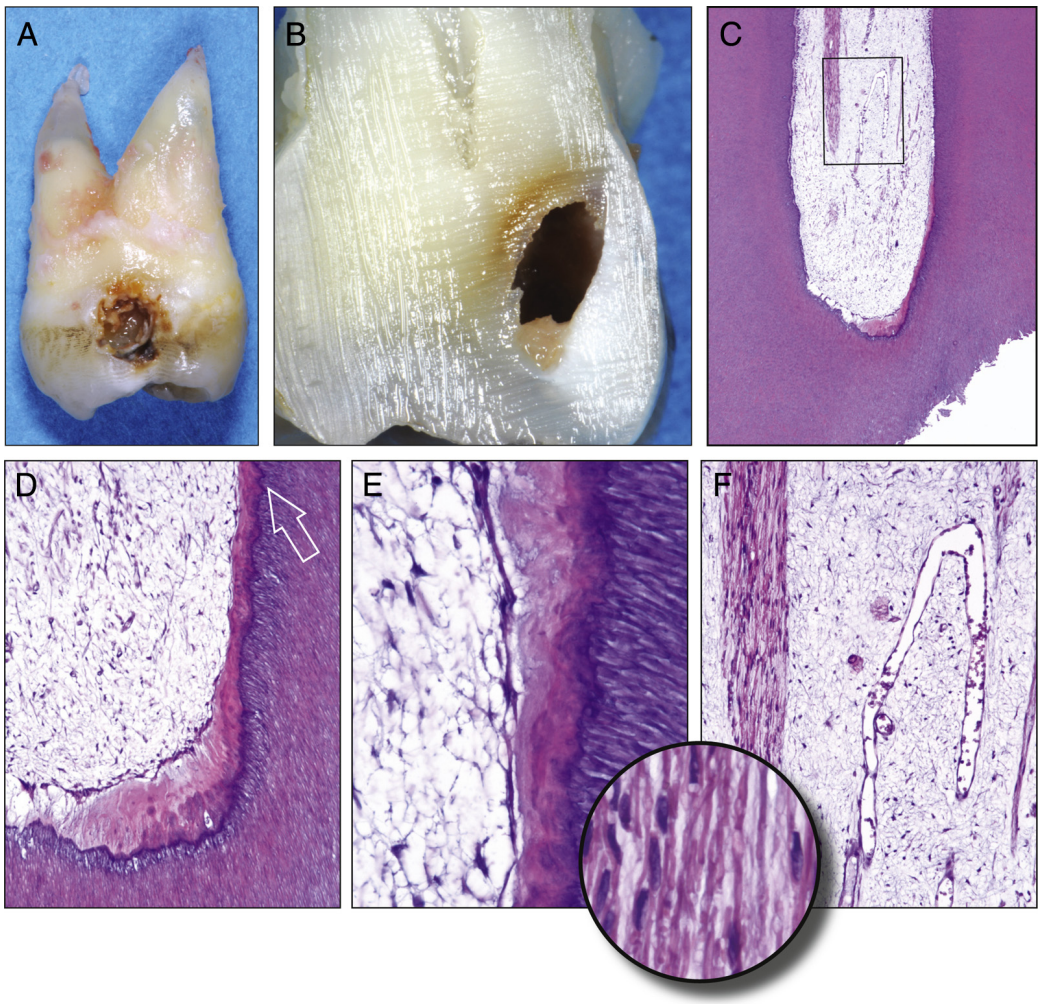

Figure 2. Clinical reversibility matching histologic reversibility (case #29, Table S1). (*A*) A maxillary third molar in a 33-year old woman with a deep mesial caries cavity and only slight sensitivity to cold stimuli. (*B*) Preparation of a mesiodistal sectioning plan. (*C*) An overview of the pulp chamber. No necrosis can be seen. Tertiary dentin is present under the carious cavity (hematoxylin-eosin, original magnification ×25). (*D*) A detailed view of the mesial pulp horn; a reduction of the odontoblast layer and the absence of inflammatory cells can be seen (original magnification ×100). (*E*) A high-power view of the area indicated by the *arrow* in *D*. An atubular tertiary dentin is layered by flattened cells resembling fibroblasts (original magnification ×400). (*F*) Magnification of the area demarcated by the rectangle in *C*. Vessels and nerve bundles in an uninflamed connective tissue (original magnification ×100, inset ×400).

accumulation of chronic inflammatory cells; in these cases, marginal leakage was evident as shown by the presence of bacterial colonization on the cavity walls and in the superficial portion of the dentinal tubules.

Irreversible Pulpitis

The clinical diagnosis of irreversible pulpitis corresponded to the histologic diagnosis for this condition in 27 (84.4%) of the 32 cases (Figs. 3*A–F* and 4*A* and *D*). In these teeth, areas of coagulation or liquefaction necrosis of varying extent could be observed in the coronal pulp (Figs. 3*C–F* and 4*C* and *D*). These areas were heavily colonized by bacteria and surrounded by severe concentrations of PMNs (Fig. 3*D–F*). Reactions were less severe in the remainder of the coronal pulp, and it was not infrequent to observe uninflamed pulp tissue with normal architecture in the contralateral pulp horn (Fig. 3*C*). For the remaining 5 teeth (15.6%), the histologic diagnosis was actually "reversible pulpitis" with localized accumulations of inflammatory cells under the affected dentinal tubules, vasodilation, and abundant tertiary dentin formation but the absence of necrosis and bacterial infection.

A case with an untreated deep caries lesion (case #3, Supplemental Table S2) is shown in Figure 5. It is a maxillary second premolar in a 52-year-old woman presenting with severe spontaneous pain in the right upper jaw. She declared to have been suffering since the evening before when pain was triggered by hot food. Pain was only mitigated by self-medication with analgesics. At inspection, both maxillary right premolars showed deep caries. These teeth were negative to percussion and palpation tests. The first premolar responded normally to thermal and electric pulp tests, whereas the second premolar was markedly hyper-responsive. A radiograph showed that the caries lesion affecting the second premolar was proximal to the pulp chamber (Fig. 5*A*). The diagnosis of irreversible pulpitis was made for tooth #4, and pulpectomy was recommended in addition to caries removal and restoration for tooth #5. The patient did not accept any treatment aimed at conservation of tooth #4 and requested its extraction. Histologic sections cut on a mesiodistal plane and passing approximately through the center of the caries lesion showed that the secondary dentin had been completely destroyed and the pulp was covered only by tertiary dentin (Fig. 5*B* and *C*). The coronal pulp tissue showed marked hyperemia with apparently empty spaces in a reduced odontoblast layer and scattered chronic

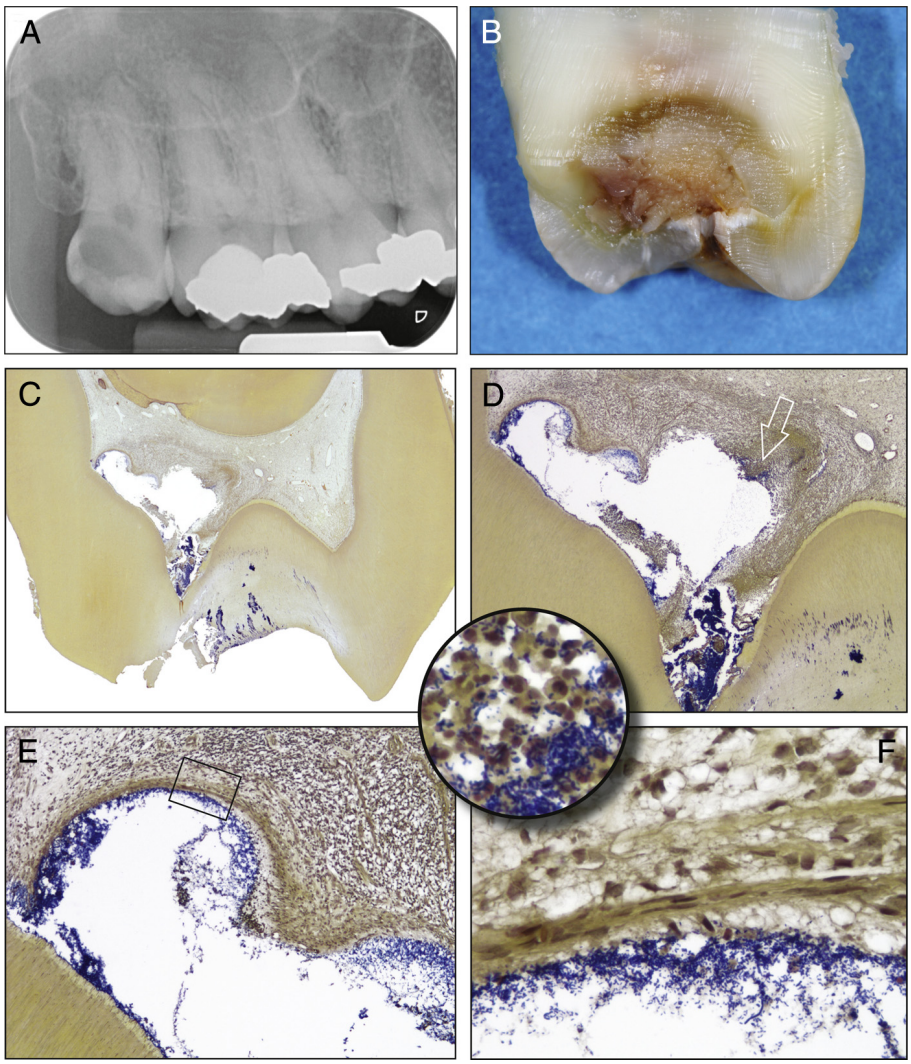

Figure 3. Clinical irreversibility matching histologic irreversibility (case #26, Table S2). (*A*) A maxillary third molar in a 30-year-old man with severe spontaneous pain. (*B*) Preparation of a buccolingual sectioning plane. (*C*) An overview of the pulp chamber. An abscess is present buccally (Taylor's modified Brown and Brenn, original magnification ×8). (*D*) A detailed view of the abscess cavity. The content has been partially washed away during processing. Necrotic debris heavily colonized by bacteria (original magnification ×16). (*E*) The left portion of the abscess. Bacteria are limited by fibrous connective tissue (original magnification ×50). (*F*) A high-power view of the rectangular area in E (original magnification ×400). (*Inset*) A high-power view from the area of the right portion of the abscess indicated by the *arrow* in *D*. Bacteria and severe accumulation of PMNs (original magnification ×400).

inflammatory cells (Fig. 5*D*). No necrosis could be seen, and bacterial stain revealed that bacteria had not penetrated the interface between secondary and tertiary dentin (Fig. 5*E*). Beyond the root canal orifice, the pulp tissue showed a completely normal aspect (Fig. 5*F*). Despite the clinical diagnosis of irreversible pulpitis, the final histologic diagnosis was that of reversible pulpitis. (Supplemental Table S2 is available online at www.jendodon.com)

Control Group

The 4 control teeth (intact teeth) showed a normal pulp microscopic architecture with normally aligned odontoblasts and the absence of inflammatory cells and other pathologic changes.

Discussion

The choice of conservative vital pulp therapy or root canal treatment for deeply carious teeth remains a matter of clinical judgment (14). To refine that judgment, this study evaluated the reliability of common clinical findings in diagnosing the present health status of the dental pulp.

The methods for the diagnosis of pulp conditions are still very limited in terms of accuracy. Basically, the clinician has to collect data from subjective examination (chief complaint and dental history), visual inspection, radiographic examination, and response to stimuli application (pulp tests) and then infer the possible diagnosis. The classification of pulpitis as reversible and irreversible based on the prognosis of treatment simplified the process, but it is important to know how well it correlates with the actual pulp conditions. In the present study, we used defined criteria for clinical and histologic classification of pulp conditions as healthy, reversibly inflamed, or irreversibly inflamed. In the 2 former conditions, the pulp can be preserved, whereas in the latter it has to be partially or totally removed for improved outcome.

Our findings revealed a high correlation between the clinical and histologic diagnosis of pulp conditions, especially for the cases with normal pulp and reversible pulpitis. This is of great clinical relevance

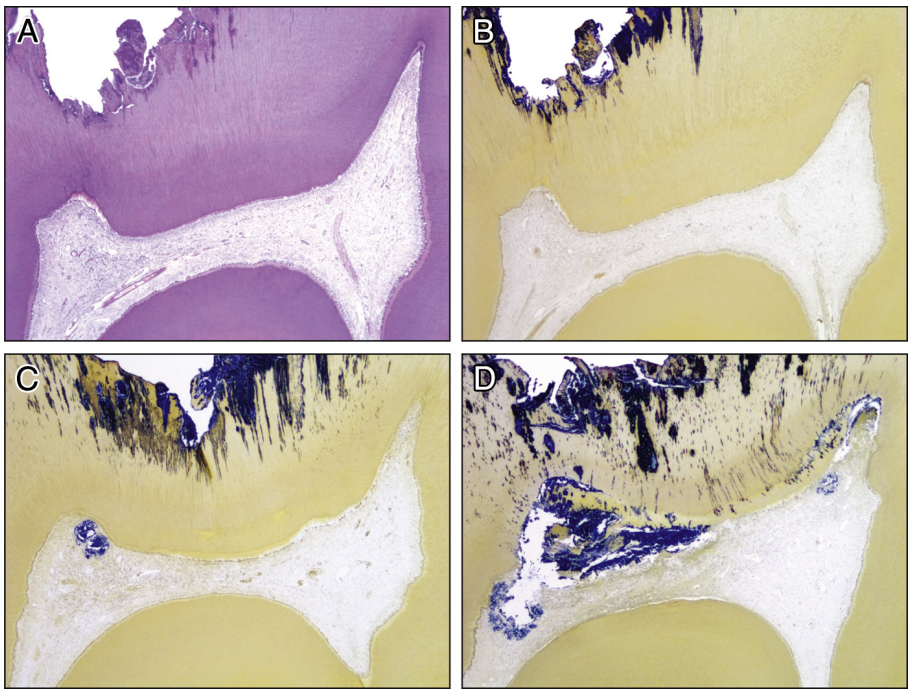

Figure 4. Clinical irreversibility matching histologic irreversibility (case #29, Table S2). A mandibular third molar with deep occlusal caries in a 42-year-old woman. Severe spontaneous pain was present. (*A*) The section passing through the buccal pulp horns. No necrosis can be seen. The picture would be indicative of reversible pulp inflammation (hematoxylin-eosin, original magnification ×16). (*B*) The section taken not far from that shown in *A* (Taylor's modified Brown and Brenn, original magnification ×16). (*C*) The section taken about 60 sections away from that in *B*. An area of necrosis heavily colonized by bacteria is now evident. Contrary to the picture in *A* and *B*, the diagnosis is now "irreversible pulp inflammation" (original magnification ×16). (*D*) After approximately another 100 sections, the pulp appears severely unstructured, with large areas of necrosis and severe bacterial colonization (original magnification ×16).

because the histologic conditions used to classify pulps as having reversible inflammation are quite compatible with tissue maintenance and survival. In other words, the clinical diagnosis of reversible pulpitis based on the criteria used in this study has great chances to accurately guide therapy toward pulp-saving approaches.

Determination of the reversibility of pulp inflammation in response to caries is usually straightforward in the presence of clinical pulp exposure (7). In other words, pulpitis is usually reversible before the pulp becomes directly exposed by the carious process. After exposure, inflammation becomes irreversible in the sense that removal of the cause (carious and infected dentin) is not sufficient to provide a predictable outcome. However, as observed in this study, in some cases of very deep and extensive caries lesions, the pulp can become irreversibly inflamed even before frank perforation. This is a result of heavy bacterial invasion of the dentinal tubules underneath the caries biofilm, which may create a "functional exposure." The success of vital pulp therapy in cases of irreversible pulpitis is low (17) because the extension of pulp degeneration and infection cannot be reliably determined in the clinical setting. In cases in which the total removal of caries or restorations is not possible, indicated, or desired, pulp exposure cannot be ascertained, and reliance on other clinical parameters is necessary for proper diagnosis. Our findings showed that the diagnostic criteria used herein may satisfactorily predict whether the pulp is savable or not.

Previous studies, most of them using old clinical classifications for pulp conditions, have not found a correlation between clinical and histologic data. In their classic study, Seltzer et al (3) concluded that the severity of pain was only partially related to the severity of the inflammatory response and was influenced by the occurrence of drainage and the patient's previous experiences. They also reported that pulp painful responses to thermal stimuli are not typical of specific types of inflammation. Baume (2) evaluated 270 pulps serially sectioned and reported the coexistence of different conditions in various parts of the same pulp. For instance, a single diagnosis for the entire pulp tissue as serous or suppurative pulpitis was not possible in most cases, and this criterion for histologic diagnosis may refer only to a distinct portion of the pulp. This author concluded that it is impossible to establish a histologic pulp diagnosis based on clinical examination. Dummer et al (18) found no clear association of signs or symptoms with pulp histologic conditions and concluded that it may be impossible to classify accurately the pulp condition of all painful teeth or to differentiate clearly between pulps that can be saved or not. Our findings differ from these studies; these differences may be related to the different clinical diagnostic criteria, established histologic criteria, and the improved histologic/histobacteriologic technique including analysis of serial sections.

Hyman and Cohen (19) evaluated the diagnostic usefulness of common endodontic tests through the retrospective analysis of studies in which histologic examinations were performed. They observed that tests were usually able to adequately identify individuals who were likely to be free of disease but were substantially less effective in identifying disease-positive individuals. Persons who were tested positive for irreversible pulp disease were frequently disease negative on the basis of histologic examination. Our results are somewhat in agreement with their observations. Although matching results were relatively high (84.4%), we found a lower correlation between clinical and histologic findings in cases of irreversible pulpitis when compared with normal/reversibly inflamed pulps. This means that a few teeth would have their canals unnecessarily treated. It is also noteworthy that all teeth clinically diagnosed with irreversible pulpitis included in this study were symptomatic, but it has been alleged that most pulps with irreversible inflammation are asymptomatic (20). Most of these teeth are only diagnosed with irreversible pulpitis after caries removal and the detection of pulp exposures. Teeth with these conditions were not included in this study.

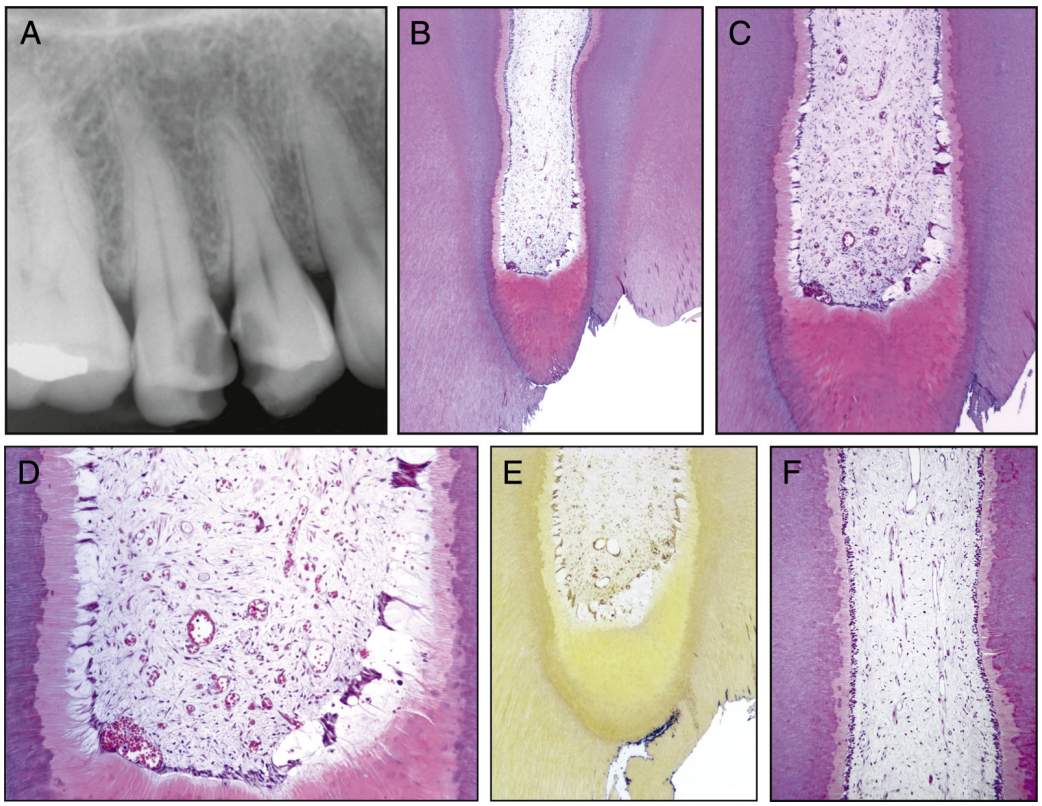

Figure 5. Clinical irreversibility not matching histologic irreversibility (case #3, Table S2). (*A*) The diagnostic radiograph. (*B–D*) Sections were cut on a mesiodistal plane. Progressive magnifications from the pulp chamber show a marked hyperemia and inflammation, but no necrosis can be seen (hematoxylin-eosin, original magnification ×25, ×50, ×100). (*E*) Bacteria are confined to the transition between secondary and tertiary dentin (Taylor's modified Brown and Brenn, original magnification ×50). (*F*) Just beyond the root canal orifice, the pulp exhibits characteristics of normality (original magnification ×50).

Findings and considerations for irreversible pulpitis cases point to a need for improvement in the pulp diagnostic means and emphasize that clinicians should use all available methods and evidence to diagnose irreversible pulpitis.

Curiously, no case considered as having clinically normal pulps exhibited histologic conditions categorized as healthy pulps. Only intact teeth included as controls evinced histologically healthy pulps. Teeth with clinically normal pulps had coronal restorations and no indication of recurrent/secondary caries. All cases were classified histologically as reversible and not healthy because even in the absence of overt inflammation, there were always deviations from normality (ie, the presence of scattered inflammatory cells, hyperemic vessels, fibrosis, a reduced odontoblastic layer, and tertiary dentin). All these features are highly likely to have been related to previous inflammation. This strongly suggests that pulps with reversible pulpitis, even after correct treatment, never regain the histological features observed in the pulps of intact teeth. However, these pulps continue to survive and function.

Most of the previous studies on this subject (3, 4, 18, 21) have not included bacterial staining or used less than optimal staining approaches (2) so that the presence/absence of bacterial colonization of the pulp tissue was not taken into consideration in the histologic diagnosis of reversibility/irreversibility. In the criteria used by Anderson et al (14), bacterial presence in necrotic areas was not highlighted either. One strength of the present study was that, in addition to the histologic evaluation, histobacteriologic processing of the specimens was performed. This is probably the first study to systematically conduct this type of analysis in association with the reversibility/irreversibility of pulp inflammatory disease. Bacterial invasion of the pulp tissue was a common finding in teeth with irreversible pulpitis but was absent in teeth with normal pulps/reversible pulpitis. The relevance of this information cannot be overstated because pulpitis is most often a sequel to caries and advancing bacteria. Moreover, the degree and depth of infection in the pulp tissue can definitely influence its healing response.

In conclusion, our findings using defined criteria for clinical and histologic classification of pulp conditions revealed a good agreement, especially for cases with no disease or reversible disease. This means that the classification of pulp conditions as normal pulps/reversible pulpitis (savable pulps) and irreversible pulpitis (nonsavable pulps) have high chances of directing the correct therapy in the large majority of cases. Despite the high number of cases matching the diagnosis of irreversible pulpitis, a few teeth would still be unnecessarily treated based on existing parameters. Therefore, there is a need for improved means for reliable pulp diagnosis.

Acknowledgments

The authors deny any conflicts of interest related to this study.

Supplementary Material

Supplementary material associated with this article can be found in the online version at www.jendodon.com (http://dx.doi.org/10.1016/j.joen.2014.08.010).

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