THE INFLUENCE OF THE PLACEMENT AND REMOVAL OF FIXED ORTHODONTIC APPLIANCES ON SENSORY DETECTION THRESHOLD RESPONSE OF TEETH AS MEASURED BY AN ELECTRIC PULP TESTER

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ABSTRACT

91 patients were examined and 1427 individual teeth were tested with the aim of the estąblishing if there were differences between the sensory detection threshold response of healthy unrestored teeth, when measured by electric vitality testing with the Analytic Technology Vitality Scanner (ATVS), as the teeth were fitted with banded and bonded orthodontic appliances or when these appliances were removed.

The following conclusions were drawn from the results of this experiment:

1. Thresholds did not seem to be stable enough to be relied upon to judge treatment effects by parametric methods.

2. No general sex differences could be demonstrated.

3. A nonparametric analysis (the Kruskal-Wallis test) attempting to distinguish treatment effects, as measured by an increased or decreased threshold, could not demonstrate a general significant difference.

4. There may be some difference in the behaviour of teeth which were banded with stainless steel orthodontic bands, but no differences could be found in the treatment groups between teeth with bonded stainless steel brackets.

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My wife, Kathryn for her patience with me during the preparation of this thesis.

DEDICATION

I wish to dedicate this work to the memory of the late

Associate Professor Barry.C.W.Barker,

HDD R.F.P.S.G. MDS, Phd, FDSRCS, FRACDS (Dental Anatomy),

a man who was the embodiment of a true academic

and whose life was sadly too brief.

TABLE OF CONTENTS

		Page
1	INTRODUCTION	1
2	ANATOMY AND PHYSIOLOGY OF DENTAL PULP TISSUE	3
	2.1 Blood Vessels and Vascular Regulation	3
	2.2 Nerves	8
	2.2.1 The response of intra-dental sensory units to stimuli	8
	2.3 The Aging of Dental Pulp Tissue	12
3	THE DAMAGED DENTAL PULP	14
	3.1 General Observations About The Damaged Pulp and its Response to Insult.	14
	3.1.1 Hyperaemia	15
	3.1.2 Inflammation	16
	3.1.3 Calcification	17
	3.1.4 Necrosis	20
	3.2 Pulpal Responses to Orthodontic Treatment	20
4	DENTAL PULP VITALITY TESTING	25
	4.1 Pulp Vitality Testing in General	25
	4.2 Methods of Pulp Vitality Testing	36
	4.2.1 Radiographic	36
	4.2.2 Temperature	37
	4.2.3 Blood flow	39
	4.2.4 Ultrasonics	41
	4.2.5 Electrical	41
	4.2.5.1 Disadvantages of electrical pulp testing	42
	4.2.5.2 Advantages of electrical pulp testing	43
	4.2.5.3 Potential risks of electrical testing	43
	4.2.5.4 The method of electrical pulp testing	44
	4.3 The Electrical Properties of Human Teeth	51
5	EXPERIMENTAL WORK	52
	5.1 Hypothesis	53
	5.2 The Experimental Method	54
6	RESULTS AND DISCUSSION	61
	6.1 Results	61
	6.2 Analysis and Discussion	64
7	SUMMARY AND CONCLUSIONS	68
	APPENDICES	72
	BIBLIOGRAPHY	93

1 INTRODUCTION

The establishment of tooth vitality as an aid to diagnosis is a routine task for dentists (Grossman et al, 1988).

Ehrmann (1977) wrote that:

"No examination of the mouth is complete without vitality tests being carried out on each tooth."

Orthodontic patients, therefore, may need to have pulp vitality tests, as part of their pretreatment assessment and from time to time during their management programme.

In addition to other traumatic insults that a patient's teeth may have experienced, orthodontic tooth movement necessitates changes in the periodontal and periapical tissues, which have the potential to cause damage, but in the large majority of patients, these changes are regarded as being a remodelling process.

Very little information is published on the possible effects of any orthodontically induced changes, or the presence of the actual appliances on the results and interpretation of various vitality testing techniques.

This study therefore has two main aims:

A. To assess the variability of observations as measured by the Analytic Technology Vitality Scanner (ATVS), in the situations to be described.

B. To acertain if there is a difference between the sensory detection threshold response of healthy unrestored teeth, as determined by electric vitality

testing with the ATVS, as the teeth were fitted with banded and bonded orthodontic appliances or when these appliances were removed.

This was carried out by testing the hypothesis that:

There would be no difference in the variation of vitality reactions, as determined by ATVS electric vitality testing, between the following four groups of patients:

1. Patients who were banded-up, bonded and fitted with initial archwires.

2. Patients, with appliances fitted, who were debanded and debonded.

3. Control patients who underwent a repeated test, which was delayed at least 45 minutes.

4. Control patients who underwent immediate repeat vitality tests.

In order to establish the basis for the experimental section of this work, the thesis firstly presents a review of anatomy and physiology of the dental pulp, with reference to vitality testing.

Secondly, details of the available methods of pulp vitality testing are then examined and discussed.

2 ANATOMY AND PHYSIOLOGY OF PULP TISSUE

Pulp tissue is composed of a layer of odontoblasts, supported by loose connective tissue, gelatinous ground substance, nerves, blood vessels and lymphatics (Warwick & Williams, 1973). From the odontoblasts, extend cellular processes, called Tomes processes, around which the dentine forms (Rowe & Pitt Ford, 1990)

2.1 Blood Vessels And Vascular Regulation.

Several arterioles enter the apical and accessory foramina, travel up the root canal and give off branches which form a subodontblastic capillary plexus. This plexus is most pronounced in the young tooth. (Warwick & Williams, 1973; Rowe & Pitt Ford, 1990)

Heyeraas (1985) found that input arterial pressure in the pulp was about 40mm Hg which, when compared with muscle mesentery (90-100mm Hg), was low and indicated that a significant reduction of arteriole pressure occurred before the vessels entered the dental pulp. The venular pressure averages 19mm Hg and this is higher than in other tissues. Therefore, there is a relatively low pressure gradient through the pulp. The significance of this is that tissue pressure in the subapical tissue, as well as within the pulp chamber, play very critical roles regulating pulpal blood flow.

Heyeraas (1985) also studied the pressure

gradients and velocity profile of the microvascular bed and demonstrated that the blood flow was slowest through the capillaries and post capillary venules. Chein (1985) when reviewing these results and those of Takahashi (1985), who demonstrated the presence of artero-venous shunts, venous-venous anastomoses and U-turn arterioles in the dental pulp, and concluded that there were still many questions unanswered about the regulation of pulpal blood flow.

Takahashi (1985) showed that the capillary network is most developed in the coronal region compared with the apical region of the canal and Kim (1985) found that blood flow through the coronal region was higher than through the apical region.

Takahashi (1985) demonstrated that there are significant changes in the microvasculature with age. In the morphogenetic stage, the terminal capillary network is 'very rich', but it becomes increasingly sparse with aging.

Chein (1985) noted that, the complete picture of pulpal haemodymnamics was not known, because the tissue perfusion pressure near the apex was unknown. Therefore, the intrapulpal vascular resistance could not be calculated. The vascular resistance needs to be known before calculations of the effects of vessel length and diameter and blood viscosity changes can be made.

The significance of understanding factors influencing intrapulpal pressure is that, because the pulp is in a rigidly entrapped environment, small changes in

volume create large changes in pressure.

In addition to the physical factors described above, pulpal vascular changes have been shown to be controlled by neural and humoral factors (Kim & Dorscher-Kim, 1989).

Pre- and post-synaptic adrenergic receptors have been shown to exert neural control of pulpal vessels by Kim (1985) who demonstrated alpha and beta receptors in the pulp vessels. He showed that alpha receptors cause a decrease in pulpal blood flow, whereas beta receptors cause a paradoxical reduction in blood flow, probably due to the low compliance within the pulp, which causes a constriction of the venules.

Electrical stimulation of the cervical sympathetic nerve has been shown to reduce pulpal blood flow according to Bergenholtz et al (1985).

Humoral regulation by either vasodilation or vasoconstiction, is by substances such as: the catecholamines, substance P, prostaglandins and bradykinins (Chein, 1985). These substances also play a role in pulpal inflammatory response.

Kim & Dorscher-Kim (1989) using 15μm radioisotope-labeled microspheres injected into the arterial vessels, found three distinct types of blood flow reductions:

A. Type 1 was a reduction due to direct stimulation of sympathetic nerve fibres; intra-arterial infusion of norepinephrine, 5-HT, or prostaglandin F2; and

infusion of norepinephrine, 5-HT, or prostaglandin F2; and indirect stimulation of the sympathetic nerve.

B. Type 2 was biphasic, with an initial increase in flow followed by a decrease. This was induced by isoproterenol, prostaglandin E2, substance P, and bradykinin.

C. Type 3 was a gradual reduction in pulpal blood flow following the administration of histamine.

Membrane permeability to proteins is another factor influencing the tissue pressure. This is also under the control of humoral factors (Chein, 1985).

Chein also described the controversy surrounding the various influences on pulpal blood flow by giving the two alternative theories of how the vasoconstrictive agent Isoproterenol causes a paradoxical reduction in the blood flow through a tooth:

1. The first theory, supported by Kim (1985), was that dilation of arterioles within the pulp causes a transient increase in flow, which causes compression of the venules, reducing subsequent drainage and hence flow.

2. The second theory, supported by Heyeraas(1985), was that there is a stealing of blood from the arterial system, before it reaches the pulp canal. The blood is diverted to the gingiva and other tissues. This lowers the surrounding tissue pressure, lowering vascular resistance, so that there is less flow through the pulp.

The oxygen requirements of the pulp have not been

quantified, because of the difficulty of observing the pulpal venous oxygen content. Chein (1985) noted that skin usually has a blood flow delivery greater than its metabolic oxygen requirement needs, the purpose of this is that it helps with the control of the body's temperature. He also postulated that pulp probably has more blood flow than needed for metabolic requirements, but at this time the required levels remain unknown. Therefore, we do not know what oxygen demands are required for dentine production, inflammatory response or nerve impulse transmission.

Establishing these basal metabolic needs of the pulp will help to explain the finding of Olgart (1985), who demonstrated that changes in pulpal blood flow can alter the oxygen supply, as well as the tissue pressure. These factors modulate nerve impulse transmission (sensory thresholds). In a complex two-way (biofeedback) relationship, neural activity (autonomic and somatosensory), in turn, determines the release of some of the local vasoactive mediators (substance P, neurokinin A, calcitonin gene-related peptide, vasoactive intestinal polypeptide and neuropeptide Y), which control blood flow.(Wakisaka & Akai, 1989; Olgart et al, 1989; and Kim & Dorscher-Kim, 1989)

2.2 Nerves

Myelinated nerve (A-delta) fibres, enter the apical foramina and give off anastomosing branches which are unmyelinated. Some fibres keep their medullary sheath until they reach the layer of Weil. (This layer supports the odontoblast layer and is approximately half its width.) There is an abundance of nerve fibres in this layer, but very few blood vessels. This subodontoblastic plexus (of Raschkow) forms a network around the odontoblasts with some unmyelinated nerve fibres passing into the predentine (Seeman, 1963). While some of these fibres loop back down to the layer of Weil (Warwick & Williams, 1973). There are greater numbers of intratubular fibres in dentine, in the vicinity of the pulp horns (Bergenholtz et al, 1985).

Unmyelinated nerves (C-fibres) of the sympathetic nervous system, involved in controlling the internal homeostasis of the pulp, are associated with the blood vessels entering via the apical foramen (Salzberg, 1985). These fibres have been demonstrated to be more resistant to pathological changes, than other pulpal structures (Salsberg, 1985).

2.2.1 The response of intra-dental sensory units to stimuli.

The protoplasmic processes of the odontoblasts within the dentine tubules were originally thought to transmit stimuli to produce afferent discharge in the sensory nerve fibres (Seeman, 1963), but this has not been

substantiated. The role of the odontoblast is still unclear according to Warwick & Williams (1973), and doubtful according to Rowe & Pitt Ford (1990), because synaptic junctions have not been observed and also dentine has still been found to be sensitive in the absence of an odontoblastic layer. Bergenholtz et al (1985) concluded that there was a proven structural association between the terminal pulpal nerves and odontoblastic processes.

There are two current theoretical explanations of stimuli transmission from dentine:

1. Direct stimulation of the nerve terminals. However, as Warwick and Williams (1973) point out, the nerve terminals are only present in 1 : 2,000 adult dentine tubules and immature teeth have no innervation of dentine.

2. The hydrodynamic theory, which requires stimuli to cause movement of fluid along the dentine tubule. This initiates the subodontoblastic nerve plexus to propagate a response (Warwick & Williams, 1973).

The hydrodynamic theory has gained most support, although the mechanism of pain transmission across exposed dentine is still incompletely understood, according to Bergenholtz et al (1985). Direct stimulation of nerve endings as the main mechanism is not supported by observations that neuroactive chemicals fail to stimulate or to anaesthetize dentine and that not all areas of dentine have nerve fibres present.

Seeman (1963) writes that:

"All stimuli result simply in pain, not

distinguishing stimuli as physical, bacterial, chemical."

He noted that pulp inflammation or even temperature changes, altered the blood circulation causing pressure changes on the rigidly enclosed pulp, which results in pain.

A more modern view is presented by Narhi (1985), who in reviewing the literature, concluded that there was variation in the perception of various sensations. The sensations experienced by a subject varied with the activation of different types and numbers of pulp nerve fibres and the different patterns of action potentials in individual nerve units. This observation explained the ability of subjects to identify cold irritation and to discriminate between the stimulation of dentine (described as being a 'sharp pain') compared with the 'dull pain' experienced by patients with advanced pulpitis.

Greenwood et al (1972), Matthews (1977) and Bergenholtz et al, (1985), report that while both unmyelinated and myelinated fibres transmit stimuli, the sensory detection threshold (S.D.T.) response of the dental pulp to electrical stimulus originates through the myelinated A- Delta nerve fibres located in the subodontoblastic layer. These fibres according to Bergenholtz et al (1985), have been identified as having the characteristics of a low sensory threshold and fast conduction of sharp pain. They are responsible for dentine sensitivity. The slow conducting unmyelinated C-Fibres are responsible for the poorly localized sensations associated

with pulpal injury (Bergenholtz et al, 1985).

Bergenholtz et al (1985), report that there is evidence suggesting that changes in blood flow can modulate sensory nerve function. A reduction in pulpal blood flow can suppress the activity of the A-fibres.

Bergenholtz et al (1985), also record that pharmacological agents, such as inflammatory mediators, can modify nerve activity by either direct or indirect means.

There is also a link between the anatomical maturation of the tooth and its response to stimuli. Bernick (1964) described that, in the pulps of unerupted third molars, the nerve fibres terminated at the pulpo-odontoblastic border, but as the tooth erupted, there was increased arborization and proliferation of nerve terminals into the odontoblastic zone and some fibres looped into the dentine tubules. Also, in general, the myelinated nerve fibres appear late in the development of the pulp, probably not becoming functional until the time of apexification.

Bernick also postulated that entrapment of nerve fibres in predentine was a result of continued deposition of calcified tissue (secondary dentine) stimulated by continued exposure to environmental stimuli.

Andreasen et al (1990) agreed with the results of Fulling and Andreasen (1976a) that increasing root development in premolar teeth was associated with a reduction of the threshold to electrometric sensitivity.

2.3 The Aging Of Dental Pulp Tissue

As described above, the pulp matures as the tooth erupts and comes into function. After this, changes continue to occur within the pulp tissue throughout life. These changes may be described as aging.

Bernick, in 1967, in a study of 150 noncarious erupted teeth, taken from people over the age of 40 years, found that more than 90% had some degree of pulpal calcification. These results were similar to those he reviewed from other studies. Bernick noted that the nerves which persisted in the pulps of old teeth demonstrated fragmentation, beading and reticulation. He described these changes as the typical signs of degeneration.

Bernick and Nedelman (1975), in an article on the aging of human dental pulp, stated that there was a continual decrease in the size of the pulp chamber due to calcification. There was progressive deposition of calcified material, in a process which originated in the root pulp and progressed to the coronal pulp. There was a decrease in the number of blood vessels and nerves, in the coronal pulp, as a result of the calcification of these structures with aging. The connective tissue sheaths persisted and this together with the decreased pulpal volume, gave the pulp a more fibrotic appearance. They also described the appearance, with aging, of Von Korff fibres, which are fine filaments extending from the predentine through the odontoblastic layer into the zone of Weil. Bernick and Nedelman found that the remaining stroma of the

pulp did not change with age. Also, the composition of fine collagenous fibres and acidic mucopolysaccharide ground substance remained constant.

3 THE DAMAGED PULP

3.1 General Observations About The Damaged Pulp and its Response to Insult.

Damage to the pulp may be categorised by the agent which produced it, such as mechanical, chemical or thermal; or by the clinical signs and symptoms present; or, histologically, by the changes seen in pulpal tissues (Howe & Pitt Ford, 1990). Attempts to correlate the clinical signs and symptoms with the histological findings have not been definitive.(Dummer et al, 1980; Olgart, 1985)

Olgart (1985) points out that pain may be present in normal pulps ("physiologic pain") as well as in damaged pulps ("pathophysiological pain"). The quality of pain may vary according to the status of the pulp. On the quality of the pain, Olgart says its diagnostic value:

".. is obscured by the fact that the function of the pain fibres is influenced by the local environment , which may or may not be associated with pulpal inflammation."

Delineating changes clinically can also be difficult, because the patient's reactions are subjective, some of the changes are time-dependent and more than one histological category of damage may be observed to be present, at the one time, in the same tooth (Dummer et al, 1980).

3.1.1 Hyperaemia.

Hyperaemia is an engorgement of the pulpal vessels, which is usually reversible. Intra-pulpal pressure is raised. Hyperaemia causes an increase in sensitivity to stimuli and the severity of response may be related to the degree of hyperaemia according to Stephan (1937).

This view has since been modified and the current belief according to Kim and Dorscher-Kim (1989), who studied hemodynamic regulation vaso-active chemicals in experimental animals, is that there are two distinct and paradoxical types of reaction to the vaso-active chemicals, which in other parts of the body would cause hyperaemia:

Firstly, there may be a transient increase in flow, followed by a decrease. This bipasic response was stimulated by isoproterenol, prosthoglandin E2, substance P and bradykinin and is probably caused by the pulp being in a low compliance, enclosed environment.

Secondly, there is a gradual single phased decrease in the pulpal blood flow caused by the administration of histamine.

Wakisaka and Akai (1989) note that not all vasoactive neuropetides are of autonomic origin and that some are somatosensory in nature.

Olgart et al (1989) agree with this observation when they found that local irritation could cause an instant vasodilation in the coronal pulp, provided that the stimulus was strong enough to activate pulpal C-fibres. They conclude that:

"Thus, only intense stimulation of the tooth crown or pulp has been shown to trigger a vasodilation in the pulp. It is therefore possible that this response is part of a nerve-induced defense reaction in the pulp."

These observations about vascular regulation help explain further the finding of Van Hassel (1971), who speculated that there were other factors contributing to pulpal haemostais than the simplistic view prevailing at that time, that the rigid structure of the pulp chamber prevented inflammatory swelling and put pressure on the apical vessels causing a 'rapid strangling' of the pulp.

3.1.2 Inflammation.

Takahashi (1985) demonstrated a loss of vasculature and changes to the capillary network in regions surrounding inflammation.

Acutely inflamed pulp may give a severely painful and prolonged response to stimuli such as heat or cold, whereas electrical stimulation generally gives an average response, but this may vary (Stephan 1937).

Chronically inflamed dental pulps generally give no severe symptoms, according to Stephan (1937), who was describing grossly carious teeth with exposed pulps.

Chambers (1982) says that:

" The old view that a lowered threshold to pulp testing indicates hyperaemia or acute pulpitis and an increased threshold indicates chronic pulpitis... should be discarded."

Dummer et al (1980) separated chronic pulpitis into two categories, partial and total, depending on the amount of tissue involved. They also identify the continuation of the disease process by describing chronic partial pulpitis with necrosis present. These features create a situation where there is a poor correlation between symptoms, vitality reactions and pulpal histopathology (Chambers, 1982).

Nahri (1985) describes a change in the type of pain experienced during the process of inflammation. The change in sensation is described as being from sharp and localized to dull and poorly localized. He postulated that the reason for this change in the type of pain is that the slow conducting C fibres of the pulp are active. Also, in general C fibres are more resistant than myelinated A fibres to pressure elevation and hypoxia.

Rowe & Pitt Ford (1990) agree with this description adding that the C fibre pain is more prolonged and of a more 'unbearable' nature.

3.1.3 Calcification.

Rowe & Pitt Ford (1990) explain that odontoblasts are capable of forming dentine throughout life. However, they are incapable of mitosis and if they become damaged, they are replaced by new cells which differentiate from precursor cells in the sub-odontoblastic layer. These new cells do not form normal dentine. The degree of irregularity in this reparative dentine depends on the

provoking stimulus.

Herbert (1953) described the progressive calcification of the pulp chamber from the coronal region towards the apex, in teeth which remain vital after trauma. He notes that calcification of the pulp occurs more commonly in teeth with wide open apices or with apices which were only recently closed.

Jacobsen and Kerekes (1977) agreed with the findings they reviewed from Magnusson and Holm, that teeth with wide open apices at the time of injury had the smallest risk of damage and subsequent pulp necrosis.

Blackwood (1957), analyzing pulpal responses, concluded that trauma-induced interferences in pulpal blood flow could cause a calcified matrix to be deposited. This matrix could have odontoblasts and other cells of the pulp embedded in it. Some time after the injury, this could lead to the obliteration of the canal. This was a reparative response rather than a true metaplasia of the pulpal tissue. Ingle and Beveridge (1976) agreed that trauma which is not sufficiently severe to devitalise the pulp may stimulate the pulp to lay down reparative dentine. This could obliterate the entire root canal.

Jacobsen and Kerekes (1977) studied 122 teeth with a history of trauma and subsequent radiographic evidence of pulpal calcification, for periods over ten years. They found that teeth which underwent rapid root canal obliteration were more likely to develop pulpal necrosis and develop periapical radiolucencies. The late

development of periapical radiolucencies was correlated also with the severity of the original injury.

Delivanis and Sauer (1982) warned that there were endodontic difficulties dealing with obliterated canals and that an early diagnosis of a narrowing canal space is desirable, so that a more conservative endodontic treatment may be initiated. They cite Ingle as recommending:

"The surgical exposure of the apex and the placement of a retrograde amalgam filling is the procedure of choice for a vital, calcified pulp."

Jacobsen and Kerekes (1977) disagreed in reviewing their results, by stating that:

"Even if 21% of the totally obliterated teeth developed necrosis, routine endodontic intervention does not seem justified."

Each case should be judged individually, but as Stephan (1937) noted:

"The atrophied and degenerated pulp is without painful symptoms, generally gives an average response to electric, hot and cold stimuli but may give variable or no response, and radiographically shows no periapical change, although it may show secondary calcification in the pulp canal. This indicates that degenerative changes cannot be reliably diagnosed clinically."

3.1.4 Necrosis.

Dummer et al (1980) deem the presence of necrosis as delineating whether the pulp is saveable or non-saveable.

In gaseous pulpal necrosis (gas gangrene), the production of gases intrapulpally exerts a pressure on the subapical tissues. This pressure may be accentuated by the application of heat (Stephen 1937).

Liquefactive necrosis involves a dissolution of all the structures of pulpal tissue.

Cahn, cited by Stephan (1937) and Mumford and Bjorn (1963), demonstrated that nerve fibres were able to survive longer into the degenerative process, with the result that the teeth could still react to electric vitality testing, while other pulpal structures were totally destroyed. This observation was still accepted recently. (Rowe & Pitt Ford, 1990)

3.2 Pulpal Responses to Orthodontic Treatment

Oppenheim (1942), reviewing the literature to that time, gave the following summary of the effect of orthodontic forces (direction and amount were not specified) on the
pulpal tissues:

1. Stasis caused by interferences with normal blood flow causes an accumulation of fluid between the pulp tissue elements and the formation of vacuoles among the

odontoblasts. This impairs the formation of 'dentinoid.'

2. Minor circulatory disturbances in the pulp vessels cause the formation of calcium salt deposits he called 'calcospherites' and 'false denticles'. He agreed with Willman that hyaline degeneration of some connective tissue fibres act as nuclei for the deposition of calcium salts and the formation of 'denticles'.

3. Small haemorrhages into the pulp or minor disturbances of the odontoblasts are considered harmless according to his review of Orban, but as larger amounts of 'escaped corpuscles' become disintegrated they change the pH of the pulp from alkali to acidic. If this acidity is prolonged, it must cause the death of the pulp.

4. The reparative capacity of pulp is very high. Oppenheim agrees with Euler-Mayer and that even if:

".. a large amount of odontoblasts are lost, they regenerate from undifferentiated mesenchyme cells of the pulp."

The ultimate fate of the previously injured pulp was summed by the conclusions Oppenheim quoted from Euler-Mayer:

"...that the once initiated atrophy of the pulp may last unchanged for a very long time, but on the whole the condition is on a perpetual decline."

Oppenheim (1942) described the divergent points of view of the clinician and the pathologist as:

"What the former would consider as normal and healthy as long as the vitality test proves normal , and no

clinical signs of abnormal conditions are present, the latter would consider as pathologic on account of his microscopic findings."

Bunner and Johnsen (1982) proposed that quantitative and qualitative assessments of nerve fibres entering the tooth, give an indication of overall change in the pulp. They found that only a small number of fibres were affected early in treatment (which they note may have been an artefact error) and no changes in long term treatment. This led them to the conclusion that:

"No irreversible insult is inflicted on the pulps of healthy teeth undergoing routine conservative orthodontic treatment."

They suggested that controlled tooth movement (in this case torquing using 75 to 120 grams), produced no inflammatory changes and that if teeth undergoing orthodontic treatment are observed to have inflammatory changes, then these may have been caused by other factors, such as caries, restorations, abrasions, or trauma.

Delivanis and Sauer (1982) using radiographic records in a sample of 46 cases, could not demonstrate a significant incidence of canal calcification as a result of orthodontic treatment.

Landay et.al.(1970) and Cooper et.al. (1971) studied the effects of various occlusal forces, applied for various periods, on the pulps of rat molars. They found that for short periods, there were no changes seen with light microscopic examination, with light or heavy forces

applied. After seven months, there were some changes noted with heavy forces, but these were possibly due to damage to the periodontal ligament in the region of the furcation being transmitted to the pulp by accessory lateral canals.

They found the "incongruity between periodontal injury and pulpal pathology" as "perplexing", because as the periodontal ligament was nearly repaired (after the seven month period), the pulp started to demonstrate pathological changes.

Bunner et.al. mentioned that all 49 teeth they examined, remained vital using electrical testing during orthodontic treatment, but that their sample size was too small to draw conclusions about vitality reactions.

Delivanis and Sauer(1982) conclude that due to the possibility of calcified canals, patients should be monitored closely during orthodontic treatment, especially if there is a history of trauma to teeth, but they do not give any guidelines as to how frequently this should be done.

Erhmann (1973 & 1977) recommends regular vitality testing for patients undergoing extensive dental procedures, including orthodontics. Chambers (1982) concurs with this view.

Ehrmann (1977) cautions that electric pulp testing cannot be used in cases of full orthodontic banding, because of the risk of conduction, but did not present any evidence to substantiate this claim.

Burnside et al (1974) noted that thresholds to

electrical stimulation in orthodontically banded patients were elevated compared to their control group.

4 PULP VITALITY TESTING

4.1 Pulp Vitality Testing in General

The screening of tooth vitality is recommended by E.H. Ehrmann (1977) before any orthodontic or major restorative procedure is undertaken. The procedure is regarded as reversible and the hazards of pulp testing are minimal in comparison with the benefits of a more comprehensive dental assessment. (McDanial et al, 1973, and Chambers, 1982)

Rowe & Pitt Ford (1990) state that:

"It is necessary to establish as accurately as possible the state of the pulp in the diagnosis of dental pain and before embarking on operative procedures."

Complications of the patient's orthodontic treatment may be avoided by establishing the vitality status of teeth prior to the commencement of treatment. Measuring vitality, at debanding, is also a benefit to the patient, because it allows judgements to be made about the patient's oral health at that point in time. Weine & Postashnick (1982) reported anecdotal cases of devitalisation occuring during the course of orthodontic treatment.

Chambers (1982) described the features of an ideal pulp tester as providing:

"..a simple, objective, standardized, reproducible, non-painful, non-injurious, accurate and inexpensive way of assessing the exact condition of the pulp tissues at any

given time."

Chambers gives comprehensive reasons for being cautious about the use of pulp testing as a method of determining the specific pathological condition of the pulp:

1. Pulp tissue vitality and nerve viability.

There are situations where the nerve is damaged, but the pulp may still be viable and other situations where the viable nerve fibres persist well into the pulpal degenerative process.

Ingle and Beveridge (1976) agree, pointing out that determining 'vitality' by using the sensory nerve response of the pulp is an incorrect premise, because 'vitality' is contingent on vascular rather than nerve supply. This must be remembered when using any technique to determine the health status of a tooth.

Pathological changes may induce disturbances in sensory perception, for example:

Brown (1967) recommended that teeth with a history of recent trauma should be examined clinically for fracture, mobility or displacement. Radiographic survey and pulp vitality testing should also be performed as reference points for future comparison. On the use of electric pulp testing, Brown (1967) wrote:

"A negative response should not be considered indicative of pulpal death at this time, since many teeth will not respond to testing until several weeks after an injury."

Jacobsen and Zachrisson (1975) recorded that teeth with root fractures, which remained vital, may initially lose their reaction to electric vitality testing. Such teeth may regain some sensation after a period of time. Teeth which were undergoing calcification gradually lost their reactions to electric stimulation.

Brown (1967) recommended a minimum waiting period of four to six weeks before the final 'status of the pulp' is ascertained. This recovery time was recommended before a permanent restoration could be placed.

Bhaskar and Rappaport (1973) also recommend a delay in the endodontic intervention of teeth which test nonvital to pulp testing immediately after trauma. They recommend radiographic assessment be carried out in order to detect narrowing of the pulp canal and periapical pathology. Teeth which initially test nonvital may take many months to recover positive vitality signs.

Bhasker and Rappaport (1973) stress that:

"the life of the pulp tissue is dependent on the blood supply."

They go on to write that:

"By contrast, however, injury that destroys the integrity or the function of a nerve has no effect on the survival of the pulp tissue."

Bhasker and Rappaport (1973) explain the different abilities of arterial vessels and nerves to withstand trauma as being that the arteries have a more coiled and tortuous character, whereas the nerves are taut

and therefore more easily damaged by traumatic movement. They did not state whether they were talking about the myelinated A-fibre or unmyelinated C-fibre nerves.

Another situation exists where Mumford and Bjorn (1963) concur with Cahn's 1930 reference showing the persistence of nerve fibres in inflamed and even totally degenerated regions of pulp, so that in these cases, positive reactions to stimulation would not equate with true vitality.

Mullaney et.al.(1970) questioned this observation concluding that it is unlikely that nerve fibres resist necrosis.

Chambers (1982) concludes that in the light of the above observations, where nerves may have greater or lesser resistance to destruction, prediction of tissue vitality based on nerve vitality measurement is open to error.

2. Lack of correlation between vitality signs and the histological condition of the pulp.

Chambers agrees with Marshall (1979), that pulp testing responses should be interpreted as 'yes'(vital) or 'no' (non-vital or severely inflamed) responses only, stating that:

"Pulp testers do not quantify disease or measure health and they should not be used to judge the degree of pulp disease."

Many other authors have failed in attempts to

correlate signs of vitality testing and the histological condition of the pulp.(Mumford and Bjorn, 1962; Reynolds, 1966; Barker and Ehrmann, 1969; Matthews, 1974; and Dummer et al, 1980)

3. Lack of objectivity in response to vitality testing.

Pain is a very subjective response which is hard to measure quantitatively or objectively. (Mumford, 1962; and Ehrmann, 1977).

Chilton and Fertig (1972) recommend the use of contra-lateral control teeth to help remove the subjectivity of a response, but Seltzer (1963) points out that there is no way of knowing if the 'control' tooth is normal.

4. Problems in reproducibility of results.

Scaffer (1958) observed that patients respond differently to pulp tests on different days and also at different times on the same day. This is controversial, because Rubach and Mitchell (1965), Friend and Glenwright (1968), Nordenram (1970), Ingle and Beveridge (1976), and Torneck and Howley (1989), have shown no great differences on different test occasions. In fact, Torneck and Howley (1989) concluded that it may be possible to use changes in electric vitality and mechanical mobility measurements to plot the maturation of newly erupting teeth.

Chambers was sceptical about the validity of

reproducibility, pointing to:

A) Poor correlation of histopathological prediction with response.

B) The response depends upon the state of the mind of the patient, which is a changeable factor (Grossman, 1978).

C) Difficulties patients have in identifying the exact threshold of sensation (Mumford, 1965).

D) The lack of intrinsic accuracy of electric pulp testers (Cooley and Robinson, 1980).

Recent developments in instrumentation may have solved some of these problems, according to Cooley et al (1984) and Torneck and Howley (1989).

5. Unpleasant sensations during testing.

All methods rely upon the patient indicating when they feel sensations. These sensations may be unpleasant; Naylor (1964) and Greenwood (1973) describe pain as the only sensation elicited by stimulation of pulpal nerves. McGrath et al (1983), in more recent work, explored the stimulus detection threshold with an instrument which varied the frequency and found that pre-pain sensations may be elicited.

6. The effect of maturation status of the tooth.

Bernick (1969) observed the maturation of the pulpal innervation with eruption and function. Adult thresholds of sensation may not be observed for several

years after a tooth enters the mouth. Fulling and Andreasen (1976a) believed that the application of carbon dioxide was the only definitive test of vitality for newly erupted teeth.

Andreasen et al (1990), found that threshold levels to electrical stimulation in 187 permanent premolars were dependent upon the stage of root development; notably, increasing root development was associated with decreasing sensitivity.

7. The effects of past trauma to the teeth.

Chambers notes that unreliable responses are found to pulp testing following trauma and that as an explanation, the pulp should be considered to be in a state of 'shock'. He did not claim to know the exact physiological reason for this. Zadick et al (1979) recommend that six months is sufficient time to wait before instigating root canal therapy in teeth which fail to respond to vitality testing. Bhasker and Rappaport (1973) are more cautious, recommending that endodontics should be postponed until other signs of non-vitality appear.

Barkin (1973) studied 31 patients with traumatized teeth over a period of one year post-trauma, using radiographs, carbon dioxide and electrical pulp testing. He made several findings: Firstly, that immediately post-trauma, patients were in a state of shock and were not reliable subjects; secondly, that no reliable pattern for prediction can be established during the first

three months; thirdly, after three months a prediction of vitality can be made in a majority of cases; fourthly, that there was a slight difference in the behaviour of teeth which had incomplete roots and mature roots at the time of the injury; 90.9% of the former and 80.9% of the latter group of teeth, which tested vital initially, still tested vital after one year.

El Deeb et al (1989) used the Analytic Technology Vitality Scanner (ATVS) to assess the vitality of three groups of maxillary canines in patients with alveolar clefts. They found that there were no significant differences in the unresponsiveness to testing between the canines which had been grafted and then exposed and orthodontically pulled into the mouth; those in noncleft patients which erupted spontaneously but required orthodontic traction for alignment; and those in noncleft patients, which were surgically exposed and bought into the mouth by orthodontic traction. They did find a trend which (was not significant) that 31% of canines that erupted through the grafted alveolar cleft in patients with unilateral cleft palate did not respond to pulp testing, where as all the canines on the contra-lateral normal side did. As a result of this finding, El Deeb et al (1989), and Turvey (1989) in his commentary on the article, recommend extended clinical and radiographic follow-up of these patients.

Andreasen et al (1990), who used electrical sensitivity tests as one of their criteria for the

assessment of autotransplanted teeth, also recommend long term followup of patients.

8. Multiple rooted teeth.

Grossman (1978) noted the problem of partial vitality within a multi-rooted tooth, where misleading vitality test results are possible. Mumford (1976) pointed out that the electric current passes through all the canals of a multi-rooted tooth, irrespective of pulp status, and cannot be localised to test one root.

9. The effect of drugs.

Sedatives, tranquilizers and analgesic medications will increase the pulpal nerve thresholds, by acting on the central nervous system.(Grossman 1978). Blair (1965) found that a placebo could also increase the threshold.

10. Age.

Chambers concludes that, with the exception of newly erupted teeth, the threshold of pain perception in pulpal nerves is stable in groups tested from 10 to 73 years.(Mumford 1963, Rubach and Mitchell 1965). Ruback and Mitchell (1965) did find an increase in the threshold to electrical stimulation associated with the deposition of secondary dentine. Ehrmann (1977) felt that this limits the validity of pulp testing in such teeth in the elderly.

11. The effect of periodontal disease.

Ruback and Mitchell (1965) found no increase in pulpal stimulus threshold associated with periodontal disease. Bender and Seltzer (1972) did find links between periodontal and pulpal inflammation.

12. Injury due to pulp testing.

There is the possibility of damage to the pulp during the testing procedure, especially with the use of carbon dioxide (Chambers 1982).

Despite the above reservations, Chambers (1982), reviewing the literature, agrees with Mumford and Bjorn (1962) and Ehrmann (1977), that pulp vitality testing is an integral part of oral diagnosis and treatment planning.

Rowe & Pitt Ford (1990) agree, but go on to emphasise the importance of careful assessment of the patient's history and clinical examination, before embarking on the special pulp vitality investigations.

Ehrmann suggested that pulp testing should be used in the following situations:

1. Prior to extensive dental procedures. Even where there are no symptoms, pulps may be nonvital or have severe disturbances. A definitive preoperative test may help to anticipate treatment complications, which could be regarded as avoidable and possibly embarrassing.

2. Diagnosis of pain. The localization of orofacial pain can be difficult and pulp vitality testing

is often necessary as an aid to diagnosis in the region served by the trigeminal nerve.

3. Differential diagnosis of radiolucent areas near the apices of teeth. Cysts, periodontal lesions, congenital abnormalities, neoplasms and anatomical structures are some of the radiographic radiolucencies seen in addition to the periapical lesions caused by a nonvital pulp. Vitality testing is a relevant procedure to help distinguish these pathologies.

Mumford and Bjorn (1962) described several more uses, for example:

4. Post-trauma assessment as an aid to treatment needs is controversial, but still important, according to Chambers (1982). Monitoring the recovery of sensation following trauma, surgical trauma, or vital transplantation is recommended.

Andreasen et al (1990a) conclude that the precision values for sensibility testing indicate that rather stable readings can be expected over a period of time. They report that this finding is useful for the evaluation of consecutive long term results and have applied this method to their longitudinal study of transplanted premolar teeth.

5. Assessment of anaesthesia prior to operative procedures and as a means of evaluating the effectiveness of anaesthetic agents.

6. On going assessment of teeth which have deep

restorations or pulp caps.

It is interesting to note the additional citations of Chambers (1982) about pulp vitality testing: Firstly, that Mumford (1965) found no definite differences in pain perception thresholds between the sexes and secondly; that lateral dominance of stimulus perception was not present. Mumford (1976) cites Wolff (1968) in expecting a higher threshold for pain on the dominant side, but could find no significant differences between the left and right sides in his study with Newton, published in 1972.

4.2. Methods of Pulp Vitality Testing

4.2.1.Radiographic

Thoma (1925) cited by Stephan (1937) relied on radiographic techniques (where he took several radiograms at different angles) as his preferred method for assessing pulpal infection.

Radiographs are still an important adjunctive diagnostic aid in most situations requiring definitive assessment of pulpal pathology (Chambers, 1982). However, Ziskin and Zegarelli (1945) state that radiographs are inadequate as a means of determining the vitality status of the pulp. Their main reasons for this statement are examples of pathological conditions which may be visible or invisible on radiographs, as well as radiographic artifacts. Farrell (1958) said that:

" Radiography can show the proximity of the carious process to the pulp, but gives little information about the probable condition of the pulp."

Rowe & Pitt Ford (1990) in their review of the role of the assessment of periapical radiographs, note that the changes observed are those of the more advanced or irreversible stages of inflammation (as in the case of internal resorption) or pulpal necrosis (as in the case of perapical bone loss or perapical condensation of bone as seen in condensing osteitis).

4.2.2.Temperature.

Chambers (1982) cites Jack (1899) as first recording the use of thermal pulp vitality tests. Naylor (1964) proposed that temperature changes caused vasomotor changes which stimulated nerves to propagate a response. Towbridge et al (1980) showed that responses were initiated before there were temperature changes recorded at the dentino-pulpal junction. They explained these results as showing that hydrodynamic pressure down the dentine tubules was responsible for activating the nerve endings. Mumford and Newtown (1970)and Horiuchi and Matthews (1973) had already demonstrated that fluid movement within the dentine tubules initiated a response.

Chambers (1982) notes that thermal tests are not quantifiable. The test merely gives a positive or negative result and the only pathologically linked correlation is between a negative result and non-vital pulps (Dummer et

al 1980). Although Ehrmann (1977) found there is anecdotal evidence that inflamed pulps have a more lasting response, after the stimulus of cold is removed. This reaction could be explained by a lack of accommodation in the vessels caused by the inflammation.

Rowe & Pitt Ford (1990) explain that negative results from thermal tests may indicate a pulpless tooth, as in the case of elderly patients, or where teeth have heavy restorations, but they may also be due to the thermal stimulus being inadequate.

Cold is regarded as superior to heat as a testing agent. Heated gutta percha, if not heated enough, may not elicit a response, thus giving a false-negative result (Lundy & Stanley 1969) and if heated too much, causes an intense pain (Mumford 1976). In the condition of gaseous necrosis, result of testing with heat may be falsely positive (Mumford 1976).

Rowe & Pitt Ford found heated gutta-percha or green-stick (with temperatures up to 150'C), to be effective in the diagnosis of irriversible pulpitis, because the pain lingers after the stimulus is removed. They cite studies illustrating the dangers of heat generating procedures: Firstly Zach (1972), who showed that a rise in temperature of 11'C is likely to cause irreversible pulpal damage; and secondly, Narhi et al (1982), who demonstrated stimulation of pulpal C fibres with temperature increases of 7'C.

Carbon dioxide ice (which has a temperature of

-78'C), or dichlorodifluoromethane, are better agents for the introduction of cold, when compared with water, ice, and ethylchloride (Chambers 1982).

Carbon dioxide ice is the only effective means of testing newly erupted teeth, but becomes unreliable in the elderly (Ehrmann 1977).

Carbon dioxide is not reliable immediately post-trauma and as Shepherd (1976) demonstrated odontoblast cell death was caused by temperatures of only -10'C in rat pulps, after application times of less than 30 seconds, so carbon dioxide should not be applied for too long.

Ehrmann (1977) was concerned that carbon dioxide testing caused cracks in enamel, but felt that these were not clinically significant. Bachman and Lutz, cited by Chambers (1982), found that carbon dioxide created more enamel cracking than dichlorodifluoromethane.

The Chloro-fluoro-carbons have been generally identified as being environmentally dangerous, causing degradation of the atmospheric ozone layer, so the use of these may soon be discontinued.

4.2.3.Blood flow.

Chambers (1982) refers to the work of Reich (1952), Taylor (1960), and Shoher et al (1973), in attempting to use the absorption of certain wavelengths of light, by the haemoglobin in pulpal blood, to demonstrate vascular viability. If the tooth is heated, the blood flow should increase, vessels should dilate and a change should

be observed. Chambers (1982) concluded that the reliability of the techniques had not yet been proven.

Advancements in laser technology have led to the development of Laser Doppler flowmetry (Wilder-Smith, 1988), where the movement of blood creates a doppler shift in the wavelenght of the scattered light beam, which can be used to determine the velocity of the blood within the pulpal vessels (Rowe & Pitt Ford, 1990). This technique was used by Olgart et al (1989), to assess the effects of neurogenic mediators in the control of pulpal blood flow, because it is a non-invasive technique.

Rowe & Pitt Ford (1990) explained that while Laser Doppler flowmetry is a good research tool, care must be taken to obtain reproducible recordings and the cost of the equipment is at present 'very expensive.'

Attempts to measure the temperature of vital teeth and compare these with nonvital teeth have been made by Howell et al (1970), Stoops and Scott (1976), and Banes and Hammond (1978). These techniques have so far not proven practical.

There are two main obstacles to these techniques:

Firstly, Brown and Goldberg (1966) showed that the periodontal tissues were the main source of heat measured at the tooth surface and that the pulpal blood flow was not as significant.

Secondly, Bernick (1976) and Takahasi (1985) have shown that there are significant vascular changes with age

in healthy teeth.

4.2.4.Ultrasonics.

Barber et al (1969) using alumínium probes (which conduct sound in a similar fashion to enamel), were able to identify the dentino-enamel and pulpo-dentine junctions, but use of this technique has not advanced to determine pulpal vitality (Seltzer and Bender, 1975).

4.2.5.Electrical.

Reynolds (1966) attributes the use of electrical stimulation of teeth for diagnostic purposes as dating back to Magitot in 1867.

Stephan (1937) reviewed the literature on the development of electrical pulp testing techniques and cites Raper (1921) as stating that:

"..the electric pulp tester is of great value in determining whether a pulp is vital or necrotic, but that crowned teeth and some teeth with large fillings, cannot be tested; that in a multi-rooted tooth with part of the pulp vital and part necrotic the reaction is the same as if all were vital; and that it is not practical to attempt to determine any particular pathologic state of a vital pulp with the electric pulp tester."

Prinz (1928) cited by Stephan (1937) introduced the threshold concept in stimulation response, recording that normal teeth responded to Faradic current at a specific "irritation point." He proposed that hyperaemic

and inflamed pulps responded at lower levels; suppurating pulps above the normal; and dead pulps not at all.

Stephan (1937) cites the findings of Reiss and Fuerdi (1933), who demonstrated that Prinz's categorization was too simplistic because the reactions among the different groups overlapped considerably. Stephan concluded that:

" A negative reaction always indicated pathology, and nearly always necrosis, but a positive reaction did not indicate a normal pulp with any certainty."

Klein in reviewing the literature put forward Schaffer's work:

"That an individual may respond differently to an electric pulp stimulator on different days and at different hours on the same day."

The modern view of electrically stimulated thresholds is presented by McGrath et al (1983), who studied the stability of electrically stimulated thresholds and showed that while there is great variation in the sensory detection thresholds of different teeth within individuals and between individuals, a single tooth will have a fairly constant threshold if observed over a period of time.

4.2.5.1 Disadvantages of electrical pulp testing.

Chambers (1982) described the disadvantages of electric pulp testing up to that time as being clumsy to use; requiring isolation; frightening to patients; painful;

and possibly eliciting responses from the periodontal ligament. Kitamura et al (1983) and Cooley et al (1984) describe the modern Analytic Technology Pulp Testers as overcoming these problems.

4.2.5.2 Advantages of electrical pulp testing.

Ingle and Beveridge (1976) agree with Degering's work (1962), that an electric apparatus was the most effective means of determining pulp vitality, when compared with air, explorer or the application of cold or heat.

Ehrmann (1977) proposed that electric pulp testers are superior compared to thermal testing for elderly patients with deposits of secondary dentine.

Fulling and Andreasen (1976a) recommend the electric pulp tester be used in the aftermath of trauma, in preference to carbon dioxide, the risk being that the use of carbon dioxide could cause pulpal damage, before a reaction, if any, was elicited.

4.2.5.3 Potential risks of electrical pulp testing.

Woolley et al (1974), in experiments on dogs, found that there may have been a danger that the electric pulp testers of that time posed a risk to patients with cardiac pacemakers fitted. Matthews and Searle (1974) concluded that healthy patients were not at risk because they would not receive an impulse current sufficient to cause ventricular fibrillation.

McDaniel et al (1973) studying sacrificed

monkey's teeth after subjecting them to five hours of stimulation at maximum current and voltage, concluded that in clinical situations electric pulp testing is unlikely to cause any pulpal damage.

4.2.5.4 The method of electrical pulp testing.

The development of electrical pulp testers has been a search to arrive at the best delivery system for the electrical stimulus, both in electrical character and the physical method of delivery, which allows accurate determination of a tooth's sensory detection threshold.

There appears to be no difference between the use of alternating or direct currents on sensory detection thresholds (Mumford 1976).

Mumford and Bjorn (1962) presented an important technique protocol when they reported that:

"It should be remembered that tissues, including teeth, have a lower threshold to cathodal than anodal stimulation and that the difference is considerable."

In recommending cathodal stimulation, they quote Bjorn as finding the difference as being 1:2.5 in current strengths required to produce a response.

The use of monopolar versus bipolar electrodes has been debated, because, theoretically, the bipolar device sends a current only through the crown of the tooth and this may reduce the risk of a false-positive reading produced by stimulation of nerves in the periodontal ligament. The disadvantage of this was that the use of the

bipolar instrument necessitated the use of much higher stimulus voltages, because two layers of high resistance enamel needed to be negotiated by the current. This reduced the accuracy of threhsold detection.

Greenwood et al (1972) found an overlap in the thresholds of nerve fibres of the pulp and periodontium in cats stimulated with a monopolar device. Nahri et al's (1979) results disagree, however, by showing that pulpal nerves responded at lower thresholds than periodontal nerves, and this was also in agreement with earlier observations by Ziskin and Wald (1938) and Bjorn (1946), who had found that with careful technique the pulpal nerves could be preferentially stimulated. Matthews and Searle (1974) could not determine the intensity threshold at which the periodontal tissues responded. This is further evidence that there is a large difference between the periodontal and pulpal thresholds.

Chambers (1982) concluded that, using monopolar probes, the technique of isolating, drying and placement of the probe in the incisal third of the tooth enamel, with a conductive medium, are all important factors if recruitment of the periodontal receptors is to be avoided.

The use of a conductive medium is necessary to ensure that the desired current is delivered to the tooth. Cooley and Robinson (1980) showed that in the absence of a conductive medium between probe and tooth, very little current or voltage was passed to the tooth. Fluoride gels are better than water or tooth paste for this purpose,

according to Cooley and Robinson (1980).

Chambers (1982) gave his opinion that recording the level at which a tooth responded to an electric stimulus was 'valueless', because of the individual's varying state and because at that time the commercial testers gave little control over the current being delivered.

Robinson and Cooley (1980), in reviewing the performance of the Digilog pulp tester, demonstrated reliability problems.

Later, however, Cooley et al (1984) reviewed the Analytic Technology pulp tester and recommended it as being more accurate (with fewer false-positives and -negatives) and dependable (by only operating when an electrical circuit is complete and delivering a reproducible stimulus), than previous electric pulp testers. The gradual increase in stimulation was more acceptable to patients, with the sensation being described as 'vibration, tingling, pulsating or like cold air'. They were still cautious in attempting to quantify the response obtained, stating that:

"An electric pulp tester should be used only to determine vitality or nonvitality and not for diagnosis of pulpal conditions. The clinician should not try to quantitate the response between teeth at any particular digital reading, as other variables may be present."

Cooley et al did not explain what they thought these variables were, nor did they include references substantiating this statement.

Ingle and Beveridge (1976) are more liberal in their interpretations, writing that:

"..to a certain extent, moreover, it is possible to quantify the responses to testing, and these figures may be consulted in follow-up examinations. This is especially true for the electric pulp tester."

They apply a 'qualifying caveat', that any testing procedure may be modified by the emotional or physical state of the patient, especially in the presence of pain. They also note that recently erupted teeth may not respond to electric stimulation. In order to accurately compare changes in vitality, the same pulp tester should always be used on the same patient.

Johnsen, et.al. (1979) compared electric pulp testing readings between the deciduous canines of children and adult permanent canines. These teeth were compared because although the deciduous canines have, on average, slightly greater numbers of myelinated and unmyelinated nerve axons, the difference was not significant. They used positive current pulp testers and write that:

"They will determine a general level of sensation, but are crude considering the number of variables which can influence readings."

They list these variables as: patient age; mental capacity; ability to cope with stress in the dental setting; and inhibitions to respond to stimuli.

Sensitivity also varies with the development of the root apex (Refer back to Chapter 4).

Ingle and Beveridge propose that the best control tooth for reference is the contralateral partner, because of variations in the enamel thickness (affecting resistance) and anatomy (the size of the pulp may affect the response.)

Andreasen et al (1990a) found in their study of 187 permanent premolars that homologous premolars gave very close sensitivity readings.

Johnsen et al (1979) wrote of the difficult area of 'interpretation of the sensory impulses .' Studies use thresholds of the sensation of discomfort. This 'first pain' threshold interpretation is related to the individual's ability to perceive pain.

Johnsen, et.al's results showed that despite all the above:

"Physiologically they (the canines) are similar in their thresholds for responses to electrical stimuli."

Fulling and Andreasen (1976b) found that thermal and electrical pulp testing procedures may be carried out in the presence of different splints and temporary crowns. Electric pulp testing, provided that it was applied to enamel and not to any metal coverage, appears to be superior to thermal stimuli for diagnosing pulp necrosis in traumatised teeth.

Jacobsen and Kerekes (1977) write that:

"When diagnosing pulp necrosis after traumatic injuries, electrometric pulp testing appears to be superior. However, a gradual narrowing of the pulp cavity

usually leads to reduced electrometric response."

Torneck and Howley (1989) found, using averaged sensory detection thresholds of pulpal reaction to electrical pulp stimulation and periodontal tactile response, that results were constant over periods of time (up to 180 days). This observation, they believed, could be used to do longitudinal studies of the maturation of the dentition. The combining of these separate sensory observations into a profile of maturation takes interpretation further than those of Cooley et al.(1984).

Andreasen et al (1990 a & b) using a cruder electric pulptester (Siemans Sirotest 2 which has a scale of 0 to 4 with half units registrable), than the one used by Torneck and Howley, studied the long-term sensitivity of 187 maturing teeth. They used these results to make comparisons with the maturation of 370 autotransplanted premolars. Their readings were taken at 6 months, 1 year, 2years, 5 years and 10 years.

Kleier et al (1982) conducted a study comparing three pulp testing methods and concluded that the Analytic Technology Pulp Tester using a negative pulse with a slow rise in voltage produced a non-painful tingling sensation at the threshold stimulus. This technique was judged to be the least painful when compared with a fast rise in voltage from the same instrument or with a similar rise in bipolar voltage wave generation, created by another instrument.

The finding of a pre-pain threshold agreed with the work of Civjan et al (1973), who reported that slow

rises in voltage stimulus reduced the amount of pain perceived, and Grayson (1977), who reported that slow rises in a monopolar negative stimulus produced a prepain sensation.

A modern electric pulp tester, the Analytic Technology tester, has been reported by several authors (Kitamura et al, 1983; Cooley et al, 1984; Abbot, 1988) as inducing a 'pre-pain' or 'tingling' sensation, when the stimulus rises to approach the pain threshold. This reduces patient discomfort and anxiety about the testing procedure, because the stimulus is detected before pain is induced.

Using this information, Torneck and Howley (1989) recommended the use of the Analytic Technology Vitality Scanner (ATVS), which produces a variable current and variable voltage in progressively increasing increments, as the most reliable instrument for detecting the 'pre-pain thresholds', which they describe as 'sensory detection thresholds.' The instrument has a cathode probe and does not begin to function until the circuit is completed by either the operator making skin contact with the patient's face or by the patient touching the handle of the probe.

4.3 The Electrical Properties of Human Teeth.

Mumford (1967) found the resistivity of enamel to be 4.5 +/- 1.58 MΩ and dentine to be 33 +/- 13.0 KΩ. This agrees with previous work done by Farrell (1958), and Ziskin and Zegarelli (1945), which also show that the resistance of enamel is much higher than dentine.

Mumford (1967) demonstrated that the resistance of enamel was increased considerably if the specimen was dried and was greatly reduced if the enamel was cracked. If the sections of enamel were allowed to imbibe saline, significant reductions in resistance were noticed within ten minutes.

5 EXPERIMENTAL WORK

The project was approved by the Human Ethical Review Committee of the University of Sydney.

The subjects of the study were patients and staff within the Orthodontic Department of the United Dental Hospital and the State Orthodontic Service at Dubbo.

The patients were verbally informed of the usual reasons for testing tooth vitality in dental practice. They were asked if they wished to be screened and informed that if they did, that the results could be used in a research study.

Informed consent was obtained verbally, because the procedure is part of standard dental practice. The patients were advised of the benefits of the analysis and function of the ATVS appliance being used for the test. They were informed of the feeling expected and the time taken for the procedure. The patient was then given the opportunity to ask questions about the procedure. It was explained to them that, if they wished, they could stop the assessment at any time.

The patient was then asked if they wanted to have their teeth checked and upon consent, a trial test was done on the upper central incisors. On completion of this procedure, the patient was asked if there were any problems and given the option of discontinuing if they wished.

The form used to record their consent, appears in Appendix 1.

The measures taken to protect the rights and

welfare of individuals in the study were that the information gathered was kept in the patient's clinical file and a copy made, which only had the patient's initials, age, sex and notes of clinical observations. Individual patients were not identified by name in the study.

No remuneration was given for participation in the study.

5.1 Hypopthesis

As mentioned in the introduction, the hypothesis tested was that:

There would be no difference in the variation of vitality reactions, as determined by ATVS electric vitality testing, between the following four groups of patients:

1. Patients who were banded-up, bonded and fitted with initial archwires

2. Patients, with appliances fitted, who were debanded and debonded.

3. Control patients who underwent a repeated test, which was delayed at least 45 minutes.

4. Control patients who underwent immediate repeat vitality tests.

5.2 The Experimental Method

In order to test the hypothesis, the following experimental method was developed:

1. The number of participitants was 91 (28 band-up, 42 deband , 21 control). There were 34 males and 57 females. The patients' ages were from 9.5 years to 35 years. Only healthy patients with asymptomatic dentitions were examined and screened.

No patients with any history of heart disease, or who were fitted with cardiac pacemakers, were tested.

No patients were tested if they were taking analgesic or narcotic drugs.

All subjects were given a dental examination, including periodontal assessment and most but not all, had a radiographic survey as well. All teeth were examined, but teeth with a history of recent trauma, teeth with periodontal lesions and teeth with caries, fissure sealants or restorations were excluded from the study. Partly erupted teeth were also excluded, on the basis that they may not have complete apexification.

2.The experimental apparatus used was the ATVS.

The specifications and performance of the ATVS were, according to Analytic Technology; Kitamura et al, 1983; Cooley et al, 1984; and Torneck and Howley, 1989:

The instrument turns itself on automatically, when the resistence across the probe mantle and probe tip falls below 5MΩ. A red light on the mantle is turned on,

and remains on, while a contact across the terminals is maintained, indicating that the instrument is activated. This eliminates the possibility of a false negitive diagnosis, where the patient does not feel the stimulus because there is a break in the electrical contact.

The range of current delivered at 1MΩ across the instrument terminals rises in increments from 15μA to a maximum of 300 μA. The output voltage is stabilised and is generated with 150 KΩ internal impedence. The use of current alone as a measurement of vitality, reduces the influence of the variable resistance of the tooth contact and the enamel. It would be very moisture dependent, and also require electronic insulation from adjacent teeth. Any moisture on the enamel surface could create shunting currents and thus affect the accuracy of the reading. This problem is created because of the high resistence of enamel and is overcome by recording the SDT voltage instead. (Analytic Technology)

The range of output voltage delivered at 2MΩ across the instrument terminals rises in increments beginning from -15V up to -300 V.(Analytic Technology)

Ten short pulses of current make up a 'train' of stimulation, and are represented on the LED display as one increment on the scale of 1 to 80, used to record the stimulus level. The delay between trains is ten pulses long and may be varied by the operator turning the rate scale on the instrument.

Torneck and Howley used a setting of 5 on the

instrument's rate scale, which gave ten pulses of current 18 msec apart, lasting 0.22 msec, making up 2.7 'trains' per second, with the delay between trains being 180 msec.

When the circuit is broken the number on the LED remains on the screen for a period of 10 to 15 seconds, after which the instrument turns itself off.

There is a period of 0.5 to 1.0 second, immediately after the probe is removed, before the reset mechanism is triggered. This feature allows for the accidental movement of the patient or probe during the test, where the contact may be broken momentarily. The reset mechanism turns the red light off and returns the instrument to its dormant state, ready for the next test procedure. This feature prevents the operator from giving the patient a jolt with a supra-thresold stimulus from the previous tooth, a fault in technique which was possible with the old manually reset pulp testers. (Analytic Technology.)

There is a low battery indicator signal.

The specifications of ATVS performance were checked for accuracy by the Biomedical Engineering Department at Westmead Hospital. The effects of different battery voltage levels and different levels of simulated patient impedance were examined.

3.The test procedure.

Each tooth examined was tested twice using the ATVS, in order to establish the variation in the pre-pain threshold response.

Four groups of patients were examined:

i) Band-up patients were tested, once prior to the band-up, and once after the bands, brackets and initial arch wires were fitted. Patients were given a few minutes to become familiar with their appliances and to allow the oral cavity to resume its normal temperature, before the second test.

ii) Deband patients were tested, once prior to the removal of the appliances and again after the bands, brackets and bonding agents were removed. Patients were given a few minutes to allow themselves to become accustomed to their new situation and to allow the oral cavity to resume its normal temperature, before the second test.

iii) Control patients were divided into two groups, both tested twice, one with a time interval between tests of at least 45 minutes, and the other having the test repeated immediately.

All patients were given the opportunity to familiarise themselves with the procedure by use of a trial test, which was always conducted on the upper central incisors, prior to the actual recording procedure.

During the procedure, patients were encouraged verbally, to concentrate on their teeth and to indicate the sensory detection threshold by raising their right hand from the instrument, thus breaking the circuit so that the machine gave a reading. This technique for the use of the ATVS was recommended by Calleteau and Ludington (1989), who

claimed that the protocol had several advantages:

A. The patient is in control of the stimulus, so they feel more relaxed. Less apprehension results in a more accurate reading being made.

B. The time lag between the patient beginning to feel the stimulus and its removal is shortened by eliminating the operator's reaction time, because it is the patient who breaks the circuit. This reduces the patient's discomfort.

C. The procedure is simplified.

D. The operator can remain gloved throughout the procedure.

Other noises, conversations and distractions were discouraged in the immediate vicinity. These procedures were standardised in an effort to focus the patient's attention and to remove extraneous factors which may have distracted the patient from determining the exact point at which the threshold was reached (Topazian, 1957).

All teeth were isolated by use of cotton rolls and dried by compressed air.

Readings from 0 to 80 on the LED display of the ATVS were recorded. The machine was set at a rate of increase of 2, which gave a slower rise in current and voltage with time, than Torneck and Howley (1989) used in their study. This gave the patient better discriminative power in determining the sensory detection threshold.

Bender et al (1989) found that the lowest threshold response to electrical stimulation was when the

probe was placed on the incisal edge of a tooth, with the highest being in the gingival one third. Placement in the gingival one third has the risk however, that there may be contact with the gingival tissues by the probe or conduction media, which would cause a leakage current, possibly distorting the reading.

In this experiment, the electrode was placed on the same position on the tooth surface on each occasion, so that the same thickness of enamel was in the path of the current. The position on which the electrode was placed was most frequently the inciso-lingual one third position of incisors, canines and premolars, and the central occlusal fossa of molar teeth. These positions were selected because of the need to have a reproducible test position on the tooth, which was not covered by the appliances and was well away from the gingival margin. Continuity of contact was aided by use of an electrolytic fluoride gel supplied by the manufacturer.

If the patient was unsure about the threshold, the observation was repeated. All non-response observations were repeated. Patients who were found to have nonvital teeth were informed of the sequelae and consequences of pulp death and offered additional followup observations and treatment as required.

All observations were made by the same operator, using the same instrument. Teeth were tested in the same order in both tests and in the same order in all patients.

The difference between the recordings for each

tooth were calculated, giving a positive or negative number, which represented a decrease or increase in the sensory detection threshold.

Statistical advice was given by Ms Adrienne C. Kirby B.Sc.(Hons 1), M.Sc, of the Department of Biometry, University of Sydney.

6 RESULTS AND DISCUSSION

6.1 Results

The ATVS was tested for its electrical characteristics using a Tektronix 50 Meg Hz Oscilloscope and Resistance Box 1051 produced by Time Electronics.

It was found that at maximum amplitude of 80 on the LED display, a 300V output was found with a 1MΛ load and an internal supply impedence of 120KΛ.

The instrument was able to maintain this output level as the battery voltage dropped from 6V to 3.7V. Below 3.7V the performance was compromised, but the instrument was still functioning. An example of this was that if the battery voltage was at 3.0V, a 170V output was recorded. The pulse width and time between trains remained accurate, so it was only the output voltage which was affected.

The low battery indicator became active as the battery voltage dropped below about 4.1V, which safely ensured the output voltage was reliable.

The output was a train of 10 positive pulses applied to the handle of the instrument, so that the probe was negative.

When the machine was set at 1,2 and 3 on the rate scale, there was a 24 millisecond gap between the 10 pulses. The gap between the pulse trains was 240 milliseconds.

When the resistance between the handle and the probe was reduced significantly below 1MΛ, the output

voltage dropped, as shown in Table 1.below.

Since the resistance of enamel remains constant at over 1MΛ, it can be assumed that the maximum output for the tests was, as expected, at 300V. From this, it can be concluded that minor differences in the skin resistence of the patient's fingers, between the two reading will not change the recorded SDT level, provided that the circuit is maintained (as shown by the red light current indicator light).

The ATVS used in this study met the characteristics of accuracy and reproducibility specified by the manufacturer and other reviewers. (Kitamura et al, 1983; Cooley et al, 1984; and Torneck and Howley, 1989)

Table 1.

RESISTANCE	OUTPUT
1MΩ	300V
900KΩ	295V
700KΩ	290V
500KΩ	270V
300KΩ	240V
100KΩ	160V
10KΩ	30V

The characteristics of the ATVS showing how changes in the resistance across the handle-probe terminals affect the maximum voltage output (read as 80 on the LED display).

6.2 Analysis and Discussion

In the 91 patients examined, 1427 individual teeth were tested.

In the results of the vitality testing there were three groups of teeth observed:

A) Teeth which reacted in both tests, giving a threshold value number on the ATVS LED.

B) Teeth which did not react in the first test (despite having the procedure repeated), which gave a threshold greater than 80 on the LED (meaning that the threshold was in excess of 300V), but did react in the second test. This indicated that the threshold had decreased. The results from these teeth were tagged and treated separately in the statistical analysis of results. See Appendix 2.

C) Teeth which did react in the first test, but did not react in the second test (despite having the procedure repeated), which gave a threshold, over 80 on the LED (meaning that the threshold was in excess of 300V). This indicated that the threshold had increased. The results from these teeth were tagged and treated separately in the statistical analysis of results. See Appendix 2.

Only Group A (called Reduced Set, without tagged teeth) data was analysed for the first statistical procedure, which was an analysis of variance procedure, with a factorial analysis of sex (male or female) and

treatment groups (bandup, deband, control 1 and control 2).

A least squares regression technique was used, to obtain a standard nonorthogonal analysis of variance. Patient age was also included as a co-variate. This analysis was rejected due to the variability of the data.

The Means and Standard Deviations of the results of Group A (the Reduced Set), appear in the tables of Appendix 2: Table 1 (Means for males); Table 2 (Standard deviations for males); Table 3 (Means for females); Table 4 (Standard deviations for females); Table 5 (Means for the combined males and females); Table 6 (Standard deviations for the combined males and females).

The statistical analysis showed that the data was extremely variable. For example, males' tooth 31 (F.D.I.) Average= 3.44 S.D.+/-9.25.

For every tooth, the error mean square was enormous compared with the difference observed between the groups. Each group was expected to have the same variance, but this was not the case. For example, males' tooth 31 variance for bandups was 85.5; for debands was 53.5; for control 1 was 0; and for control 2 was 2.3.

The Means of Groups A, B and C, together (called the Complete set) are included in Appendix 2, in tables 11, 12 and 13. It was concluded that an analysis of variance of this data was not necessary.

The next step in the analysis of the data, was to use a nonparametric technique called a Kruskal-Wallis test.

This test cannot be used for factorial analysis, so separate tests were done to evaluate sex differences and treatment group differences.

The first use of the Kruskal-Wallis test was an analysis for sex differences in the results of Group A (Reduced set). These results appear in Appendix 3, Table 7.

Looking for the possibility of sex differences, a probability of less than .05 was set for the level of significance. Sex differences were not generally found, with the exceptions of the 47,46,45 & 41, where slight differences were found. Two examples were the 47 where p= .024 (Medians were: males 8.0, and females -2.5), and 46 where p= .039 (Medians were: males -4, and females 2). These differences in the medians were not consistent and lead to the conclusion that they were spurious changes. If this assumption is correct then both sexes can be grouped together, ignoring a sex effect, for an assessment of treatment effects.

The second use of the Kruskal-Wallis test was for treatment effects in Group A (Reduced set). These results appear in Appendix 3, Table 8.

There was no generalised difference between any of the bandup-bondup, deband, control 1 or control 2 treatment groups.

Only two specific teeth demonstrated significant differences. These were the 26 (p = 0.004) and the 36 (p = 0.006), with the 46 (p = 0.057) being almost significant. In these teeth it was found that the deband/bebond group

had a slightly lower ranking, than the bandup group or the control groups.

In order to study the influence, if any, of the teeth which did not react in one of the tests, Groups A, B and C were grouped together (called the Complete Set, all tagged teeth were included) and the Kruskal-Wallis tests for sex differences and treatment differences were repeated.

The results of the Kruskal-Wallis test for sex differences appear in Appendix 3, Table 9. They show no generalised differences between the sexes, with only the 41 demonstrating a significant difference ($p = 0.014$) and the 45 being almost significant ($p = 0.054$)

The results of the Kruskal-Wallis test, when the complete sets of males and females were grouped together and analysed for treatment differences, appears in Appendix 3, Table 10. They show that there was no generalised difference between any of the bandup-bondup, deband, control 1 or control 2 treatment groups. Teeth 16 ($p = 0.016$), 26 ($p = 0.00$), 36 ($p = 0.00$) and 46 ($p = 0.003$), were the only teeth with significant differences, with the deband group ranking lower than the bandup and control groups. This means that changes observed between the readings taken from the specific teeth which had stainless steel orthodontic bands removed, ranked lower than changes from teeth which were banded-up with stainless steel orthodontic bands.

7 SUMMARY AND CONCLUSIONS

91 patients (28 band-up, 42 deband and 21 control, of whom, 34 were males and 57 females, with ages from 9.5 years to 35 years), were examined with the aim of the establishing if there was a difference between the sensory detection threshold response of healthy unrestored teeth, as determined by electric vitality testing with the Analytic Technology Vitality Scanner (ATVS), as the teeth were fitted with banded and bonded orthodontic appliances or when these appliances were removed. 1427 individual teeth were tested.

The hypothesis tested was that:

There would be no difference in the variation of vitality reactions, as determined by ATVS electric vitality testing, between the four groups of patients.

The following conclusions can be drawn from the results of this experiment:

1. Thresholds did not seem to be stable enough to be relied upon to judge treatment effects by parametric methods.

2. A nonparametric analysis (the Kruskal-Wallis test) attempting to find sex differences, could not demonstrate their general presence.

3. A nonparametric analysis (the Kruskal-Wallis test) attempting to distinguish treatment effects, as measured by an increased or decreased threshold, could not demonstrate a general significant difference, so the

hypothesis is accepted. There was no general demonstrable difference between the groups.

4. There may be some difference in the behaviour of teeth which were banded with stainless steel orthodontic bands, but no differences could be found in the treatment groups between teeth with bonded stainless steel brackets.

This study casts doubts about the assumption that sensory detection thresolds (SDT's) can be averaged in the way that some authors have done. An analysis of variance on this data, which was based on a larger sample of patients than other studies, showed that averaging SDT's was not reliable.

The results of this study agree with those of Lado et al (1988), who found that in a sample of eight patients, thresholds were not reproducible in repeat tests.

This study did not find any difference in the proportions of teeth which did not react during the test period in the different treatment groups, and as no differences or trends were found in the rankings of the threshold changes teeth bonded with stainless steel brackets, it may be concluded that the electric vitality testing in the circumstances described is no less reliable than at any other time.

Future studies using Laser Doppler flowmetry (LDF), studying the dental pulp blood flow in similar situations as those examined in this thesis, may be a more useful means of assessing any possible effects of

orthodontic treatment. A longitudinal study by LDF could be undertaken to study the long term effects of orthodontic treatment and may reveal information about any possible links between orthodontic tooth movement and such phenomena as internal root resorption and root canal calcification.

APPENDIX 1.

PATIENT CONSENT FORM:

I, ...,

of

(address).....,

have had the project explained to me and have had the opportunity to ask questions. I understand the procedures involved and their potential risks and benefits.

I wish to participate in the study and consent to my records being used for this research purpose.

I understand that the confidentiality of my records will be preserved.

I also understand that I have the right to stop the procedure and withdraw my consent at any time.

S i g n e d . . . . . .

Witnessed.....

Date.....

I give my consent for my child to participate in this study.

Signed..... (Parent or Guardian.)

Witnessed.....

Date.....

APPENDIX 2

STATISTICAL TEST RESULTS

The abbreviations used are:

d.f. — Degrees of freedom

Rx — Treatment groups:
1. Band-up
2. Deband
3. Control Group 1
   (second test at 45 minutes)
4. Control Group 2
   (second test was repeated immediately)

p — Probablility for obtaining the H value for the number of degrees of freedom specified.

S. — Significant probability (<0.05) result

A.S. — Almost Significant result (Close to 0.05)

N.S. — Not Significant result (>0.05)

H — The value calculated in the Kruskal-Wallis test, which given a number of degrees of freedom and probability, determines whether the null hypothesis is accepted or rejected.
H values may be compared with the Chi square tables.

Z — Where a significant difference between the four treatment groups was found, a multiple comparison analysis was done. In each case, the Bandup was significantly higher than the Deband, as shown by Z > 1.96 (0.05 level of p).

TREATMENT GROUPS

1. BANDUP/BONDUP
2. DEBAND
3. CONTROL 1. (45 min. repeat test)
4. CONTROL 2. (Immediate repeat test)

Complete Set - All data was used, including the tagged Group B and Group C teeth.

Reduced Set - Group A teeth only.

TABLE 1.

MEANS FOR MALES (REDUCED SET)

Tooth	Treatment Group				
	1	2	3	4	ALL
17	-18.000	-11.000	1.000	7.000	-6.400
16	7.500	-2.333	--	0.000	0.643
15	7.000	5.111	0.000	-8.500	3.125
14	0.0000	-4.1250	-12.0000	5.0000	-2.6923
13	0.300	3.462	-1.500	5.333	2.179
12	2.1250	-1.3846	1.5000	10.0000	1.2308
11	4.0000	0.3636	-8.0000	5.3333	2.2222
27	6.0000	-7.0000	0.5000	-3.0000	-1.6667
26	21.500	-4.222	12.000	-1.000	5.611
25	9.3333	0.1429	1.0000	-1.0000	2.4167
24	0.0000	-1.8750	-32.0000	5.0000	-3.0833
23	-0.6000	0.0000	-1.5000	0.3333	-0.2963
22	4.7000	1.0714	-5.5000	7.0000	2.4828
21	-0.4545	3.2500	-0.5000	4.3333	1.6429
37	8.000	-2.333	-5.000	5.000	0.167
36	15.500	-3.000	--	5.000	1.091
35	-1.250	-4.571	0.000	2.000	-2.692
34	6.600	0.222	1.500	2.000	2.333
33	4.111	-1.667	-0.500	13.000	1.680
32	6.900	1.417	2.000	-2.333	3.074
31	3.4444	3.0909	3.0000	-4.3333	2.3200
47	--	1.0000	10.0000	8.0000	6.3333
46	-1.500	-7.667	-3.000	-4.000	-5.455
45	-9.000	-7.125	-2.500	3.000	-6.333
44	-1.600	1.286	-1.000	-1.000	-0.187
43	-3.2857	-2.5833	1.0000	1.6667	-1.9583
42	0.9000	-1.0000	6.5000	8.0000	1.2593
41	6.800	3.333	-0.500	0.667	4.037

TABLE 2,

STANDARD DEVIATIONS FOR MALES

Tooth	Treatment Group				
	1	2	3	4	ALL
17	--	14.142	--	--	12.361
16	39.077	5.268	--	--	19.751
15	11.136	9.117	8.485	13.435	10.118
14	4.2426	10.1198	--	16.9706	10.2419
13	17.366	8.705	4.950	5.508	11.898
12	5.4363	7.5004	3.5355	8.1854	7.3610
11	6.7689	4.3191	--	4.1633	5.9957
27	--	12.7279	2.1213	--	7.6594
26	8.240	7.742	--	2.828	14.017
25	7.6376	7.0576	--	--	7.4402
24	--	4.9117	--	2.8284	10.2908
23	6.7032	12.3067	9.1924	5.0332	9.2229
22	14.5071	3.6682	4.9497	3.6056	9.2297
21	5.5743	5.3279	2.1213	3.7859	5.3279
37	--	4.041	--	--	5.707
36	28.991	6.949	--	--	13.202
35	18.927	13.214	--	--	13.493
34	17.141	4.842	0.707	0.000	9.381
33	7.079	14.361	3.536	4.243	11.463
32	12.801	9.913	4.243	6.028	10.590
31	9.2481	7.3137	0.0000	1.5275	7.5703
47	--	--	--	--	4.7258
46	0.707	12.848	--	0.000	9.470
45	10.033	11.192	3.536	--	9.788
44	2.191	11.828	--	6.557	8.052
43	10.0948	8.7330	1.4142	5.0332	8.2803
42	8.1982	7.4346	13.4350	11.3578	8.5602
41	9.438	10.790	3.536	5.859	9.448

TABLE 3.

MEANS FOR FEMALES (REDUCED SET)

Tooth	Treatment Group				
	1	2	3	4	ALL
17	--	-4.667	0.667	-7.000	-3.250
16	13.000	-3.077	6.750	-5.333	2.296
15	2.000	6.824	1.200	1.800	4.333
14	2.1429	4.0000	-1.5714	-2.8333	1.4865
13	-1.364	0.478	-7.000	0.429	-0.915
12	3.2500	-1.6667	4.3333	-0.8000	0.5909
11	3.5000	0.0455	0.6667	-1.0000	0.8913
27	--	-7.2500	-1.2500	5.6667	-1.5455
26	16.429	-3.308	-2.000	-2.000	2.148
25	2.6667	-2.6471	2.6000	4.0000	0.0000
24	0.4286	2.2857	-1.2000	-1.5714	0.5455
23	3.1538	-1.0000	-3.0000	0.8333	0.1042
22	-0.0833	-3.7391	1.5000	-0.1667	-1.6809
21	2.2500	-1.0909	4.0000	1.4000	0.6818
37	--	-2.333	6.500	21.000	6.857
36	15.400	-7.778	6.250	4.333	2.143
35	0.833	0.583	1.571	3.143	1.406
34	-5.375	1.467	-0.250	5.143	0.342
33	0.417	-0.571	1.250	4.250	0.755
32	0.750	0.333	1.125	-0.500	0.429
31	1.0000	-4.4286	-2.6250	0.7143	-2.0851
47	--	-4.0000	-2.3333	1.0000	-2.1250
46	11.875	-6.714	-1.000	7.000	2.737
45	4.500	0.500	4.167	-3.286	1.129
44	-3.333	-2.333	5.875	-4.286	-1.139
43	1.9167	0.1500	-1.6250	-2.5000	-0.1458
42	4.5000	-2.1579	0.6250	-0.2500	0.3404
41	-1.273	-1.650	-2.000	-0.625	-1.447

TABLE 4.

STANDARD DEVIATIONS FOR FEMALES (REDUCED SET)

Tooth	Treatment Group				
	1	2	3	4	ALL
17	--	4.726	4.163	8.485	5.751
16	15.460	15.080	6.551	10.408	15.114
15	18.633	6.701	3.493	5.805	9.449
14	9.0079	7.7217	2.9921	2.0412	7.0774
13	11.218	14.206	9.011	7.115	12.059
12	7.7004	8.6622	12.2746	12.0706	9.3692
11	8.1854	8.6821	1.8619	4.1952	7.5195
27	--	4.9917	6.5511	7.5056	7.7636
26	25.139	22.991	14.782	2.828	22.319
25	9.7297	8.5363	6.2290	6.3246	8.3743
24	3.5989	9.8405	3.6332	4.6853	7.0935
23	6.7435	9.3079	6.9282	6.1779	8.1116
22	6.2444	7.8406	5.6833	2.7142	6.8876
21	4.9749	8.3661	13.6931	6.2290	8.0746
37	--	2.887	7.778	25.456	15.159
36	14.450	9.985	8.617	5.859	13.731
35	8.035	20.668	4.541	7.734	13.363
34	10.378	7.918	3.240	13.297	9.315
33	9.867	11.952	4.432	6.756	9.707
32	6.943	12.990	7.338	4.751	9.631
31	9.3167	9.3732	7.7263	2.2887	8.5359
47	--	3.4641	2.3094	7.0711	4.0510
46	12.392	13.659	5.292	--	14.149
45	8.849	5.992	6.113	9.482	7.697
44	14.379	12.265	11.307	5.090	11.811
43	6.9473	9.6588	9.3034	5.5549	8.3028
42	8.7438	9.2332	8.0877	7.3241	8.7707
41	8.742	12.071	7.764	3.159	9.364

TABLE 5.

MEANS FOR COMBINED MALES & FEMALES (REDUCED SET)

Tooth	Treatment Groups				
	1	2	3	4	ALL
17	-18.000	-7.200	0.750	-2.333	-4.462
16	11.000	-2.773	6.750	-4.000	1.732
15	3.667	6.231	0.857	-1.143	3.939
14	1.6667	1.4000	-2.8750	-0.8750	0.4000
13	-0.571	1.556	-5.625	1.900	0.240
12	2.8000	-1.5588	3.6250	3.2500	0.8286
11	3.7500	0.1515	-0.5714	1.1111	1.3836
27	6.0000	-7.1667	-0.6667	3.5000	-1.5882
26	18.769	-3.682	0.333	-1.500	3.533
25	4.8889	-1.8333	2.3333	3.0000	0.6591
24	0.3750	0.7727	-6.3333	-0.1111	-0.4222
23	1.5217	-0.6571	-2.6250	0.6667	-0.0400
22	2.0909	-1.9189	-0.2500	2.2222	-0.0921
21	0.9565	0.4412	2.7143	2.5000	1.0556
37	8.000	-2.333	2.667	15.667	3.769
36	15.429	-5.529	6.250	4.500	1.781
35	0.000	-1.316	1.375	3.000	0.222
34	-0.769	1.000	0.100	4.444	0.982
33	2.000	-0.970	0.900	6.000	1.068
32	3.545	0.727	1.300	-1.000	1.368
31	2.1000	-1.8437	-1.5000	-0.8000	-0.5556
47	--	-2.7500	0.7500	3.3333	0.1818
46	9.200	-7.154	-1.500	-0.333	-0.267
45	-0.900	-2.550	2.500	-2.500	-1.304
44	-2.714	-1.000	5.111	-3.300	-0.846
43	0.0000	-0.8750	-1.1000	-1.3636	-0.7500
42	2.8636	-1.7097	1.8000	2.0000	0.6757
41	2.571	0.219	-1.700	-0.273	0.554

TABLE 6.

STANDARD DEVIATIONS FOR COMBINED MALES & FEMALES (REDUCED SET)

Tooth	Treatment Group 1	2	3	4	ALL
17	--	8.556	3.403	10.066	8.530
16	24.682	11.860	6.551	8.907	16.610
15	15.945	7.485	4.525	8.821	9.584
14	8.0000	9.1969	4.6117	7.5676	8.1190
13	14.119	12.447	8.245	6.790	12.013
12	6.7481	8.1210	10.5416	11.5604	8.6275
11	7.3499	7.4377	3.6904	5.0360	6.9814
27	--	6.8823	5.2409	7.5056	7.4838
26	18.740	18.030	14.404	2.380	19.319
25	9.2120	8.0847	5.6095	5.9161	8.1181
24	3.3354	8.4961	12.9872	5.0854	8.1058
23	6.8415	10.2611	6.8439	5.5000	8.4684
22	10.7964	6.9297	6.0886	4.5491	8.0650
21	5.3298	7.6443	11.4268	5.3452	7.1066
37	--	3.141	8.622	20.232	11.854
36	16.712	8.776	8.617	4.796	13.346
35	12.508	18.049	4.241	7.171	13.379
34	14.054	6.833	2.961	11.599	9.298
33	8.781	12.668	4.149	7.149	10.264
32	10.262	11.812	6.634	4.879	9.993
31	9.1243	9.3329	7.2150	3.1552	8.4285
47	--	3.7749	6.4485	6.4291	5.6182
46	12.300	12.740	4.435	6.351	13.088
45	11.210	9.035	6.164	9.055	9.045
44	11.378	11.907	10.822	5.417	10.724
43	8.3732	9.2762	8.2926	5.5186	8.2816
42	8.4982	8.4703	8.7788	8.8431	8.6470
41	9.765	11.689	6.977	3.771	9.701

TABLE 7.

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (REDUCED SET)

Tooth	Result
17	13 CASES WERE USED H = 0.02 d.f. = 1 p = 0.883 (adj. for ties) N.S.
16	41 CASES WERE USED H = 0.53 d.f. = 1 p = 0.466 (adj. for ties) N.S.
15	49 CASES WERE USED H = 0.44 d.f. = 1 p = 0.508 (adj. for ties) N.S.
14	50 CASES WERE USED H = 1.04 d.f. = 1 p = 0.308 (adj. for ties) N.S.
13	75 CASES WERE USED H = 1.95 d.f. = 1 p = 0.162 (adj. for ties) N.S.
12	70 CASES WERE USED H = 0.27 d.f. = 1 p = 0.605 (adj. for ties) N.S.
11	73 CASES WERE USED H = 0.73 d.f. = 1 p = 0.394 (adj. for ties) N.S.
27	17 CASES WERE USED H = 0.12 d.f. = 1 p = 0.724 (adj. for ties) N.S.
26	45 CASES WERE USED H = 0.93 d.f. = 1 p = 0.336 (adj. for ties) N.S.
25	44 CASES WERE USED H = 0.71 d.f. = 1 p = 0.399 (adj. for ties) N.S.
24	45 CASES WERE USED H = 0.41 d.f. = 1 p = 0.520 (adj. for ties) N.S.
23	75 CASES WERE USED H = 0.00 d.f. = 1 p = 0.947 (adj. for ties) N.S.
22	76 CASES WERE USED H = 3.48 d.f. = 1 p = 0.063 (adj. for ties) N.S.
21	72 CASES WERE USED H = 0.96 d.f. = 1 p = 0.328 (adj. for ties) N.S.

TABLE 7.(Cont.)

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (REDUCED SET)

Tooth Result

37 13 CASES WERE USED
H = 1.01 d.f. = 1 p = 0.316 (adj. for ties) N.S.

36 32 CASES WERE USED
H = 0.10 d.f. = 1 p = 0.751 (adj. for ties) N.S.

35 45 CASES WERE USED
H = 0.32 d.f. = 1 p = 0.572 (adj. for ties) N.S.

34 56 CASES WERE USED
H = 1.47 d.f. = 1 p = 0.225 (adj. for ties) N.S.

33 74 CASES WERE USED
H = 0.76 d.f. = 1 p = 0.385 (adj. for ties) N.S.

32 76 CASES WERE USED
H = 0.94 d.f. = 1 p = 0.333 (adj. for ties) N.S.

31 72 CASES WERE USED
H = 2.54 d.f. = 1 p = 0.111 (adj. for ties) N.S.

47 11 CASES WERE USED
H = 5.09 d.f. = 1 p = 0.024 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK
MALE	3	8.000	9.7
FEMALE	8	-2.500	4.6
OVERALL	11		6.0

46 30 CASES WERE USED
H = 4.28 d.f. = 1 p = 0.039 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK
MALE	11	-4.000	11.1
FEMALE	19	2.000	18.0
OVERALL	30		15.5

45 46 CASES WERE USED
H = 6.07 d.f. = 1 p = 0.014 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK
MALE	15	-7.000	16.5
FEMALE	31	-1.000	26.9
OVERALL	46		23.5

TABLE 7. (Cont.)

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (REDUCED SET)

Tooth	Result
44	52 CASES WERE USED H = 0.05 d.f. = 1 p = 0.827 (adj. for ties) N.S.
43	72 CASES WERE USED H = 0.47 d.f. = 1 p = 0.495 (adj. for ties) N.S
42	74 CASES WERE USED H = 0.10 d.f. = 1 p = 0.753 (adj. for ties) N.S.
41	74 CASES WERE USED H = 6.09 d.f. = 1 p = 0.014 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK
MALE	27	3.000	45.6
FEMALE	47	-1.000	32.8
OVERALL	74		37.5

TABLE 8.

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES (REDUCED SET)

Tooth Result

17 13 CASES WERE USED
H = 4.39 d.f. = 3 p = 0.223 (adj. for ties) N.S.

16 41 CASES WERE USED
H = 6.93 d.f. = 3 p = 0.075 (adj. for ties) N.S.

15 49 CASES WERE USED
H = 4.69 d.f. = 3 p = 0.196 (adj. for ties) N.S.

14 50 CASES WERE USED
H = 3.91 d.f. = 3 p = 0.272 (adj. for ties) N.S.

13 75 CASES WERE USED
H = 3.51 d.f. = 3 p = 0.319 (adj. for ties) N.S.

12 70 CASES WERE USED
H = 3.69 d.f. = 3 p = 0.297 (adj. for ties) N.S.

11 73 CASES WERE USED
H = 3.59 d.f. = 3 p = 0.310 (adj. for ties) N.S.

27 17 CASES WERE USED
H = 5.42 d.f. = 3 p = 0.144 (adj. for ties) N.S.

26 45 CASES WERE USED
H = 13.32 d.f. = 3 p = 0.004 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK	Z
BANDUP	13	18.000	34.1	3.63
DEBAND	22	-3.500	17.7	
CONT.1	6	-5.000	20.5	
CONT.2	4	-1.500	20.1	
OVERALL	45		23.0	

25 44 CASES WERE USED
H = 5.16 d.f. = 3 p = 0.161 (adj. for ties) N.S.

24 45 CASES WERE USED
H = 1.82 d.f. = 3 p = 0.611 (adj. for ties) N.S.

23 75 CASES WERE USED
H = 1.81 d.f. = 3 p = 0.613 (adj. for ties) N.S.

TABLE 8.(Cont.)

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES (REDUCED SET)

Tooth Result

22 76 CASES WERE USED
H = 3.73 d.f. = 3 p = 0.292 (adj. for ties) N.S.

21 72 CASES WERE USED
H = 1.65 d.f. = 3 p = 0.648 (adj. for ties) N.S.

37 13 CASES WERE USED
H = 6.41 d.f. = 3 p = 0.094 (adj. for ties) N.S.

36 32 CASES WERE USED
H = 12.40 d.f. = 3 p = 0.006 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK	Z
BANDUP	7	15.000	24.0	3.06
DEBAND	17	-3.000	11.1	
CONT.1	4	3.000	21.8	
CONT.2	4	3.500	21.1	
OVERALL	32		16.5	

35 45 CASES WERE USED
H = 1.77 d.f. = 3 p = 0.623 (adj. for ties) N.S.

34 56 CASES WERE USED
H = 1.95 d.f. = 3 p = 0.584 (adj. for ties) N.S.

33 74 CASES WERE USED
H = 2.91 d.f. = 3 p = 0.406 (adj. for ties) N.S.

32 76 CASES WERE USED
H = 3.33 d.f. = 3 p = 0.344 (adj. for ties) N.S.

31 72 CASES WERE USED
H = 0.84 d.f. = 3 p = 0.840 (adj. for ties) N.S.

47 11 CASES WERE USED
H = 1.61 d.f. = 2 p = 0.447 (adj. for ties) N.S.

46 30 CASES WERE USED
H = 7.56 d.f. = 3 p = 0.057 (adj. for ties) A.S.

45 46 CASES WERE USED
H = 2.38 d.f. = 3 p = 0.498 (adj. for ties) N.S.

TABLE 8.(Cont.)

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES (REDUCED SET)

Tooth	Result
44	52 CASES WERE USED H = 2.82 d.f. = 3 p = 0.421 (adj. for ties) N.S.
43	72 CASES WERE USED H = 0.51 d.f. = 3 p = 0.916 (adj. for ties) N.S.
42	74 CASES WERE USED H = 2.40 d.f. = 3 p = 0.493 (adj. for ties) N.S.
41	74 CASES WERE USED H = 1.93 d.f. = 3 p = 0.587 (adj. for ties) N.S.

TABLE 9.

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (COMPLETE SET)

Tooth	Result
17	14 CASES WERE USED H = 0.15 d.f. = 1 p = 0.697 (adj. for ties) N.S.
16	46 CASES WERE USED H = 0.32 d.f. = 1 p = 0.572 (adj. for ties) N.S.
15	53 CASES WERE USED H = 0.18 d.f. = 1 p = 0.668 (adj. for ties) N.S.
14	50 CASES WERE USED H = 1.04 d.f. = 1 p = 0.308 (adj. for ties) N.S.
13	76 CASES WERE USED H = 2.61 d.f. = 1 p = 0.106 (adj. for ties) N.S.
12	70 CASES WERE USED H = 0.27 d.f. = 1 p = 0.605 (adj. for ties) N.S.
11	73 CASES WERE USED H = 0.73 d.f. = 1 p = 0.394 (adj. for ties) N.S.
27	17 CASES WERE USED H = 0.12 d.f. = 1 p = 0.724 (adj. for ties) N.S.
26	50 CASES WERE USED H = 0.49 d.f. = 1 p = 0.484 (adj. for ties) N.S.
25	47 CASES WERE USED H = 1.10 d.f. = 1 p = 0.295 (adj. for ties) N.S.
24	46 CASES WERE USED H = 0.60 d.f. = 1 p = 0.437 (adj. for ties) N.S.
23	76 CASES WERE USED H = 0.03 d.f. = 1 p = 0.855 (adj. for ties) N.S.
22	76 CASES WERE USED H = 3.48 d.f. = 1 p = 0.063 (adj. for ties) N.S.
21	72 CASES WERE USED H = 0.96 d.f. = 1 p = 0.328 (adj. for ties) N.S.
37	15 CASES WERE USED H = 2.64 d.f. = 1 p = 0.105 (adj. for ties) N.S.

TABLE 9.(Cont.)

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (COMPLETE SET)

Tooth Result

36 40 CASES WERE USED
H = 0.12 d.f. = 1 p = 0.733 (adj. for ties) N.S.

35 47 CASES WERE USED
H = 0.72 d.f. = 1 p = 0.398 (adj. for ties) N.S.

34 57 CASES WERE USED
H = 2.18 d.f. = 1 p = 0.140 (adj. for ties) N.S.

33 75 CASES WERE USED
H = 1.25 d.f. = 1 p = 0.263 (adj. for ties) N.S.

32 76 CASES WERE USED
H = 0.94 d.f. = 1 p = 0.333 (adj. for ties) N.S.

31 72 CASES WERE USED
H = 2.54 d.f. = 1 p = 0.111 (adj. for ties) N.S.

47 14 CASES WERE USED
H = 0.82 d.f. = 1 p = 0.365 (adj. for ties) N.S.

46 35 CASES WERE USED
H = 0.31 d.f. = 1 p = 0.578 (adj. for ties) N.S.

45 50 CASES WERE USED
H = 3.72 d.f. = 1 p = 0.054 (adj. for ties) A.S.

	NOBS	MEDIAN	AVE. RANK
MALE	15	-7.000	19.4
FEMALE	35	-1.000	28.1
OVERALL	50		25.5

44 56 CASES WERE USED
H = 0.02 d.f. = 1 p = 0.902 (adj. for ties) N.S.

43 73 CASES WERE USED
H = 0.15 d.f. = 1 p = 0.701 (adj. for ties) N.S.

42 75 CASES WERE USED
H = 0.21 d.f. = 1 p = 0.647 (adj. for ties) N.S.

TABLE 9.(Cont.)

KRUSKAL-WALLIS TESTS FOR SEX DIFFERENCES (COMPLETE SET)

Tooth Result

41 74 CASES WERE USED
H = 6.09 d.f. = 1 p = 0.014 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK
MALE	27	3.000	45.6
FEMALE	47	-1.000	32.8
OVERALL	74		37.5

TABLE 10.

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES
(COMPLETE SET)

Tooth Result

17 14 CASES WERE USED
H = 4.86 d.f. = 3 p = 0.183 (adj. for ties) N.S.

16 46 CASES WERE USED
H = 10.41 d.f. = 3 p = 0.016 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK	Z
BANDUP	15	25.000	31.8	2.98
DEBAND	23	-1.000	18.5	
CONT.1	4	6.500	28.1	
CONT.2	4	-1.000	16.7	
OVERALL	46		23.5	

15 53 CASES WERE USED
H = 4.05 d.f. = 3 p = 0.257 (adj. for ties) N.S.

14 50 CASES WERE USED
H = 3.91 d.f. = 3 p = 0.272 (adj. for ties) N.S.

13 76 CASES WERE USED
H = 3.87 d.f. = 3 p = 0.277 (adj. for ties) N.S.

12 70 CASES WERE USED
H = 3.69 d.f. = 3 p = 0.297 (adj. for ties) N.S.

11 73 CASES WERE USED
H = 3.59 d.f. = 3 p = 0.310 (adj. for ties) N.S.

27 17 CASES WERE USED
H = 5.42 d.f. = 3 p = 0.144 (adj. for ties) N.S.

26 50 CASES WERE USED
H = 20.08 d.f. = 3 p = 0.000 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK	Z
BANDUP	17	21.000	38.2	4.35
DEBAND	23	-4.000	17.9	
CONT.1	6	-5.000	21.5	
CONT.2	4	-1.500	21.1	
OVERALL	50		25.5	

25 47 CASES WERE USED
H = 5.25 d.f. = 3 p = 0.155 (adj. for ties) N.S.

TABLE 10. (Cont.)

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES
(COMPLETE SET)

Tooth Result

24 46 CASES WERE USED
H = 2.11 d.f. = 3 p = 0.551 (adj. for ties) N.S.

23 76 CASES WERE USED
H = 1.57 d.f. = 3 p = 0.667 (adj. for ties) N.S.

22 76 CASES WERE USED
H = 3.73 d.f. = 3 p = 0.292 (adj. for ties) N.S.

21 72 CASES WERE USED
H = 1.65 d.f. = 3 p = 0.648 (adj. for ties) N.S.

37 15 CASES WERE USED
H = 3.80 d.f. = 3 p = 0.284 (adj. for ties) N.S.

36 40 CASES WERE USED
H = 21.22 d.f. = 3 p = 0.000 (adj. for ties) S.

	NOBS	MEDIAN	RANK	Z
BANDUP	12	36.0	30.9	4.40
DEBAND	19	-4.0	11.9	
CONT.1	4	3.0	23.8	
CONT.2	5	5.0	25.7	
OVERALL	40		20.5	

35 47 CASES WERE USED
H = 2.35 d.f. = 3 p = 0.503 (adj. for ties) N.S.

34 57 CASES WERE USED
H = 2.88 d.f. = 3 p = 0.411 (adj. for ties) N.S.

33 75 CASES WERE USED
H = 4.13 d.f. = 3 p = 0.248 (adj. for ties) N.S.

32 76 CASES WERE USED
H = 3.33 d.f. = 3 p = 0.344 (adj. for ties) N.S.

31 72 CASES WERE USED
H = 0.84 d.f. = 3 p = 0.840 (adj. for ties) N.S.

47 14 CASES WERE USED
H = 3.59 d.f. = 3 p = 0.310 (adj. for ties) N.S.

TABLE 10. (Cont.)

KRUSKAL-WALLIS TESTS FOR TREATMENT DIFFERENCES
(COMPLETE SET)

Tooth Result

46 35 CASES WERE USED
H = 13.71 d.f. = 3 p = 0.003 (adj. for ties) S.

	NOBS	MEDIAN	AVE. RANK	Z
BANDUP	14	16.000	25.7	3.58
DEBAND	14	-4.500	11.8	
CONT.1	4	-1.000	14.9	
CONT.2	3	-4.000	15.2	
OVERALL	35		18.0	

45 50 CASES WERE USED
H = 2.35 d.f. = 3 p = 0.504 (adj. for ties) N.S.

44 56 CASES WERE USED
H = 2.96 d.f. = 3 p = 0.399 (adj. for ties) N.S.

43 73 CASES WERE USED
H = 1.02 d.f. = 3 p = 0.796 (adj. for ties) N.S.

42 75 CASES WERE USED
H = 2.99 d.f. = 3 p = 0.393 (adj. for ties) N.S.

41 74 CASES WERE USED
H = 1.93 d.f. = 3 p = 0.587 (adj. for ties) N.S.

TABLE 11.

MEANS FOR MALES (Complete Set)

Tooth	Treatment Groups 1	2	3	4	ALL
17	-18.000	-11.000	1.000	24.500	1.667
16	14.200	2.600	--	0.000	6.062
15	7.000	3.000	0.000	-8.500	2.000
14	0.0000	-4.1250	-12.0000	5.0000	-2.6923
13	0.300	6.143	-1.500	5.333	3.517
12	2.1250	-1.3846	1.5000	10.0000	1.2308
11	4.0000	0.3636	-8.0000	5.3333	2.2222
27	6.0000	-7.0000	0.5000	-3.0000	-1.6667
26	26.429	-4.222	12.000	-1.000	8.263
25	9.3333	0.7500	1.0000	-1.0000	2.3571
24	0.000	-1.875	-32.000	5.000	-3.083
23	-0.6000	1.3846	-1.5000	0.3333	0.3571
22	4.7000	1.0714	-5.5000	7.0000	2.4828
21	-0.4545	3.2500	-0.5000	4.3333	1.6429
37	8.000	-4.750	-5.000	-6.000	-3.500
36	35.250	-8.444	--	5.000	5.000
35	-1.250	-4.571	0.000	2.000	-2.692
34	6.600	0.222	1.500	10.000	3.579
33	4.111	-1.667	-0.500	26.667	3.692
32	6.900	1.417	2.000	-2.333	3.074
31	3.4444	3.0909	3.0000	-4.3333	2.3200
47	52.000	-5.500	10.000	-10.500	5.000
46	28.600	-7.667	-3.000	-4.000	6.143
45	-9.000	-7.125	-2.500	3.000	-6.333
44	-1.600	0.556	-1.000	-1.000	-0.389
43	0.1250	-2.5833	1.0000	1.6667	-0.9200
42	0.900	-1.000	6.500	8.000	1.259
41	6.800	3.333	-0.500	0.667	4.037

TABLE 12.

MEANS FOR FEMALES (Complete Set)

Tooth	Treatment Groups 1	2	3	4	ALL
17	--	-4.667	0.667	-7.000	-3.250
16	24.800	-3.077	6.750	-5.333	7.300
15	-3.286	5.833	-3.000	1.800	2.028
14	2.1429	4.0000	-1.5714	-2.8333	1.4865
13	-1.364	0.478	-7.000	0.429	-0.915
12	3.2500	-1.6667	4.3333	-0.8000	0.5909
11	3.5000	0.0455	0.6667	-1.0000	0.8913
27	--	-7.2500	-1.2500	5.6667	-1.5455
26	29.100	-6.786	-2.000	-2.000	5.871
25	2.6667	-4.6111	2.6000	4.0000	-1.1515
24	0.429	4.667	-1.200	-1.571	1.647
23	3.1538	-1.0000	-3.0000	0.8333	0.1042
22	-0.0833	-3.7391	1.5000	-0.1667	-1.6809
21	2.2500	-1.0909	4.0000	1.4000	0.6818
37	--	-2.333	6.500	21.000	6.857
36	27.500	-10.300	6.250	15.250	7.808
35	9.250	0.583	1.571	3.143	3.353
34	-5.375	1.467	-0.250	5.143	0.342
33	0.417	-0.571	1.250	4.250	0.755
32	0.750	0.333	1.125	-0.500	0.429
31	1.0000	-4.4286	-2.6250	0.7143	-2.0851
47	--	-4.000	-2.333	1.000	-2.125
46	14.667	-10.875	-1.000	7.000	2.333
45	-2.286	-1.067	4.167	-3.286	-0.857
44	-3.333	5.214	5.875	-4.286	1.579
43	1.9167	0.1500	-1.6250	-2.5000	-0.1458
42	4.500	-4.700	0.625	-0.250	-0.771
41	-1.273	-1.650	-2.000	-0.625	-1.447

TABLE 13.

MEANS FOR COMBINED MALE & FEMALE (Complete Set)

Tooth	Treatment Groups 1	2	3	4	ALL
17	-18.000	-7.200	0.750	8.750	-1.143
16	21.267	-0.609	6.750	-4.000	6.870
15	-0.200	4.821	-2.250	-1.143	2.019
14	1.6667	1.4000	-2.8750	-0.8750	0.4000
13	-0.571	2.622	-5.625	1.900	0.776
12	2.8000	-1.5588	3.6250	3.2500	0.8286
11	3.7500	0.1515	-0.5714	1.1111	1.3836
27	6.0000	-7.1667	-0.6667	3.5000	-1.5882
26	28.000	-5.783	0.333	-1.500	6.780
25	4.8889	-2.9615	2.3333	2.3333	-0.1064
24	0.375	2.391	-6.333	-0.111	0.413
23	1.5217	-0.1389	-2.6250	0.6667	0.1974
22	2.0909	-1.9189	-0.2500	2.2222	-0.0921
21	0.9565	0.4412	2.7143	2.5000	1.0556
37	8.000	-3.714	2.667	7.500	1.333
36	30.083	-9.421	6.250	13.200	6.825
35	5.750	-1.316	1.375	3.000	1.681
34	-0.769	1.000	0.100	6.600	1.421
33	2.000	-0.970	0.900	10.364	1.773
32	3.545	0.727	1.300	-1.000	1.368
31	2.1000	-1.8437	-1.5000	-0.8000	-0.5556
47	52.000	-4.600	0.750	-4.750	0.929
46	19.643	-9.500	-1.500	-0.333	3.857
45	-4.727	-3.174	2.500	-2.500	-2.500
44	-2.714	3.391	5.111	-3.300	0.946
43	1.2000	-0.8750	-1.1000	-1.3636	-0.4110
42	2.864	-3.312	1.800	2.000	-0.040
41	2.571	0.219	-1.700	-0.273	0.554

BIBLIOGRAPHY

Abbot P V (1988) "Product review: A Combined vitality scanner/Apex locater." J.Endodontics. Vol.14, No.11: pp 8-9.

Analytic Technology Corporation. "Vitality Scanner Model 2007 Instruction Guidelines." 3301 181st Place N.E., Redmond, W.A.98052.

Anderson R W & Pantera E A (1988) "Influence of a Barrier Technique on electric pulp testing." J.Endodontics. Vol.14, No.4: pp 179-180.

Andreasen J O (1970) "Etiology and pathogenesis of traumatic dental injuries. A clinical study of 1,298 cases." Scand.J.Dent.Res. Vol.78: pp 329-342.

Andreasen J O, Paulsen H U, Yu Z, et al. (1990a) "A Long-term study of 370 Autotransplanted Premolars. Part 1 Surgical procedures and standardized techniques for monitoring healing." Europ.J.Orthod. Vol.12, No.1: pp 3-13.

Idem. (1990b) "A Long-term study of 370 Auto transplanted Premolars. Part 2 Tooth survival and pulp healing subsequent to transplantation." Europ.J.Orthod. Vol.12, No.1: pp 14-25.

Anstendig H S & Kronman J H (1972) "A Histologic Study of pulpal reaction to Orthodontic Tooth Movement in Dogs." Angle Orthod. Vol.42, No.1: pp 50-55.

Austin L T & Waggener D T (1941) "Vitality tests with particular reference to the use of ice." J.Amer.Den.Ass. Vol.28: pp 1044-1049.

Avery J K, Cox C F, Corpron R E & Trefz B R (1974) "Distribution of Adrenergic and Cholinergic Nerve Endings in Mouse Molars using Cytochemical analysis."

I.A.D.R. Abst. No.79: pp 74.

Banes J D & Hammond H L (1978) "Surface temperatures of vital and nonvital teeth in humans." J.Endodontics Vol.4, No.4: pp 106-109.

Barber F E, Lees S & Lobene R R (1969) "Ultrasonic pulse-echo measurements in teeth." Arch.Oral Biol. Vol.14: pp 745-760.

Barker B C W & E H Ehrmann (1969) "Human pulp reactions to a glucocorticosteroid antibiotic compound." Aust.Dent.J. Vol.14, Apr: pp 104-119.

Barkin P R (1973) "Time as a factor predicting the vitality of traumatized teeth." J.Dent.Child. May-June: pp 188-192.

Baume L J (1966) "Clinical and pathological aspects of current endodontic therapy." Int.Dent.J. Vol.16: pp 30-34.

Bawden J W (1985) "Reaction Paper" J.Den.Res. Vol.64 (Spec.Iss.): pp 628-630.

Bender I B & Seltzer S (1972) "The effect of periodontal disease on the pulp." Oral Surg. Vol.33, No.3: pp 458-474.

Bender I B (1986) "Pain Conference Summary." J.Endodontics Vol.12, No.10: pp 509-517.

Bender I B, Landau M A, Fonsecca S & Towbridge H O (1989) "The optimum placement-site of the electrode in electric pulp testing on the 12 anterior teeth." J.Amer. Dent. Ass. Vol.118: pp 305-310.

Bergenholtz G, Mjor I A, Cotton W R, Hanks C T, Kim S, Torneck C D & Towbridge H O (1985) "Consensus Report." J.Dent.Res. Vol.64 (Spec.Iss.): pp 631-633.

Bernice S (1964) "Differences in nervous distribution between erupted and

non-erupted human teeth."
J.Dent.Res. Vol.43, No.3:
pp 406-411.

Bernick S & Levy B M (1968) "Studies on the biology of the periodontium of marmosets: IV. Innervation of the periodontal ligament."
J.Dent.Res. Vol.47: pp 1158-1165.

Bernick S (1967a) "Age changes in the blood supply to human teeth."
J.Dent.Res. Vol.46, No.1:
pp 544-550.

Bernick S (1967b) "Effect of aging on the nerve supply to human teeth."
J.Dent.Res. Vol.46, No.4:
pp 694-699.

Bernick S (1969) "Differences in nerve distribution between erupted and non-erupted human teeth."
J.Dent.Res. Vol.43, No.3:
pp 406-411.

Bernick S & Nedelman C (1975) "The effect of aging on the human pulp."
J.Endodontics Vol.1, No.2:
pp 88-94.

Blackwood H J (1957) "Metaplasia or repair of the dental pulp as a response to injury."
B.J.O. Vol.102, No.8: pp 87-92.

Blair A E (1965) "The efficacy of placebo on pain perception threshold."
J.Oral Surg.Oral Path. Vol.20, No.3: pp 384-391.

Bhasker S N & Rappaport H M (1973) "Dental vitality tests and pulp status."
J.Amer.Dent.Ass. Vol.86:
pp 409-411.

Bolden T E, Lemeh D H, Seibert W & Stewart E B (1975) "Effect of prolonged use of analgesics on pulpal response: A preliminary investigation."
J.Den.Res. Vol.54 (Spec.Iss.):
pp B198-B200.

Brown W E (1967) "The management of injuries to young teeth."
Aust.Dent.J. April: pp 99-104.

Brunner M & Johnsen D (1982) "Quantitative assessment of intrapulpal axon response to orthodontic movement." Am.J.Orthod. Vol.82, No.3: pp 244-250.

Burnside R R, Sorenson F M & Buck D L (1974) "Electric vitality testing in orthodontic patients." Angle Ortho. Vol.44, No.3: pp 213-217.

Burrill D Y (1949) "Fusion frequency as an indication of pulp condition." J.Den.Res. Vol.28: pp 652.

Burrill D Y (1962) "Pulp testing by determination of fusion frequency." J.Dent.Res. Vol.41, No.2: pp 437-445.

Cailleteau J G & Ludington J R (1989) "Using the Electric pulp tester with gloves: A simplified approach." J.Endodontics Vol.15, No.2: pp 80-81.

Castelli W C (1962) "The vascular structure of dental pulps in cats." J.Dent.Res. Vol.41: pp 213.

Chambers I G (1982) "The role and methods of pulp testing in oral diagnosis: a review." Int.Endo.J. Vol.15: pp 1-5.

Chatrian G E, Canfield R C, Lettich E & Black R G (1974) "Cerebral responses to electrical stimulation of tooth pulp in man." J.Dent.Res. Vol.53, No.5: pp 1299.

Chien S (1985) "Haemodynamics of the dental pulp." J.Dent.Res. Vol.64 (Spec.Iss) pp 602-606.

Chilton N W & Fertig J W (1972) "Pulpal responses of bilateral intact teeth." Oral Surg. Vol.33, No.5: pp 797-800.

Civjan S, Barone J J & Vaccaro G J (1973) "Electric Pulp Vitality Testers." J.Dent.Res. Jan-Feb: pp 120-126.

Clarke A M, Moon P C, Sanders E C & Serene T P (1972) "Danger to patients." J.Amer.Dent.Ass. Vol.85: pp 232.

Cooke G H (1982) "Reversible pulpitis with etiology of bruxism." J.Endodontics Vol.8, No.10: pp 280-281.

Cooley R L & Barkmeier W W (1977) "An alternative to electric pulp testing." Quint.Int. Report 1573, Dec: pp 23- 25.

Cooley R L & Robinson S F (1980) "Variables associated with electric pulp testing." Oral.Surg. Vol.50, No.1: pp 66-73.

Cooley R I, Stilley J & Lubow R M (1984) "Evaluation of a digital pulp tester." Oral.Surg. Vol.58, No.4: pp 437-442.

Cooper M B, Landay M A & Seltzer S (1972) "The effects of excessive occlusal force on the pulp. Number two: Heavier and long term forces." J.Perio. Vol.42, No.6: pp 353-359.

Crandell C E & Hill R P (1966) "Thermography in dentistry: A pilot study." Oral Surg., Oral Med. & Oral Path. Vol.21, No.3: pp 316-320.

Dachi S F, Haley J V & Sanders J E (1967) "Standardization of a test for dental sensitivity to cold." Oral.Surg., Oral Med., Oral Path. Vol.24, No.5: pp 687-692.

Dale J G (1982) "Trauma: Its influence on Orthodontic Treatment Planning." Symposium on Dentofacial Trauma. Dent.Clin.North.Amer. Vol.26, No.3, July: pp 565-590.

Degering C I (1962) "Physiological evaluation of dental pulp testing methods." J.Dent.Res. Vol.41: pp 695-700.

Delivanis H P & Sauer G J H (1982) "Incidence of canal calcification in the orthodontic patient." Am.J.Orthod. Vol.82, No.1: pp 58-61.

Dreven L J, Reader A, Beck F M, Meyers W J & Weaver J (1987) "An evaluation of an Electric Pulp Tester as a measure of Analgesia in Human Vital Teeth." J.Endodontics. Vol.13, No.5: pp 233-238.

Dummer P M H, Hicks R & Huws D (1980) "Clinical signs and symptoms in pulp disease." Int.Endo.J. Vol.13: pp 27-35.

Dummer P M H & Tanner M (1986) "The response of caries-free, unfilled teeth to electrical excitation: a comparison of two new pulp testers." Int.Endodontic J. Vol. 19: pp 172-177.

Ehrmann E H (1973) "Electric Pulp testers." Letter to the Ed. J.Amer.Dent.Ass. Vol.87: pp 786-787.

Ehrmann E H (1977) "Pulp testers and pulp testing with particular reference to the use of dry ice." Aust.Dent.J. Vol.22, No.4: pp 272-279.

El Deeb M E, El Deeb M E, Bevis R R & Gomez-Marin O (1989) "Canines erupted through grafted alveolar cleft defects in patients with alveolar clefts: A pulp testing evaluation study." Cleft Pal. J. Vol.26; No.2: pp 100-104.

England M C, Pellis E G & Michanowicz A E (1974) "Histopathologic study of the effect of pulpal disease upon nerve fibres of the human dental pulp." Oral Surg. Vol.38, No.5: pp 783-790.

Farrell T H (1958) "A preliminary investigation into electrical properties of human teeth." Brit.Dent.J. Vol.104, No.10: pp 353-359.

Friend L A & Glenwright H D (1969) "An experimental investigation into the localization of pain from the dental pulp." Oral Surg., Oral Med., Oral Path. Vol.25, No.5: pp 765-774.

Fulling H J & Andreasen J O (1976a) "Influence of maturation status and tooth type of permanent teeth

upon electronic and thermal pulp testing."
Scand.J.Dent.Res. Vol.84:
pp 286-290.

Fulling H J & Andreasen J O (1976b) "Influence of splints and temporary crowns upon electric and thermal pulp-testing procedures."
Scand.J.Dent.Res. Vol. 84:
pp 291-296.

Fuss Z, Trowbridge H, Bender I B, Rickoff B & Sorin S (1986) "Assessment of reliability of electrical and thermal pulp testing agents."
J.Endodontics Vol.12, No.7:
pp 301-305.

Garfunkel A, Sela A & Ulmansky M (1973) "Dental pulp pathosis: Clinicopathological correlations based on 109 cases."
Oral Surg. Vol.35, No.1:
pp 110-117

Gargiulo A V (1984) "Diagnostic Procedures" Endodontic-Periodontic Interrelationships.
Dent. Clin.North Amer.
Vol.28, No.4: pp 779-781.

Greenwood F, Horiuchi H & Matthews B (1972) "Electrophysiological evidence on the types of nerve fibres excited by electrical stimulation of teeth with a pulp tester."
Arch.Oral.Biol. Vol.17:
pp 701-709.

Greenwood F (1973) "An electrophysiological study of the central connections of primary afferent nerve fibres from the dental pulp in the cat."
Arch.Oral Biol. Vol.18:
pp 771-785.

Grossman L I, Oliet S & del Rio C E (1988) "Endodontic Practice."
11th Ed. Pub. Lea & Febiger,
Philadelphia.

Guthrie T J, McDonald R E & Mitchell D F (1965) "Dental Pulp Hemogram."
J.Dent.Res. Vol.44, No.4:
pp 678-682.

Hannam A G (1968) "The conduction velocity of nerve impulses from dental mechanoreceptors in the dog."
Arch.Oral Biol. Vol.13:

pp 1377-1383.

Hannam A G, Siu W & Tom J (1974) "A comparison of monopolar and bipolar pulp-testing." J.Canad.Den.Ass. Vol.40: pp 124-128.

Hare G C (1962) "The Management of Traumatic Injuries to Children's Teeth." J.Canad.Dent.Ass. Vol.28, No.2: pp 114-121.

Hare G C (1969) "Diagnostic value of vitality tests." J.Cana.Dent.Ass. Vol.35: pp 495.

Harris W E (1973) "Endodontic pain referred across the midline: a report of case." J.Amer.Dent.Ass. Vol.87: pp 1240-1243.

Harris W E (1982) "Electric pulp testing as an aid in endodontic diagnosis." J Endodontics Vol.8, No.4: pp 171-174.

Hemphill B F (1959) "Preservation of pulp vitality." M.D.S. Thesis University of Sydney, Sydney, Australia.

Herbert W E (1953) "Calcification of pulp following trauma." Brit.Dent.J. March: pp 127-169.

Heyeraas K J (1985) "Pulpal, Microvascular, and Tissue Pressure." J.Dent.Res. Vol.64 (Spec.Iss.): pp 585-589.

Horiuchi H & Matthews B (1973) "In-vitro observations on fluid flow through human dentine caused by pain-producing stimuli. Arc.Oral Biol. Vol.18: pp 275-294.

Howell R M, Duell R C & Mullaney T P (1970) "The determination of pulp vitality by thermographic means using Cholesteric liquid crystals: A preliminary study." J.Oral Surg. Vol.29, No.5: pp 763-768.

Ingle J I & Beveridge E E (1976) "Endodontics." 2nd Ed. Pub. Lea & Febiger.

Jacobsen I & Kerekes K (1977) "Long-term prognosis of traumatized permanent anterior

teeth showing calcifying processes in the pulp cavity."
Scand.J.Dent.Res. Vol.85: pp 588-598.

Jacobsen I & Zachrisson B U (1975) "Repair characteristics of root fractures in permanent anterior teeth."
Scand.J.Dent.Res. Vol.83: pp 355-364.

Jacobson J J (1984) "Probe placement during electric pulp-testing procedures."
Oral Surg. Vol.58: pp 242-247.

Johnsen D & Karlsson U (1974) "Development of innervation patterns in cat primary and permanent incisors."
I.A.D.R. Abst. No.80, pp 74.

Johnsen D & Johns S (1978) "Quantitation of nerve fibres in the primary and permanent canine and incisor teeth in man."
Arch.Oral Biol. Vol.23: pp 825-829.

Johnsen D C, Harshbarger J & Nash D A (1979) "Vitalometer testing of primary and permanent canine teeth."
Ped.Dent. Vol.1, No.1: pp 27-30.

Johnsen D C (1985) "Innervation of teeth: Qualitative, Quantitative, and Developmental Assessment."
Session 3: Innervation of the Dentin, Predentin, and Pulp - H.O.Towbridge Chairman.
J.Dent.Res.(Spec.Iss.) Vol.64: pp 555-563.

Johnson J V & Hinds E C (1969) "Evaluation of teeth vitality after subapical osteotomy."
J.Oral Surg. Vol.27: pp 256-257.

Jones E H (1967) "Battery powered vitality testers."
Aust.Dent.J., April: pp 147-151.

Kaletsky T & Furedi A (1935) "Reliability of various types of pulp testers as a diagnostic aid."
J.Amer.Dent.Ass. Vol.22: pp 1559-1574.

Kaletsky T (1936) "Further studies on the reliability of various types of electric pulp testers."

Dental Cosmso. Vol.78: pp 732-742.

Kaletsky T (1937) "An additional report on further studies in electric pulp testing." New York J.Dent. Vol.7, No.3: pp 81-84.

Kaletsky T (1938) "The pulp tester in the conservative management of oral surgical lesions." J.Amer.Dent.Ass. Vol.25: pp 700-706.

Karlsson U, Johnsen D & Herman A M (1974) "Early degenerative nerve alterations in feline resorbing deciduous incisors as observed by electron microscopy." J.Dent.Res. Vol.53, No.6: pp 1429-1431.

Khayat B G, Byers M R, Taylor P E, Mecifi K & Kimberly C L (1988) "Response of nerve fibres to pulpal inflammation and periapical lesions in rat molars demonstrated by calcitonin gene-related peptide immunocytochemistry." J.Endodontics Vol.14, No.12: pp 577-587

Kim S (1985) "Regulation of pulpal blood flow." J.Dent.Res. Vol.64 (Spec.Iss.): pp 590-596.

Kim S & Dorscher-Kim J (1989) "Hemodynamic regulation of the dental pulp in a low compliance environment." J.of Endo. Vol.15, No.9: pp 404-408.

King D R & King A C (1977) "Use of the Vitalometer with gloves." J.Can.Dent. Ass., No.4: pp 182-186.

Kitamura T, Takahasi T & Horiuchi H (1983) "Electrical characteristics and clinical application of a new automatic pulp tester." Quintessence International, Report 2167, January: pp 45-53.

Kleier D J, Sexton J R & Averbach R E (1982) "Electronic and Clinical Comparison of Pulp Testers." J.Dent.Res.Vol.61, No.12: pp 1413-1415.

Klein H (1978) "Pulp response to an electric pulp stimulator in the developing

permanent anterior dentition."
J.Dent.Child. Vol.45: pp 199-200.

Lado E A, Richmond A F & Marks R G (1988) "Reliability and validity of a digital pulp tester as a test standard for measuring sensory perception."
J.Endodontics Vol.14, No.7: pp 352-356.

Landay M, Nasimov H & Seltzer S (1970) "The effects of excessive occlusal force on the pulp."
J.Perio. Vol.41: pp 3-11.

Lees S & Barber F E (1969) "Looking into teeth with ultrasound."
Abstract by J A Salzmann.
Am.J.Ortho. Vol.55, No.5: pp 527.

Lilja E, Bjornestedt T & Lindskog S (1983) "Cellular enzyme activity associated with tissue degradation following orthodontic tooth movement in man."
Scand.J.Dent.Res. Vol.91: pp 381-390.

Linden R W A & Millar B J (1988) "The response characteristics of mechanoreceptors related to their position in the cat canine periodontal ligament."
Arch.Oral Biol. Vol.33, No.1: pp 51-56.

Lundy T, Stanley H R & (1969) "Correlation of pulpal histopathology and clinical symptoms in human teeth subjected to experimental irritation."
Oral Surg. Vol.27, No.2: pp 187-201.

McDanial K F, Rowe N H & Charbeneau G T (1973) "Tissue response to an electric pulp tester."
J.Pros.Dent. Vol.29, No.1: pp 84-87.

McGrath P A, Gracely R H, Dudner R & et al (1983) "Non-pain and pain sensations evoked by tooth pulp stimulation."
Pain Vol.15, pp 377-388.

Machida K & Omori K (1979) "Recovery of nerve action potentials and vascularization after inferior alveolar nerve sectioning."
Bull.Tokyo Dent.Coll. Vol.20, No.3: pp 101-114.

Marshall F J (1979) "Planning endodontic treatment." Dent. Clin. Nth. Am. Vol.23: pp 495-518.

Manly R S & Ruffle R H (1966) "Influence of microampere current flow on tooth enamel porosity." Arc.Oral Biol. Vol.11: pp 931-936.

Matthews B, Horiuchi H & threshold Greenwood F (1974) "The effects of stimulus polarity and electrode area on the to monopolar stimulation of teeth in human subjects, with some preliminary observations on the use of a bipolar pulp tester." Arch.Oral Biol. Vol.19: pp 35-42.

Matthews B & Seale B N (1974) "Some observations on pulp testers." Brit.Dent.J. Vol.137: pp 307-312.

Matthews B, Seale B N, Adams D & Linden R. (1974) "Thresholds of vital and non-vital teeth to stimulation with electric pulp testers." Brit.Dent.J. Vol.137: pp 352-355.

Matthews J L, Dorman H L & Bishop J G (1959) "Fine structures of the dental pulp." Oral Surg. Vol.38, No.5: pp 940-946.

Mendoza M M, Reader A, Meyers W J & Foreman D W (1987) "An ultrastructural investigation of the human apical pulp in irreversible pulpitis. 1.Nerves." J.Endodontics Vol.13, No.6: pp 267-276.

Michaelson R E, Seldberg B H & Guttuso J (1975) "An in vivo evaluation of interface media used with the electric pulp tester." J.Amer.Dent.Ass. Vol.91: pp 118-121.

Millard H D (1973) "Electric pulp testers." J.Amer.Dent.Ass. Vol.86: pp 872-873.

Mjor I A (1985) "Dentin-predentin complex and its permeability: Pathology and treatment overview." J.Dent.Res. Vol.64 (Spec.Iss.): pp 621-627.

Moss-Salentijn L & Hendricks-Klyvert M "Calcified structures in human dental pulps."

(1988) J.Endodontics Vol.14, No.4: pp 184-189.

Mostafa Y A, Weakes-Dybvig M & Osdoby P (1983) "Orchestration of tooth movement." Am.J.Orthod. Vol.83, No.3: pp 245-250.

Mount G J (1972) "Idiopathic internal resorption: A report of fifteen cases." Oral Surg. Vol.33, No.5: pp 801-809.

Mullancy T P, Howell R M & Petrich J D (1970) "Resistance of nerve fibres to pulpal necrosis." Oral Surg. Vol.30, No.5: pp 690-693.

Mumford J M (1956) "Direct-current electrodes for pulp testing." Dent.Pract. Vol.6, No.7: pp 235-238.

Mumford J M (1959) "Path of direct current in electric pulp testing." Brit.Dent.J. Vol.106: pp 23-26.

Mumford J M (1960) "Reproducibility and discrimination in electric pulp-testing." J.Dent.Res. Vol.39, no.6 art.39: pp 1111-1112.

Mumford J M (1963) "Pain threshold of normal human anterior teeth." Arch.Oral Biol. Vol.8: pp 493-501.

Mumford J M (1964) "Evaluation of guttapercha and ethyl chloride in pulp testing. Brit.Dent.J. Vol.116: pp 339-342.

Mumford J M (1965) "Pain perception threshold and adaptation of normal human teeth." Arch.Oral Biol. Vol.10: pp 1965.

Mumford J M (1967) "Resistivity of human enamel and dentine." Arch.Oral.Biol. Vol.12: pp 925-927.

Mumford J M (1971) "Pain perception threshold of retained deciduous teeth." J.Den.Res. Vol.50, No.2: pp 506.

Mumford J M & "Problems in electric pulp testing

Bjorn H (1962) and dental algesiometry." Int.Dent.J: pp 161-179.

Mumford J M & Newton A V (1969) "Transduction of hydrostatic pressure to electric potential in human dentition." J.Den.Res. Vol.48, No.2: pp 226-229.

Mumford J M & Newton A V (1971) "Convergence in the trigeminal system following stimulation of teeth." Arch.Oral Biol. Vol.16: pp 1089-1097.

Mumford J M & Newton A V (1974) "Trigeminal convergence from human teeth: Influence of contralateral stimulation and stimulus frequency on the pain perception threshold." Arch.Oral Biol. Vol.19: pp 145-149.

Narhi M V O, Virtanen A, Kuhta J & Huopaniemi T (1987) "Electrical stimulation of teeth with a pulp tester in the cat." Scand.J.Den.Res. Vol.87: pp 32-38.

Narhi M V O (1985) "The characteristics of intradental sensory units and their responses to stimulation." J.Dent.Res. Vol.64 (Spec.Iss.) pp 564-571.

Natkin E & Ingle J (1963) "A further report on alveolar osteoporosis and pulpal death associated with compulsive bruxism." Periodontics. Vol.1, No.6: pp 260-263.

Naylor M N (1964) "Studies on sensation to cold stimulation in human teeth." Brit.Dent.J. Dec: pp 482-486.

Neidle E A & Liebman F M (1964) "The effects of vasoactive drugs and nerve stimulation on blood flow in the tooth pulp and allied structures in the cat." J.Dent.Res. Vol.43, No.3: pp 412-422.

Newton A V & Mumford J M (1969) "Current paths through analogues of human teeth." Arc.Oral Biol. Vol.14: pp 699-705.

Newton A V & "Lateral dominance, pain

Mumford J M (1972) perception and pain tolerance." J.Dent.Res. Vol.51, No.4: pp 940-942.

Nordenram A (1970) "Dental sensitivity to electrical excitation threshold values of caries-free nonfilled teeth." Acta Odont.Scand. Vol.28: pp 233-242.

Oehlers F A C (1951) "A case of internal resorption following injury." Brit.Dent.J. Jan: pp 13-16.

Olgart L M (1985) "The role of local factors in dentin and pulp in intradental pain mechanisms." J.Dent.Res. Vol.64 (Spec.Iss.): pp 572-578.

Olgart L M, Edwall B & Gazelius B (1989) "Neurogenic mediators in control of pulpal blood flow." J.of Endo. Vol.15, No.9: pp 409-412.

Olgart L M, Gazelius B & Lindh-Stromberg U (1988) "Laser doppler flowmetry in assessing vitality in luxated permanent teeth." Int.Endo.J. Vol.21: pp 300-306.

Oppenheim A (1942) "Human tissue response to orthodontic intervention of short and long duration." Am.J.Orthod. Vol.28, No.5: pp 263-301.

Pashley D H (1985) "Dentin-Predentin complex and its permeability: Physiologic overview." J.Dent.Res. Vol.64 (Spec.Iss.): pp 613-620.

Peters D D, Mader C L & Donnelly J C (1986) "Evaluation of the effects of carbon dioxide used as a pulpal test 3. In vivo effect on human enamel." J. Endodontics Vol.12, No.1: pp 13-20.

Reader A & Foreman D W (1981) "An ultrastructural quantitative investigation of human intradental innervation." J.Endodontics. Vol.7, No.11: pp 493-499.

Reich J (1952) "A preliminary note on a new

approach to the determination of the vitality of the dental pulp in intact teeth."
Aust.J.Dent. Vol.56, June: pp 148.

Reiss H L & Furedi A (1933) "Significance of the pulp test as revealed in a microscopic study of the pulps of 130 teeth."
Dental Cosmos Vol.75: pp 272-280.

Reynolds R L (1966) "The determination of pulp vitality by means of thermal and electrical stimuli."
Oral Surg.,Oral Med.,Oral Path. Vol.22, No.2: pp 231-240.

Roane J B, Foreman D W, Melfi R C & Marshall F J (1973) "An ultrastructural study of dentinal innervation in the adult human tooth."
Oral Surg. Vol.35, No.1: pp 94-104.

Roberts W E, Goodwin W C & Heiner S R (1981) "Cellular response to Orthodontic Force."
Symposium on Orthodontics. Dent. Clin. North Amer. Vol.25, No.1: pp 3-17.

Rosenthal A B, Taintor J F & Karp G (1984) "Human Pulp's electrical responsiveness in Hodgkin's disease patients who are undergoing Chemotherapy."
J.Endodontics Vol.10, No.1: pp 28-33.

Rowe A H R & Pitt Ford T R (1990) "The assessment of pulpal vitality."
Int. Endo. J. Vol.23: pp 77-83.

Rubach W C & Mitchell D F (1965) "Periodontal disease, age, and pulp status."
Oral Surg.,Oral Med.,Oral Path. Vol.19, No.4: pp 482-492.

Salzberg B M (1985) "New Approaches to the Neurophysiology of the dental pulp."
J.Dent.Res. Vol.64 (Spec.Iss.): pp 597-601.

Sapone J, Simon J H S & Lieber W S (1984) Chapter 20 "Orthodontic-endodontic treatment."
Pathways of the Pulp. 3rd Ed. Edited by S Cohen & R C Burns. Pub. C V Mosby.

Schaffer J (1958) "Pulp testing." New York Dent.J Vol.28, No.2: pp 48-61.

Seeman J (1963) "Evaluation of pulp reaction: A critical review." M.D.S.Thesis, University of Sydney, Sydney, N.S.W., Australia.

Seltzer S (1972) "Classification of pulp pathosis." Oral Surg. Vol.34. No.2: pp 269-287.

Seltzer S (1986) "Pain in Endodontics." J.Endodontics Vol.12, No.10: pp 505-507.

Seltzer S, Bender I B & Ziontz M (1961) "The Dynamics of pulp inflammation: correlations between diagnostic data and actual histologic finding in the pulp." Oral Surg.,Oral Med.,Oral Path. Vol.16, No.7: pp 846-871, 969-977.

Seltzer S & Bender I B (1984) "The dental pulp: biologic considerations in dental procedures." Pub. J B Lippincott.

Shepherd J P (1976) "Effects of lowered temperatures on rat pulp and gingivae." Oral Surg.,Oral Med.,Oral Path. Vol.45, No.3: pp 388-394.

Stanley H R, Weisman M I, Michanowicz A E & Bellizzi R (1978) "Ischaemic infarction of the pulp: Sequential degenerative changes of the pulp after injury." J.Endo.Vol.4,No.11: pp 325-335.

Stark M M, Kempler D, Pelzner R B, Rosenfeld J, Leung R L & Mintatos S (1977) "Rationalization of electric pulp testing methods." Oral Surg.,Oral Med.,Oral Path. Vol.43, No.4: pp 598-606.

Stenvik A & Mjor I A (1970) "Pulp and dentine reactions to experimental tooth intrusion." Am.J.Orthod. Vol.57, No.4: pp 370-385.

Stephen R M (1937) "Correlation of clinical tests with microscopic pathology of the dental pulp." J.Dent.Res. Vol.16: pp 267-278.

Stoops L C & Scott D (1976) "Measurement of tooth temperature as a means of determining pulp vitality." J.Endodontics Vol.2 No.5: pp 141-145.

Takahashi K (1985) "Vascular architecture of dog using corrosion resin cast examined under scanning electron microscope." J.Dent.Res.64 (Spec.Iss.): pp 579-584.

Tamse A, Kaffe I, Littner M M & Shani R (1982) "Statistical evaluation of radiologic survey of pulp stones." J.Endodontics Vol.8, No.10: pp 455-458.

Taylor P P (1953) "The evaluation and use of the high frequency vitalometer." Oral Surg. Vol.6: pp 1020-1024.

Taylor P P (1960) "Pulp testing: an evaluation of various technics." J.Dent.Child. Vol.27: pp 107-112.

Teitler D, Tzadik D, Eidelman E & Chosack A (1972) "A clinical evaluation of vitality tests in anterior teeth following fracture of enamel and dentin." Oral Surg. Vol.34, No.4: pp 649-652.

Ten Cate A R (1985) "Reaction paper: Session 1. Odontoblasts." J Dent.Res. Vol.64 (Spec.Iss.): pp 549-551.

Topazian R G (1957) "Pain thresholds and factors which modify them." Oral Surg. Vol.10, No.11: pp 1192-1203.

Torneck C D & Howley T P (1989) "A comparison of pulpal and tactile detection threshold levels in young adults." Am.J.Orthod. Vol.96, No.4: pp 302-311.

Trowbridge H O, Franks M, Korostoff E & Emling R (1980) "Sensory response to the thermal stimulation in human teeth." J.Endodontics Vol.6, No.1: pp 405-412.

Turvey T A (1989) "Commentary" Cleft.Pal.J. Vol.26, No.2: pp 104.

Upthegrove D D, Bishop J G & Dorman H L (1966) "A method for detection of blood flow in the dental pulp." J.Dent.Res. Vol.45, No.4: pp 1115-1119.

van Hassel H J & Harrington G W (1969) "Localization of pulpal sensation." Oral Surg.,Oral Med.,Oral Path. Vol.28, No.5: pp 753-760.

van Seenberghe D & de Vries J H (1978) "Psychophysical threshold level of periodontal mechanoreceptors in man." Arch.Oral Biol. Vol.23: pp 1041-1049.

Vargas F (1956) "Accommodation of normal and pathologic pulp nerves." J.Dent.Res. Vol.35, No.5: pp 723-733.

Vargas F & Vivaldi L (1957) "Correlation between nervous accommodation symptomatology, and histology of normal and pathologic tooth pulp: Its application to electrodiagnosis." J.Den.Res. Vol.38: pp 866-880.

Wahab M H A & Kennedy J G (1987) "The effect of rate of increase of electrical current on the sensation thresholds of teeth." J.Dent.Res. Vol.66: pp 799-801.

Wakisaka S & Akai M (1989) "Immunohistochemical observation on neuropeptides around the blood vessel in feline dental pulp." J. of Endo. Vol.15, No.9: pp 413-416.

Walker A (1933) Ed. Discussion of H L Reiss and A Furedi's article. Dental Cosmos Vol.75: pp 281-283.

Walker J A, Tanzer F S & Harris E F (1987) "The enkephalin response in human tooth pulp to orthodontic force." Am.J.Orthod. Vol.92, No.1: pp 9-16.

Warwick R & Williams P L Ed.(1973) "Gray's Anatomy" 35th Ed. Pub. Longman, pp 1224-1225.

Weine F S & Potashnick S R (1982) Chapter 13 "Endodontic-orthodontic relationships." Endodontic Therapy. Edited by Weine, Pub. C V Mosby.

White J H & Cooley R L (1977) "A quantitative evaluation of thermal pulp testing." J.Endodontics Vol.3, No.12: pp 453-457.

Wilder-Smith P F F B (1988) "A new method for the non-invasive measurement of pulpal blood flow." Int.Endo.J. Vol.21: pp 307-312.

Woolley L H, Woodworth J & Dobbs J L (1974) "A preliminary evaluation of the effects of electrical pulp testers on dogs with artificial pacemakers." J.Am.Dent.Ass. Vol.89: pp 1099-1011.

Yamamoto H, Gomi H, Kozawa Y, Yamaura Y, Matsushima K & Yamazaki M (1987) "A comparative study between clinical and pathological diagnosis using extirpated pulps." J.Nihon Univ.Sch.Den. Vol.29: pp 196-202.

Yoshikawa K D (1981) "Biomechanical principles of tooth movement." Dent.Clin.North Am. Vol.25, No.1 pp 19-26.

Zadik D, Chosack A & Eidelman E (1979) "The prognosis of traumatized permanent anterior teeth with fracture of the enamel and dentin." Oral Surg.,Oral Med.,Oral Path. Vol.47, No.2: pp 173-175.

Ziskin D E & Wald A (1938) "Observations on electrical pulp testing." J.Dent.Res. Vol.17, No.2: pp 79-89.

Ziskin D E & Zegarelli E V (1945) "The pulp testing problem: The stimulus threshold of the dental pulp and the peridental membrane as indicated by electrical means." J.Amer.Dent.Ass. Vol.32: pp 1439-1449.