# QUALITATIVE AND QUANTITATIVE EVALUATION OF PULPAL PATHOLOGY AND ITS RELATIONSHIP TO PULPAL HEALING FOLLOWING VITAL PULPOTOMY

By

Richard M. Adams, B. A., D. M. D.

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APPROVED

[redacted]

Donald R. Porter, D.D.S., M.S.

[redacted]

Norman R. Rickles, D.D.S., M.S.
Professor of Oral Pathology

[redacted]

Mills B. Jump, A.B., D.M.D., Ph.D.
Chairman, Graduate Education Committee

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# Qualitative and Quantitative Evaluation of Pulpal Pathology and Its Relationship to Pulpal Healing Following Vital Pulpotomy

The stumbling block in consistently successful therapy of the dental pulp is faulty diagnosis. The problem centers around an inability to assay clinically the degree of inflammation and degeneration present in the pulp tissue at the time of treatment. Thermal and electric tests and evaluation of radiographs are unreliable indicators of the pulp status (44, 58, 64, 78). The more precise histopathologic method has been used for evaluating pulpal tissue in extracted teeth — in effect, a "post-mortem" diagnosis of the sacrificed unit.

It is noteworthy that the most important aspect of cancer therapy is early and accurate diagnosis. Tissue biopsy and subsequent histopathologic examination determine not only the identity of the neoplasm but, to a significant degree, the course of treatment. This technic is used to verify the clinical diagnosis of other diseases. In operative dentistry, however, it has never been used despite the fact that for over one-hundred years biopsies have been available from teeth subjected to vital pulp therapy.

In the vital pulpotomy technic, the coronal portion of the pulp that is removed represents a biopsy specimen, available for histopathologic evaluation. The presence or absence of inflammation

and/or degeneration can be determined. Utilization of such information suggests a possible diagnostic method. Any means of establishing more accurate criteria for the treatment of diseased dental pulps deserves consideration.

The purpose of this study was 1) to make a quantitative and qualitative histopathologic evaluation of the inflammatory status of the coronal pulpal tissue in primary molars, and 2) to determine their relationship to the healing response following vital pulpotomy using calcium hydroxide.

## Review of the Literature

The historical background of vital pulpotomy therapy provides a basis for understanding its rationale. An examination of the literature also permits an assessment of the criteria utilized in selecting teeth for this therapy as well as the basis for judging success of the treatment. Knowledge of the response of dental pulp to calcium hydroxide, the influence of environmental factors on the response, and significance of cellular changes within the tissue, as reported in the literature, helps us comprehend the events following vital pulp therapy.

The vital pulpotomy method dates back to the empirical efforts of such men as W.W. Allport, whose work was reported by Allen (1). Allport amputated the exposed portion of the pulp and peeled it away from the margins in order to cause its recession within the chamber. Atkinson in the discussion following Allen's paper advocated this

technic and advised the careful treatment of amputated pulp horns by temporary fillings. Permanent restorations were recommended after healing is assured. He concluded that "the great majority of exposed pulps will be preserved for indefinite periods!" Brockway (16) in 1868 was more specific in explaining the use of treatment filling, recommending capping with a small piece of lead to encourage a new deposit of dentin. In the same year Atkinson (3) advised using creosote followed by oxychloride of zinc cement over the pulp before placing a restoration.

Excision of the pulp was favored by another contemporary, G. F. Foote, who contributed to the discussion of Allen's paper (1). Foote's concept of the physiology of healing the pulp, particularly as related to blood clot formation, was remarkably astute. He believed that the pulp's best covering is the blood, which he regarded as a non-conductor lying "in perfect harmony with the pulp." His warning that all escharotics should be avoided represents a modern concept in pulpal therapy. Perhaps Foote was influenced by Wadsworth (86) who wrote, in 1852, that the best method of treatment is to eliminate the pulp mechanically without using any chemical agents.

About 1890, Davis (26) inadvertently began to perform vital pulpotomies while engaged in root canal treatments. Thirty years later he evaluated the results by radiographic examination. Of 100 root canal treatments recorded, 12 had a pulp canal filling which did not extend to the apex, nor in some instances, even into the root canal itself. The pulpal tissue was removed surgically and no devitalizing agent was used.

Leonard (54) was impressed by the "inherent recuperative powers in the dental pulp." Beginning about 1905, he treated several hundred primary teeth by extirpating only the coronal portion of the pulp, dressed the remaining stumps with an aseptic and mildly disinfectant paste, and sealed the cavity with a permanent restoration at the same sitting. Application of the paste gently and without undue pressure was emphasized. In view of the recent interest in physiologic (indirect) pulp capping, Leonard's recommendations stamp him as a pioneer in enlightened pulp therapy. A part of good surgical judgment, according to Leonard, includes the use of a pre-operative palliative to reduce inflammation and congestion. This, in turn, would encourage the resumption of normal function so that the operation may be performed as nearly as possible in healthy tissue.

Considering the meager scientific information available at that time, Leonard's rationale for vital pulpotomy technic is unusually perceptive. He stressed the following advantages in cutting away the coronal portion of the pulp: 1) most of the infected tissue is removed; 2) the site of the operation and healing is extended into a sterile area of the dentin; 3) the remaining vital pulp tissue in the root canals is relieved from back pressure of the congested coronal portion; and 4) the same vascularity in the root canals supports a greatly lessened amount of pulp tissue.

In the 1920's Hopewell-Smith (39) and Thoma (81) advocated the opposite in pulp treatment. The former believed that, since the injured or degenerated pulp had little chance of recovering and could never be completely rehabilitated, it should be extirpated. Thoma believed that,

except for the early stages of hyperemia, there is only one treatment for diseased dental pulp and that is removal.

A specific explanation of the basic purpose of vital pulpotomy was given in 1939 by Zander (88), namely, that it causes the pulp to produce dentin at the amputation site in order to insulate the pulp canals from the cavity. Support for vital pulpotomy procedure appeared in a 1950 statement by Hess (37) that every method of treatment which maintains vitality of the dental pulp is better than the best root canal filling because it keeps the periapical tissues healthy.

Rationale and effectiveness of calcium hydroxide in vital pulpotomy therapy: Since its introduction in the literature in this country by Teusher and Zander (80) in 1938, calcium hydroxide has been commonly used as a pulpal dressing. Brindsen (15) noted that it was first used for this purpose by Hermann in Germany in 1936. The medicament was compounded with salts known to be present in the human blood serum, according to Castagnola and Orlay (23) and was marketed under the name Calxyl. Hermann (1930) cited by Berman (10) also first demonstrated the formation of a true dentin bridge by means of histologic sections.

Zander (88) in 1939, on the basis of histologic evidence, suggested the use of calcium hydroxide in deciduous teeth where resorption is taking place because of the rapid formation of the dentin barrier. He suggested that this medicament provided a source of easily ionized calcium ions which, when in contact with vascular tissue whose blood is normally saturated with $Ca^{+}$ and $PO_4^{\equiv}$ ions, caused a precipitation of calcium salts. Calcium hydroxide also created an alkaline reaction

due to its pH of 12.4. Since bone alkaline phosphatase acts best in an alkaline medium and this enzyme liberates phosphate ions from the blood or tissue, "rapid precipitation of calcium phosphate might be expected."

Berman and Massler (11), on the other hand, stated that "all evidence points to the vital pulp tissue as the 'reactive' zone and the source of the calcium ions necessary for the calcification of both primary and permanent (dentin) bridges." They found only minor differences in pulp reactions under calcium hydroxide and zinc oxide-eugenol in rat molars subjected to experimental pulpotomy. "These differences were confined entirely to the first 14 days and were related to the rate of healing — not the quality of healing."

The findings of Berman and Massler (11) contradict those of O'Malley (65) who also used rats in performing experimental pulpotomies. O'Malley implanted the blood clot over the pulpal tissue with gelatin sponge, various calcium compounds, and zinc oxide-eugenol paste. Histologic study 14 days after treatment showed that direct injury alone, and all medicaments tested except zinc oxide-eugenol caused similar changes. The zone adjacent to the amputation site was an area of coagulation necrosis, while the subjacent zone showed acute inflammation and scar tissue which included formation of an osteodentin that tended to close the pulp cavity. The zone basal to it was mildly inflamed, but the rest of the pulpal tissue was essentially normal. The zinc oxide-eugenol paste produced a moderate inflammatory and hyperplastic response, but did not lead to the formation of osteodentin.

Extensive dentin bridging was seen by Hunter (41) to occur under zinc oxide-eugenol in tests with dogs; histologic studies resulted in more

frequent bridging with both calcium and magnesium hydroxide, which he attributed to an elevated pH.

Berman and Massler's study on rat molars was essentially the same as that done by Glass and Zander (32) who used 40 human teeth subjected to pulp capping. Both the rat and human pulps revealed odontoblastic differentiation in 14 days and new dentin formation in four weeks with calcium hydroxide. Zinc oxide-eugenol produced results similar to calcium hydroxide in rats; however, in human teeth there was no evidence of bridging even eight weeks after the operation, only a wall of chronic inflammatory cells on a base of normal pulp.

Mitchell (61) noted that "calcium hydroxide, magnesium hydroxide, and plaster of Paris have a peculiar osteogonic potential which other drugs studied do not."

Calcium hydroxide with radioactive $Ca_{45}$ was used as a pulp dressing after pulpotomy treatment in dog's teeth by Fletke (31). The $Ca_{45}$ ions penetrated the necrotic layer and were present in the newly formed bridge of secondary dentin, but not observed in the undifferentiated pulp tissue or odontoblasts.

Other investigators have reported the clinical effectiveness of calcium hydroxide in vital pulpotomy therapy in permanent teeth (24, 28, 75). Similar reports regarding both permanent and primary teeth have been made by Brown (17) and Strange (79). Based on histologic studies, additional favorable evidence has been submitted by Restarski (71) in 1940, Bork (7) in 1950, Brindsen (15) in 1955, and others. (8, 9, 37, 45, 64)

Conversely, studies within the last seven years by Via (84), Law (53), and Porter (66) indicate that calcium hydroxide has been associated with a high rate of failure in vital pulpotomy in permanent teeth and a higher rate in deciduous teeth.

Shroff (76) in 1959 contended that "the clinical problems associated with healing of pulp wounds are threefold: 1) removal of existing irritants, mainly bacterial; 2) the provision of a seal which will protect the healing wound from the oral environment; and 3) the incorporation, either in or beneath such a seal, of a really biologic wound dressing which will encourage normal and natural growth and healing processes." He declared that calcium hydroxide falls far short of the ideal because it actually destroys more tissue rather than acting as a biologic dressing.

In challenging the rationale of using calcium hydroxide in vital pulpotomy, Buonocore (18) in 1960 commented on the "relatively large amount of tissue destruction which results from its application." Such destruction is an important factor in attempts to save the small strands of pulp tissue in the root canals and may superimpose on the existing injury such irritants as the indigenous bacteria and tissue toxins from the area of necrosis. The result is "further tissue loss and/or the establishment of a chronic inflammatory condition."

Methods of evaluating success and failure of vital pulpotomy treatment: Wide differences in the success rate of vital pulpotomy in deciduous teeth have been reported by various investigators (6, 53, 54, 66, 84, 87), owing partly to differences in criteria used for

accepting teeth for treatment. By the same token, meaningful evaluation of the teeth after treatment is jeopardized by different standards of success and failure.

Pretreatment criteria which contraindicate pulpotomy: Radiographic findings such as the presence of calcified masses in the pulp indicates pulpal degeneration, according to recent investigators (58, 68, 87). Law (53) rejected teeth with this finding in his 1956 study. Hill (38) in 1934 and Langeland (52) in 1947, however, showed that calcific masses are found in asymptomatic, apparently normal permanent teeth from an early age. Thickened periodontal membrane and periapical or bifurcation bone loss factors, apparently, are regarded as contraindicating factors by all investigators, but very few instances are found in the literature where these changes are established as criteria. McDonald (59) cited their importance and Strange (79) used these criteria in her pulpotomy study. Internal resorption detected radiographically has been used as a treatment-rejecting criterion (66) but rarely stated. Guthrie (34) in 1960 found by histologic examination that internal resorption was associated with a degree of pulpal inflammation which rendered such pulps "poor candidates" for treatment. Deciduous teeth showing advanced root resorption are not recommended for vital pulp therapy (14). The radiograph, although a valuable aid, unfortunately does not show evidence of early periapical infection (78, 58).

In 1955 Prophet and Miller (68) compared the histologic findings in 55 primary teeth with a history of pain. Intact odontoblasts and no

inflammatory signs were found in 10 asymptomatic teeth (carious or filled). Twenty teeth with severe pain of less than 24 hours duration had dilated blood vessels and destroyed odontoblasts. The authors stated (without providing evidence) that clinical experience shows that a sedative dressing relieves pain but will not save the pulp. Twenty-five teeth with pain of more than 24 hours duration exhibited acute and chronic pulpal inflammation and necrosis.

Teeth judged severely inflamed by histologic examination were found by Guthrie (34) to have a higher incidence of pain than less inflamed teeth. Law (53) and McDonald (59) stated that spontaneous pain during day or night, or prolonged pain after thermal stimulation, contraindicates vital pulpotomy. Although supportive evidence is lacking, pain associated with mastication does not rule out accepting teeth for treatment (53, 59, 87). In order to test the reaction of cariously exposed pulps to various calcium and antibiotic compounds, James, Englander, and Massler (45) in 1956 accepted teeth with or without a history of pain. Ishibashi (44) in his histologic study of clinical material demonstrated a lack of conformity between pain and pulp tissue status attributable to the subjective expression of pain by children and their parents.

A pin point exposure is accompanied by less pulpal inflammation than a larger exposure, according to the histologic study by McDonald (58). Wittich (87) advocated (without supporting evidence) that a pulp with one millimeter or more exposure, or multiple exposures, qualifies for vital pulpotomy.

Normal bleeding at the site of amputation was a prerequisite of vital pulpotomy by Englander, Massler and Carter (29); Rapaport and Abramson (70); and Wittich (87). Law (53) noted that healthy pulps yield little hemorrhage. Excessive bleeding was associated with hyperemia and inflammation in studies by Guthrie (34) and McDonald (58). These investigators, therefore, considered pulps with profuse bleeding poor risks for vital pulpotomy.

A tooth which yields a watery exudate or pus at the exposure site has been shown to exhibit, by histologic examination, severe inflammatory and degenerative changes which contraindicate vital pulpotomy treatment (34, 58). This diagnostic criterion was used by Castagnola and Orlay (23), Law (53) and Strange (79) in their studies. A few contemporary investigators, however, have accepted for treatment teeth with necrosis in the coronal portion of the pulp (63), a procedure reported as acceptable by Crowell (25) in 1868.

Sensitivity to percussion indicates inflammatory changes in the periapical region and, therefore, is another criterion for excluding a tooth for vital pulpotomy procedure (59). Ishibashi (44) in his histologic study showed that a negative response to percussion is not necessarily associated with absence of inflammation.

Mobility is a criterion so commonly accepted and used that it rarely is mentioned specifically (59).

Gross clinical soft tissue changes which represent one or more cardinal signs of inflammation dictate against pulpotomy treatment of deciduous teeth, according to Prophet and Miller (68).

Regional lymphadenitis may be associated with an infectious process of dental origin (74). Apparently Prophet and Miller (68) are the only investigators who have suggested this criterion.

Chronic illness and/or debilitating diseases have long been considered unfavorable conditions in accepting teeth for pulp therapy, but specifically stated as criterion (59, 79) only occasionally. Animal studies by Glickman and Skhlar (33) in 1954 provided evidence of the influence of systemic disturbances on the pulp cells.

In the realm of objective clinical tests electricity is widely used. Cartledge, Cooke and Rowbotham (22) stated that "the pulp tester is the method of choice for all teeth", whereas Thoma (81) asserted that "the vitality test is of very little value." Stephan (78) in 1937 reported the following responses of pulps to electrical stimuli: normal pulp, variable response; atrophied and degenerated pulp, average response (but may give variable or no response); acutely inflamed pulp, average response, but may vary; necrotic pulp, may test positive if canals contain liquid. Vargus and Vivaldi (83) using two currents of different time-constants, measured the two thresholds for each tooth tested and designated the ratio between the thresholds as the accommodation index. In teeth judged normal and pathologic (by subsequent histologic examination) the accommodation indices were shown to be statistically different for each group. Via (85) pointed out the many variables in electrically testing tooth vitality; namely, thickness of enamel and dentin, position of electrode, moisture on teeth and electrode, and metallic restorations. McDonald (58)

asserting that children are apprehensive of this testing procedure, showed that the electric test is an unreliable means of predicting early inflammatory and degenerative changes in the pulp. Schaeffer (72) found primary teeth especially difficult to test and the results inconclusive. This information supports the observation by Hartsock (36) that failure of an injured tooth to respond to electric tests is not an "adequate criterion of pulp death."

In 1941 Austin and Waggener (4) recommended the use of ice in testing pulp vitality, especially in children because it does not make the patient apprehensive. Other advantages are its availability, constancy of the temperature delivered to the tooth, and fewer false responses elicited. Via (85) also preferred the use of cold (to heat) because of fewer negative responses, but neither investigator presented supporting evidence. Nevertheless, Via (85) in 1957 advocated the use of a thermal agent in diagnosing pulp necrosis caused by gas-producing bacteria, the resultant pain being relieved by cold. He concluded that "the use of thermal stimulants is the method of choice for pulp testing." Ishibashi's (44) study in 1958 revealed that pain in response to thermal stimuli does not provide an accurate means of diagnosing pulpitis.

Prader (67), originator of the hemogram, cited his 1949 study in which he analyzed the white blood cells from the exposure site to estimate pulp tissue status. In 1960 Guthrie (34) used this technic and found a significant relationship between high neutrophil counts and marked pulpal inflammation. Therefore, the pulpal hemogram was considered an aid in diagnosis, although it could not accurately determine

pulp health. This study indicates that when the pulp is not severely inflamed the white blood cell count has no diagnostic value. The pulp tissue, then, may show a mild or moderately acute inflammation, or any degree of chronic inflammation, without significantly detectable changes in the number of white blood cells at the site of exposure. Apparently, the hemogram does not give any indication of the degree of degeneration in pulp tissues.

The literature reveals with regrettable frequency such nebulous terms as "vital teeth", asymptomatic", or "normal response" as criteria for accepting teeth for treatment.

Post-operative criteria for judging success or failure of vital pulpotomy treatment: Thickened periodontal membrane, periapical or bifurcation bone loss, and abnormal root resorption are recommended criteria for judging success or failure. Brown (17) considered abnormal bone and root loss as criteria of failure. Via (84) used "loss of periradicular bone to judge failure and normal width of periodontal membrane, normal lamina dura and bone to judge success." Nyborg (64) noted that of 23 teeth judged histologically as exhibiting an unfavorable healing response, 16 had no radiographic change. Out of nine teeth judged to have healed, one had a thickened periodontal membrane and another root resorption by radiographic examination. Nyborg based success or failure on histologic, not radiographic, findings and demonstrated that permanent teeth showing root resorption may have normal cementum and periodontal membrane. McDonald (59) questioned an observer's ability to distinguish between abnormal and

physiologic bone loss in primary teeth.

Internal root resorption was found by Via (84) in 69 percent of the teeth which failed when treated with calcium hydroxide. Cabrini, Maisto, and Manfredi (19) drew attention to the high rate (28.5 percent) of internal resorption of dentin which coincides with pulp amputation in teeth treated with calcium hydroxide. Rabinowitch (69) classified (without supporting evidence) internal resorption following pulpotomy as a sequel of excessive trauma, infection, or strong medications, but did not associate it with success or failure. Massler, James and Englander (56) found that an increase in severity of inflammation was associated with increased internal resorption. These findings were not reported as determining success or failure. Buonocore (18) pointed out than many clinical successes show evidence of internal resorption.

Formation of a dentin bridge was first suggested as evidence of success by Glass and Zander (32). Viewed radiographically, this structure indicates a successful pulpotomy according to Cooke and Rowbotham (24), Brown (17), Kalnins (48), Via (84), and James, Englander and Massler (45). However, by probing the amputation site Brindsen (15) found only 66 percent agreement between radiographic and clinical evaluation of evidence of bridging.

All these recommendations and findings, reported during the past 15 years, suggest that there are no clearly defined quantitative radiographic means of judging the status of dental pulp. However, no investigator has suggested that the radiographic evidence of thickened periodontal membrane, periapical or bifurcation bone loss, abnormal root resorption and internal resorptioh, individually or in combination,

warrents judging the treatment successful.

Acute or prolonged post-operative pain is interpreted as failure of the treatment, and absence of pain as success. Mitchell and Tarplee (62) verified histologically the value and accuracy of post-operative pain. They found microscopic evidence of pulpitis in every tooth which had a history of pain after treatment, but no correlation between severity of pain and extent of pulpal involvement. A significant finding by Mitchell and Tarplee was occasional evidence of pulpitis in asymptomatic teeth. In his histologic study of permanent non-carious teeth treated with calcium hydroxide, Nyborg (64) found that of 46 teeth with abscess formation or marked inflammation 41 gave no history of pain. In a microscopic study of pulp changes after cavity preparation, Hori (40) found that lack of pain for a long period afterwards does not always mean that the pulp tissue is healed or is healing. The absence of pain, then, either pre-operatively or post-operatively, is not a reliable diagnostic tool.

Nyborg (64) found no instance of a marked response to the percussion test. In 31 teeth showing histologic evidence of failure after pulpotomy treatment, 27 gave no response and only four exhibited slight tenderness.

The presence of mobility, ipso facto, suggests failure. It is a universal criterion with no known attempt having been made to correlate it with failure.

Leonard (54) in 1925 indicated that redness or swelling of soft tissue was the major criterion to judge success of vital pulpotomy in primary teeth.

No author has been found who specifically mentions using involvement of lymph nodes as a post-operative criterion, although it may be assumed that Prophet and Miller (68) who suggested its use pre-operatively, would assent to its inclusion.

No coronal tissue is present in the pulpotomized tooth, and the well-insulated radicular tissue is remote from the site of testing. Nevertheless, the electric test was used to evaluate the success of vital pulpotomies by Cooke and Rowbotham (24 and Kalnins (48). A tendency for lower threshold values was noted in teeth with favorable tissue healing after pulp capping treatment with calcium hydroxide by Nyborg (64), but he concluded that "some inconsistency is evident."

Although dentin has a low thermal conductivity, according to Lisanti and Zander (55) and the potentially reactive pulp tissue is insulated and lies well beyond the confines of the crown after pulpotomy treatment, thermal testing was used by Englander, Massler and Carter (29) in 1956. Even after pulp capping, a process which leaves most of the coronal tissue intact, pulpal injury could rarely be detected by thermal test according to Nyborg (64). Vital pulps , confirmed by histologic examination, always responded to thermal stimuli.

The bacteriologic status at the amputation site one year after treatment with calcium hydroxide was reported by Easlick (27). Eight asymptomatic teeth had negative cultures while the two painful teeth had positive cultures.

Cooke and Rowbotham (24) judged a vital pulpotomy successful if it met certain criteria one or more years after treatment. Via's study (84) established a minimum interval of nine months. Histologic

studies have been made at various intervals after treatment in order to study the stages of healing (32, 64).

As noted previously, numerous investigators have made microscopic examination of pulpal tissue in extracted teeth for the purpose of evaluating dentin bridge formation and status of the underlying tissue (7, 8, 9, 15, 37, 45, 64, 71, 80, 88).

Since no two investigators have used the same criteria for accepting teeth for treatment, or judging success or failure, and since a large subjective factor is inherent in judging each criterion, it is small wonder that diverse manifestations of healing are reported following the pulpotomy technique.

Evaluation of the factors involved in the healing response of the deciduous pulp: As in tissues elsewhere in the body, consideration of the healing process in the dental pulp involves an evaluation of the role of bacterial, chemical, and mechanical irritants and their inter-relationship with certain "resistance" factors. The unique aspect of the mature dental pulp is its environment, the unyielding walls of dentin. Let us examine the answers given by various investigators to the question, "What factors affect pulpal healing?" and particularly, healing of the primary pulp after vital pulpotomy using calcium hydroxide.

In enumerating reasons for failure after pulp therapy, Hess (37) stated that an aseptic (sterile cavity, instruments, and capping materials) must be maintained. Perhaps he was influenced by Carrel, cited by Cameron (21), who stressed the importance of sepsis as a

retarding factor in any part of the animal body. Bacteremia as a possible sequela of vital pulpotomy was investigated by Beechen, Laston and Garbarino (5) in 1956. The coronal portion of 22 primary and one permanent teeth tested produced positive cultures. Blood cultures taken one and ten minutes post-operatively were negative for all except one tooth. In this instance, contamination was suspected because different bacteria were cultured from the blood than from the pulp. Easlick, Wilbur and Crowley (28) claimed complete correlation between bacteriologic examination and previous clinical and radiographic findings in ten teeth. Brown (17) and Porter (66) however, found no significant relationship between maintenance of asepsis and the outcome of vital pulpotomy. Contamination by saliva was not considered by Bergh and Martensson (6) as a contraindication in treating 1,100 deciduous teeth by vital pulp amputation. Nevertheless, all these authors agreed that aseptic methods are advisable in order to keep bacteria at the amputation site to a minimum. No dissenting opinions have been noted in the literature.

Another factor contributing to failure, according to Hess (37), is the "use of caustic agents or of agents damaging to the tissue..." He cited Zander (88) as having shown that zinc oxide-eugenol paste was a damaging agent (based on the presence of chronic inflammation in the pulp eight weeks after treatment). Paradoxically, Zander and Glass (89), using histologic sections, found that "the use of phenol (a caustic agent) prior to capping of exposed dental pulps (with either zinc oxide-eugenol or calcium hydroxide) does not interfere with nor

does it enhance the healing process."

With the possible exception of Kalnins and Frisbie (49), investigators have not engaged in histologic studies of sufficient numbers of deciduous teeth to offer valid conclusions regarding healing factors. This team, however, used 22 normal primary and 24 normal permanent teeth in an attempt to learn the effect on healing of dentin fragments accidentally inserted into the pulp wound during application of calcium hydroxide paste. Their findings indicated a 50 percent chance of dentin fragments contaminating the pulp during the pulpotomy treatment. Only one of the 22 teeth showed an absence of reaction to dentin chips, and only five had non-inflamed pulps. The authors stated, "it is possible that the contradictory reports... for the effectiveness of various pulp capping agents may largely be occasioned by the presence or relative absence of dentin chips left in the pulp while operating." The status of those pulps without dentin chips was not discussed. Huysen and Boyd (42) found pulp inflammation associated with impaction of calcium hydroxide or dentin chips directly into the pulp. In the experimental pulpotomies in normal rat molars, Berman and Massler (11) admitted that their attempts to remove dentin splinters with an air spray were revealed as unsuccessful by the histologic sections. In a clinical study of primary and permanent teeth exposed by caries, Hess (37) found healing of pulps treated with dentin powder in only 50 percent of the teeth treated. He conjectured that the failures were due to contamination, noting that in practice sterility is not always possible.

A significantly higher rate of healing in primary teeth treated by vital pulpotomy was found by Porter (66) in the three to five year old group than six to eight year olds.

According to Cameron (21), local resistance would be reflected by the "vascularity of the wounded tissue with maintenance of the integrity of the blood supply to the damaged area, and the occurrence of secondary infection." These factors plus the "state of the general circulation and the blood... all play their part in wound healing", the author concluded. It is apparent, however, that local resistance is not a factor separate from general resistance since the blood is the common denominator of each.

Glickman and Shklar (33) noted that degenerative changes occurred in the pulps of non-carious teeth in animals exhibiting induced systemic illnesses, such as protein deficiency, alloxan diabetes, starvation, and response to repeated "alarm reactions." Ireland (43) suggested (without presenting evidence) that a relationship exists between a child's health and ability of the pulp to form secondary dentin. McDonald (58) influenced perhaps by the findings of Glickman and Shklar and Ireland stated that "children with chronic illness or a lowered resistance should not be considered for vital pulp therapy." Some additional support was provided for this tenet by two recent investigators. Kozan and Burnett (51) examined the pulps of unhealthy animals and found circulation usually subnormal. Seltzer (73) in discussing the nutritional factors in "reparative dentinogenisis", stated that vitamin A deficiency causes a failure of pulp cells to differentiate into odontoblasts, or

failure of proper alignment of the cells resulting in irregular dentin. Lack of vitamin C leads to irregular and delayed dentin formation, due to poor formation of intercellular substances.

To summarize: the foregoing survey indicates that surprisingly little work has been done to evaluate the healing response of the primary pulp, and even less regarding the response after vital pulpotomy treatment using calcium hydroxide. The points in question have been largely discussed by the authorities with unsupported evidence.

Significance of white blood cells in damaged pulpal tissue: It has been noted that the diagnostic method used in selecting teeth for vital pulpotomy included history of pain, radiographs, size of exposure, hemorrhage, necrosis, mobility, redness or swelling of soft tissue, involvement of lymph nodes, percussion, electric tests and the hemogram. The purpose of these tests is to select teeth whose pulp tissue is as free as possible from gross inflammatory and degenerative changes in order to attain as high a success rate as possible in the healing response. Since any one or combination of these tests does not offer a reliable means of judging the status of the pulp, an additional diagnostic method might prove useful.

Teeth selected for vital pulpotomy treatment are assumed to have damaged pulps. An injured tissue responds, irrespective of the damaging agent, by exhibiting inflammation. Haden (35) stated that "in evaluating a white blood count, we try to determine from the number and type of cells present how great the stimulus for the formation of new cells is and thus determine the severity of a toxic process."

Nyborg (64) in his histologic study of teeth treated with calcium hydroxide remarked that "most important is the inflammatory state expressed in terms of the type and degree of cellular infiltration and of the consequent damage to the tissue." Smith and Gault (77) defined inflammation as "the sum total of action and reactions of the body against an irritant introduced from without or generated within, with the purpose to fix in situ, destroy, neutralize, or remove the injurious agent, thus paving the way for restitution to normal by regeneration or repair." They added that inflammation therefore includes, among other factors, "the complicated vascular and cellular response locally at the site of injury."

A description of the various inflammatory cells serves as 1) a brief review of the cytologic and pathologic aspects, and 2) a frame of reference for consideration of procedures employed, findings, and discussion related to this study.

Three types of cells may be identified in the inflammatory tissue cellular response. The total number of these cells in the normal blood varies from 5,000 to 9,000 per cmm. A differential count shows distribution as follows: I. Granulocytes: polymorphonuclear neutrophils, 60-70 percent; polymorphonuclear eosinophils, 2-4 percent; polymorphonuclear basophils, 0.5-1 percent; II. Lymphocytes, 20-30 percent; and III. Monocytes, 5-10 percent.

Haden (35) states that the granulocytes, ranging from 10 to 12 microns in diameter, are the most important white cells; furthermore, that the components of white blood cells are only using the

blood stream as a highway to travel from marrox, spleen, or lymphoid tissues to the site of localization and function in the tissues. They constitute the first line of defense against bacterial invasion. The exclusive presence of granulocytes in the tissue is pathognomonic of acute inflammation except in such cases as leukemia or allergy.

Granulocytes, and particularly neutrophils, have the ability to ingest and digest microorganisms and foreign particles, provided that these are of small size (12). Some of these cells can ingest other cells, and even particles of very large size. Erythrophagocytosis and leukophagocytosis have been noted microscopically.

Granulocytes also have the ability to undergo diapedesis and exhibit a special chemotaxis which allows them to pass into tissue spaces and so to be drawn to pathogenic bacteria (57); they are no doubt attracted by one or more of the constituents of the bacteria (i.e., polysaccharides). The length of life of the mature granulocytes is estimated to be three days (12).

Neutrophils may be recognized readily by their deep-staining segmented and lobed nuclei. The cytoplasm stains a faint grayish pink with eosin-hemotoxylin. The cells contain a proteolytic enzyme, leukoprotease, which is effective only in a narrow amphoteric pH range.

The nucleus of eosinophiles is usually composed of two (occasionally three) lobes connected by rather broad bands. The easily identified cytoplasm contains coarse, intensely red, eosin-staining granules. Their presence is of diagnostic value in Hodgkin's disease

and various parasitic and allergic states (77). Eosinophil granulocytes have the ability to ingest particles, although to a lesser extent than neutrophils; their ability to act as phagocytes, however, can be quite clearly demonstrated (12).

The basophils are more difficult to recognize, being morphologically similar to eosinophils, and exhibit eosin-staining cytoplasmic granules which are larger but less numerous than in eosinophils. To date, the function of the basophils is quite obscure (12, 77).

Whether $8_u$ or $15_u$, small and large lymphocytes may be identified readily by their nuclei which tend to be centrally placed. The chromatin stains intensely with hematoxylin and exhibits a peripheral distribution, giving the nucleus a typical "cart-wheel" appearance. The cytoplasm, barely visible in the small lymphocytes but more prominent in the large form, stains rather clear bright pink with the eosin-hematoxylin method. Their functions are not well understood, but occur in all chronic inflammatory reactions. Lymphocytes possess a digestive ferment, lymphoprotease, which acts only in an acid medium (77) and is very sensitive to variations of pH (12). They may be transformed into macrocytic phagocytes. Some authorities attribute the production of immune bodies to these cells (77).

In a normal individual 10 billion lymphocytes are formed daily. Their life is short, not over 24 hours, and even much less. Plasma cells, for practical purposes, may be considered as modified lymphocytes. They are $10\text{-}12_u$ in diameter with the "cart-wheel" nucleus surrounded by a pale crescentic zone of cytoplasm (77). More recent evidence indicates

that the formation of antibodies and other gamma globulins is associated with the plasma cells rather than the lymphocytes (12, 13, 47, 82).

Monocytes, 15-20 microns in diameter, are usually identified by the nucleus which has a horseshoe shape in the adult form. The cytoplasm stains a grayish pink similar to that of neutrophils. They originate from the reticuloendothelial apparatus of the bone marrow.

The monocytes' functions are similar to those of lymphocytes with the added capacity of phagocytosis. They act as scavenger cells which come into almost every inflammatory situation following acute leukocytic response. They phagocytize and digest the dead or wounded polymuclears and lymphocytes. When distended with ingested dead cells, bacteria, cell debris, or other foreign material, these cells may enormously increase in size up to 40 or 50 microns in diameter (77).

The type and severity of cellular response are considered indicative of the type and severity of the inflammation (35). Therefore, to obtain a quantitative evaluation of the inflammatory status of pulp tissue, it would be necessary to identify and count the white blood cells in the sections obtained from the pulp biopsy specimens.

# MATERIALS AND METHODS

Selection of the Sample: Primary teeth treated with vital pulpotomy therapies by undergraduate students in the Dental Clinic were selected when they met all of the following criteria: (a) pre-operative radiographs showed no abnormal bone changes in the periapical, radicular, or bifurcation areas, nor evidence of internal resorption of the roots (Figure 1); (b) radiographs were no more than three months old at the time of treatment; (c) teeth exhibited no mobility, sensitivity to percussion, redness or swelling of soft tissue on clinical examination, nor history of severe or prolonged pain; (d) calcium hydroxide paste was used to cover the pulp stumps; (e) treated teeth were examined clinically and radiographically one year or more after treatment, or a record was available of the clinical and radiographic evidence which led to the extraction of treated teeth where loss occurred prior to the one-year interval; and (f) specimens of the pulp tissue were available for histologic examination. A sample of fifty-eight primary molars met the criteria described.

Criteria for determining success of treatment: The clinical and radiographic evidence used in selecting teeth for treatment was also employed to determine success or failure of the pulpotomy treatment. Examples of variation in treatment outcome are presented in Figures 2 through 5.

Figure 2 shows a radiograph of a lower left first primary molar

with subtle evidence of bifurcation bone changes three months after the operation. Thickened periodontal membrane and decreased bone density in this area are noted.

Figure 3 depicts a sequela of vital pulpotomy therapy using calcium hydroxide. The distal root of the first primary molar exhibits internal resorption six weeks after treatment.

Figure 4 is a radiograph taken seven months post-operatively, of a molar which was accepted for treatment and shown previously in Figure 1. There is evidence of internal resorption and bone loss.

Figure 5 demonstrates a successful vital pulpotomy treatment in an upper first primary molar viewed one year post-operatively.

Distribution of treatment failures by diagnostic criteria is presented in Table I-A; these 25 failures represented 43 percent of the sample.

In the belief that many teeth exhibiting internal resorption are clinically successful (18), an alternate set of criteria was used to evaluate the data previously presented in order to rule out this bias. Teeth showing radiographic evidence of internal resorption were changed from failure to success category. The resulting distribution is shown in Table I-B. The 20 failures represented 34 percent of the sample. The data in Tables I-A and I-B are not suitable for statistical evaluation.

The sample was evaluated to determine if any clinical factors, such as age, sex, location of treated teeth, or extent of restoration had influenced the treatment outcome. Using the Chi square criterion, the factors were found not to be significant in their effect on the

treatment outcome (Table II-A). The same data were tested using internal resorption as a criterion of success instead of failure in order to rule out bias (Table II-B). The result failed to demonstrate significant difference in outcome when internal resorption was used as a criterion of success.

Histological treatment of the sample: During treatment the coronal portion of the pulp was removed with a spoon excavator and placed in a solution of ten percent formalin. The specimens were imbedded in paraffin, sectioned at six microns, and stained with Hematoxylin and Eosin as well as by the Brown and Brenn modification of the Gram stain for bacteria in tissues.

Qualitative evaluation of the dental pulp pathology: Qualitative evaluations of the tissue sections were recorded on an analysis form (Figure 6). A scale of 0 (zero or none), 1+ (mild), 2+ (moderate), or 3+ (severe) was used to judge the following characteristics: (a) new hemorrhage, "laking" of red blood cells; (b) disintegrated red blood cells and/or hemosiderin deposits; (c) degree and prevalence of engorged and dilated blood vessels; and (d) inflammation (relative amounts of cellular elements in the tissue).

The numerous granulocytes often seen in a meshwork of fibrin were considered a result of surgical hemorrhage and not included in the analysis.

Seven non-carious primary teeth were removed (for orthodontic reasons) and sectioned for histologic evaluation in order to serve as controls for the qualitative evaluation. Figures 7, 8 and 9 represent typical sections examined. Figures 10 through 20 are

photomicrographs of tissue sections representative of various qualitative evaluations.

Significance of the differences in the treatment outcome in relation to the qualitative measures was tested using the Chi-square criterion. The investigator's ability to make qualitative judgments was tested and the coefficient of consistency was considered satisfactory.*

Quantitative evaluation of dental pulp pathology: The number and relative proportions of white blood cells are assumed to be related to severity of pulp tissue change (64, 77). In order to assess these changes, a quantitative system was devised using the actual count of 1) acute inflammatory cells (neutrophils, eosinophils and basophils) and 2) chronic inflammatory cells (small and large lymphocytes, plasma cells, monocytes and macrophages) within a standard area. A representative tissue section was selected from the 12 to 16 Hemotoxylin and Eosin stained sections of each pulp specimen after scanning with low (approx. 50X) and high (approx. 430X) magnification.

An arc-microprojector was first used to project the sections to facilitate cell identification and count. Despite enlargement, cells were difficult to differentiate, particularly fibrocytes (in cross section), lymphocytes, and plasma cells. The need to record the cell image became apparent.

A mirror-prism was then adjusted to project the slide material

* Lu, K.H., Personal communication, May 23, 1962.

on a paper placed next to the microscope. Gross sections were outlined at low power (approx. 50X) and representative sites of inflammation magnified about 430X. White blood cells were outlined, but identification of cell types again became a problem. Attempts to verify the identity of recorded cells through the ocular eyepiece required such drastic changes in the mirror-prism system that it was virtually impossible to reproduce the original location and clarity of the image.

The method finally adopted was to view each section through a camera lucida attachment in order to record a low power outline of the tissue on graph paper (Figure 21). Areas considered most representative of the inflammatory tissues were recorded at high power on another paper (Figure 22). Outlines of the white blood cells and landmarks were recorded and verified by alternate use of the camera lucida and ocular eyepiece.

By employing a grid in the ocular eyepiece with the same degree of magnification, the same area was recorded for each specimen. Exact location of each camera lucida recording was noted using the field-finder coordinates. At low power the camera lucida produced an outline of about 67X magnification (Figure 21); using a 43X lens and 10X eyepiece resulted in an enlargement of about 577 times (Figure 22).

Relationship of the number of acute and chronic inflammatory cells to success or failure of treatment was evaluated by "discriminant analysis." Certain weights were applied to factors related to success or failure and all possible combinations of cell counts that

might indicate a significant relationship to the healing response were tested.

The relationship of the number of inflammatory cells associated with each of the three degrees of degeneration categories was tested using the Chi square criterion in a Poisson type distribution.

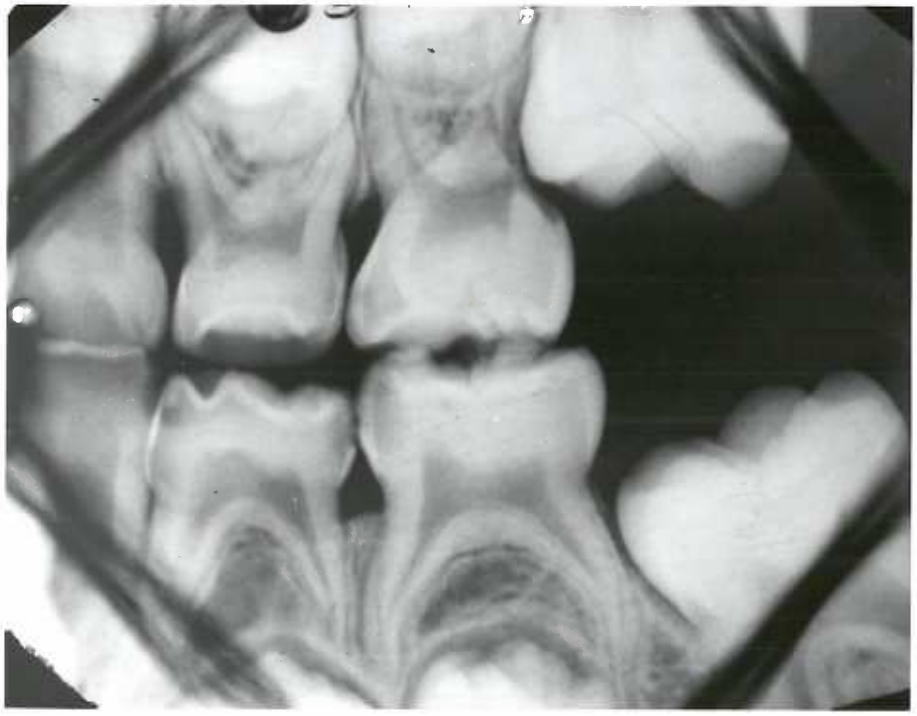

Figure 1

Lower second primary molar evaluated as

"normal" prior to treatment.

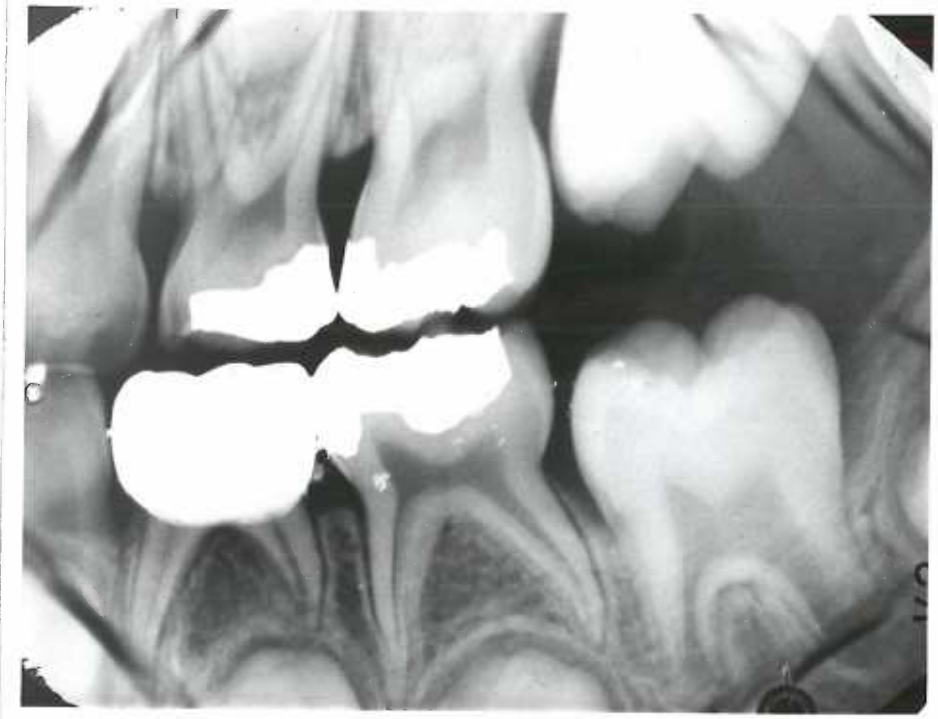

Figure 2

Lower first primary molar three months after treatment. Subtle bifurcation bone changes.

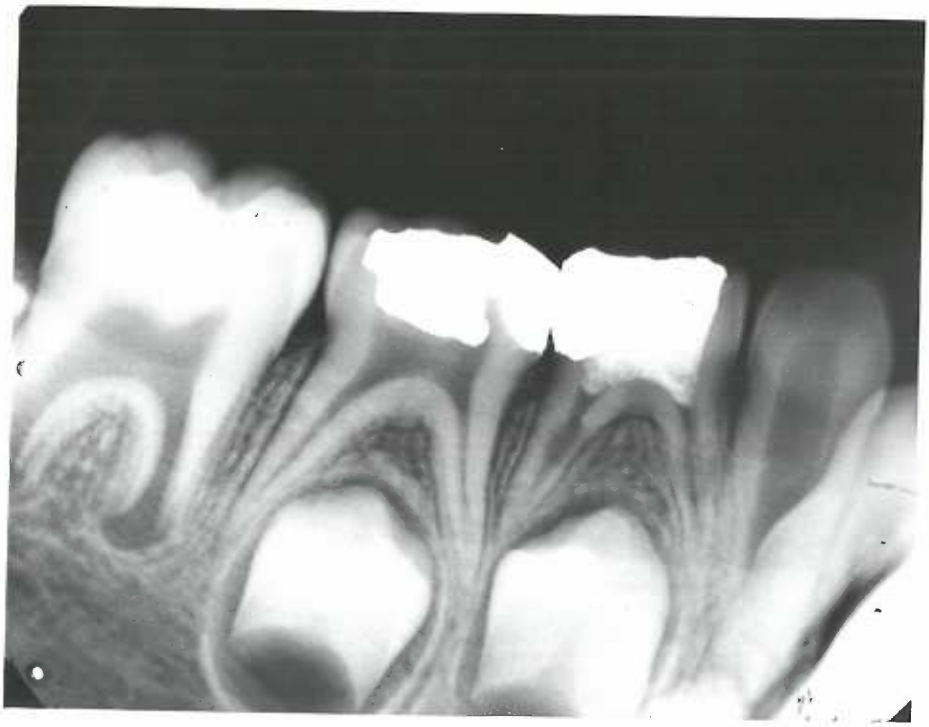

Figure 3

Lower first primary molar six weeks after treatment. Internal resorption of distal root.

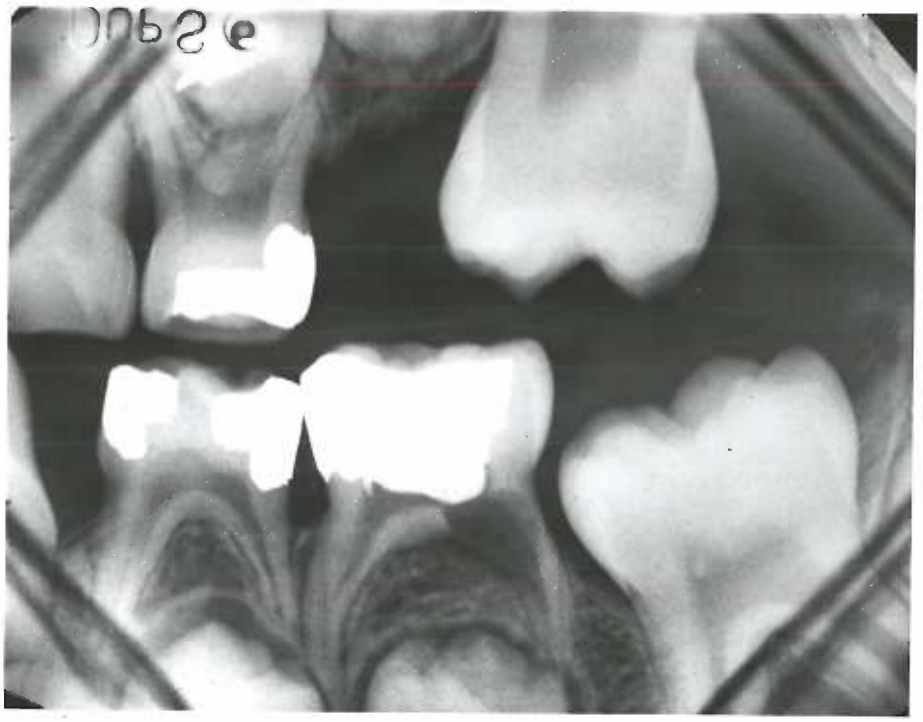

Figure 4

Lower second primary molar. Bone loss and internal resorption. Seven months after treatment.

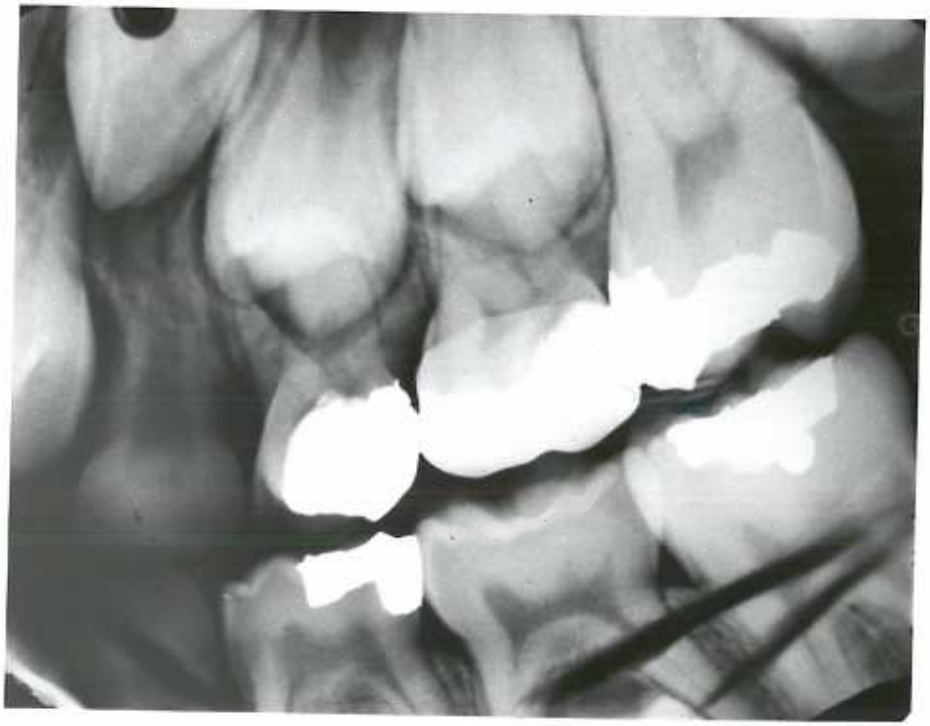

Figure 5

Upper first primary molar. Successful vital pulpotomy one year after treatment.

Table I-A

DISTRIBUTION OF RADIOGRAPHIC DIAGNOSIS AND TIME OF RECOGNITION OF PULPOTOMY FAILURES

(Teeth with internal resorption alone included in failure category.)*

| Radiographic Diagnosis | Time of Radiographic Recognition in Mos. | | | | | Total Number Failures |
|---|---|---|---|---|---|---|
| | 0-3 | 3-6 | 6-9 | 9-12 | 12-24 | |
| Internal Resorption (alone) | 1 | 1 | - | 1 | 2 | 5 |
| Internal Resorption and Bone Loss | - | 1 | 3 | - | 3 | 7 |
| Bone Loss (alone) | 1 | 3 | - | 5 | 4 | 13 |
| Total | 2 | 5 | 3 | 6 | 9 | 25 |

*Represent 43 percent of the 58 treated teeth.

Table I-B

DISTRIBUTION OF RADIOGRAPHIC DIAGNOSIS AND TIME OF RECOGNITION OF PULPOTOMY FAILURES

(Teeth with internal resorption alone included in success category.)*

| Radiographic Diagnosis | Time of Radiographic Recognition in Months | | | | | Total Number Failures |
|---|---|---|---|---|---|---|
| | 0-3 | 3-6 | 6-9 | 9-12 | 12-24 | |
| Internal Resorption & Bone Loss | - | 1 | 3 | - | 4 | 8 |
| Bone Loss (alone) | 1 | 3 | - | 4 | 4 | 12 |
| Total | 1 | 4 | 3 | 4 | 8 | 20 |

*Represents 34.48 percent of the 58 treated teeth.

Table II-A

TREATMENT OUTCOME IN RELATION TO UNCONTROLLED CLINICAL FACTORS

(Teeth with internal resorption alone included in failure category)

| Clinical Factors | Treatment Outcome | | Chi-square Value and Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|
| | Success | Failure | | |
| Primary Molar: | | | | |
| First . . . . . | 9 | 10 | | |
| vs | | | 1.018 | none |
| Second. . . . . | 24 | 15 | (1df) | |
| Arch Location: | | | | |
| Maxillary . . . . | 16 | 10 | | |
| vs | | | 0.411 | none |
| Mandibular . . . | 17 | 15 | (1df) | |
| Molar Location: | | | | |
| Right 1 . . . . . | 13 | 13 | | |
| vs | | | 1.349 | none |
| Left . . . . . | 20 | 12 | (1df) | |
| Age at Treatment: | | | | |
| 3-6 years . . . . | 21 | 16 | | |
| vs | | | 0.001 | none |
| 6-9 years . . . . | 12 | 9 | (1df) | |
| Gender: | | | | |
| Male . . . . . | 20 | 14 | | |
| vs | | | 0.124 | none |
| Female . . . . . | 13 | 11 | (1df) | |
| Surfaces Restored: | | | | |
| Two or less . . . | 16 | 12 | | |
| vs | | | 0.000 | none |
| Three or more . . | 16 | 12 | (1df) | |

Table II-B

TREATMENT OUTCOME IN RELATION TO UNCONTROLLED CLINICAL FACTORS

(Teeth with internal resorption alone included in success category)

| Clinical Factors | Treatment Outcome | | Chi-square Value & Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|
| | Success | Failure | | |
| Primary Molar: | | | | |
| First . . . . . | 10 | 9 | | |
| vs | | | 2.0767 | none |
| Second . . . . | 28 | 11 | (1df) | |
| Arch Location: | | | | |
| Maxillary . . . | 18 | 8 | | |
| vs | | | 0.2876 | none |
| Mandibular . . | 20 | 12 | (1df) | |
| Molar Location: | | | | |
| Right . . . . . | 16 | 10 | | |
| vs | | | 0.6231 | none |
| Left . . . . . | 22 | 10 | (1df) | |
| Age at Treatment: | | | | |
| 3-6 years . . . | 26 | 11 | | |
| vs | | | 1.0218 | none |
| 6-9 years . . . | 12 | 9 | (1df) | |
| Gender: | | | | |
| Male . . . . . | 22 | 12 | | |
| vs | | | 0.0239 | none |
| Female . . . . | 16 | 8 | (1df) | |
| Surfaces Restored: | | | | |
| Two or less . . | 19 | 10 | | |
| vs | | | 0.0084 | none |
| Three or more . | 18 | 9 | (1df) | |

Findings

DENTIN: Carious ☐ Non-carious ☐

PULP: Old hemorrhage ☐ New hemorrhage ☐
Dilated blood vessels ☐
Engorged blood vessels ☐

INFLAMMATION: Polys, numerous ☐
few ☐
scattered ☐
concentrated ☐

Lymphocytes, Numerous ☐
Plasma cells, few ☐
Macrophages scattered ☐
concentrated ☐

DEGENERATION: Nucleus fragmentation ☐
Cellular fragmentation ☐

Granular appearance ☐
Homogenous masses (necrosis) ☐

BACTERIA:
Peripheral: value = 1 if this is only distribution.
Intracellular: single ☐ colonies ☐
Extracellular: single ☐ colonies ☐

Cocci ☐ Rods ☐ Vibrio ☐ Filamentous ☐
None ☐

Name Tooth Slide # Chart #

Figure 6

Analysis form for qualitative evaluation
of pulp tissue sections.

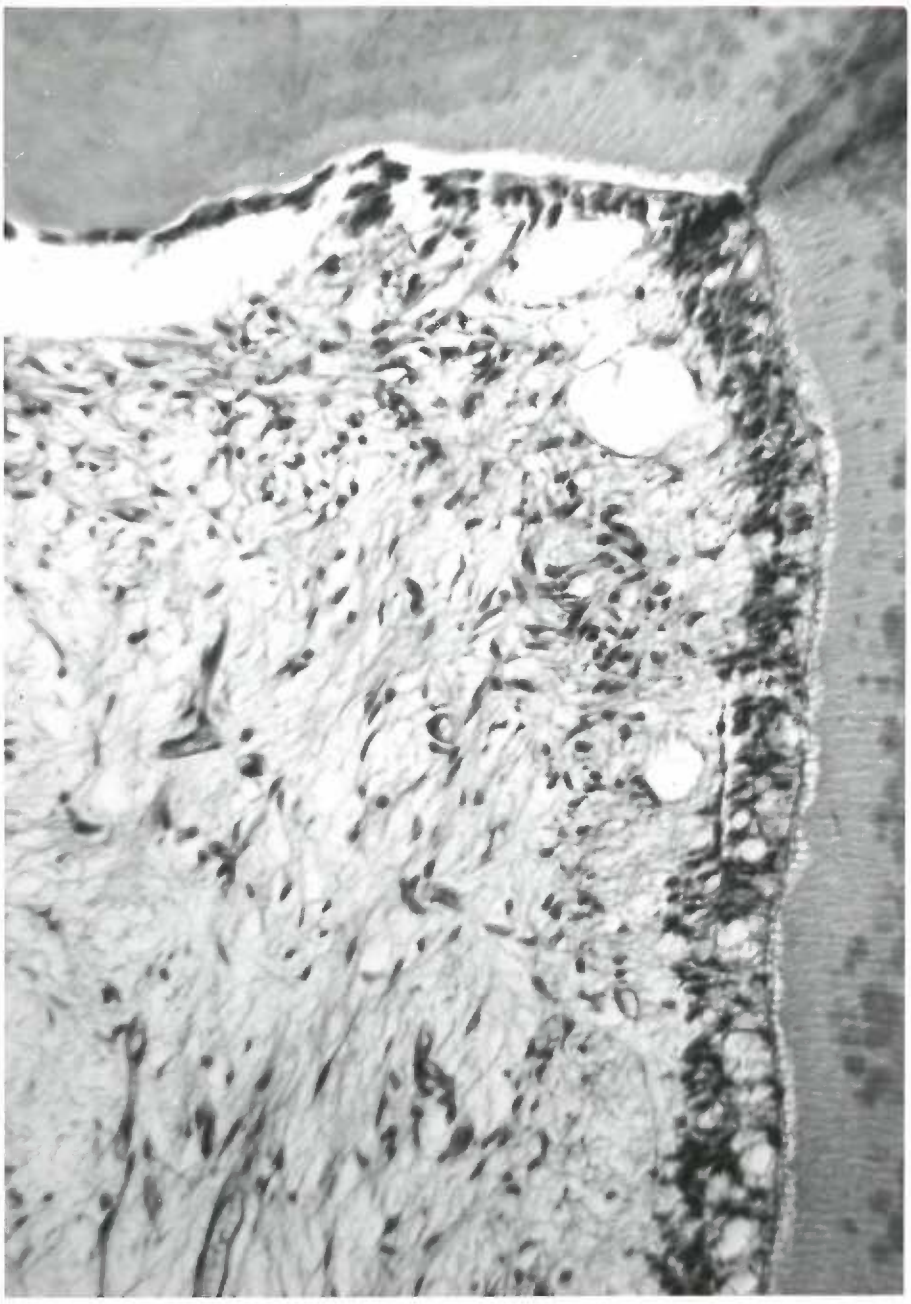

Figure 7

Control #1. Extracted primary cuspid (coronal portion). Normal young type connective tissue. (Approx. 106X)

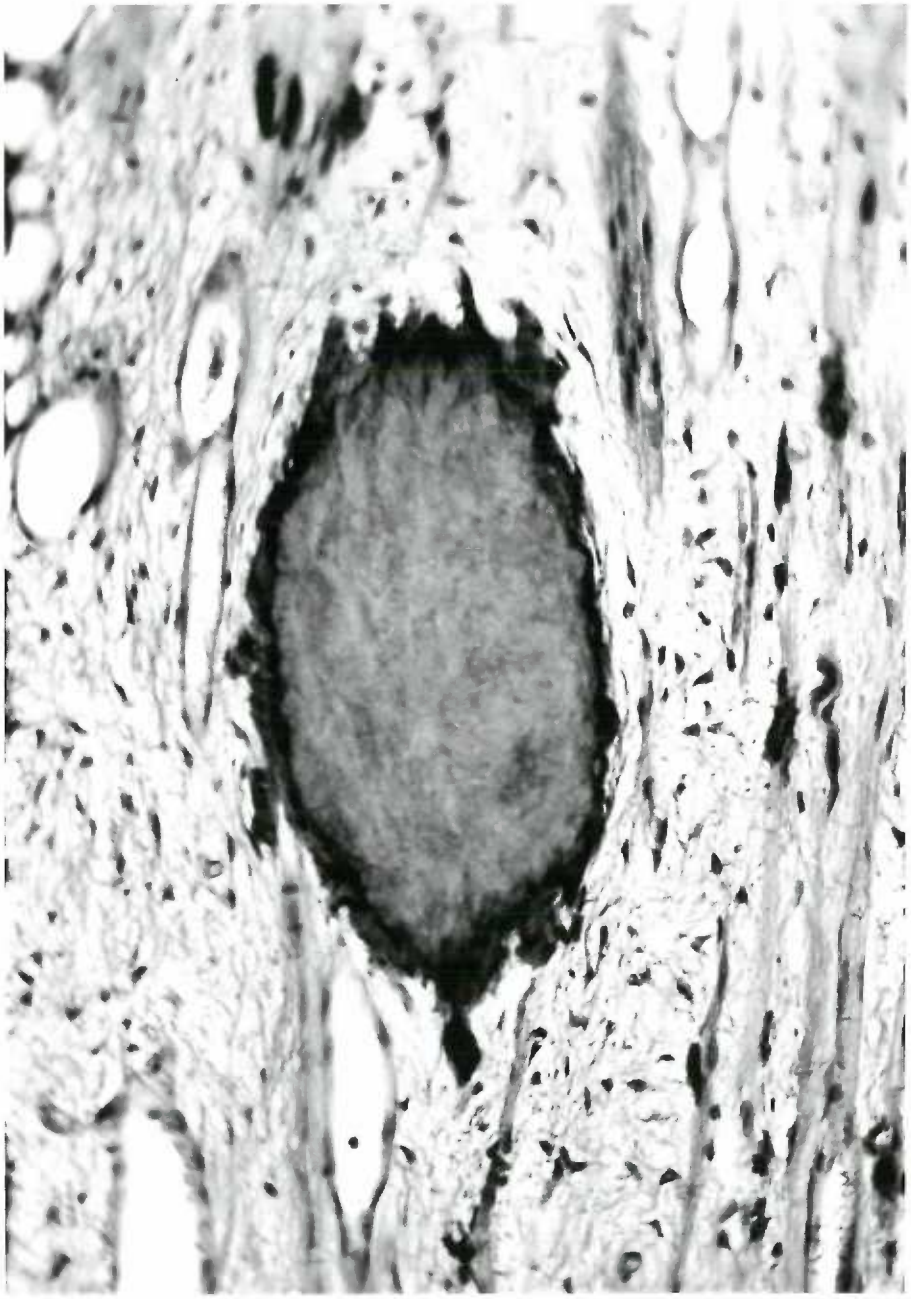

Figure 8

Control #5. Extracted primary cuspid. Radicular portion showing normal young type connective tissue surrounding large pulp stone and numerous dilated blood vessels. (Approx. 204X)

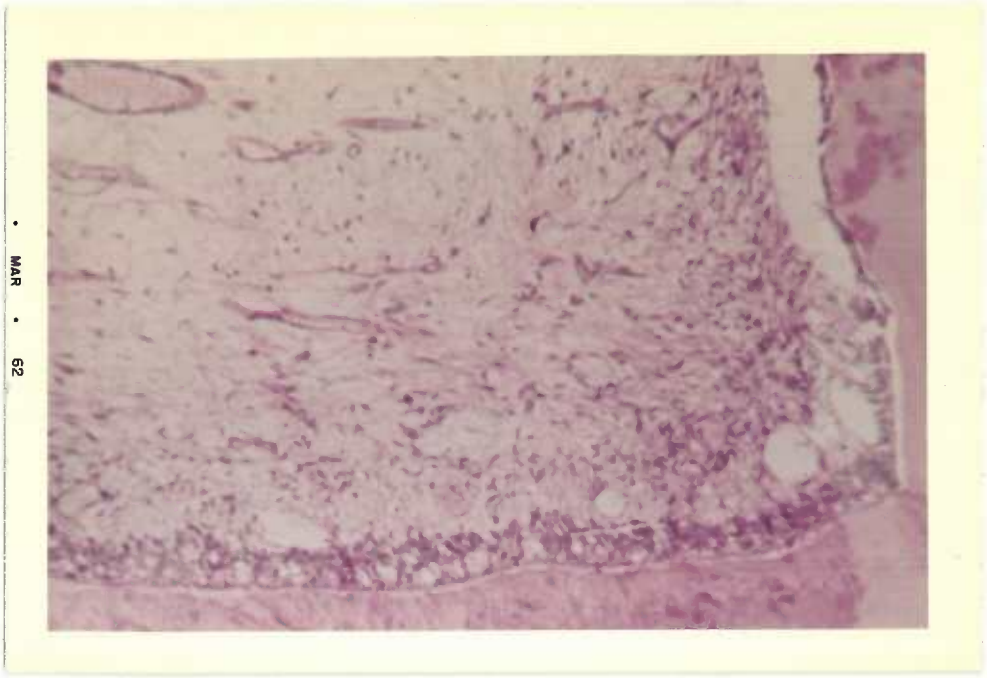


Figure 9.

Control #1. Extracted primary cuspid. Normal young type connective tissue. (Approx. 47X)

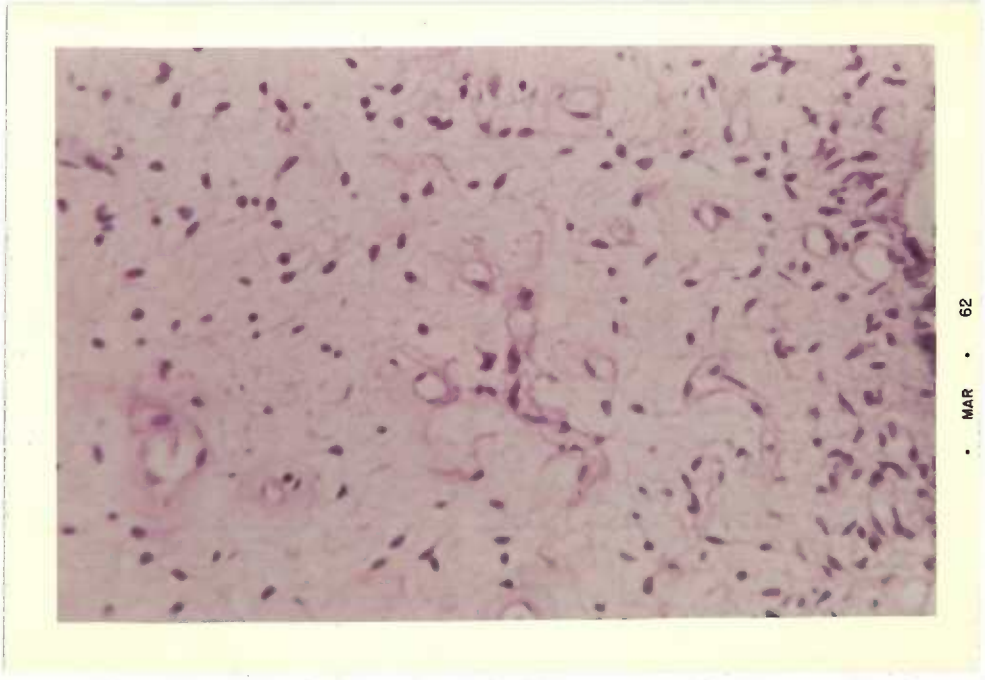


Figure 10

#60-177-1. Normal young type connective tissue. (Approx. 112X)

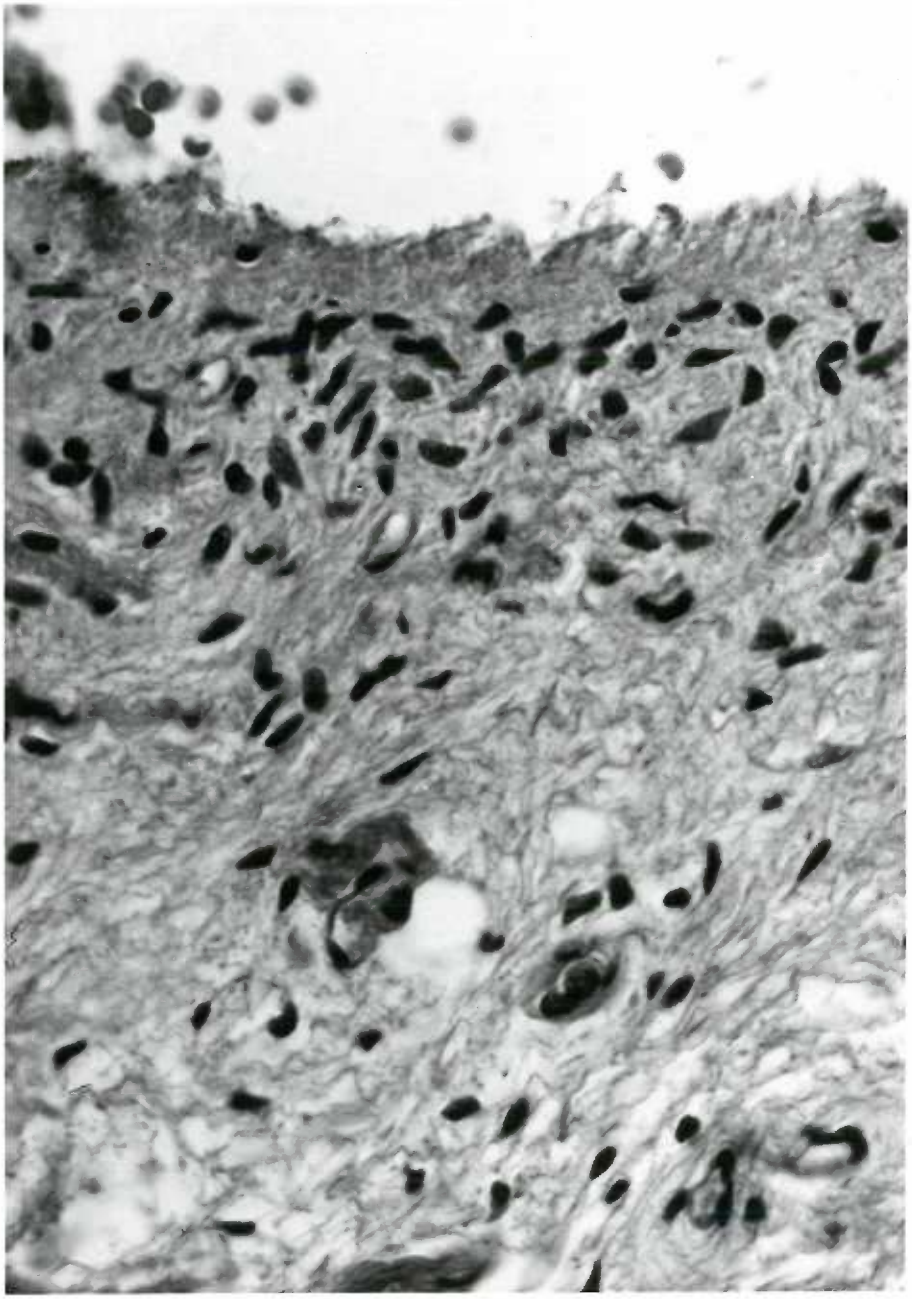

Figure 11

#59-178B. An example of "normal" pulp tissue. Dense fibrous connective tissue. No inflammation. Little or no degeneration. (Approx. 204X)

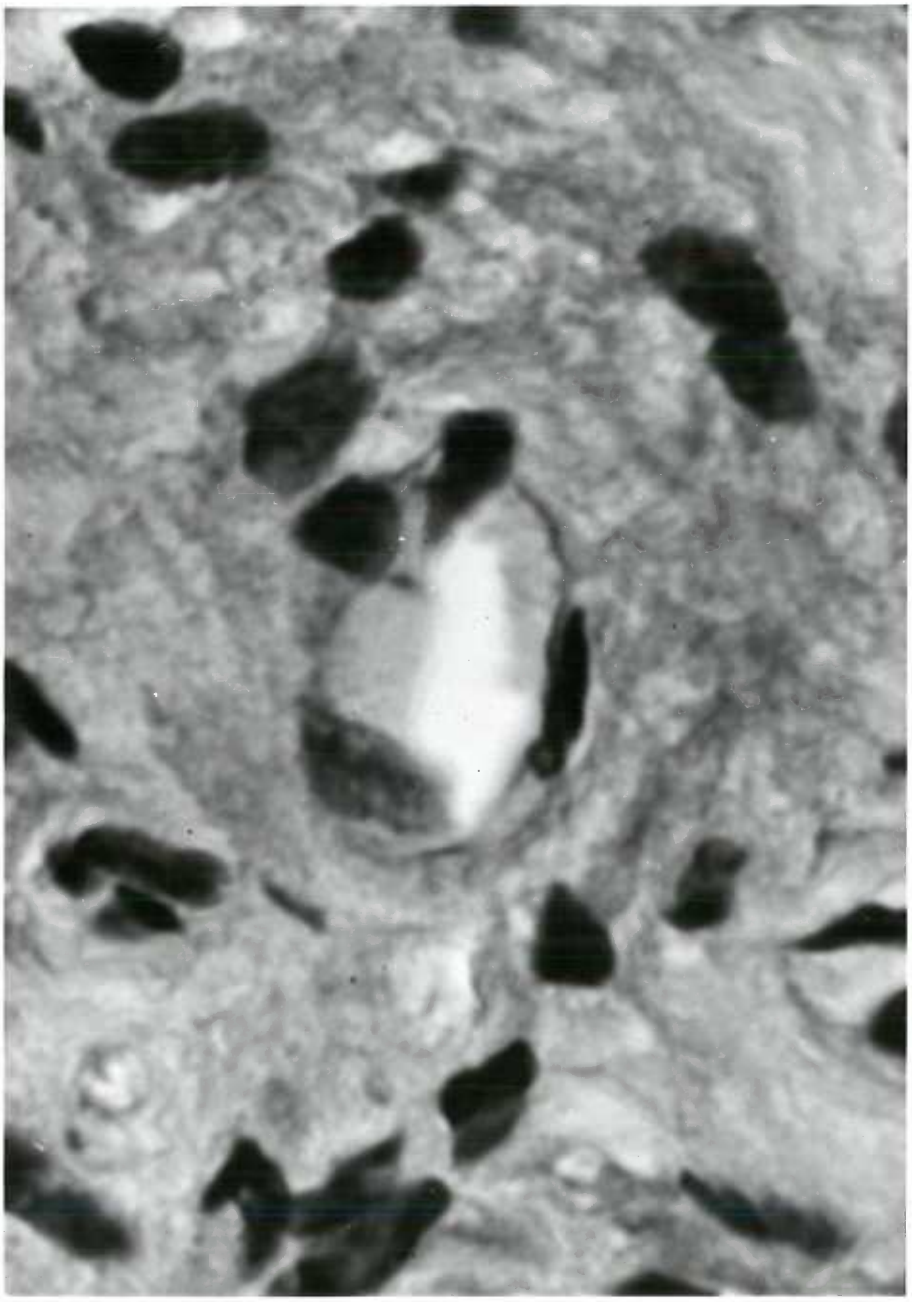

Figure 12

#60-176-1
Inflammation: 0
Degeneration: 1+
- granular cytoplasm
(Approx. 770X)

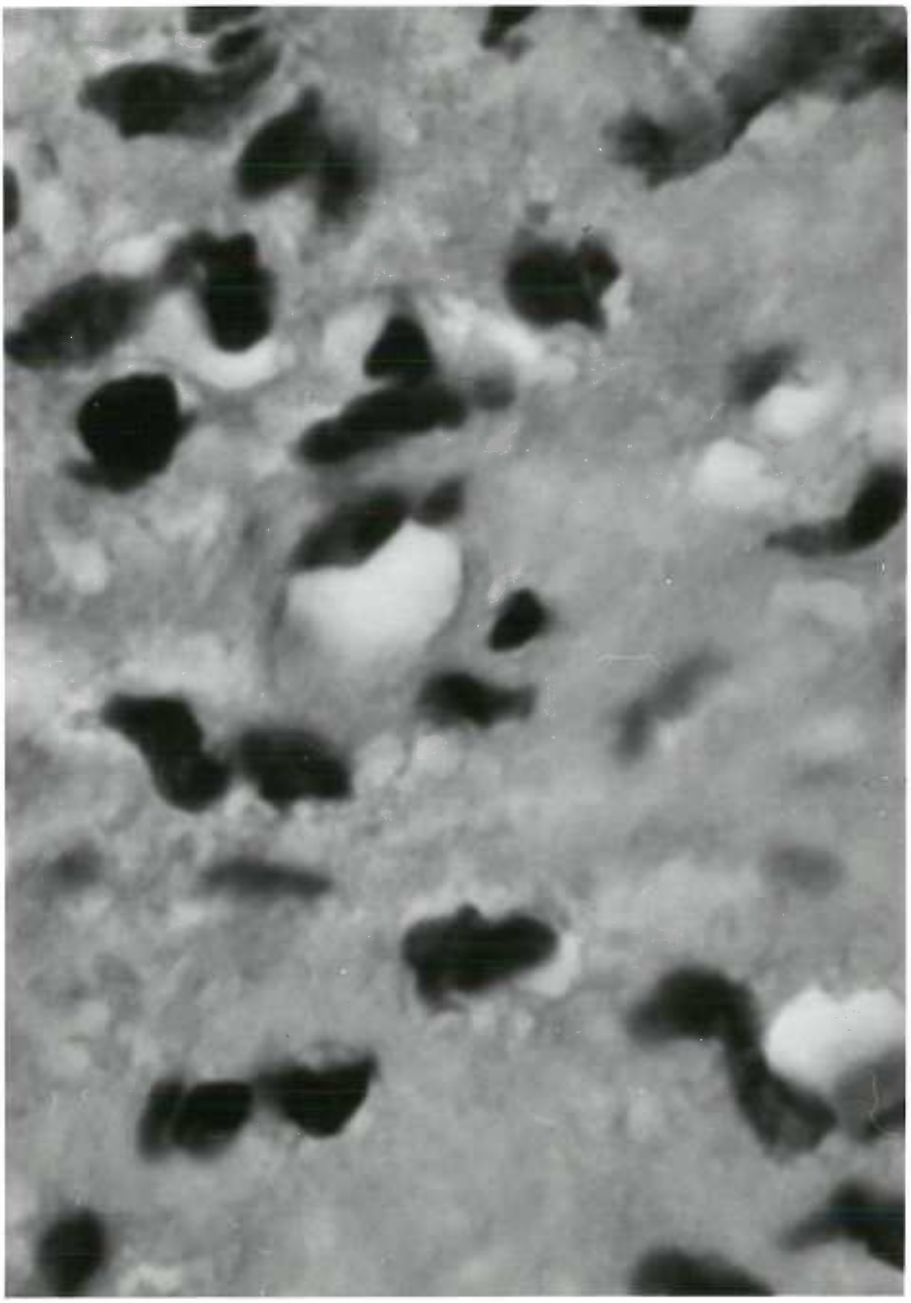

Figure 13

#60-103B-1
Inflammation: 0
Degeneration: 2+
- granular cytoplasm
- some karyorrhexis
(Approx. 770X)

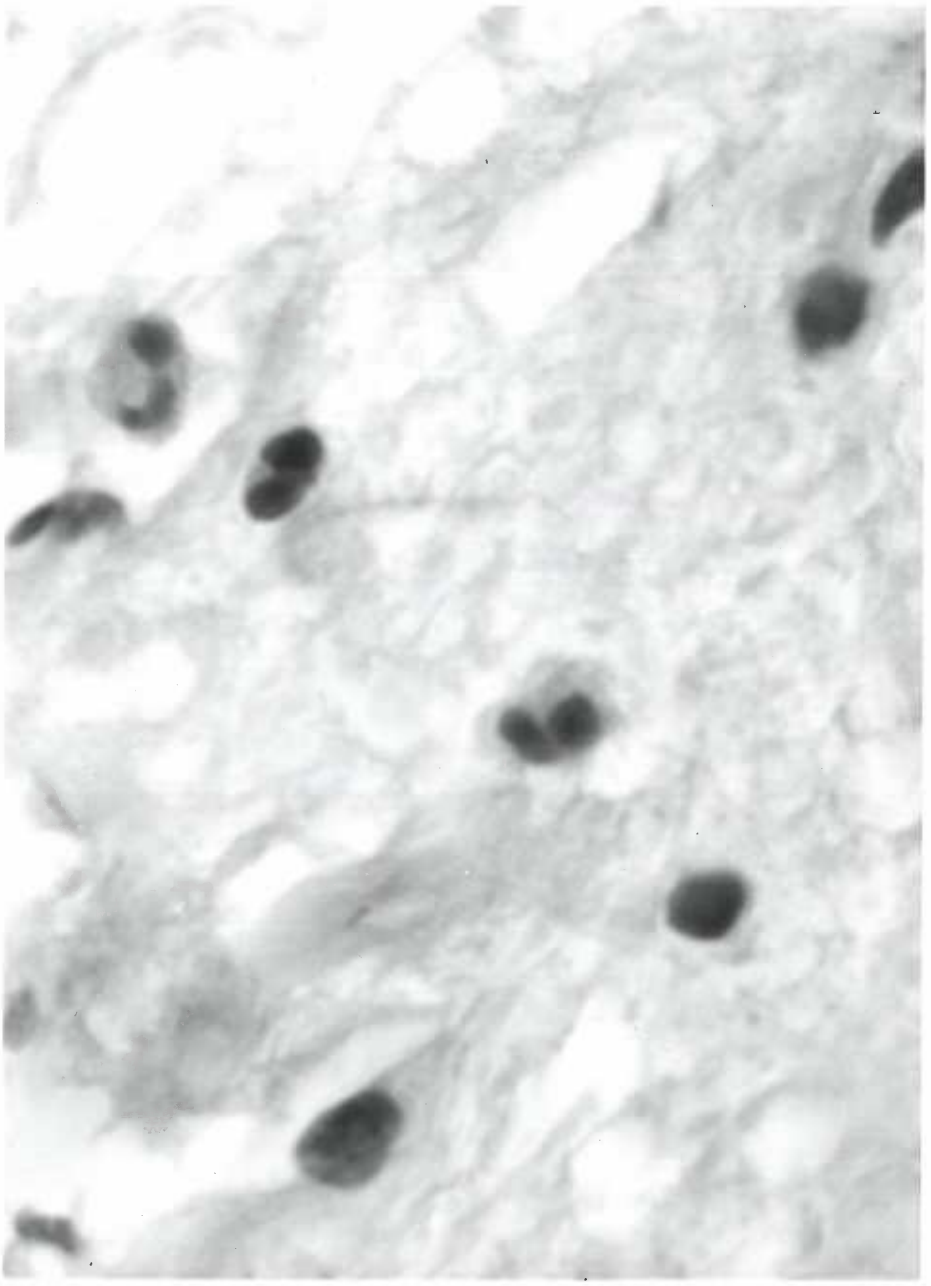

Figure 14

#60-128-1
Inflammation, subacute: 1+
- scattered neutrophils and lymphocytes
Degeneration: 1+
(Approx. 315X)

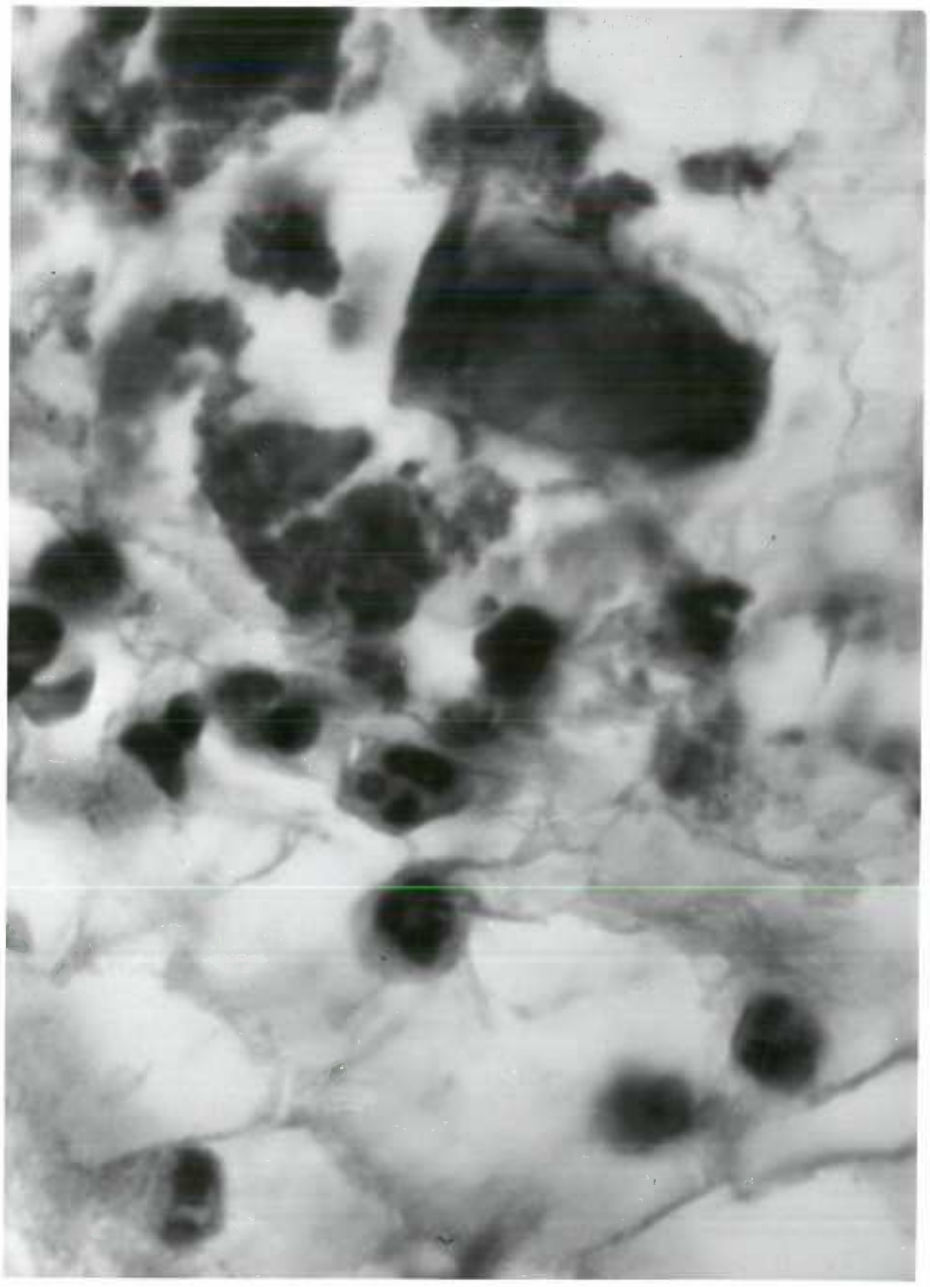

Figure 15

#60-130-1
Inflammation, subacute: 1+
Degeneration: 2+
(Approx. 520X)

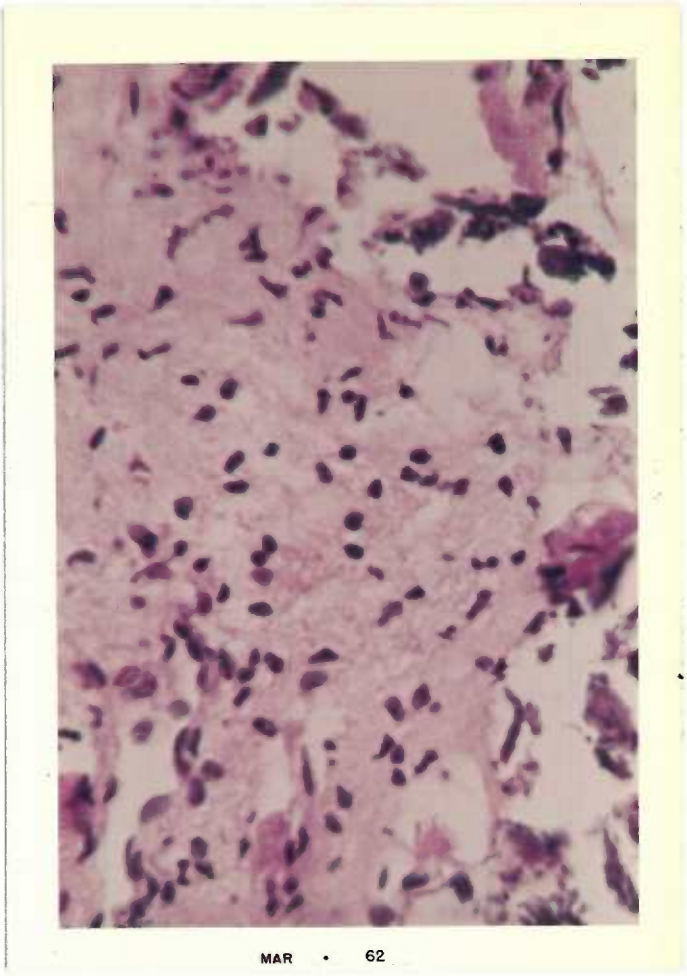


Figure 16-a

#60-1-1
Inflammation, chronic: 1+
Degeneration: 1+
(Approx. 136X)

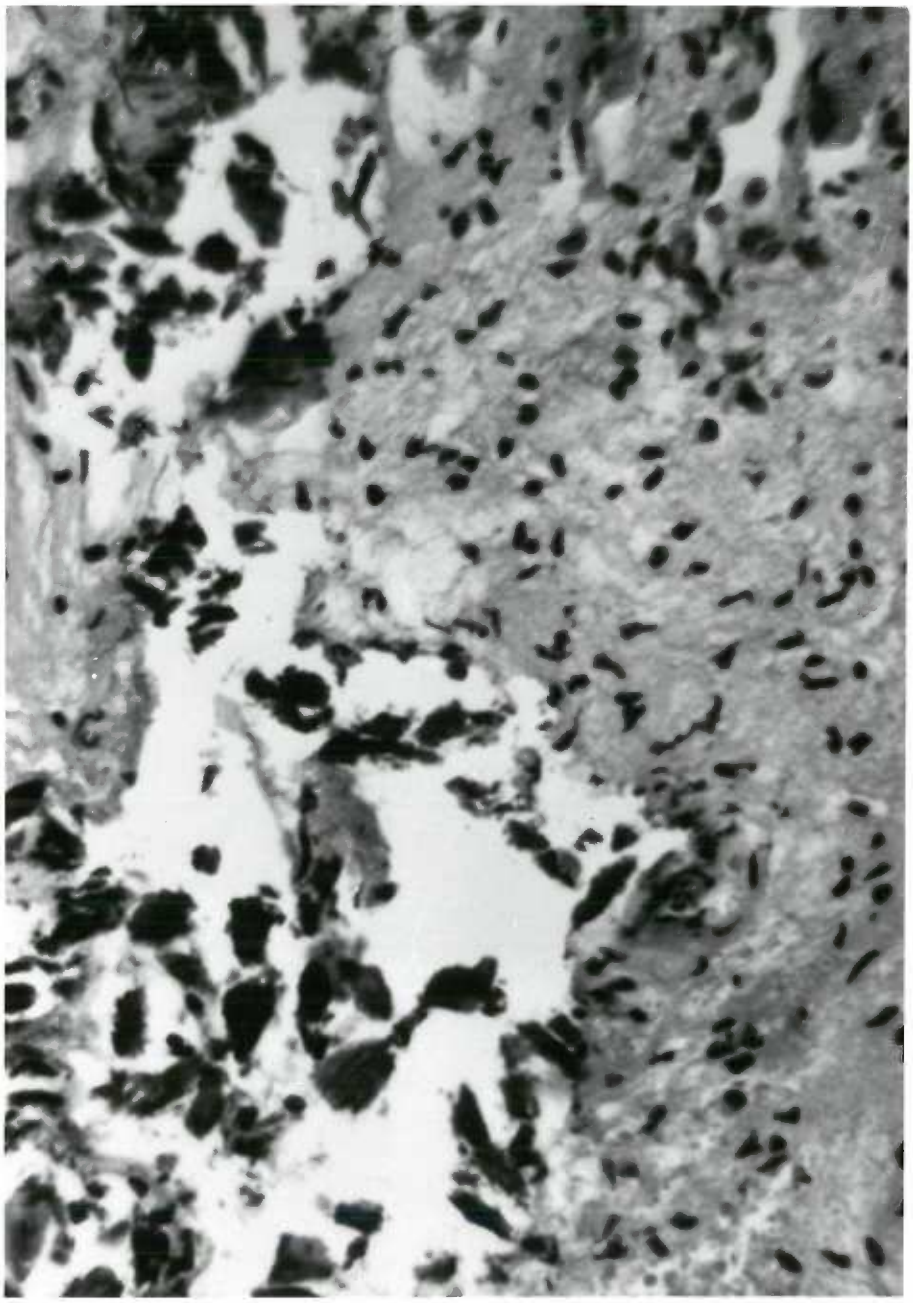

Figure 16-b

#60-1
Inflammation, chronic: 1+
- scattered lymphocytes
Degeneration: 1+
(Approx. 315X)

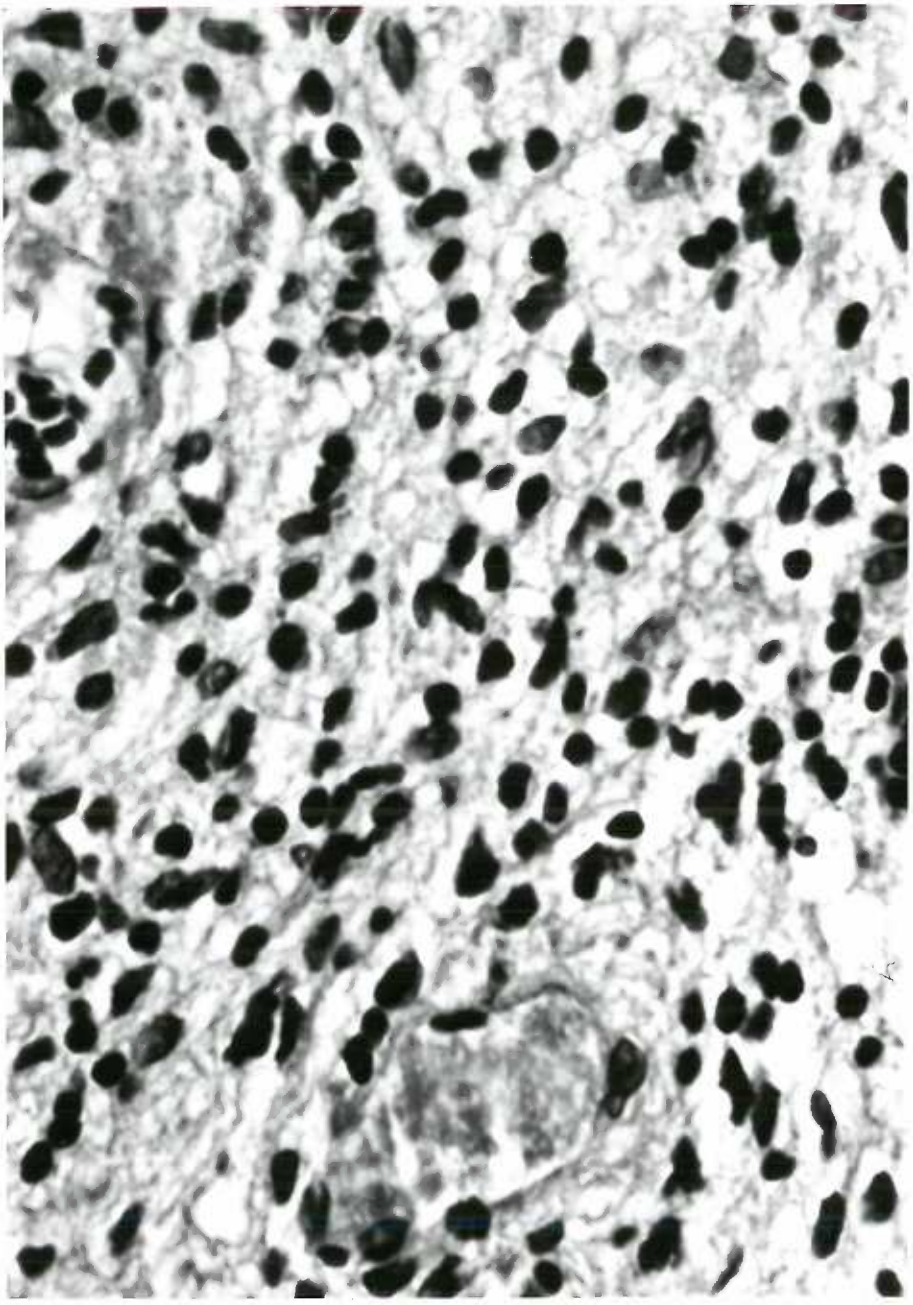

Figure 17

#59-147-1
Inflammation, chronic: 2+
Degeneration: 2+
(Approx. 315X)

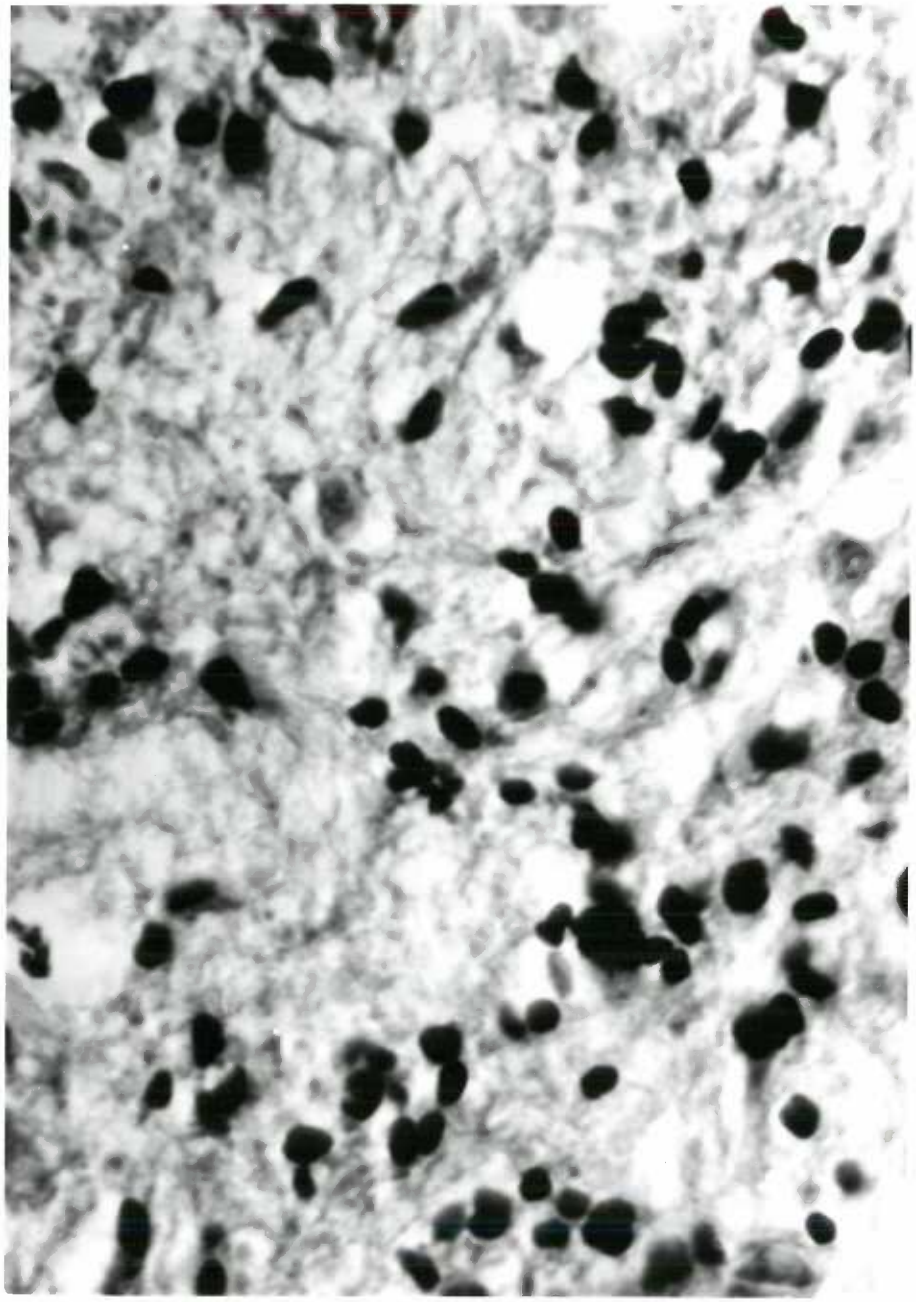

Figure 18

#59-237-1
Inflammation, chronic: 2+
Degeneration: 3+

(Approx. 315X)

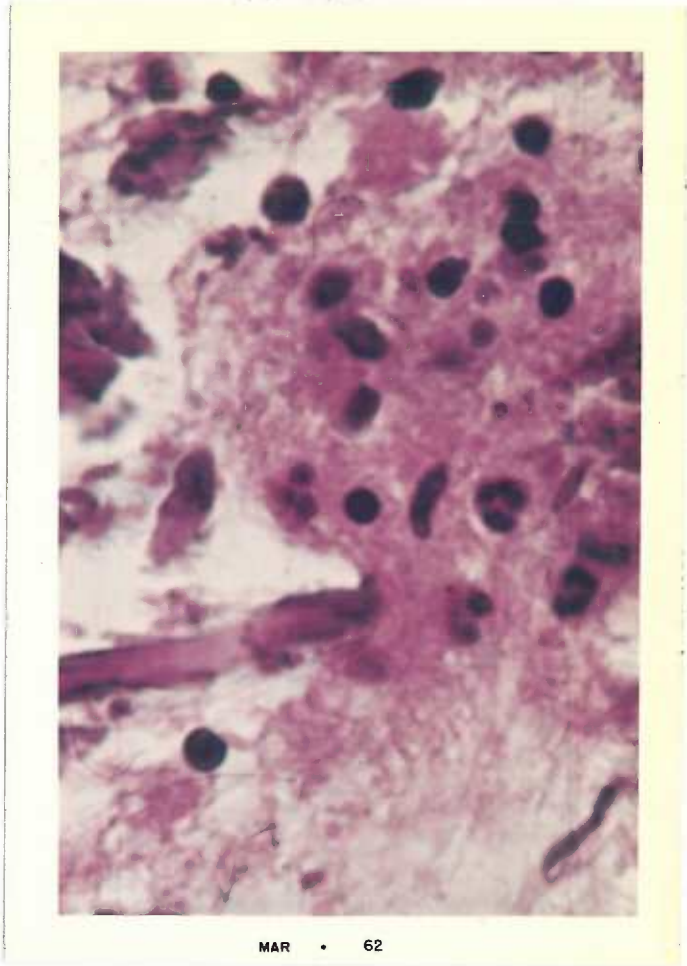


Figure 19-a

#60-18-1
Inflammation, subacute: 2+
Degeneration: 3+
- homogeneous pink connective tissue
- disintegrated fibrocytes
(Approx. 310X)

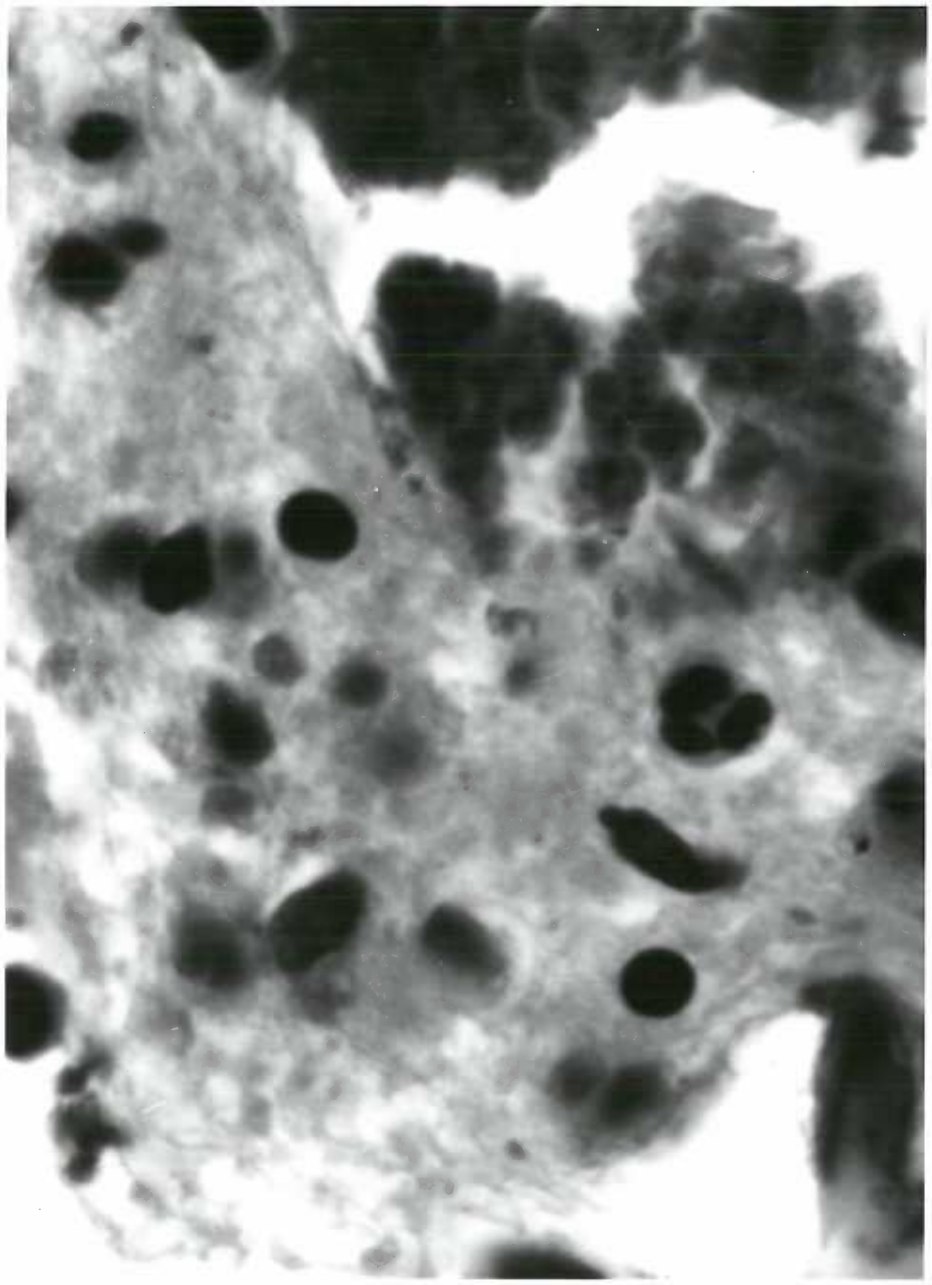

Figure 19-b

#60-18-1
Inflammation, subacute: 2+
 - moderately heavy scattering
   of neutrophils and lymphocytes
Degeneration: 3+
(Approx. 520X)

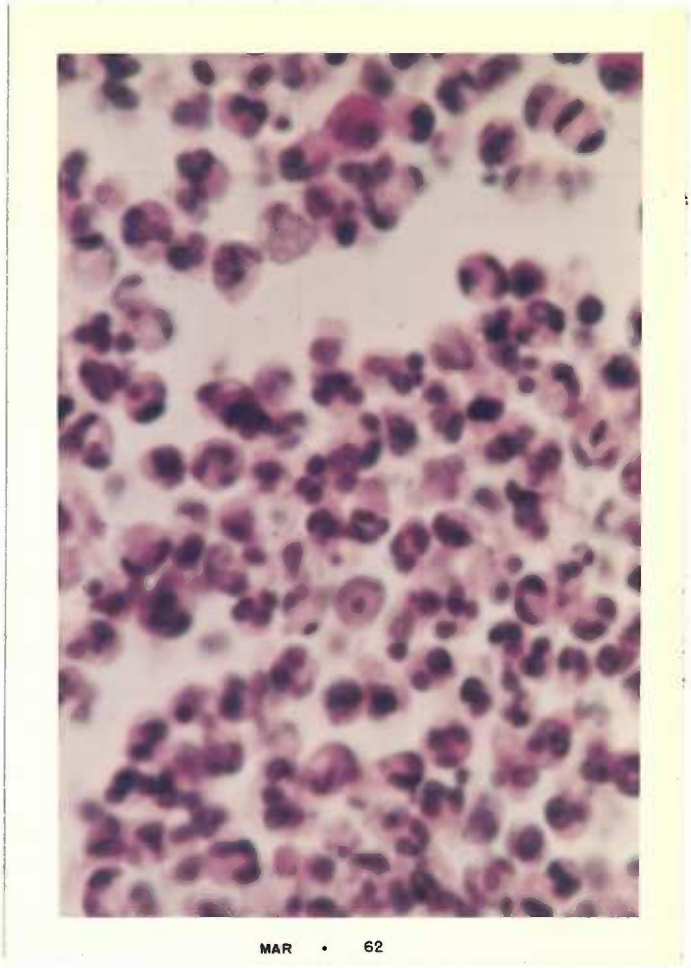


Figure 20-a

#60-38
Inflammation, acute: 3+
Degeneration: 3+
(Approx. 310X)

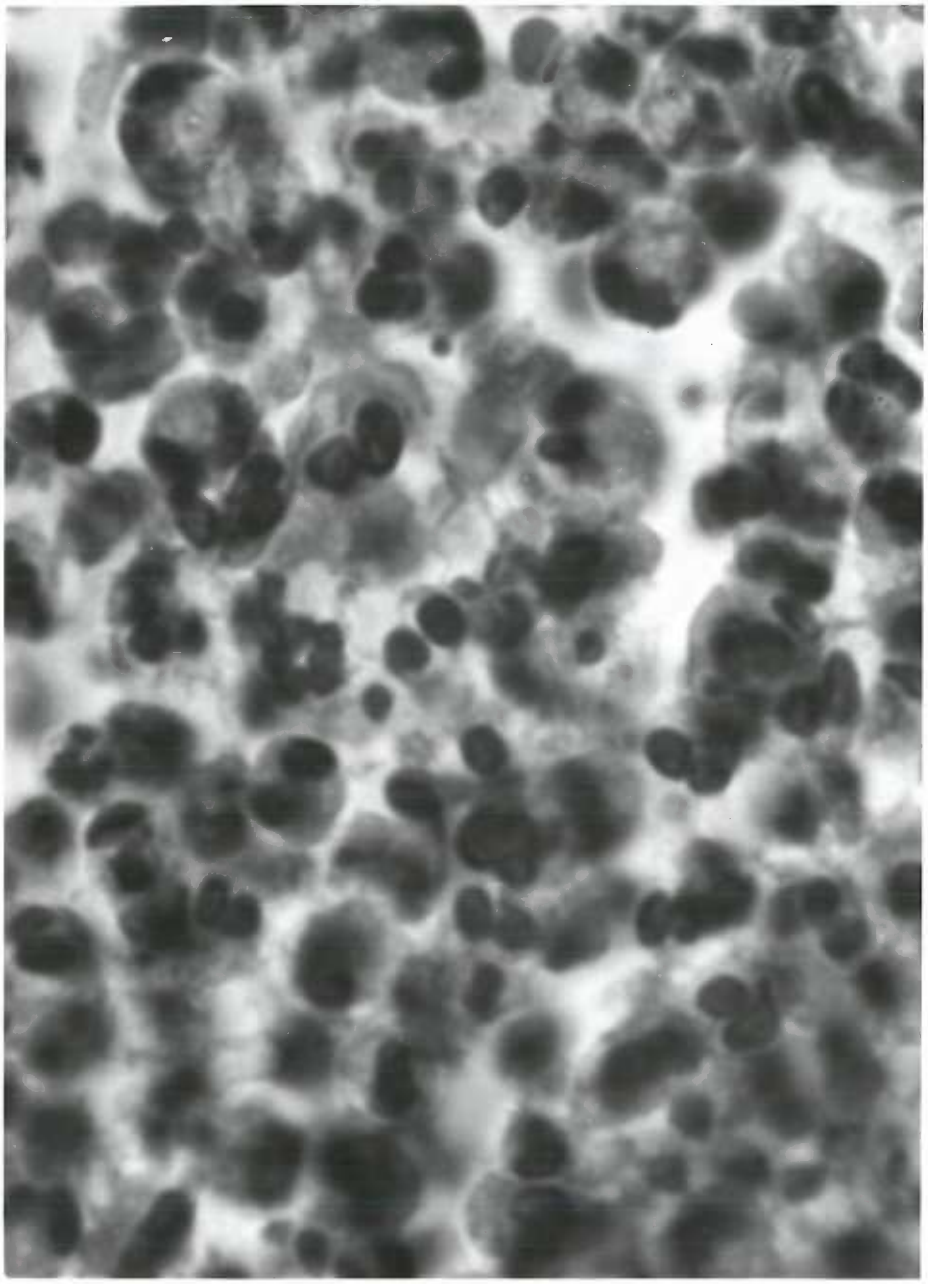

Figure 20-b

#60-38-1
Inflammation, acute: 3+
Degeneration: 3+
(Approx. 520X)

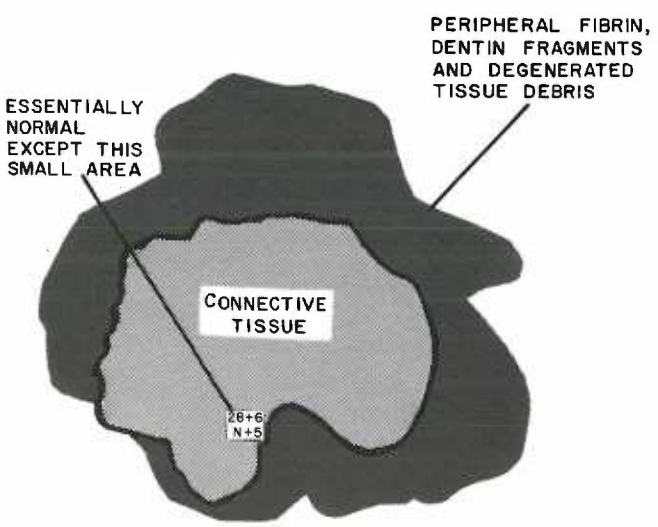


Figure 21

Low power outline (one-half scale) of pulp tissue section obtained by means of the camera lucida attachment to the microscope.

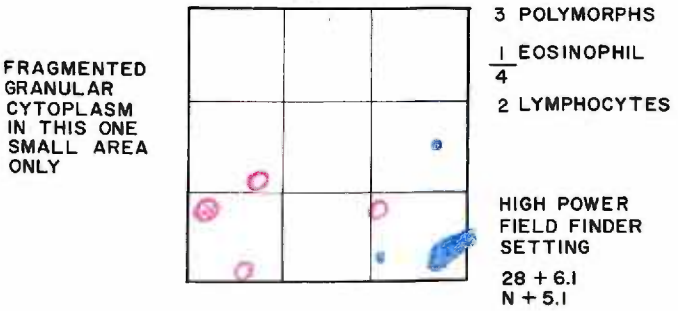


Figure 22

Camera lucida high power outline (one-half scale) of area of inflammation. Small blue markings represent lymphocytes. The large blue marking is a dentin fragment. Polymorphonuclear leucocytes are represented by red circles; neutrophils, small; eosinophils, large.

# FINDINGS

The relationship of the qualitative factors (new and old hemorrhage, dilated and engorged blood vessels, bacterial invasion, and degree of degeneration) to success and failure of the vital pulpotomy treatment is shown in Table III-A with internal resorption as a criterion of failure, and in Table III-B with internal resorption included in the success category. Using the Chi square criterion, the presence or absence of each of the qualitative factors, evaluated in the tissue sections, was shown to bear no significant relationship to success or failure of treatment.

The schematic distribution curves, Figure 2, illustrate the distribution of data relating the number of acute and chronic inflammatory cells in samples of coronal pulp to success or failure of vital pulpotomy treatment.

One curve represents the distribution of cell counts (either acute or chronic inflammatory cells or their combinations) in tissue sections from teeth which exhibited successful treatments. Similarly the other curve represents the distribution of cell counts in the failure group. The midpoints of the distribution curves, the means, are in close proximity to each other with severe overlapping. The horizontal solid line labelled "DM" represents the difference of the two means. The two horizontal dotted lines labelled SEM represent the standard error of the mean for success (S) and failure (F). The standard error of the means was shown to be greater than the difference of the means by analysis. This finding demonstrates that the distribution

of inflammatory cell counts in one group cannot be distinguished from the other.

Tables IV-A and IV-B present the relationship of the number of the acute and chronic inflammatory cells to the degree of degeneration of coronal pulp tissue. The differences between means indicate significant differences between the number of acute inflammatory cells in each of the categories of degeneration (Table IV-A). The mean number of chronic inflammatory cells of category 1+ (Table IV-B) is significantly different from those of 2+ and 3+. The number of chronic inflammatory cells in categories 2+ and 3+ do not differ significantly. The Chi square values for the acute and chronic inflammatory cells in each of the three categories of degeneration demonstrate that there is great lack of uniformity of cell counts within each category. This is easily visualized in the raw data in Table V in the appendix.

Figure 25 shows data about patients who have received more than one vital pulpotomy treatment. Six of the seven patients exhibit either all successful or all unsuccessful treatments.

Table III-A

TREATMENT OUTCOME IN RELATION TO QUALITATIVE EVALUATION OF CORONAL PULP TISSUE AT TIME OF TREATMENT

(Teeth with internal resorption included in failure category)

| Tissue Factors Evaluated | Treatment Outcome | | Chi-square Value & Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|
| | Success | Failure | | |
| New Hemorrhage | | | | |
| Absent. . . . . | 13 | 16 | | |
| vs | | | 2.7663 | none |
| Present . . . . . | 16 | 8 | (1df) | |
| Old Hemorrhage | | | | |
| Absent. . . . . | 20 | 16 | | |
| vs | | | 0.0549 | none |
| Present . . . . . | 10 | 7 | (1df) | |
| Dilated & Engorged Blood Vessels | | | | |
| Absent or Slight. . . | 18 | 18 | | |
| vs | | | 0.7767 | none |
| Moderate or Severe. . | 10 | 6 | (1df) | |
| Bacterial Invasion: | | | | |
| Absent or Peripheral. | 13 | 11 | | |
| vs | | | 0.5176 | none |
| Intra- and/or Extracellular | 21 | 12 | (1df) | |
| Degree of Degeneration | | | | |
| Mild (1+) . . . . . | 9 | 7 | | |
| vs | | | | |
| Moderate (2+) . . . . | 13 | 14 | 2.4977 | none |
| vs | | | (2df) | |
| Severe (3+) . . . . . | 11 | 4 | | |

Table III-B

TREATMENT OUTCOME IN RELATION TO QUALITATIVE EVALUATION OF CORONAL PULP TISSUE AT TIME OF TREATMENT

(Teeth with internal resorption included in success category)

| Tissue Factors Evaluated | Treatment Outcome | | Chi-square Value & Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|
| | Success | Failure | | |
| New Hemorrhage | | | | |
| Absent . . . . . | 17 | 12 | | |
| vs | | | 1.5708 | none |
| Present . . . . . | 18 | 6 | (1df) | |
| Old Hemorrhage | | | | |
| Absent . . . . . | 24 | 12 | | |
| vs | | | 0.0198 | none |
| Present . . . . . | 11 | 6 | (1df) | |
| Dilated & Engorged Blood Vessels | | | | |
| Absent or Slight. . . | 24 | 13 | | |
| vs | | | 0.3481 | none |
| Moderate or Severe. . | 11 | 4 | (1df) | |
| Bacterial Invasion | | | | |
| Absent or Peripheral. | 13 | 11 | | |
| vs | | | 2.1016 | none |
| Intra- and/or Extracellular . . . . | 24 | 9 | (1df) | |
| Degree of Degeneration . | | | | |
| Mild (1+) . . . . . | 11 | 5 | | |
| vs | | | | |
| Moderate (2+) . . . . | 17 | 11 | 0.5764 | none |
| vs | | | (2df) | |
| Severe (3+) . . . . . | 10 | 4 | | |

Table IV-A

RELATIONSHIP OF NUMBER OF ACUTE INFLAMMATORY CELLS

TO DEGREE OF DEGENERATION

| Degeneration | Total Acute Inflammatory Cells | Sample Size | Mean | Chi-square Value and Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|---|
| 1+ | 9 | 16 | 0.56 | 39.1741 (15df) | Highly |
| 2+ | 177 | 28 | 6.32 | 618.117 (27df) | Highly |
| 3+ | 229 | 14 | 16.36 | 863.644 (13df) | Highly |
| Total | 415 | 58 | | 1520.935 (55df) | Highly |
| | | Inter-category value for mean = | | 191.152 (2df) | Highly |

Table IV-B

RELATIONSHIP OF NUMBER OF CHRONIC INFLAMMATORY CELLS

TO DEGREE OF DEGENERATION

| Degeneration | Total Chronic Inflammatory Cells | Sample Size | Mean | Chi-square Value and Degrees Freedom | Significance of Difference 5% Level |
|---|---|---|---|---|---|
| 1+ | 33 | 16 | 2.0625 | 64.9697 (15df) | High |
| 2+ | 239 | 28 | 8.5357 | 260.8997 (27df) | High |
| 3+ | 107 | 14 | 7.6429 | 235.1495 (13df) | High |
| Total | 379 | 58 | | 561.0189 (55df) | High |
| | | Inter-category value for mean = | | 172.3905 (2df) | High |

# DISCUSSION

In evaluating the healing response of amputated pulp tissue treated with calcium hydroxide, the interaction of multiple variables as well as the reliability of methods used must be considered. Much is known about the effect of calcium hydroxide on amputated "normal" pulp tissue (11, 31, 65, 71, 88). However, in tissue which is not "normal" the response to this medicament is a matter of speculation. Furthermore, status of the radicular portion of the pulp is not known when cariously involved coronal tissue is removed prior to using calcium hydroxide. One of the purposes of this study was to test the relationship of the coronal pulp tissue to the healing response of the remaining radicular pulp tissue.

The course of pulp healing under calcium hydroxide in "normal" human teeth has been followed for various intervals. Glass and Zander (32) examined 40 pulps from one day to 12 months after treatment. In a study of 77 permanent teeth Nyborg (64) evaluated six after one and one-half years and one after 32 months. Although these investigators have reported satisfactory healing after prolonged treatment periods, it must be emphasized that a) only a few teeth were tested, and b) the treatments were on normal, surgically traumatized pulp tissue. The findings contribute little to answer the question, "How does the acutely or chronically inflamed pulp respond to calcium hydroxide after amputation?" Zander (88) reported treating 150 carious teeth, but histologic evidence was presented for only one primary tooth three and one-half months after treatment. To date, no

controlled study has been performed to evaluate the response of inflamed or degenerated pulp tissue to vital pulpotomy. The deterrent aspect is loss of the control conditions once the tooth is treated.

The status of the coronal tissue is best evaluated by the histologic method; however, this necessitates sacrifice of the treated teeth. Although removal of teeth was unnecessary with the pulp biopsy method, inherent limitations were present. Tissue sections lacked peripheral dentin landmarks for orientation to sites of exposure and root stumps. The desirability of this orientation has been demonstrated by Mitchell and Tarplee (62). It was impossible to determine, for example, if an acutely inflamed portion of tissue was from an area remote from or adjacent to the root.

Pulp biopsy may not include all the coronal tissue. Tissue may be lost while dentin is removed to gain access to the pulp chamber. Curettage may produce fragmentation of degenerated pulp tissue and even normal tissue, resulting in further tissue loss. Other portions may be lost in processing. Of 300 or more sections examined while obtaining material for this study, specimens were noted to vary greatly in size. This may reflect the range in pulp chamber size as well as problems inherent in pulp biopsy. Nevertheless, it was assumed for the purpose of this investigation that a given tissue section was representative of coronal pulp.

To compare the results of this investigation with those studies utilizing permanent teeth is hazardous. A physiologic difference between permanent and primary teeth is the latter's resorptive process

leading to exfoliation. Vital pulpotomy failures in primary teeth are often characterized by marked internal resorption. This osteoclastic activity represents a pathologic process which may be considered an exaggerated response of the normal resorptive process, triggered by the action of calcium hydroxide, with or without the presence of inflammation.

Clinical assessment of pulp status is characterized by further variables. Previous investigators have shown that no objective or subjective clinical diagnostic test is capable of reliably determining degree or type of pathosis in pulp tissue. Radiographic evidence is considered a prerequisite of valid pulp studies. It serves a dual purpose for judging teeth for eligibility and outcome of treatment. Yet radiographic evidence has been shown to be neither a good detector of early periapical infection associated with inflamed and degenerated pulp tissue (78), nor of success and failure based on histologic evidence (64). Two investigators (17, 84) obtained grossly different evaluations of the same pulpotomy data because of the effect of time, use of different sets of radiographic criteria, or both. Thus, the variables are manifold: poor correlation of radiographic evidence with histopathologic status of pulp, lack of standardization of radiographic criteria for success and failure, and lack of standardized quality of radiographs themselves. The question arises whether the success or failure of the vital pulpotomy using calcium hydroxide is determined more by the actual tissue response or by its radiographic interpretation.

Shortcomings of all other clinical tests were evident in the

review of the literature. However, certain areas hold promise for the future. The method of electric pulp testing of Vargas and Vivaldi suggests better correlation with histologic findings than has been presented by other investigators who have not used their apparatus or accommodation index. Further testing by this method is to be encouraged. Thermal tests in children have definite advantages (4), but their reliability is poor (44, 64). Reference to the relationship of pulp response to pulp pathosis using devices capable of delivering measurable thermal stimuli has not been found in the literature. Such a study might result in a valuable addition to diagnostic methods, especially as a pre-treatment test for teeth which are candidates for vital pulpotomy.

Further speculation regarding vital pulpotomy outcome centers around calcium hydroxide-treated pulp tissue. Zander (88) believed that alkalinity and calcium ions of the drug contribute to the rapid formation of the dentin bridge. Cameron (21, p.220) pointed out that alkaline solutions with pH 9-9.6 dissolve nuclear membrane, and that with increasing alkalinity the nucleolus disappears and the cell persists in an abnormal form for hours after alkali removal. Calcium hydroxide used in vital pulpotomy therapy is not only left in place but produces alkalinity of 12.5 (88), perhaps higher (60). The action of calcium hydroxide then produces necrotic tissue.

Cameron (21, p. 348) referred to the phenomenon in which calcium is deposited in dead or dying cells or intercellular structures as "dystrophic calcification". He noted that calcium salts infiltrate any devitalized tissue which is not rapidly absorbed, and that

scar tissue is an ideal site for calcification. This phenomenon is explained as a deposition of calcium phosphate crystals which, once begun, upsets the equilibrium in the body fluids resulting in further precipitation. Acceptance of his statement that "the emphasis in pathological calcification is on damaged or dead cells or their products" warrants classifying formation of the primary dentin bridge, formed under a caustic dressing such as calcium hydroxide (with or without prior application of another caustic agent such as phenol), as a pathologic phenomenon.

Fletke, Hayden and Winkler (31), using radioisotopes, showed that calcium ions from the calcium hydroxide dressing appeared in the dentin bridge but not in the adjacent pulp tissue. Berman and Massler (11) observed dentin bridge formation under zinc oxide-eugenol in rat molars and concluded that the only source of calcium ions necessary are those derived from the viable pulp tissue. Shroff (76) showed that in untreated teeth the pulp attempts to wall off the inflamed tissue at the exposure site by means of dentin bridge formation. Hence, the production of the dentin bridge cannot be attributed to the direct action of calcium hydroxide.

In this regard an interesting observation by Davis (26) in 1921 was, "In all cases of partial pulp canal filling with an absence of rarefied areas, there seemed to be an obliteration of the pulp canal." This phenomenon was mentioned by other investigators (28). Postoperative radiographs in this study revealed complete or partial obliteration of a number of canals of primary teeth treated by vital pulpotomy with calcium hydroxide. However, Davis used no calcium hydroxide;

therefore, once again the justifiable conclusion is that calcific deposits may, and often do, occur without the use of this drug.

The nature of the test animal and its tissue sensitivity may also interfere with assessment of healing. Pulp tissue of rats may react quite differently than human pulp to calcium hydroxide or zinc oxide-eugenol. Kiryati (50) found that cortisone did not impair healing in rat molars, but he did not state that human teeth might react differently. However, Ragan, cited by Asboe-Hansen (2, p. 133), found rats the most resistant and humans the most sensitive to effects of cortisone on wound healing. Cortisone has been shown by Ragan to delay wound healing in connective tissue (2, p. 171). If connective tissue of rats and humans differ in response to cortisone, then might not they differ in response to calcium hydroxide, zinc oxide-eugenol, or any other test drug? Despite the fact that all tissues in the animal kingdom show identical wound healing characteristics (2, p.162), we are not justified in assuming that the pulp tissue of rat, human, or other test animal will respond similarly to the same drugs. Cabrini, Maisto, and Manfredi (19) investigated histochemical differences in pulpal healing under both calcium hydroxide and zinc oxide-eugenol. Glycogen was found in the healing area under calcium hydroxide, but not in the corresponding cells under zinc oxide-eugenol. Whether or not this finding reflects a physiologic or pathologic environmental change is a moot question.

The difference in pulpal response to various dressings may be related to oxygen consumption of treated tissue. Oxygen consumption of rabbit tissue treated with cortisone was reduced 66 percent (21).

Fisher, et al (30) showed that eugenol significantly reduced oxygen consumption of bovine pulp tissue, surpassing calcium hydroxide in this respect. Composition of the two drugs suggests a probable difference in duration of effect; the alkaline calcium hydroxide stimulates buffering action and forms a necrobiotic barrier while the aromatic hydrocarbon (even as a constituent of zinc oxide-eugenol paste) persists as an insoluable and volatile oil. If these drugs act in a comparable manner on human pulp tissue, the delayed or ineffective dentin bridge formation may be a manifestation of decreased oxygen consumption. The drugs may exert different "anti hyaluronidase effects" (2, p. 108) on the adjacent tissue resulting in varying degrees of "spreading" of tissue and bacterial toxins or the drugs themselves.

During vital pulpotomy treatment, dentin chips often contaminate and irritate the treated pulp (11, 48); hence they represent another variable in the healing response.

A variable which characterizes this study, as well as others (17, 85), is the multiplicity of operators. Admittedly, this situation is not conducive to consistent handling of coronal and/or radicular pulp tissue. A single operator is recommended for studies of this type in order to assure optimum reliability.

In summary, the variables inherent in the evaluation of the response of amputated pulps treated with calcium hydroxide in this study may be related to: pretreatment status of radicular pulp ("normal" or pathologic), amount and location of tissue available (whole or coronal portion), root resorption, effect of dentin chips, and manifold operators. Additional variables which may effect different results

between this and other studies are: time interval between treatment and evaluation, tissue sensitivities of test subjects, altered oxygen consumption, and different spreading reactions.

In the course of making entries on the IBM data sheet (Appendix) several patients were noted to have received more than one pulpotomy treatment. An additional separate data sheet was prepared for these patients (Figure 23, Appendix). The results were provocative, because all categories except success and failure lacked uniformity. Six patients had either all successes or all failures, including one patient with three treatments. The sample size was too small to permit statistical evaluation, but the data suggests that other factors, perhaps those related to "general resistance" may be the determining factors in pulp response. Of interest in this regard is the comment made by Zander (88) over 20 years ago after evaluating 150 pulps treated with calcium hydroxide. "It is not known whether it was the condition of the pulp at the time pulpotomy was performed, the technique employed or the difference in the vital reaction of the individual." Future studies should be designed to evaluate the relationship of pulp response to specific systemic factors that are known to affect tissue healing, such as endocrine imbalances and avitaminoses (33; 2, p. 170).

If standard textbook description of inflammation and degeneration coincides with the interpretation of the data in Tables IV-A and IV-B, then the histopathologic findings may be assumed to be valid.

That "the exception proves the rule" is evident in the finding that a similar mean number of chronic inflammatory cells exists in 2+ and 3+ degeneration, whereas the mean number of acute inflammatory

cells is significantly different for all categories of degeneration.

In the severe acute inflammatory reaction, 3+ degeneration is likely to result because this reaction is usually elicited by bacteria which cause rapid and extensive tissue breakdown. Chronic inflammation is a response to less virulent and/or less numerous bacteria or other mild irritants. This chronic situation may be seen as an original response or as a late development of acute inflammation ("proliferative" fibrosis). This response to milder irritants would be expected to be related to a milder degree of degeneration. It is possible that the cell counts in tissues with severe 3+ degeneration are comparable in number to those at the lower level of 2+ degeneration, because they are on either side of what is a "peak" of reaction for chronic inflammatory cells. We may speculate that in coronal pulp tissue this "peak" occurs in tissues which border between 2+ and 3+ degeneration.

The use of the 0, 1+ , 2+, 3+ scale is a common method of designating the severity of a condition which has been subjectively evaluated. It should be emphasized that although pulp response has been assessed using various indexes, such as the I. D. Index of James, Schour and Spence (46), the application of a number to a qualitative factor does not render the data quantitative in character.

The findings pertaining to the quantitative aspect of this study were disappointing, but negative findings are not without value. The information pertaining to the number of acute and chronic inflammatory cells in samples of coronal pulp as shown in Figure 23 indicates that there is no specific number of cells which can demarcate the dividing zone between treatment success and failure. The reason relates to the

fact that many of the successful pulpotomies exhibited coronal pulps which had high cell counts. Conversely, many of the failures had few, if any, acute and/or chronic inflammatory cells. The statistical method used evaluated every possible cell combination as they related to success and failure and no significant differences were found. Therefore, this study provided no quantitative means to predict the success or failure of teeth treated with vital pulpotomy using calcium hydroxide.

# SUMMARY

Specimens of coronal pulp tissue from 58 primary molars were qualitatively and quantitatively analyzed by histopathologic methods. Ability of the investigator to make reliable qualitative judgments was tested.

The histopathologic findings were compared to treatment outcome after vital pulpotomy therapy using calcium hydroxide. Success and failure were judged by clinical and radiographic criteria.

Effects of clinical factors (age, gender, tooth location, extent of restoration) were tested and found non-contributors to treatment outcome. Internal resorption as a criterion of success or failure was also eliminated as a possible biasing factor.

From the histologic findings, a determination was made of the relationship of each degree of degeneration to the number of acute and chronic inflammatory cells in that category.

A discussion was presented concerning some of the pharmacologic, physiologic, and pathologic aspects of vital pulpotomy therapy using calcium hydroxide, as well as variables inherent in this and other similar studies.

# CONCLUSIONS

Healing response following vital pulpotomy using calcium hydroxide (judged by clinical and radiographic criteria) cannot be predicted by histopathologic evaluation of coronal pulp tissue on the basis of:

1. The presence of new or old hemorrhage.
2. Dilated and engorged blood vessels.
3. The extent of bacterial invasion.
4. The degree of degeneration.
5. The number of acute inflammatory cells.
6. The number of chronic inflammatory cells.

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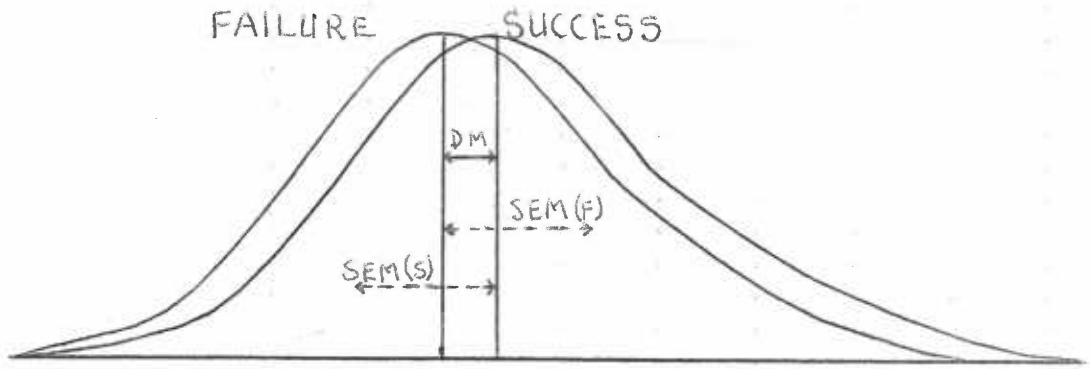
# APPENDIX




Schematic Distribution Curves of Inflammatory Cell Counts

in Successful and Unsuccessful Pulpotomy Groups

DM..... Difference of Means
SEM (S).... Standard Error of Mean: Success Group
SEM (F).... Standard Error of Mean: Failure Group

Figure 24

FINDINGS IN PATIENTS RECEIVING MULTIPLE PULPOTOMIES

| Patient | Age | Sex | Race | Type Restoration | Number Polys | Number Plasma and Lymphos | Degree of Hemorrhage Old | Degree of Hemorrhage New | Degree of Vessel Engorgement | Degree of Degeneration | Bacteria Location | Bacteria Type | Success | Failure | Etiology of Failure | Time Interval (in months) After Treatment | Tooth Treated |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1) Bass, B. | 7 | M | W | 3 surf. alloy | 26 | 6 | 0 | 3+ | 3+ | 2+ | Periph. | T & B | | X | | 12-24 | D/ |
| | 7 | | | 2 surf. alloy | 1 | 6 | 0 | 1+ | 2+ | 2+ | Periph. | C | | X | | 9-12 | E/ |
| 2) Bertrand, N. | 6 | M | W | 2 surf. alloy | 63 | 15 | 1+ | 0 | 2+ | 2+ | Int. Ext. | B & C | X | | | 36+ | D/ |
| | 6 | | | 2 surf. alloy | none | 4 | 1+ | 1+ | 1+ | 1+ | Ext. | C | X | | | 36+ | E/ |
| 3) Knox, C. | 6 | F | W | 2 surf. alloy | none | 1 | 0 | 0 | 1+ | 1+ | Periph. | C | | X | Bone loss Int. Res. | 6-9 | E/ |
| | 6 | | | 2 surf. alloy | none | 1 | 0 | 0 | 1 | 2+ | Int. Ext. | B & C | | X | Int. Res. | 9-12 | E/ |
| 4) Kuhn, S. | 6 | M | W | Crown | 3 | 5 | 0 | 0 | 1+ | 1+ | Ext. | C | X | | | 24-36 | E/ |
| | 6 | | | Crown | 0 | 16 | 0 | 1+ | 2+ | 2+ | None | None | X | | | 12-24 | E/ |
| | 6 | | | Crown | 4 | 42 | 0 | 0 | 1+ | 3+ | Int. Ext. | C | X | | | 36+ | E/ |
| 5) Maxon, N. | 6 | F | W | 3 surf. alloy | 0 | 0 | 1+ | 0 | 1+ | 2+ | Ext. | C | | X | Bone loss | 12-24 | E/ |
| | 6 | | | 2 surf. alloy | 2 | 2 | 0 | 1+ | 1+ | 2+ | Int. Ext. | B & C | X | | | 12-24 | E/ |
| 6) McKinnery, M. | 6 | M | N | 2 surf. alloy | 1 | 7 | 2+ | 3+ | 3+ | 2+ | Int. Ext. | B & C | X | | | 24-36 | D/ |
| | 6 | | | 2 surf. alloy | 0 | 3 | 0 | 1+ | 1+ | 1+ | Int. | C | X | | | 12-24 | E/ |
| 7) Winkler, K. | 7 | M | W | Crown | 0 | 1 | 1+ | 0 | 1+ | 1+ | none | none | | X | Int. Res. Bone loss | 12-24 | E/ |
| | 7 | | | Crown | 0 | 0 | 1+ | 0 | 1+ | 1+ | Ext. | C | | X | Bone loss | 12-24 | E/ |

Figure 25

Table V

RELATIONSHIP OF DEGREE OF DEGENERATION TO NUMBER OF ACUTE AND CHRONIC INFLAMMATORY CELLS

| Degree of Degeneration | Type of Inflammatory Cell | Cell Counts | Total No. of Cells | Sample Size | Mean Number of Cells Representative Areas | Standard Deviation |
|---|---|---|---|---|---|---|
| 1+ | Acute | 0-0-0-0-0-0-0-0-3-3-0-0-0-0-0-3 | 9 | 16 | 0.56 | 1.22 |
| | Chronic | 4-4-0-0-0-3-0-1-5-9-3-3-0-1-0-0 | 33 | 16 | 2.06 | 2.5 |
| 2+ | Acute | 26-1-63-6-3-0-3-10-0-0-0-8-0-0-2 1-8-0-23-0-6-0-0-0-10-0-0-7 | 177 | 28 | 6.32 | 12.95 |
| | Chronic | 6-6-15-14-33-0-17-5-0-1-16-23-0-0 2-7-5-3-23-3-7-1-0-11-16-23-0-2 | 239 | 28 | 8.52 | 9.1 |
| 3+ | Acute | 80-0-0-105-0-6-4-0-3-18-2-5-6-0 | 229 | 14 | 16.36 | 30.7 |
| | Chronic | 2-0-0-5-0-11-42-23-0-5-1-8-9-1 | 107 | 14 | 7.64 | 10.95 |

| Name | Slide Ident. Number | Tooth Number | Age | Sex | Race | Type Rest'n | Success or Failure: 0-3 mos. | 3-6 mos. | 6-9 mos. | 9-12 mos. | 12-24 mos. | 24-36 mos. | 36+ mos. | Number of Polys | Number of Lymphos | Number of Plasma Cells | Old Hemor. | New Hemor. | Vessels | Degen. | Loc.Bact. | Type Bact. | Sp.M.Abut. | Prev.Cap Rx | Prev.Rest Rx |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Bass, B. 1 | 001 | 02 | 07 | 1 | 1 | 3 | - | - | - | + | 3 | - | - | 026 | 006 | 000 | 0 | 3 | 3 | 2 | 4 | | 0 | 0 | |
| Bass, B. 2 | 002 | 01 | 07 | 1 | 1 | 2 | - | - | - | 3 | 3 | - | - | 001 | 006 | 000 | 0 | 1 | 2 | 2 | 4 | 3 | 0 | 0 | |
| Beckwith, D 3 | 003 | 12 | 05 | 2 | 1 | 4 | - | 3 | - | - | 2 | - | - | 080 | 002 | 000 | 0 | 3 | 2 | 3 | 4 | 3 | 0 | | 1 |
| Bertrand, N 4 | 004 | 09 | 06 | 1 | 1 | 2 | - | 0 | - | - | - | - | 0 | 063 | 015 | 000 | 1 | 0 | 2 | 2 | 3 | 6 | 0 | 0 | 0 |
| Bertrand, N 5 | 005 | 10 | 06 | 1 | 1 | 2 | - | - | 0 | - | - | - | 0 | 000 | 004 | 000 | 1 | 1 | 1 | 1 | 2 | 3 | 0 | 0 | 0 |
| Brown, M.A. 6 | 006 | 12 | 03 | 2 | 1 | 4 | - | - | - | - | 2 | - | - | 006 | 013 | 001 | 0 | 0 | 1 | 2 | 3 | 6 | 0 | 0 | 0 |
| Brown, S. 7 | 007 | 09 | 06 | 2 | 1 | 2 | 0 | 0 | - | - | 0 | 0 | - | 000 | 004 | 000 | 1 | 1 | 2 | 1 | 3 | 3 | 0 | 0 | 0 |
| Castor, B.A 8 | 008 | 10 | 04 | 2 | 1 | 3 | - | 0 | 0 | - | 0 | - | - | 000 | 000 | 000 | 2 | 0 | 0 | 3 | 4 | 3 | 0 | 0 | 0 |
| Ceuci, K. 9 | 009 | 10 | 04 | 2 | 1 | | 0 | - | - | 3 | 1 | - | - | 003 | 033 | 000 | 0 | 0 | 1 | 2 | 3 | 3 | 0 | 0 | 0 |
| Clark, D. 10 | 010 | 20 | 07 | 1 | 1 | 2 | 0 | - | - | - | - | 0 | - | 000 | 000 | 000 | - | - | - | 2 | 2 | 3 | 0 | 0 | 0 |
| Clark, M. 11 | 011 | 12 | 06 | 1 | 1 | 4 | - | - | 2 | - | 3 | - | 3 | 000 | 000 | 000 | 0 | 1 | 1 | 1 | 3 | 6 | 0 | 0 | 0 |
| Coffey, B. 12 | 012 | 10 | 06 | 2 | 1 | 3 | - | - | 3 | - | - | 0 | - | 003 | 006 | 011 | 0 | 0 | 1 | 2 | 4 | 3 | 0 | 0 | 0 |
| Daniels, D. 13 | 013 | 19 | 06 | 1 | 1 | 2 | - | 0 | 0 | 0 | 3 | - | - | 010 | 002 | 003 | 0 | 0 | - | 2 | 3 | 6 | 0 | 0 | 0 |
| Ediger, B. 14 | 014 | 09 | 08 | 2 | 1 | 4 | - | 0 | - | - | 0 | - | - | 000 | 000 | 000 | 0 | 2 | 1 | 2 | 0 | 0 | 0 | 0 | 0 |
| Faulkner, T. 15 | 015 | 20 | 07 | 1 | 1 | 4 | - | - | - | - | 0 | - | - | 000 | 000 | 000 | 2 | 0 | 1 | 1 | 3 | 4 | 0 | 0 | 0 |
| Franz, R. 16 | 016 | 02 | 06 | 1 | 1 | 3 | - | 0 | - | - | - | 0 | - | 000 | 000 | 000 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| Gilbert, K. 17 | 017 | 09 | 07 | 2 | 1 | 2 | - | - | - | - | 0 | - | - | 000 | 000 | 000 | 0 | 0 | 2 | 3 | 4 | 3 | 0 | 0 | 0 |
| Gordon, J. 18 | 018 | 20 | 05 | 1 | 1 | 4 | - | - | - | - | 0 | - | - | 007 | 002 | 000 | 0 | 1 | 0 | 2 | 1 | 3 | 0 | 0 | 0 |
| Granshaw, P. 19 | 019 | 02 | 07 | 1 | 1 | 4 | - | - | - | - | 0 | - | - | 105 | 004 | 001 | 0 | 2 | 0 | 3 | - | - | 0 | 1 | 0 |
| Graves, S. 20 | 020 | 20 | 04 | 2 | 1 | 2 | - | - | - | 0 | 0 | - | 0 | 000 | 003 | 000 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| Gurske, L. 21 | 021 | 11 | 04 | 2 | 1 | 2 | - | - | - | - | 0 | - | - | 000 | 000 | 000 | 0 | 0 | 1 | 3 | 2 | 3 | 0 | 0 | 0 |
| Haider, R. 22 | 022 | 19 | 06 | 2 | 1 | 2 | 1 | - | - | 1 | - | - | - | 000 | 000 | 000 | 0 | 0 | 1 | 1 | 2 | 3 | 0 | 0 | 0 |
| Jemtegard 23 | 023 | 02 | 07 | 1 | 1 | 2 | - | - | - | 3 | 2 | - | - | 006 | 011 | 000 | 1 | 0 | 1 | 3 | 2 | 3 | 0 | 0 | 0 |
| Knox, C. 24 | 024 | 11 | 06 | 2 | 1 | 2 | - | - | 2 | - | - | - | - | 000 | 001 | 000 | 0 | 0 | 1 | 1 | 4 | 3 | 0 | 0 | 0 |
| Knox, C. 25 | 025 | 20 | 06 | 2 | 1 | 2 | - | - | - | 1 | 1 | - | 3 | 000 | 001 | 000 | 0 | 0 | 1 | 2 | 3 | 6 | 0 | 0 | 0 |
| Kuhn, S. 26 | 026 | 20 | 06 | 1 | 1 | 4 | - | - | - | - | 0 | 0 | - | 003 | 005 | 000 | 0 | 0 | 1 | 1 | 2 | 3 | 1 | 0 | 0 |
| Kuhn, S. 27 | 027 | 10 | 06 | 1 | 1 | 4 | - | - | 0 | - | 0 | 4 | - | 000 | 016 | 000 | 0 | 1 | 2 | 2 | 0 | 0 | 1 | 0 | 0 |
| Kuhn, S. 28 | 028 | 01 | 06 | 1 | 1 | 4 | - | - | - | 0 | - | - | 0 | 004 | 036 | 006 | 0 | 0 | 1 | 3 | 3 | 3 | 0 | 0 | 0 |
| Lackey, S. 29 | 029 | 20 | 04 | 1 | 1 | 1 | - | - | - | - | 0 | - | 2 | 003 | 009 | 000 | 0 | 0 | 1 | 1 | 3 | 6 | 0 | 0 | 0 |
| Laurion, M. 30 | 030 | 11 | 08 | 1 | 1 | 4 | - | - | - | 0 | 0 | 0 | - | 008 | 021 | 002 | 1 | 1 | 2 | 2 | 3 | 3 | 1 | 0 | 0 |
| LeDoux, J. 31 | 031 | 09 | 07 | 1 | 1 | 2 | - | 1 | - | - | 2 | - | - | 000 | 000 | 000 | 0 | 0 | 0 | 2 | 4 | 6 | 0 | 0 | 0 |
| Loveland, C. 32 | 032 | 20 | 07 | 2 | 1 | 2 | - | 2 | - | - | - | - | - | 000 | 003 | 000 | 2 | 3 | 3 | 1 | 0 | 0 | 0 | 0 | 0 |
| Maxon, N. 33 | 033 | 11 | 06 | 2 | 1 | 3 | - | - | - | - | 3 | - | - | 000 | 000 | 000 | 1 | 0 | 1 | 2 | 2 | 3 | 0 | 0 | 0 |
| Maxon, N. 34 | 034 | 20 | 06 | 2 | 1 | 2 | - | - | - | - | 0 | - | - | 002 | 002 | 000 | 0 | 1 | 1 | 2 | 3 | 6 | 0 | 0 | 0 |
| Maxon, R. 35 | 035 | 10 | 08 | 1 | 1 | 3 | 0 | - | - | - | 0 | - | - | 000 | 023 | 000 | 0 | 1 | 2 | 3 | 3 | 3 | 0 | 0 | 0 |
| McKinney, M. 36 | 036 | 12 | 06 | 1 | 2 | 2 | - | - | - | - | 0 | 0 | - | 001 | 006 | 001 | 2 | 3 | 3 | 2 | 3 | 6 | 0 | 0 | 0 |
| McKinney, M. 37 | 037 | 11 | 06 | 1 | 2 | 2 | - | - | - | - | 0 | - | - | 000 | 003 | 000 | 0 | 1 | 1 | 1 | 1 | 3 | 0 | 0 | 0 |
| Mitchell, H. 38 | 038 | 11 | 06 | 2 | 1 | 4 | - | - | - | - | 0 | 0 | - | 008 | 005 | 000 | 0 | 0 | 1 | 2 | 3 | 6 | 0 | 0 | 0 |
| Mitchell, M. 39 | 039 | 11 | 08 | 1 | 1 | 4 | - | - | - | - | 0 | - | - | 003 | 000 | 000 | - | - | - | 3 | 4 | 3 | 0 | 0 | 1 |
| Moore, Dale 40 | 040 | 10 | 08 | 1 | 1 | 2 | - | - | - | - | 0 | - | - | 018 | 005 | 000 | 0 | 2 | 1 | 3 | 3 | 3 | 0 | 1 | 0 |

Questionable = 5
Success = 0
Int. Res.=1
I.R. + Bone loss = 2
Bone Loss = 3
Ext'd = 4

1 Restoration
2 occlusal=1
2 surf. =2
3 3 surf. =3
4 crown = 4
temp. = 5
5

6 Location of
7 Bacteria:
8 Clear = 0
Intracell=1
9 Extracell=2
10 Int & Ext=3
11 Periph. = 4

12 Type Bact:
13 None = 0
14 Thread = 1
Bacillus=2
15 Cocci = 3
16 I & B = 4
I & C = 5
17 B & C = 6
18 T,B,C = 7

19 Space Maint
20 Abutment
21 No = 1
22 Yes = 2

23 Prev. Pulp
24 Cap Treatmn
25 No = 1
26 Yes = 2

27 Prev. Rest.
28 Treatment
29 No = 1
30 Yes = 2
31
32
33
34
35
36
37
38
39
40

Success or Failure

| Name | Slide Ident. Number | Tooth Number | Age | Sex | Race | Type Rest'n | 0-3 mos. | 3-6 mos. | 6-9 mos. | 9-12 mos. | 12-24 mos. | 24-36 mos. | 36+ mos. | Number of Polys | Number of Lymphos | Number of Plasma Cells | Old Hemor. | New Hemor. | Vessels | Degen. | Loc. Bact. | Type Bact. | Sp.M. Abut. | Prev.Cap.Rx | Prev.Rest.Rx |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Moore, R. 1 | 041 | 11 | 09 | 1 | 1 | 4 | 00 | - | - | - | -● | - | | 002 | 001 | 000 | - | - | - | 3 | 4 | 6 | | | |
| Morgan, B. 2 | 042 | 20 | 04 | 1 | 1 | 4 | - | - | - | - | 1- | 3 | | 000 | 000 | 000 | 0 | 0 | 1 | 1 | 4 | 7 | 0 | 0 | 0 |
| Neufer, J. 3 | 043 | 01 | 05 | 1 | 1 | 4 | - | - | - | - | 2- | - | | 000 | 003 | 000 | 2 | 1 | 1 | 2 | 2 | 7 | 0 | 0 | 0 |
| Oakley, P. 4 | 044 | 11 | 09 | 2 | 1 | 2 | - | - | - | 0 | -● | - | | 023 | 018 | 005 | 0 | 2 | 2 | 2 | 3 | 6 | 0 | 0 | 0 |
| O'Neal, S. 5 | 045 | 20 | 05 | 2 | 1 | 4 | - | - | 2 | 4 | -- | - | | 005 | 008 | 000 | - | - | - | 3 | 0 | 0 | 0 | 0 | 0 |
| Parker, J. 6 | 046 | 10 | 06 | 2 | 1 | 2 | - | - | - | - | 0● | - | | 000 | 003 | 000 | 2 | 1 | 2 | 2 | 4 | 3 | 0 | 0 | 0 |
| Price, H. 7 | 047 | 11 | 06 | 1 | 2 | 5 | 3 | - | - | - | -- | - | | 006 | 009 | 000 | 0 | 0 | 0 | 3 | 3 | 6 | 0 | 0 | 0 |
| Raab, T. 8 | 048 | 10 | 04 | 1 | 1 | 1 | - | - | 0 | 0 | 1- | - | | 006 | 007 | 000 | 2 | 1 | 1 | 2 | 3 | 3 | 0 | 0 | 0 |
| Remme, S. 9 | 049 | 01 | 07 | 1 | 1 | 2 | - | 3 | - | - | -- | - | | 000 | 001 | 000 | 0 | 0 | 1 | 2 | 2 | 3 | 0 | 0 | 0 |
| Rennie, S. 10 | 050 | 20 | 05 | 1 | 1 | 4 | - | 2 | 2 | - | -● | - | | 000 | 001 | 000 | 1 | 2 | 0 | 3 | 2 | 6 | 1 | 0 | 0 |
| Robedeau, M 11 | 051 | 09 | 09 | 2 | 1 | 3 | - | - | - | - | 0- | - | | 000 | 000 | 000 | 0 | 0 | 1 | 2 | 4 | 3 | 0 | 0 | 0 |
| Rivelli, P. 12 | 052 | 01 | 05 | 1 | 1 | 2 | - | - | - | - | 0- | - | | 003 | 000 | 000 | 0 | 2 | 2 | 1 | 4 | 3 | 0 | 0 | 0 |
| Slaughter, B. 13 | 053 | 09 | 06 | 2 | 1 | 2 | - | 3 | - | - | -- | - | | 000 | 011 | 000 | 0 | 0 | 1 | 2 | 4 | 3 | 0 | 0 | 0 |
| Taylor, D. 14 | 054 | 01 | 09 | 2 | 1 | 3 | - | - | - | 3 | -- | - | | 010 | 016 | 000 | 0 | 0 | 1 | 2 | 4 | 3 | 0 | 0 | 0 |
| Warren, C. 15 | 055 | 19 | 05 | 2 | 1 | 2 | 0 | - | - | - | 0- | - | | 000 | 023 | 000 | 1 | 3 | 2 | 2 | 3 | 6 | 0 | 0 | 0 |
| Winkler, K. 16 | 056 | 20 | 07 | 1 | 1 | 4 | - | - | - | - | 24 | - | | 000 | 001 | 000 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| Winkler, K. 17 | 057 | 11 | 07 | 1 | 1 | 4 | - | - | - | - | 3- | - | | 000 | 000 | 000 | 1 | 0 | 1 | 1 | 2 | 3 | 0 | 0 | 0 |
| Wood, J. 18 | 058 | 12 | 06 | 1 | 1 | 2 | - | - | - | 3 | 22 | - | | 000 | 000 | 000 | - | - | - | 2 | 4 | 3 | 0 | 0 | 0 |
| 19 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 20 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 21 | | | | | | | | | | | | | | | | | | | | | | | | | |
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| 34 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 35 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 36 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 37 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 38 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 39 | | | | | | | | | | | | | | | | | | | | | | | | | |
| 40 | | | | | | | | | | | | | | | | | | | | | | | | | |

Success = 0
Int.Res.= 1
I.R. + Bone Loss = 2
Bone Loss = 3
Ext'd. = 4

[1] Restoration
2 occlusal=1
3 2 surf. =2
4 3 surf. =3
5 crown = 4
6 temp. = 5

[7] Location of
[8] Bacteria:
[9] Clear = 0
[10] Intracell=
[11] Extracell=
[12] Int & Ext=
[13] Periph. =

[14] Type Bact.
[15] None = 0
[16] Thread = 1
[17] Bacillus=2
[18] Cocci = 3
[19] T & B = 4
[20] T & C = 5
[21] B & C = 6
[22] T,B,C = 7

[23] Space Maint.
[24] Abutment:
[25] No = 1
[26] Yes = 2

[27] Prev. Pulp
[28] Cap Treatmnt
[29] No = 1
[30] Yes =2

[31] Prev. Rest.
[32] Treatment:
[33] No = 1
[34] Yes = 2
35
36
37
38
39
40