AN INVESTIGATION OF THE DENTAL PULP HEMOGRAM

AS A DIAGNOSTIC AID FOR VITAL PULP THERAPY

by

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INTRODUCTION
Introduction

A dilemma which long has perplexed those keenly interested in vital pulp therapy, is when to institute therapy and when to resort to other types of treatment. There is little doubt that vital pulp therapy can save thousands of teeth. The literature is replete with testimony on the virtues of conservative pulp treatment. However, many clinicians are still far from content with vital pulp therapy, due to the number of failures which occur. The present trend in vital pulp therapy seems to be toward adherence to strict biological and surgical principles. These principles are becoming recognized as the foundation of vital pulp therapy. With these principles in mind, the need for an accurate diagnosis of the true pulpal condition cannot be over emphasized.

The enigma facing the diagnostician is that at the present time the only method of precisely determining the pulpal status of a tooth is to remove the tooth and prepare histological sections of the pulp. Obviously, this is not the method of choice when we desire to keep the tooth intact. Noonan" emphasizes the problem when he states, "The exact nature of the pulp cannot be determined with the present day pulp testers or roentgenograms; until more accurate means are developed, there will be failures." In a similar statement, Shroff in discussing vital pulp therapy stated, "Such therapy must remain a hit and miss procedure until more accurate and scientific methods of pulp diagnosis are found." The search for an accurate means of evaluating the pulp condition is of utmost importance if we are to improve the results of conservative pulp treatment. It has been suggested that the local blood picture from the pulp of a tooth may be an important clue as to the condition of that tissue.
REVIEW OF THE LITERATURE
Review of the Literature

Apparently there are only two significant references to the pulp hemogram as a diagnostic aid in vital pulp therapy. Therefore, it was decided to review a portion of the literature concerning some of the related aspects of this study. The following topics will be reviewed: (1) pulp anatomy, physiology and characteristics (2) pulp pathology (3) methods of determining pulp vitality (4) studies correlating clinical symptoms and histological diagnosis of the pulp (5) indications for vital pulp therapy (6) the white blood cells and (7) the pulp hemogram in diagnosing pulpal conditions.

Pulp Anatomy, Physiology and Characteristics

Orban\(^3\) points out that the dental pulp is of mesenchymal origin and contains most of the cellular and fibrous elements which are present in other connective tissue. Schaffer\(^4\) describes the pulp as appearing similar to embryonal tissue and he notes that it is often likened to Wharton’s jelly of the umbilical cord or to myxomatous tissue. Nevertheless, it is an adult connective tissue that is gelatinous or loosely packed. He also calls our attention to the fact that the pulp is encased by hard tissue with no possibility to expand and without much collateral circulation. Ciampa\(^5\) describes the pulp as a specialized connective tissue which is composed of cells and intercellular substance rich in blood vessels and nerves. The pulp is covered by a layer of columnar cells, the odontoblasts. Essentially it consists of a homogenous jelly-like ground substance in which cells and fibers are suspended along with other elements which may be found in connective tissue. The zone which immediately borders the dentin contains odontoblasts which are highly specialized connective tissue
cells, columnar in shape and which present long cytoplasmic processes (Tomes fibrils) extending into the dentinal tubules.

Korff's fibrils are collagenous and precollagenous fibrils which arise from deeper layers of the pulp and unite at the base of the odontoblasts to form relatively thick bundles. They then extend between the odontoblasts and unravel in the layer of ground substance. The layer of Weil is a cell-free zone situated immediately beneath the pulpal end of the odontoblasts. This area contains many nerve fibrils of the subodontoblastic plexus. It is found only in teeth of the aged and more in the coronal portion. Deep to the layer of Weil, for a space about twice as wide as the odontoblasts are long, is a cell rich zone. In this zone we find a dense arrangement of fibrocytes, the dominant cells found in all connective tissue. Their cytoplasm and fiber-like processes give the cells a stellate shape.

In describing the vascular arrangement of the pulp, Ciampa states that one artery enters the apex of the tooth and passes upward in the center of the pulp together with the nerve trunk, giving off many lateral branches along the course. These form a rich plexus of capillary loops immediately beneath the odontoblasts. Here minute vessels gather the venous blood, converge into larger and larger vessels and finally pass out through the apical foramen, either as one or two veins.

Russell and Kramer, in a report on the vascular architecture of the dental pulp, mention that permanent teeth often have numerous lateral canals. Also that in deciduous teeth large vessels were noted passing through the walls of the root canals and having branches running in both coronal and apical directions.
Boling, studying the blood supply in animals' teeth, found that five to eight arterioles enter each root and that veins leaving the apex are more numerous. The arterioles entering the side of the apex are usually distributed to the pulp of the root, while the larger central vessels pass to the pulp chamber. Russel and Kramer and Boling all mention that anastomoses between arteries entering through separate roots occurs commonly in the pulp chamber. Boling noted that the diameter of vessels in the pulp was much greater in those vessels entering the pulp than those leaving the apex, suggesting a sluggish flow within the pulp. Manley has stated that the pulp has poor collateral circulation. Cheng and Provenza found in all cases that the veins and arteries seen in the middle regions of the pulp were greater in diameter than those observed apically or coronally.

Barr mentions that only the larger arteries of the pulp show muscle fibers and that there is only slight condensation of fibrous tissue for the tunique adventitia. He relates this peculiarity of the blood vessel wall to the unusual susceptibility of the pulp tissue to hyperemia and inflammation. Kozan and Burnett observed the blood circulation through windows cut in rat incisors. They found that in normal pulp the arterioles and venules were easily identifiable and that blood flowed rapidly and was free from intra-vascular sludge.

Ciampa states the nerve trunk entering the pulp through the apical foramen divides into branches containing eight to forty medullated nerve fibers. Most nerve elements which enter the pulp are of the medullated type; but there are also non-medullated sympathetic fibers for blood vessels. Usually the nerve bundles follow the blood vessels into the root canal and in their branchings. Most branches of the
nerves lose their medullary sheath very soon after leaving the trunk; others retain the myelin sheath until they reach the layer of cells where they lose the myelin. From the plexus non-myelinated fibers are given off, passing between and around the odontoblasts. They form a network around each cell and even pass between the dentinal tubules. The passage of the nerve fibrin into the dentinal tubule has not yet been demonstrated.

Provenza and Biddington found that the larger nerve trunks are intimately associated with accompanying blood vessels in the radicular pulp, but that the smaller nerve fibers in the coronal portion need not be associated with accompanying blood vessels. Wasserman has expressed doubt that nervous tissue is present in the developing tooth germ.

The presence of lymph in the pulp is controversial; however, references to its presence appear frequently in the literature. Ciampa describes a lymphatic system in the pulp tissue which anastomoses with vessels of the periodontal membrane. Haldal reports that he was able to measure the pressure of pulpal lymph in dogs' teeth. He claimed the fluid to be normal lymph. Bodecker claims that the dentinal tubules contain a fluid, the dental lymph. Orban tells us that the presence of dental lymph vessels has been demonstrated by the application of dyes into the pulp.

Many of the criteria which were thought to be indications of pathology in histological sections of pulp are now being re-evaluated. Langeland reports that he found such phenomena as eosinophilic zones, vacuoles, reticular atrophy, deviations of the odontoblastic layer, extravasation of erythrocytes and calcifications all to be present in clinically normal teeth. Nyborg believes that signs
of persistent or subsiding inflammation such as dilated vessels, pulpal hyperemia, local extravasation of erythrocytes, blood pigment and fibrin-like substances, are common in histological sections of both clinically sound and amalgam filled teeth. Stephan has reported that histologic sections of normal teeth may show signs of degeneration such as vacuolization of the odontoblastic layer and pulp calcification. Lefkowitz, et al. noted that histologic sections showing pathologic entities such as hydropic degeneration, vacuoles, which are sometimes interpreted as old abscesses, and reticular atrophy may be recognized as artefacts. They further mention that freshly ruptured capillaries in the presence of normally extravasated erythrocytes found in the pulp are due to extraction. Noonan has emphasized that there is no difference between the pulp tissue of the deciduous and the permanent teeth.

The pulp serves to produce dentin, to preserve the vitality of the dentin and to conduct sensory stimuli. The pulp produces the classical physiological response to irritation that other tissues do, i.e., inflammation. Most of the changes of the pulp due to trauma and irritation are reversible and fortunately seldom serious enough to produce necrosis. With inflammation there is dilatation of the vessels, an outpouring of serum and some cellular exudate into the pulp stroma. The loose architecture of the pulp allows for an increase in pressure so that ordinarily no permanent damage is acquired.

Manley states that the pulp reacts to injury like other tissue. However, this reaction is modified by the anatomical environment and a lack of collateral circulation in it's vascular system both of which render the pulp extremely susceptible to injury. He emphasizes that the power to lay down calcific material is a most important defense measure and that we should remember the pulp is the formative organ of
the dentin. He mentions that an injury to the dentin will cause a reaction in the pulp. He advocates that the two tissues be considered as one.

The laying down of secondary dentin is the first reaction in the pulp to peripheral injury of the dentin. It is brought about by mild irritation such as slow caries and attrition. Zander relates that with an advance in age the size of the pulp chamber is reduced by secondary dentin and that pathologic stimuli such as caries, cavity preparation, drugs and thermal stimuli, all accelerate this dentin formation. Hill mentions that throughout life there is a gradual histologic change in the pulps of teeth. The embryonal tissue and the number of stellate cells become reduced. There is increased fibrosis which he felt is hastened by caries, abrasion, etc. Nyborg believes that fibrosis of the pulp and the formation of denticles may result from an earlier inflammation. Via mentions that age changes in the pulp reduce its sensitivity to irritants. Stephan noted that teeth with regressive changes could not be distinguished clinically from normal teeth.

Although some authors have expressed doubt that the deciduous teeth can deposit secondary dentin, Ireland and also Prophét and Miller have shown the deciduous tooth is capable of depositing secondary dentin. In many instances they found that the deciduous teeth could lay down a greater amount than is usual for permanent teeth. In addition, Bevelander and Benszer found that secondary dentin occurs more frequently in anterior teeth than in molars and that the deciduous specimens had a higher ratio of adventitious dentin than the permanent carious teeth.

Austin and Waggener believe that sensation in the pulp is caused by stimulation of the dentinal fibrils or by a vasomotor disturbance
which may cause interstitial pressure and directly affect the sensory 
endings. The dental pulp and dentin have no sensory function other 
than pain; therefore, response to stimuli and injury is pain of varying 
degrees. The application of thermal stimuli to a tooth can 
cause hyperemia and hence pain. The pulp, in regard to sensation, 
resembles an internal organ. It has no sense of touch or 
localization and responds to various stimuli only by the sensation of 
pain. The pain is usually located correctly only with reference to 
the midline. The pain originating from a tooth pulp may be referred 
to almost any point on the same side supplied by the fifth cranial 
nerve.

The capillaries rupture in the presence of inflammation long 
before the nerve fibers do; therefore, a pulp which gives a vital 
response to stimuli may be a normal pulp or may be a diseased pulp with 
the nerve fibers still functioning. Thomas mentions that as 
long as metabolism takes place we may speak of the pulp as vital, but 
at the same time it may be diseased just as a person may be alive and 
at the same time be sick.

Fohto and Scheinin microscopically used thin dentin windows in 
rat incisors and observed the circulation in the vital pulp. They 
found that retraction stress applied to the incisor could stop blood 
circulation. Electrical stimulation of the cervical sympathetic nerve 
was found to arrest the flow of blood in the pulp. Taylor in using 
a similar technic found almost the same results. He adds that his ob-
servations suggest that there may exist some special mechanism for 
controlling blood circulation in teeth. Kozam and Burnett noted 
that the incisal capillaries were the most easily damaged of all pulpal 
vessels. Provenza and Biddington found that vasodilatation of all
muscle bearing components of the vascular system in the pulp can be affected by nitroglycerin. Also, they found that vasoconstriction can be induced by topical application of epinephrine. Because of the alteration of the blood supply by these drugs they suggest the possibility of their use in pulp disease. Coy mentions that anesthetized pulps do not hemorrhage as freely as those not anesthetized. He attributes this to a restriction of the blood flow due to the action of vasoconstrictor in the anesthetic. Hellner claimed that local injections of procaine-epinephrine for dental purposes can cause a reaction hyperemia and hence increase the likelihood of pathological processes in the pulp.

Zander describes two types of phagocytes in the dental pulp. One he calls the microphages which he considers to be the polymorphonuclear leucocytes, the activities of which are usually confined to smaller particles. The other group he calls macrophages which are characterized by their ability to take up large particles. Orban describes the presence of the "cells of defense" in the pulp. He has shown histiocytes and macrophages in human pulp. He describes how resting wandering cells are gradually transformed into round mobile elements by the retraction of their long branching cytoplasmic processes and assume the characteristic features of macrophages. Orban emphasized that like other connective tissue the pulp will overcome some types of injury. Adams states that phagocytes will quickly destroy bacteria when circulation is present. He summarized the pulp defense mechanism as consisting of several complicated processes, the purpose of which is to destroy the microorganisms, neutralize the toxins and remove foreign matter.
The entire basis of vital pulp therapy rests upon the fact that the pulp, under certain circumstances, can repair itself. However, it is interesting to note that as late as 1928 Grove doubted if the pulp could heal. He thought that it was impossible for the odontoblasts to regenerate, hence, pulp capping would be impossible. Zander expressed belief that nature has a way of repairing damage to odontoblasts. He believes that the undifferentiated mesenchymal cell and the young fibroblast are capable of changing their function and form, and migrating through the layer of Weil and transform into odontoblasts. Zander has also mentioned that the pulp tissue in a young patient is more embryonic in nature and probably can differentiate into odontoblasts better. Teushner and Zander were among the first to demonstrate histologically that healing takes place over root stumps in pulpotomized teeth. They showed the actual formation of dentin-like material by what appeared to be odontoblasts. The odontoblasts were undoubtedly regenerated from cells of the pulp.

**Pulp Pathology**

No discussion of pulp pathology should be made without focusing attention on the dynamic process of inflammation. Boyd defines inflammation as the local reaction of the body to injury. Barr mentions that all of the cardinal signs of inflammation occur in the inflamed pulp; however, he mentions that only pain and disturbances of function are recognized unless the periapical tissue is involved.

The symptoms of inflammation are due to the vascular changes which occur in the following sequence. (1) A brief constriction of the vessels which is followed by dilatation. (2) Temporary acceleration of the blood stream which is followed by deceleration. At this stage vascular dilatation is very great and many capillaries which were
at rest are filled with blood. Pain is caused by extravascular pressure on the nerve endings. If the injury is sufficiently great there may be complete stoppage of circulation. The result is necrosis of the part supplied by the occluded vessel. Loss of function is due partly to pain and partly to tissue destruction. (3) The white blood cells instead of flowing intermingled with the red blood cells, line the walls of the vessels. This is called pavementing or margination of the leucocytes. (4) The leucocytes pass through the vessel walls by an ameboid movement or diapedesis. The vascular changes are not the entire story because wandering cells also act against the irritant. It is necessary for both the white blood cells and the blood plasma to escape from the interior of the vessels in order that they reach the irritant. This escape is brought about by the vascular phenomena described above.

Barr believes the most important cause of inflammation in the pulp is bacteria. Bacteria may enter the pulp by (1) direct invasion from the mouth, i.e., caries, fractures, etc. or (2) through lymphatic channels associated with periodontal disease, or (3) by way of the blood stream. Robinson and Boling point out the possibility of anachoresis as a cause of pulpitis. Hill mentions that hyperemia may be produced by thermal shock, but there is evidence that bacterial invasion is the most important etiological factor in the death of pulps.

Shroff divides the changes of the pulp into active and passive changes. Under the active changes he lists inflammation. He reports that in both acute and chronic inflammation there is an increase in local cell population. In acute inflammation, the increase in volume of the extravascular fluid is considerable, while in the chronic it is slight. The fixed volume of the pulp cavity cannot support the great
increase in the visiting cell population which accompanies such an inflammatory change. This however is partly offset by death of local pulp cells. He mentions that the ability of the pulp to survive an inflammatory reaction of this type is almost entirely dependent upon its capacity to deal with the great increase in its fluid content. This capacity is in turn dependent upon the nature of the blood supply and lymphatic drainage, physiological nature of the periapical tissue, the size of and number of the peripheral foramina and the presence or absence of an opening into the oral cavity.

Barr reports that as soon as any marked enlargement of the arteries occur, which he calls active hyperemia, there occurs a strangulation of the veins. This strangulated state he notes is sometimes called passive hyperemia. He prefers not to differentiate between the two types of pulp hyperemia. Boling and Robinson have shown that much discussed "self strangulation" of the pulp is invalid because, (1) the pulp may have several afferent and efferent vessels and separate foramina; (2) this theory does not explain the changes in a multi-rooted tooth and (3) the pulpal blood flows through a closed hydrostatic system and the pressure in the vein will soon equal that in the artery. In inflammation the permeability of the vessel walls increase and the osmotic phenomena tend to cause edema. The edematous cells enclosed in a rigid dentinal box can swell only against vessel walls and retard the flow of blood and perhaps lead to thrombosis.

Schröff points out that the increased hydrostatic pressure also affects the pulp cells per se. He continues, that in chronic inflammation there is a less acute increase in cell population and a minimal amount of fluid exudation takes place slowly over a considerable period of time. These changes are not sufficient to cause a wide spread death
of all cells, but they are sufficient to interfere with the more complicated system of specialized cells. In most tissues, therefore, chronic inflammation is typically characterized by degeneration of specialized cells and proliferation of the more humble fibroblasts. In the closed dental pulp proliferation of any extent is rendered impossible by the limits imposed on the cell population due to the limited space available. Degeneration of specialized cells (odontoblasts) proceeds, but instead of proliferation of the fibroblasts they gradually atrophy over a long period of time. In multi-rooted teeth this atrophic process may be extremely slow and one canal may become completely atrophic, while others maintain life. Occasionally hyperplasia does develop in a closed pulp. When this occurs, however, the proliferation process is accompanied by enlargement of the pulp cavity through the activity of osteoclasts which resorb the hard dentinal walls. Shroff mentions that if an opening into the oral cavity exists early in the process of pulpitis, the pulp may survive a considerable degree of injury and manifest less pain on account of the absence of pressure. In this case inflammation may then have some of the beneficial effects that it has on other injured lesions. Fortunately most inflammation of the pulp commences as a chronic form. If an opening is established before the tissue has sustained a great deal of damage, there is a release of pressure, subsidence of pain, drainage of metabolites and autolytic products, and the tissue may enter a reparative phase. Such pulpitis with an opening may take two forms: (1) chronic ulcerative, or (2) chronic hyperplastic. In the chronic ulcerative type the destructive influences predominate, while in the latter the proliferative phenomena are dominant. In both cases the pulp remains vital, though the prognosis
of the ulcerative type is less favorable. In the ulcerative type the picture is similar to a chronic ulcer found in other tissue. Hyperplastic pulpitis commences as the ulcerative type, but owing to the mild nature of the irritant and a better internal state of the pulp, there is proliferation of granulation tissue through the opening.

Sommer and Crowley report that the first reactions to injury of the pulp are a disturbance of the circulatory vessels together with the usual degenerative changes manifested in the form of calcium deposits throughout the pulp tissues. Zander states that if the inflammatory response is acute we find a predominance of polymorpho-nuclear leucocytes. They may be so numerous that we can speak of localized abscesses. If the reaction is a chronic one, the plasma cells and the lymphocytes are the predominating cells found in the pulp near the irritation.

Several investigators have pointed out that exposed dentinal tubules will cause irritation of the pulp. Ross noted that upon exposure of the pulp tissue, the pulp is immediately infected. There is copious extravasation of plasma from the walls of the blood vessels followed by the migration of polymorpho-nuclear leucocytes, histiocytes and round cells into the tissue. As a result the organisms become effectively confined by the leucocytes to a small area in the pulp around the exposure and this area is walled off by round cells, while the rest of the pulp remains histologically normal. If the carious exposure becomes large, allowing the escape of inflammatory exudate, the localized abscess may remain for some time. However, Ross states that a sudden invasion of organisms into the barrier of leucocytes as a result of trauma during mastication would
quickly produce an acute inflammation from which the pulp could not recover.

Bender, Seltzer and Kaufman suggest that bacteria can penetrate the dentinal tubules. They recommend avoiding excessive pressures and heat so as not to force microorganisms into the pulp during operative procedures.

Thoma believes that certain microorganisms can infect a dental pulp leaving it chronically infected for long periods of time. He suggests that such a tooth may be a factor in focal infection.

Kreshover and Bevelander studied the pulp tissue of dogs' teeth following exposure. The pulp tissue showed a gradation of inflammatory response ranging from acute to chronic, characterized by necrosis, abscess formation, vascular response with exudation and granulation tissue formation proceeding to complete fibrosis.

Fischer illustrating that the clinical findings may not reflect the histological picture of the pulp, mentions an example of a symptomless tooth. Histologically most of the pulp was normal; however, secondary caries was noticed under a filling. Beneath the caries was a layer of adventitious dentin and beneath this in the pulp there was extreme vacuolization. Around this area were seen dilated vessels and cellular infiltration. Numerous round cells, histiocytes and polymorphonuclear leucocytes were recognized. Fischer interpreted these as being the first changes due to the toxic effect of the bacteria from the progressive carious process.

Urban has said that in conditions of acute inflammation the leucocytes may migrate into the dentinal tubules. Eosinophils, as well as neutrophils could be observed migrating into the tubules.
Langeland found that inflammatory cells appear in the pulp of clinically intact teeth and he explains that their presence is probably caused by undetected caries. He believes that the migration of the odontoblast nuclei into the dentinal tubuli must be considered the initial reaction to injury.

Methods of Determining Pulp Vitality

Before taking up the individual methods of determining pulp vitality, some general concepts should be considered.

It seems generally agreed that no single diagnostic technique can accurately indicate the extent of pathology in a pulp. 

Barr mentions that hyperemia is characterized by a sharp pain lasting but a moment. It does not occur spontaneously and does not continue after the cause is removed. In acute pulpitis the pain is more severe, sharp, stabbing and it lasts longer. A definite characteristic of pulpitis is that the pain is intermittent and it does not have to be initiated by a stimulus, as in the case of hyperemia.

The time necessary for an uncomfortable sensation to disappear after the application of a test stimulus is an indication of the condition of the pulp. If it disappears promptly, the pulp is probably normal. If the uncomfortable sensation disappears gradually, the pulp is likely to be diseased.

Lowenstein and Faivovich compared the excitability of normal teeth and those with pulpitis. They found no significant difference between the two groups.

Herbert explains that an unusually quick response may mean hyperemia or an acutely inflamed pulp and that a diminished response may mean a thick layer of secondary dentin, interstitial calcification,
or the destruction of a portion of the pulp from an inflammatory process. He reports that the dental pulp usually will continue to give a vital response as long as it contains some vital tissue. It is not uncommon for a pulp containing a chronic abscess to give a normal response.

It is well recognized that there is a great deal of difference between the way two individuals may respond to a stimulus. These variations are due to a number of factors such as phobias, environment, experiences, etc. The individual variation should be kept in mind during pulp testing procedures.\textsuperscript{4,22,51}

Schaffer\textsuperscript{1} mentions that one cannot expect to get accurate results when testing deciduous teeth.

Many workers have reported that multi-rooted teeth may have one canal non-vital and the others vital, thus causing inaccuracy in pulp testing.\textsuperscript{2,48,52,53}

A. The Electric Pulp Test - Schaffer\textsuperscript{1} reports that Magitot in 1867 advocated the use of inducted current to localize carious teeth. He cites both John S. Marshall in 1891 and Woodward in 1896 as using electricity as a means of demonstrating vitality and non-vitality of the dental pulp. Schaffer\textsuperscript{1} also reports that a non-vital tooth, with or without fillings, which has an excess of moisture on the crown during testing will usually respond. The current in such an instance is short-circuited by the presence of moisture along the crown to nerve fibers of the periodontal membrane. He further mentions that false responses are much higher in multi-rooted teeth than they are in single-rooted teeth.

Many workers have reported that secondary dentin and degenerative changes of the pulp will cause a higher threshold of response.\textsuperscript{4,22,52,54,55}
Ziskin and Zegarelli found that the periodontal membrane is an irritable tissue and is capable of stimulation by electricity. Stimulation of this tissue may be a source of error in pulp testing. They recommend a current-measuring device instead of the voltage-regulating type of apparatus to distinguish between the stimulation of the periodontal membrane and that of the pulp tissue.

Reiss and Prieblu found that there is no normal or average that can be applied universally in electrical testing of pulps.

Ziskin and Wald suggested that electrical frequencies of one thousand to five thousand seemingly separate the pulpal from the periodontal membrane responses, this range being favorable for responses of the pulp tissue.

Cartledge et al suggest that if a tooth is symptom-free, it usually means a normal pulp; however, at certain stages of acute and chronic pulpitis, it may give a normal reading to electric pulp tests. They conclude that the electric pulp test will not aid in diagnosing chronic pulpitis.

Cooke and Rowbotham found that the absence of any reaction or even a doubtful reaction at a high reading invariably indicates the death of the pulp. They found that a dead tooth may give a "pins and needles" response to a maximum stimulus. This means that the periodontal tissues have been stimulated. Also they found that in a certain number of young children there has been no response to the pulp tester, even though the pulps were normal. They relate that such teeth show widely open spines on the radiographs. Frequently these are upper anterior teeth in the first year after eruption. They suggest that an explanation might be that the innervation of the dental pulp occurs in the later
stages of root development. They further mention that whenever possible, the electrode should be applied to sound enamel.

B. The Use of Cold for Pulp Testing - Green states that ice will afford relief from pain when the pulp is putrescent and there is little or no reaction to stimulation in chronic ulcerative pulpitis. A totally gangrenous pulp will give no response to ice.

Mitchell mentions that the non-vital tooth will give no response to ice. The tooth with pulpitis may give an extremely early response. This response may be relieved upon removal of the ice. However, the ice may induce pain which persists long after its removal.

Austin and Wagganer advocate the use of ice in the form of pointed cones as an accurate means of testing pulp tissue. They mention that ice is a substance known to all patients, therefore, its application to teeth does not cause anxiety. They state that it is probably the most useful of all means in examining children's teeth.

Via points out that false negative responses are possible with ice and the greater the amount of adventitious dentin formed, the greater the chance for the false negative response. However, he emphasizes that false positive responses are impossible.

C. The Use of Heat in Pulp Testing - Herbert suggests that heat is perhaps the most reliable pulp test, although a positive response may be obtained from expansion of inflammatory products and from pressure upon vital tissue remaining in the pulp chamber, or upon periapical tissue.

Nyborg believed heat might be a more reliable test than cold.

Mitchell mentions that heat may induce pain stimulating the toothache and help one to locate the offending tooth. He also suggests using hot water or a hot wax spatula for certain testing procedures.
Via suggests that a normal pulp does not react as well to heat as it does to cold. He mentions that the greatest number of false negative responses are obtained by the use of heat. He emphasizes that probably the greatest value of heat in pulp testing is in the differential diagnosis of the more advanced pathological conditions. A pulp undergoing necrosis caused by bacteria is a good example; an increase in pain with heat will be relieved by the application of cold.

D. Radiographic Interpretation and the Diagnosis of Pulpitis - Thomas in 1928 claimed that, as seen in roentgenograms, a heavy deposit of adventitious dentin in the pulp canal and enlargement of the periodontal membrane space can be aids in making the diagnosis of pulp pathology. In 1929, he expressed the opinion that the roentgen picture is the most reliable means of diagnosing pulp infections and that they will show secondary decay under fillings, as well as periapical radiolucencies.

Ross claims that bone resorption will always occur in those areas which are contaminated by the toxic products of bacteria and never at the place where the bacteria are located. Hence, a blurred or uneven surface of the periapical bone with the absence of an intact lamina dura will indicate bacteria in the canal and a discharge of their toxic products into the periapical tissue. In such a case the thickening or sclerosis of the bone may also be seen.

Sommer and Crowley concluded that many types of bone characteristics which appear to be abnormal may be normal for certain individuals or certain specific regions. They also concluded that too much emphasis should not be placed on the roentgenogram as a diagnostic aid.

Periapical involvement often is seen as a thickening of the periodontal membrane at the root apex. Also a zone of sclerotic bone may sometimes be seen. This indicates that the infection is a slowly progressive one of long standing.
B. Other Diagnostic Tests and Signs — Barr emphasizes that in both hyperemia and pulpitis the teeth respond normally to percussion tests. Mitchell explains that tenderness to percussion, carefully evaluated and compared from tooth to tooth, indicates inflammation of the periodontal membrane and most often is a sequel to pulpitis or pulp death. Shafer et al concur that the first evidence of pulpal inflammation spreading beyond the confines of the tooth pulp may be a noticeable sensitivity of the involved tooth to percussion.

Schaffer concludes that discoloration should be observed and noted; however, it is not necessarily of great importance in diagnosing the condition of the pulp.

Studies Correlating Clinical Examinations and Histological Sections of Teeth

Thoma in 1929 correlated roentgen pictures and clinical histories of teeth with microscopical findings. He noted that in fourteen cases of adult patients the pulp inflammation was of the chronic type. The symptoms generally were indefinite and occurred only occasionally. The microscopic picture showed that the inflammation was characterized by the infiltration of mononuclear cells. The largest number of cases were caused by secondary caries under fillings. He felt that the roentgen picture is the most reliable means of diagnosing pulp infection.

In 1933 Reiss and Fureidi attempted to correlate the significance of the pulp test with the microscopic study of one hundred and thirty teeth. They found that out of fourteen cases of very marked pulpal fibrosis and calcification, ten had given a normal response to the electric test. Also, that teeth with large calcific depositions in otherwise normal or moderately atrophied pulps did not give any unusual response. In teeth testing normal, it was found that 63.5% were found to be practically normal and that 36.5% were pathological. In teeth with
moderately high thresholds, 69% were shown to be normal and 31% pathological. Teeth which responded only with a very high threshold were found to be pathological in 90% of the cases. Of the teeth which gave a negative response, 100% proved to be pathological. They observed that teeth which responded painfully to stimuli were almost always pathological. They concluded that a tooth which failed to respond or only responded at a high threshold was most likely to have a necrotic pulp or the root canal filled by root canal therapy.

Stephan in 1937 correlated clinical tests with microscopic studied of thirty-seven teeth. He found that the normal dental pulps are without painful symptoms and give a response to the electric tester at a variable index. He found that pulps which were markedly atrophied and degenerated showed no evidence of inflammation. These pulps responded normally to all tests and manifested no painful symptoms. Stephan believed this to show that regressive and degenerative changes of the pulp cannot be diagnosed clinically by his methods. He found that the acutely inflamed pulps give painful symptoms and may give average reactions to hot and cold, but generally these pulps give severely painful reactions which last longer than the normal reaction. To the electrical stimulus these teeth generally respond normally, but this may vary. Stephan noted that the chronically inflamed pulp generally gave no severe symptoms and it was usually exposed by caries. He mentions that probably vascular changes produce painful symptoms which may vary considerably. He concludes that a necrotic pulp gives no response to heat and cold and that if the contents of the canal are liquid it might respond to the electric test.
Herbert in 1945 attempted to correlate the clinical signs and histologic conditions of fifty-two teeth. He found that twenty-one of the fifty-two cases were chronic pulpal abscesses. The majority of these were associated with exposures of the pulp. Of the twenty-one chronic abscesses, nine were without symptoms; nine gave a history of pain lasting for various periods of time when stimulated by heat, cold, or pressure of food in the cavity. Three ached without stimulus. Thus a small chronic abscess may easily pass unsuspected or give the symptoms of hyperemia. He noted that pulp stones occurred in fifteen out of the fifty-two cases and relates these as a response of the pulp to irritation.

In 1955 Prophet and Miller correlated the type of pain and the duration of pain in deciduous teeth with the histologic picture. They concluded that the signs and symptoms bear no precise relation to the pathological condition of the pulp and that the onset of pain indicates that marked inflammatory changes have been taking place in the pulp. They believe that the invasion of secondary dentin by bacteria and their products results in inflammatory changes in the pulp and the onset of pain; also, that the penetration of the secondary dentin and infection of the pulp can result in severe local inflammatory changes with subsequent involvement of the whole pulp. They found that pain in a deciduous tooth indicates a dying pulp and that the absence of pain does not insure a healthy pulp.

Ishibashi compared the clinical diagnosis with histological diagnosis of pulp disease in 136 deciduous teeth. The clinical diagnosis of hyperemia of the pulp in fifteen teeth was confirmed histologically in 6 teeth, or in 40%. The clinical diagnosis of acute simple pulpitis in 29 teeth was confirmed in 10 teeth, or 34% of the
cases. Of the 23 teeth in which acute suppurative pulpitis was
diagnosed clinically, 12 were proved so by histological examination,
or 52%. Twenty-eight were diagnosed clinically as having ulcerative
pulpitis and 11 were confirmed by the sections for a 39% accuracy.
The 3 instances of chronic hyperplastic pulpitis were correct. Of 40
teeth clinically diagnosed as having pulp gangrene, histologic
diagnosis was confirmed in 31, or 77.5% of the 40. He concluded that
diffuse spontaneous pain often indicates extensive enlargement of
acute inflammation of the whole pulp, but this is not always true.
Local spontaneous pain is not necessarily an indication of slight
changes in the parts of the pulp. The pain felt by patients upon
thermal stimulation of the pulp does not provide an accurate method
for the diagnosis of pulpitis. The value of percussion in diagnosing
pulpal diseases was found to be less dependable than previously thought.
The gangrenous odor provides good grounds for diagnosis of a gangrenous
change in the pulp. He concluded that the tendency for lack of con-
formity between histological and clinical diagnosis is much greater in
instances involving deciduous teeth than in permanent teeth.

Indications for Vital Pulp Therapy

Teuscher and Zander\textsuperscript{38} reported successful healing and dentin
bridging in pulpotomized root stumps in 1936. They hoped that with
improvement of technics and selection of cases, vital pulpotomy would
become a useful means of saving chidren’s teeth. Since that time a
search for more adequate diagnostic aids has been conducted.

Rosenstein\textsuperscript{62} in 1942 reported 90% success in a series on pulp
capping in which he evaluated his teeth by clinical signs and symptoms.
He emphasized that selection of cases was probably more important for
success than the capping material. At this time he pointed out two
important factors in case selection: the exposure site should be sensitive to touch and should bleed. In 1949 he stated that proper evaluation of the status of the pulp is necessary. He emphasized at this time four criteria which should be present before capping an exposure. These included a slight hemorrhage and sensitivity at the exposure site, normal color of adjacent tooth structure, and a small or moderate size of exposure.\textsuperscript{63}

Herbert\textsuperscript{51} in 1945 mentioned that, if softened dentin comes away in one piece leaving the pulp exposed, the pulp is clearly unsavable. Teuscher\textsuperscript{61} in 1948 mentioned that for vital pulp therapy there should be little hemorrhage from the exposure site. Also, that when doing a pulpotomy, the pulp should exhibit a clean, glistening appearance when the roof of the chamber is removed.

Orban\textsuperscript{65} in discussing vital pulp therapy, mentions the complexity of the problem. He believes that when a pulp is inflamed, one cannot expect a favorable reaction in a high percentage of cases.

Wittich\textsuperscript{66} in 1952 claimed 87\% success in capping exposures. He emphasized that he capped only teeth which had a small exposure and no history of pain except perhaps when eating; and which, after exposure, bled and were painful at the exposure site.

Patterson and Van Huyse\textsuperscript{67} in 1954 pointed out in 1954 that for successful capping the pulp must be healthy and must not be permitted to dry or be injured by chemical irritants. Van Huyse\textsuperscript{68} in 1955 stated that a pulp exposure which is preceded or accompanied by a toothache should not be capped. These are signs of pulp inflammation and one should not attempt to cap a pulp with any degree of pulpitis.

Ireland\textsuperscript{23} mentioned that there is a direct relationship between the health of the child and the ability of the pulp to form secondary
dentin. Clickman showed that systemic disturbances could severely affect the health of the dental pulp. He noted that certain systemic illnesses, such as alloxan diabetes, starvation, protein deficiency, induced in animals would cause the pulps of carious teeth to show degenerative changes. Kozam and Burnett mentioned that the pulpal circulation was invariably subnormal in animals which were obviously unhealthy.

Via in a study which did much to cause vital pulp therapy enthusiasts to re-evaluate their methods, reported a success of only 31.1% in pulpotomized teeth. He mentions that the most important factor in increasing the chance of success is the determination of the exact condition of the pulp tissue at the time of the operation.

McDonald in January, 1956, emphasized that diagnosis is much more important than the material used in vital pulp therapy. He mentioned seven items which should be considered in establishing a diagnosis: (1) history of a toothache, (2) radiographic examination, (3) size of the exposure, (4) amount of hemorrhage, (5) age of the patient, (6) pulp testing, (7) the physical condition of the patient.

In July, 1956, McDonald mentions that we cannot tell the true condition of the pulp from the roentgenogram. He noted that when the pulp shows calcified masses in the chamber, it indicates pulpal degeneration. This can be found in teeth which have never ached. He describes the success of vital pulp therapy as dependent upon accurate preoperative diagnosis. He found that the electric pulp test was not reliable in determining a pulpal exposure. He suggests that pinpoint exposures may be associated with a mild, probably reversible degree of inflammation. He observed that profuse hemorrhage from exposed pulps is evidence of extensive inflammation and hyperemia and is a contraindication to vital pulp therapy. He concludes that children with
chronic illness or lowered resistance should not be considered for
vital pulp therapy, and that one procedure should not be adapted for
treatment of all pulp exposures.

Messerer et al.73 mention that there are indications that pulpal
healing following amputation occurs more frequently in young permanent
teeth than in primary teeth. They believe that internal resorption
occurs more commonly in primary teeth than permanent teeth. They con-
clude that age is not a valid contra-indication to the undertaking
of vital pulp therapy.

McDonald74 in 1959, states that it is not uncommon to find a
badly degenerated deciduous pulp and no history of pain. He reviews
a number of factors which should be considered before deciding to
undertake conservative treatment. He believes that a history of a
toothache at night, or spontaneous pain, means that the degeneration
has gone too far. The pinpoint size of exposure should be considered
the most favorable for treatment. A thickening of the periodontal
membrane and/or excessive bleeding of the exposure, he considers to
be contra-indications for vital pulp therapy. McDonald concludes that
the physical condition of the patient should always be considered.

Shroff2 seems to summarize the entire problem by stating, "It is
obviously of paramount importance to know the state of the tissue we
are attempting to treat".

The White Blood Cells

Langley and Cheraskin75 describe the white blood cells as follows.
The leucocytes are larger than the red blood cells and contain no
hemoglobin. They do contain a nucleus and are classified by their
nucleus, the granular or agranular character of their cytoplasm and
the staining properties of the cell.
The lymphocyte (Fig. 7) is a small cell with a dark staining nucleus which occupies the greater portion of the cell. Around the nucleus may be seen a small area of clear cytoplasm which is basophilic. Because of the clear or agranular cytoplasm, this cell represents one of the two kinds of agranulocytes.

The monocytes are, also, agranulocytes. They are different from the lymphocytes, in that they are larger, their nuclei tend to be kidney-shaped and there is a greater portion of clear basophilic cytoplasm.

The neutrophils or polymorphonuclear leucocytes (Fig. 7) are the more common of the granulocytes. They are large cells and each has a multi-lobed nucleus. Most commonly, they are referred to as "polys".

In addition to the neutrophil's multi-lobed nucleus the cytoplasm is granular and it stains a light purplish color because it takes up both the acid and the basic stains.

The eosinophil resembles the neutrophil, in that it has a many-lobed nucleus. It differs from the neutrophil, in that the granules take an acid stain. Consequently the cytoplasm granules are bright pink.

The basophil is a polymorphonuclear granulocyte, the granules of which have an affinity for the basic stain. They are the least common of the leucocytes.

The prime function of the leucocytes is one of protection. They are capable of destroying invading microorganisms and phagocytosis. The neutrophil is the most active of the phagocytes. The white cells pass through the capillary wall by diapedesis. They are attracted to the site of invasion and surround the foreign substance and destroy it. They then carry off the debris.
The part played by each individual type of cell has not been clearly determined. It has been demonstrated that the neutrophils increase in number during certain bacterial infections. The eosinophils increase in number during certain parasitic infestations and allergic states. The basophils continue to remain a mystery. There still is no particular situation which excites basophils enough for their number to increase in the circulatory blood.

Zoethout and Tuttle mention that phagocytosis and diapedesis are characteristic of neutrophils and of the monocytes. These cells perform their function only after they have left the blood stream. They engulf bacteria and debris. The phagocytic leucocytes may be killed by toxins from the bacteria which they engulf. These dead leucocytes plus erythrocyte debris make up pus. They further mention that lymphocytes do not function as phagocytes, but probably function in the formation of immune bodies.

Stern mentions that all of the white blood cells are found in the capillaries of the pulp. The granular neutrophils are considered to be the active agents in acute inflammatory reactions.

The Pulp Hemogram

Prader in 1949 advocated the local pulp hemogram as a diagnostic aid for vital pulp therapy. He recommended using the first drop of blood from the exposure site in preparing a blood smear. From this blood smear a white blood cell differential count could be made. He claims that the white blood cell differential count can be used as an index to the health of the pulp tissue. He mentions that the white blood cell hemogram of the intact healthy pulp without inflammation is identical with the circulating blood of the patient. He claims that with a pure toxic inflammation of the pulp we see lymphocytosis and a
monocytosis. Thus the amount of neutrophils stays constant, but later may decrease somewhat. He believes such a blood picture justifies vital amputation. The increase in monocytes with a constant or decreasing number of neutrophils he calls a "monocytic defense phase." He mentions that as the first microorganisms reach the pulp, the amount of neutrophils slowly rise without an increase in the band cells. He advocates that clinically such a pulp is suitable for devitalization, while a slight increase in neutrophils is suitable for amputation. He mentions that if the invasion of the pulp is stronger, or of longer duration, the picture changes considerably. In this phase the number of segmented leucocytes increases rapidly. He further emphasizes that qualitative changes of the neutrophils are noted in this phase and nearly every cell shows vacuolization. Also, he noted that the nucleus many times breaks into numerous separate nuclei. He concludes that these pulps are unsuitable for amputation. Prader advocates that pulp hemogram as a means of pulp diagnosis and an important sign in determining the type of treatment to be given to the pulp.

Bevilaqua in 1958, claimed to have corroborated Prader's findings with the pulp hemogram. He states that massive neutrophil counts are interpreted as a sign of tissue breakdown and suppuration. He noted that perhaps more significant than the quantitative changes were the qualitative alterations of the neutrophils. The presence of toxic granulations, vacuolization of the cytoplasm and nucleus, chromatin changes and atypical nuclear segmentation, are all indicative of a severe toxic process and suppurative degeneration. He noted, as Prader had, that when the mononuclear forms (lymphocytes and monocytes) predominate, that this denotes a favorable response to vital pulp therapy. He states there is evidence that examination of the exudate will give a picture of the pulp status, but he fails to mention what this evidence is.
STATEMENT OF THE PROBLEM
Statement of the Problem

The local pulp hemogram, although recently advocated as a diagnostic aid in the selection of teeth for vital pulp therapy, has never been compared with the histologic picture of the same pulp tissue. This seemed to be the logical method of determining the diagnostic value of the local pulp hemogram. The purpose of this investigation was to determine if any relationship existed between the white blood cell differential count obtained from the first drop of blood from an exposed pulp, and the histologic condition of the pulp tissue. Another purpose was to compare the white blood cell differential count from normal pulp tissue and from the peripheral blood. In addition, an attempt was made to note any relationship between the clinical signs, the dental pulp hemogram, and the histologic picture of the tooth.
MATERIALS AND METHODS
Materials and Methods

The sample for this study consisted of 44 primary and 9 permanent teeth with carious pulp exposures. Also included were 14 primary and permanent teeth with normal pulps which served as controls in the blood study portion of the experiment. The teeth were removed from 27 boys and 13 girls between the ages of 4 and 11 years.

The author selected for extraction and observation carious teeth which soon would be exfoliated; those indicated for removal because of discomfort to the patient; and those which were indicated for removal during the course of orthodontic therapy. All of the intact, non-caries teeth included in the control group for blood study comparison were slated for extraction prior to the correction of irregularities in the occlusion.

Before carrying out any operative or surgical procedures the parent and the child were interrogated to determine if the tooth had been painful in the past. The exact type of pain, if any, and the duration were recorded.

A pulp test was carried out on each tooth included in the study in an effort to determine the true status of the pulp. It was felt that it would be interesting to attempt a comparison between the results of the various pulp tests and the histological picture of the pulp.

A small piece of ice was used on the teeth to determine if the tooth was sensitive to cold. A stick of gutta percha was flamed and used in testing the tooth's response to heat. The results of these two tests were recorded as either a vital or a non-vital response. An electric uni-polar vitality tester (Ritter) was used to test the tooth in question. The response was recorded as vital or non-vital.
and usually whether the response was in a high, medium, or low range. While using these various vitality tests the tooth was compared with a similar normal tooth for a control.

The tooth also was subjected to percussion with a metal instrument and the reaction was recorded. The tooth in question was subjected to finger pressure and it was recorded whether the tooth was mobile or non-mobile, when compared to a control tooth. The preoperative radiograph for each tooth was observed and any sign of pathology was recorded.

The tooth was anesthetized, isolated with the rubber dam and the decay removed. Whenever possible the caries removal was accomplished with a spoon excavator, although occasionally a rotary instrument was used to gain access. In the normal tooth, without clinical decay, the pulp was exposed entirely by a rotary bur instrument (Fig. 10). Upon exposure of the pulp and the appearance of the first drop of blood, cotton pliers were carried into contact with the blood (Fig. 2). The blood was attracted between the beaks of the cotton pliers and was then carried to the end of a clean glass slide upon which it was allowed to stand (Fig. 3). It was not uncommon to transport several droplets of blood from the cavity to the slide before obtaining a suitable amount.

Next a second glass slide was held at a thirty degree angle against the original horizontal slide on which the blood was standing and the blood was spread evenly over the surface of the slide. The blood smear was allowed to dry (Fig. 4). Then it was stained with Wright's stain (Fig. 1). Immediately after preparing a blood smear from the dental pulp a second smear from the patient's finger was made. The patient's finger was cleaned with alcohol, pricked, and the resultant hemorrhage was used to make a stained blood smear by
the identical procedure previously described (Fig. 5 and 6). Thus a blood smear was obtained from the peripheral blood which could be compared with the local dental pulp smear.

Subsequently, the stained blood smear slides were studied microscopically and 100 white blood cells were counted (Figs. 7, 8 and 9). While counting these, a notation was made as to the number of each type of leukocyte so that a percentage of the total white blood cell count was obtained for each leukocyte type. Thus, the author obtained a white blood cell differential count for the exposed bleeding pulp and for the peripheral blood. A comparison between the two differential counts was made and the findings were recorded.

Immediately after obtaining the blood smears, the tooth in question was extracted. The apical one-third of the roots of the tooth were removed with a diamond disc to aid in adequate fixation. The tooth was then placed in a 10% formalin solution.

Later the teeth were decalcified in 5% formic acid. The teeth were trimmed to facilitate sectioning through the area of pulp exposure. They were then embedded in paraffin and four to eight sections, 6 to 10 microns thick, were cut. The sections were stained with hematoxylin and eosin and prepared for microscopic observation.

The histopathologic findings were then noted and compared with the changes of the white blood cell differential counts and with the various clinical signs and symptoms.
Figure 1. The armamentarium used in preparing the stained blood smears.
Figure 2. The cotton pliers being applied to the first drop of blood from an exposed pulp.

Figure 3. The transfer of the blood from between the cotton plier beaks to a clean glass slide.
Figure 4. The blood smear after it has been spread and allowed to dry.

Figure 5. The lancet used in making a finger stick.
Figure 6. A drop of blood from the finger stick which was utilized in making a peripheral blood hemogram.
RESULTS
Results

The histopathological findings of 53 teeth with carious pulp exposures were evaluated. The teeth were divided into two groups. One group, in the opinion of the author, would respond to vital pulpotomy and maintain vitality. The second group was thought to have undergone degenerative changes to the extent that the procedure would not be indicated. The criterion used for this evaluation was the extent of inflammatory cell infiltration of the dental pulp. If the inflammatory cells extended into a root canal beyond the point where we normally amputate during pulpotomy, the tooth was considered as one which would not have been a good candidate for vital pulpotomy. (Fig. 12 and 13). If the inflammatory cells were localized in the pulp chamber, the tooth was considered as a "good candidate" for vital pulpotomy. (Fig. 11 and 14). These two groupings will be referred to as "good candidates" and "poor candidates" throughout the remainder of this paper.

In the "poor candidate" classification there were 21 deciduous and 4 permanent teeth making a total of 25 (Table I). In the "good candidate" group there were 23 deciduous and 5 permanent teeth, making a total of 28 teeth (Table II).

Blood Findings

The blood findings were evaluated using the white blood cell differential count (hemogram) of the peripheral blood as a base line. Arbitrarily a rise of 10% in the number of lymphocytes or neutrophils was considered as a significant change. This amount of increase is considered as evidence of significant change in hematological studies. When the dental pulp hemogram did not show a rise of 10% or more in the number of lymphocytes or neutrophils, it was considered
to fall within the range of normal, and such a finding was classified as normal. When the dental pulp hemogram showed a rise of 10% or more neutrophils, it was classified as a plus neutrophil count. When the dental pulp hemogram showed a rise of 10% or more lymphocytes, it was classified as a plus lymphocyte count.

In the "good candidate" group 10 teeth had a normal count, 18 had a plus lymphocyte count, and none had a plus neutrophil count, (Table II).

In the teeth classified as "poor candidates" 9 had a normal count, 7 had a plus lymphocyte count, and 9 had a plus neutrophil count, (Table I).

Fourteen non-exposed histologically normal teeth were evaluated by the pulp hemogram (Fig. 10). Seven showed a plus lymphocyte count and 7 a normal count.

**Amount of Hemorrhage**

The amount of hemorrhage from the exposed pulp was arbitrarily evaluated by the author as slight, moderate, or profuse. An exposure which did not bleed or in which only a drop or two of blood appeared was classified as slight hemorrhage. Where several drops of blood appeared from the exposure, but the bleeding quickly ceased, it was classified as moderate hemorrhage. When the bleeding quickly filled the cavity and continued to bleed extensively, it was classified as profuse hemorrhage (Fig. 16).

In the "good candidate" group, 12 teeth bled only slightly, 12 were classified as moderate hemorrhage, and 3 were classified as bleeding profusely (Table II). One exposure of this group was not classified as to the amount of hemorrhage.
In the "poor candidate" group, 2 teeth showed slight bleeding, 9 were moderate, and 7 classified as profuse hemorrhage (Table I). Seven exposures of this group were not classified as to the type of hemorrhage.

**History of Pain**

An inquiry was made as to the history of pain in 27 of 28 teeth in the "good candidate" group. Twenty-one of these had no history of pain. Six teeth had a history of pain. In 4 of these, the pain had occurred only when eating (Table II).

In the "poor candidate" group, 9 teeth had no history of pain. The history of pain was not recorded for one tooth. Fifteen teeth had a history of pain. Of these 15 teeth one had hurt while eating and 6 had a history of pain at night (Table I).

**Response to Heat**

In the "good candidate" group, 25 teeth responded to heat. One tooth failed to respond and the findings were not recorded in two teeth of this group (Table II).

The recordings of 21 teeth in the "poor candidate" group showed that 20 teeth responded normally and one failed to respond (Table I).

**Response to Cold**

Cold, in the form of ice cones, was applied to 27 teeth in the "good candidate" group. Twenty-three of these teeth responded as vital teeth and 4 failed to respond (Table II).

Twenty-one teeth in the "poor candidate" group were tested as to their reaction to cold. Nineteen of these teeth gave a positive response and 2 a negative response (Table I).
Response to the Electric Pulp Tester

The electric pulp test was applied to 25 teeth in the "good candidate" group. As a result of this, all 25 of these teeth were classified as vital (Table II); however, 3 of these responded only at a very high reading on the pulp tester scale and 4 at a very low reading.

Twenty teeth of the "poor candidate" group were tested with the electric pulp tester. Nineteen responded to the test while one did not (Table I). One tooth responded at a very high reading and another at a very low reading on the pulp tester scale.

Response to Percussion

Of 26 teeth tested in the "good candidate" group, 9 were tender to percussion and 17 responded normally to percussion (Table II). In the "poor candidate" group, 21 teeth were tested by percussion. Fifteen of these were tender to percussion (Table I).

Tooth Mobility

Mobility was checked in 26 teeth of the "good candidate" group. However, only two showed any deviation from normal (Table II). This mobility was recorded as slight in both cases.

Mobility was checked in 20 teeth in the "poor candidate" group. Nineteen were firm and only one showed a slight mobility (Table II).

Incidence of Internal Resorption

An attempt was made to detect histologic evidence of internal resorption in the coronal and root canal areas of the 53 teeth (Fig. 15). It should be pointed out that in order to make an accurate determination
of the incidence of internal resorption, it would have been necessary to have made serial sections of each tooth.

There was no evidence of internal resorption in the group of teeth classified as "good candidates" for vital pulp therapy (Table II). However, 5 of the 25 teeth in the "poor candidate" group exhibited some evidence of internal resorption in the histologic sections (Table I).
Table I
"POOR CANDIDATE" GROUP RESULTS

<table>
<thead>
<tr>
<th>Tooth No.</th>
<th>Type of tooth</th>
<th>Hemo-gram</th>
<th>Amount of hemorrhage</th>
<th>History of pain</th>
<th>Reaction to heat</th>
<th>Reaction to cold</th>
<th>Elect. Pulp</th>
<th>Test</th>
<th>Per-</th>
<th>Mobil-</th>
<th>Age</th>
<th>Internal yrs.</th>
<th>Resor-</th>
<th>tion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>decid.</td>
<td>L. plus</td>
<td>-</td>
<td>No</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7</td>
<td>Neg.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>decid.</td>
<td>N. plus</td>
<td>-</td>
<td>No</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>Neg.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>perm.</td>
<td>normal</td>
<td>-</td>
<td>Yes(nt)</td>
<td>neg.</td>
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L. plus = hemogram with high lymphocyte count
decid. = deciduous
N. plus = hemogram with raised neutrophil count
perm. = permanent
(mod.) = moderate
neg. = negative
(pos.) = positive
prof. = profuse
(nt) = pain at nighttime

---

L. plus = hemogram with high lymphocyte count
decid. = deciduous
perm. = permanent
mod. = moderate
neg. = negative
pos. = positive
prof. = profuse
(nt) = pain at nighttime
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L.plus - pulp hemogram with raised lymphocytes
M.plus - pulp hemogram with raised neutrophils
(m) - indicates pain at mealtime

decid. - deciduous
perm. - permanent
mod. - moderate
neg. - negative
pos. - positive
prof. - profuse
Figure 7. A photomicrograph of a stained peripheral blood smear showing numerous red blood cells, a round dark-stained lymphocyte (A), and a multi-lobed neutrophil (B).

Figure 8. A photomicrograph of a stained dental pulp blood smear showing many red blood cells, a normal lymphocyte (A), and a neutrophil (B) with vacuolization of the cell.
Figure 9. A photomicrograph of a stained dental pulp blood smear showing a massive number of neutrophils.

Figure 10. A photomicrograph showing a normal tooth and pulp which has been mechanically exposed. A pulp hemogram was obtained from the subsequent hemorrhage. The pulpal destruction resulted from the insertion of a round bur into the pulp tissue to stimulate hemorrhage.
Figure 11. A photomicrograph illustrating a small carious pulp exposure. The pulpal inflammation was well localized. This is an example of a tooth considered as a "good candidate" for vital pulp therapy.

Figure 12. A photomicrograph illustrating a tooth with pulpal inflammation extending into the pulp canal adjacent to the exposure. There is relatively normal pulp tissue in the opposite canal. This is an example of a tooth considered as a "poor candidate" for vital pulp therapy.
Figure 13. A photomicrograph of a root canal involved by the extension of inflammation. (Notice the excessive number of inflammatory cells present).

Figure 14. A photomicrograph of a tooth with considerable pulpal inflammation which had not extended into the root canals.
Figure 15. Photomicrographs showing inflammation
A. & B. throughout the pulp tissue.

Figure 15. Photomicrographs showing high magnification of the internal resorption process frequently associated with massive inflammation of vital pulp tissue.
Figure 16. A tooth with an exposed pulp which bled profusely. Histopathologically the pulp exhibited massive inflammation and degeneration.
DISCUSSION
Discussion

It was necessary to compare the dental pulp hemogram with the peripheral blood hemogram to evaluate the local change, if any, of the dental pulp. This was especially true with children because they have variable peripheral white blood cell differential counts. This type of comparison seemed to be more accurate than comparing the dental pulp hemogram to an average peripheral blood hemogram for the particular age group of the patient. Prader in his study evidently compared adult dental pulp hemograms to an adult peripheral blood hemogram average.

In the present study all of the teeth with normal pulp tissue or those with well localized carious pulp exposures and mild inflammation were found to have pulp hemograms exhibiting either normal counts or counts with increased lymphocytes. This finding agreed with the observations of Prader. He believed a normal pulp hemogram or one with increased lymphocytes indicated that the pulpal inflammation was well localized and that the tooth was suitable for vital pulp therapy.

The dental pulp hemogram of teeth with extensive inflammation were found to vary considerably. Some of these dental pulp hemograms showed normal counts, some showed increased lymphocytes, while some showed increased neutrophil counts.

The teeth with pulp hemograms demonstrating increased neutrophils were all found in the group classified as "poor candidates" for vital pulp therapy. This indicated that a pulp hemogram with increased neutrophils was evidence of extensive pulpal inflammation. This partially corroborated Prader's belief that a pulp hemogram showing a
high neutrophil count was indication that the tooth would not respond favorably to vital pulp therapy. However, the present investigation also showed that some teeth with extensive inflammation exhibit hemograms with either normal counts or with increased lymphocyte counts. This finding disagrees with the work of Prader. He found that teeth with pulp hemograms exhibiting increased lymphocytes or normal counts had undergone only mild localized inflammation. For this reason he considered a tooth with a normal hemogram or a high lymphocyte hemogram as a "good candidate" for vital pulp therapy.

Prader did not indicate his method of evaluating the extent of pulp pathology in the teeth of his observation group. The histopathological findings which were utilized in the present investigation, however, are generally accepted as the most accurate means of evaluating the status of the pulp tissue.

In many of the teeth in which the pulp hemograms exhibited an increase in neutrophils, there were signs that the neutrophils were undergoing degeneration. The neutrophils many times showed vacuoles in the nuclei and cytoplasm (Fig. 8). The nuclei often appeared to be breaking apart (karyolysis) and sometimes were seen outside of the cell walls. Prader and Bevilacqua both noticed this phenomenon of neutrophil degeneration.

The pulp hemogram apparently was not an accurate diagnostic aid in selecting teeth for vital pulp therapy since slightly more than one-third of the "poor candidates" for vital pulp therapy were found to have a high neutrophil count. The remainder of the "poor candidate" group had pulp hemograms corresponding to teeth with apparently excellent prospects for vital pulp therapy.
One-half of the normal non-exposed teeth were found to have pulp hemorrhages within the range of normal and the other one-half had hemorrhages with increased lymphocytes. The sample of normal non-exposed pulps consisted of 14 teeth.

A possible explanation for the increased lymphocyte count from the smears of normal teeth might be that many times the larger cells (such as the neutrophils) tend to collect at the edge and tail of the smear. It was difficult to obtain adequate hemorrhage from normal teeth. Thus, the smears were thin and scattered over the slides. The cells in such cases were counted in the thicker portion of the smear. It is possible that the smaller lymphocytes were more concentrated in the thicker portion of the smear. This could have caused the hemorrhage to be inaccurate. Thus, technical difficulty in preparing pulp smears may be another disadvantage of this technique.

The amount of hemorrhage seemed to give some indication of the nature of the pulp tissue. In the "good candidate" group, 11.1% bled profusely, while in the "poor candidate" group, 36.9% bled profusely. The majority of the normal unexposed pulps used in the control series exhibited very little bleeding upon exposure. This substantiated to a limited degree, McDonald's belief that profuse bleeding is an indication of pulpal degeneration. 72,74

A history of pain is believed to be a contra-indication for vital pulp therapy. 66,68,71,72,74 In this study 62.5% of the "poor candidate" group had a history of pain and 22.2% of the "good candidate" group had a history of pain. However, the pain in the "good candidate" group almost always was associated with eating. This seemed to substantiate the view that pain during mastication indicates a less serious pulpal inflammation than spontaneous pain. All six teeth which ached at night were in the "poor candidate" group. This suggests that teeth which ache
at night have undergone extensive degeneration and almost always vital pulp therapy is contra-indicated. A history of pain is a significant signpost. However, it is only one of several guides which should be used in evaluating a tooth for vital pulp therapy.

Response to heat and cold was of little or no value in determining the degree of pulp pathology. Both tests resulted in false negative responses from vital teeth. In this study cold gave false negative responses more frequently than heat. However, negative responses from heat and cold in the majority of instances were found to indicate a necrotic pulp.

The teeth in both the "good candidate" and "poor candidate" group responded to the electric pulp tester at variable readings on the pulp tester scale. Only one tooth with an exposed vital pulp was recorded as non-vital to the electric pulp test. The histopathological findings showed this tooth to have massive destruction of the pulp tissue although it bled profusely. The tooth tested vital to heat and non-vital to cold. The electric pulp test seemed to give no definite indication as to the extent of pulp pathology in an exposed tooth.

A high percent (71.4) of the teeth in the "poor candidate" group showed tenderness to percussion. However, a considerable number (34.6%) of the teeth in the "good candidate" group also were recorded as tender to percussion. This indicated that extensive pulpal inflammation might cause tenderness to percussion, but the psychological response of the child made this response quite unreliable in evaluating the pathology of the pulp.

Although teeth with necrotic pulps often were found to exhibit mobility, only three teeth in the sample of exposed vital pulps showed
excessive mobility. In all three of these cases the mobility was described as slight. Tooth mobility was not an aid in determining the degree of pulp vitality.

No one sign or test indicated the extent of pulpal involvement. However, it appeared that by using several diagnostic aids a high percent of teeth not acceptable for vital pulp therapy could be eliminated.

The incidence of internal resorption was related to the extent of inflammation in the pulp. Internal resorption was observed only in the "poor candidate" group. Twenty percent of this group exhibited some degree of internal resorption.

Although the pulp hemogram per se did not give a clear indication of the status of the pulp, there is promise that expansion of this work may develop a more exact diagnostic tool. Wied mentions the use of cytological smears for observing changes in the leukocytes and inflammatory changes in tissue cells. It is hoped that this preliminary work with the pulp hemogram will encourage further effort in the pursuit of a diagnostic tool for selection of cases in vital pulp therapy.
Summary

Fifty-three cariously exposed deciduous and young permanent teeth were evaluated in regard to the amount of bleeding, the reaction to cold and heat, the response to the electric pulp test, the response to percussion, the history of pain, and the amount of abnormal mobility.

The first drop of blood from the exposure was used to prepare a stained blood smear. A white blood cell differential count (hemogram) was then made from the stained smear. Next the dental pulp hemogram was compared with a peripheral blood hemogram made from a finger stick. Any significant rise in the number of lymphocytes or neutrophils was noted.

The teeth were extracted, decalcified and histologic sections prepared. The histopathological findings were classified according to the extension of inflammatory cells into the pulpal tissue. If the inflammatory cells extended into the pulp canal, the inflammation was considered too extensive for vital pulp therapy. If the inflammation was limited to the pulp exposure site and well localized, it was assumed that the pulp could have survived following vital pulpotomy. On this basis, the teeth were divided into "good candidates" and "poor candidates" for vital pulp therapy. Twenty-eight of the teeth were classified as "good candidates" and 25 were considered as "poor candidates".

The teeth in the "poor candidate" group failed to exhibit a constant blood picture. Some pulp hemograms were within the range of normal. However, others had elevated lymphocyte counts and still others had elevated neutrophil counts. All of the dental pulp hemograms with elevated neutrophil counts were in the "poor candidate" group, but the elevated neutrophil hemogram cannot be relied upon to indicate a
"poor candidate" because many teeth in this group exhibited normal hemograms or hemograms with elevated lymphocyte counts. In teeth with high neutrophil counts the neutrophils many times were found to exhibit signs of degeneration and karyolysis. The teeth with localized inflammation, "good candidates", were found to have either a normal pulp hemogram or one with elevated lymphocytes.

To serve as a control group, 16 non-exposed teeth with normal pulps were opened and pulp hemograms made. The dental pulp hemogram was compared with the hemogram made from the child's peripheral blood. The dental pulp hemograms showed that one-half of the counts were within the normal range and one-half exhibited counts with elevated lymphocytes.

The "poor candidate" group showed profuse bleeding in 38.9% of the cases, while the "good candidate" group had profuse bleeding in 11.1% of the cases.

In the "good candidate" group, 6 teeth presented a history of pain, but 4 of these had hurt only during eating. A history was taken of 24 teeth in the "poor candidate" group. Fifteen of these had a history of pain. Of the 15 teeth which had a history of pain, 6 had ached at night. It was apparent that pain other than at mealtime indicated extensive pulpal inflammation.

Heat and cold were found to be of little value in determining the degree of pulpal involvement. Most of the exposed vital teeth exhibited a positive response. However, false negative responses were noted from both heat and cold.

The electric pulp tester showed that teeth responded at quite variable readings on the pulp tester scale in both the "poor candidate" and "good candidate" group. The electric pulp tester does not seem to indicate the degree of pulp pathology in an exposed vital pulp.
The teeth were subjected to percussion and 71.4% of the "poor candidate" group were tender to percussion, while 34.6% of the "good candidate" group were tender to percussion. Tenderness to percussion was of little aid in determining the degree of vitality of exposed teeth.

Mobility was tested in 16 teeth. Slight mobility was noticed in 3 of the teeth. Mobility did not reveal any information regarding the degree of pathology in these exposed vital teeth.

The histologic sections were examined for evidence of internal resorption. Five teeth, all in the "poor candidate" group, were noticed to exhibit some degree of internal resorption. It seems from this study that internal resorption occurs more frequently in teeth involved with considerable inflammation.

No clear-cut relationship was found between the pulp hemogram and the extent of pulpal inflammation. However, some degree of relationship was noticed between the pulp hemogram with an elevated neutrophil count and teeth with extensively involved pulps. More study along similar lines seems indicated in our search for an accurate diagnostic aid to be used in vital pulp therapy.
CONCLUSIONS
Conclusions

1. The dental pulp hemogram is not a precise diagnostic aid in selecting teeth for vital pulp therapy.

2. A dental pulp hemogram with an elevated neutrophil count indicates extensive pulpal inflammation.

3. The presence of neutrophils exhibiting degeneration and karyolysis is indicative of a pulp showing extensive inflammation or degeneration.

4. The presence of profuse bleeding at the exposure site many times is associated with extensive inflammation of the pulp.

5. Pulp testing by heat, cold, or electricity is unreliable in determining the degree of pulpal inflammation.

6. Tenderness to percussion is of little value in determining the degree of inflammation in the dental pulp.

7. Most vital teeth with exposed pulps show no abnormal mobility.

8. A history of pain during mastication is found to be associated with a less extensive degree of inflammation than a tooth with a history of spontaneous pain.

9. A tooth with a history of pain at night has a considerable degree of pulpal inflammation.

10. Internal resorption occurs more frequently in teeth with extensive inflammation.
References


42. Van Huysen, Grant and Gurlay, W. B.: Histological changes in dogs teeth following preparation of cavities of various depths and their exposure to mouth fluids. J.A.D.A., 26:87, 1939.


80. Langeman, R. B.: Personal communication.


BIBLIOGRAPHY
Bibliography


Lingeman, R. B.: Personal communication.


Van Huyse, Grant and Gurley, W. B.: Histological changes in dog teeth following preparation of cavities of various depths and their exposure to mouth fluids. J.A.D.A., 26:37, 1939.


Vita

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The Oklahoma State Dental Association

The American Society of Dentistry for Children
### APPENDIX (Continued)

#### BLOOD HEMOGRAMS

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* Indicates a dental pulp hemogram from a normal non-exposed pulp.
## APPENDIX

### BLOOD HEMOGRAMS

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ABSTRACT
Abstract

The first drop of blood from the carious exposure site of 53 deciduous and permanent teeth was used for counting a white blood cell differential count (hemogram). The dental pulp hemogram was compared to a peripheral blood hemogram and the elevation, if any, of the lymphocytes or neutrophils was noted.

The teeth were evaluated as to history of pain, response to the electric pulp test, reaction to cold and heat, response to percussion, abnormal mobility and the amount of hemorrhage from the exposure site.

After obtaining the blood for the hemogram the teeth were extracted and histologic sections prepared. The histopathological findings were evaluated and if the inflammation extended into a root canal, the tooth was considered a "poor candidate" for vital pulp therapy. If the inflammation was localized to the pulp chamber, the tooth was considered a "good candidate" for vital pulp therapy. Twenty-eight teeth were found to be "good candidates" and 25 "poor candidates".

The "good candidates" exhibited pulp hemograms with both normal counts and with raised lymphocyte counts. The "poor candidates" were found to have pulp hemograms with normal counts, some with raised neutrophils and some with elevated lymphocytes. It was noticed that the neutrophils from teeth with elevated neutrophil counts often showed degeneration and karyolysis.

All of the pulp hemograms with raised neutrophil counts were found in the "poor candidate" group. However, the high neutrophil pulp hemogram cannot be relied upon to diagnose a "poor candidate"
for vital pulp therapy because only slightly over one-third of the hemograms in the "poor candidate" group were found to have an elevated neutrophil count. The remainder of the "poor candidates" had raised lymphocyte counts or normal counts.

A control group of 14 non-exposed normal pulps were opened and used for making dental pulp hemograms. One-half of these showed hemograms with a normal count and one-half showed hemograms with raised lymphocytes.

Cold, heat, and electric pulp tests were found to be of little value in determining the extent of inflammation. Also, percussion and mobility exhibited little correlation with the extent of pulp inflammation.

Of the "good" candidate group, 22.2% had a history of pain, most of which had occurred at mealtime. Of the "poor candidates", 62.5% had a history of pain. All of the teeth which had ached at night were in the "poor candidate" group.

Profuse bleeding was observed in 38.2% of the "poor candidates" compared to 11.1% in the "good candidates".

Twenty percent of the "poor candidates" had some degree of internal resorption. None of the "good candidates" exhibited any internal resorption.

It is suggested that similar methods be explored for finding an adequate diagnostic aid for vital pulp therapy.