PULP REACTIONS FOLLOWING PARTIAL PULPECTOMY

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PULP REACTIONS FOLLOWING PARTIAL PULPECTOMY

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I, GEOFFREY SINCLAIR HEITHERSAY, declare that this thesis contains no material which has been accepted for the award of any other degree or diploma in any University and further that to the best of my knowledge and belief, no material is contained which has been previously published or written by another person except when due reference has been made in the text.

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SUMMARY

The repair reactions of a pulp amputated 2 - 5 mms from the apex have been described by several workers during this century. The histological study of partial pulpectomy (or sub-total pulp amputation) which forms the content of this thesis was initiated to add to the relatively limited histological material dealing with this form of therapy.

The study is based on animal and human material. The animal experiments, performed on 15 hooded rats, acted as a methodological study and serves as an adjunct to the human material. The human study consisted of a pilot investigation into the method of amputation, the type of wound produced and possible experimental errors. The main investigation was carried out on 9 patients (10-16 years) who formed the "younger group" and 4 patients (40-46 years) who constituted an "older group". 43 roots were treated in the younger group - mainly on first premolars requiring extraction for orthodontic reasons, while 24 roots of teeth to be removed for prosthetic reasons constituted the material in the "older group".

Calcium hydroxide in the form of Calxyl was used throughout the experiments as a dressing and root filling material. A Hedström file with a flattened tip was the instrument used to effect the amputation in both rat and human teeth.

The amputation wound produced in the rat was of a "lacerated type". Following an initial acute inflammation due to the trauma, the vital pulp tissue, under the superficial necrotic layer produced

by the action of calcium hydroxide, showed a series of connective tissue changes leading eventually to repair by calcific bridging. Initially a "dense zone" of fibrous tissue and hyalinised fibrous tissue was found. Further changes within this dense zone resulted in calcification.

Although the histological preparation of material presented difficulties, it was shown that the rat could be used to study the repair reactions following partial pulpectomy.

The pilot study in humans showed that the usual type of wound produced using a flattened Hedström file was a "lacerated" type with some degree of twisting of the remnant. It was shown that the pulp remnant in experimental teeth with open apices could remain attached to the periapical tissues at the time of extraction. The loss of the remnant was indicated by a layer of odontoblasts remaining on the dentinal wall of the canal.

Two types of reaction following partial pulpectomy were found in the younger group in the main human study:

(1) Resolution of the acute inflammation under a zone of superficial necrosis resulted in normal pulp tissue lying in direct contact with this layer. No attempt at fibrous or calcific repair was evident.

(2) Repair of the superficial pulp tissue under the layer of superficial necrosis resulted in fibrosis, hyalinisation and finally calcification in the "dense zone."

The older material showed only the second response and seemed to produce calcific changes more readily. Complete bridging was a sign of one response by a healthy pulp, while partial bridging was often associated with localised chronic inflammation. No canalised dentine was observed in the bridge, and the bordering pulp cells, which were orientated parallel to the bridge, were apparently fibroblasts. Internal resorption, usually followed by repair by cellular secondary dentine (calcific scar tissue) was a common finding in both groups.

Histological results bore little relationship to the clinico-radiographic evaluation. In the young group 38°/₀ of cases were histologically successful and 33°/₀ doubtful. Correspondingly 48°/₀ of the older group were successful and 13°/₀ doubtful.

The therapy is considered, despite the prognosis assessment, to have definite clinical indications in special cases. The repair reactions observed in this study showed that a biological apical seal can be attained following partial pulpectomy and dressing with calcium hydroxide.

INTRODUCTION

Vital pulp therapy, in its various forms, aims at a biological repair of the pulp or periodontal tissues. The concept that the pulp is a highly specialised connective tissue structure containing all the cells of defense and repair found in other connective tissue was established by Orban (1929). In the light of this knowledge, the surgical treatment of the vital pulp, in common with all living tissue, should be based on established biological principles so that the mechanisms of defence and repair will attain the desired healing response.

A thorough understanding of the anatomy, physiology and pathology of the pulp and surrounding structures is an essential basis for any rational therapy. Vital pulp therapy includes surgical procedures in varying locations in the pulp and differences in response may be due to differences in the anatomy, physiology and pathology of these regions within the same organ. For example, variations in blood supply may influence the pattern of healing. A section is included to discuss the anatomy and physiology of the pulp from general and regional aspects.

Massler (1958) has formulated a glossary of terms for endodontic therapy and these include the procedures of pulp capping, partial pulpotomy, total pulpotomy, partial pulpectomy and total pulpectomy. In the interests of uniformity this glossary of terms will be used throughout this thesis, although the writer feels that

the term "partial pulpectomy" is somewhat ambiguous, and this is supported by the fact that the literature already contains major works dealing with "pulpotomy" which have been described under the term of "partial pulpectomy" (e.g. Jeffrey, 1962). The writer prefers "sub-total pulp amputation" as a descriptive term for the therapy intended by the term "partial pulpectomy."

Some of the different treatment methods and materials have been extensively studied on human and animal material and a vast wealth of knowledge is available.

Calcium hydroxide in varying forms has been shown to produce the most beneficial effects in promoting healing in pulpal tissues. A review of investigations and theories dealing with the actions of calcium hydroxide will form portion of this thesis. Calcium hydroxide in the form of calxyl has been the material used in the present study.

Pulp capping has been studied in detail by many workers on a clinical and histological basis.

The histological evaluations have established the indications for treatment, expected reactions within the pulp and the prognosis for this therapy.

Pulpotomy has also been studied extensively, both clinically and histologically, and although deficiencies in diagnostic methods make follow-up examinations of this treatment somewhat difficult, nevertheless the indications, results and prognosis are well established.

Healing following total pulpectomy has been described on histological grounds but the majority of studies have been clinical in nature.

A review of the repair reactions which occur with these three forms of therapy will be discussed in this thesis as it would be of some interest to contrast differences in the healing pattern with the different forms of pulp therapy.

Partial pulpectomy was first described as a therapy by Nygaard-Ostby (1939) although it had been discussed by Davis (1921). This therapy was designed to avoid the problems associated with treatment of the apical region of root canals. In this region irregularities and the presence of accessory canals may be such that adequate conservative endodontic treatment in the form of total pulpectomy may be impossible. If a small remnant of vital pulp tissue could undergo repair then a biological seal would result. The position of this biological seal would have the advantage over a seal following pulpotomy in that the root canal above the amputation site could be utilised in the construction of a post crown if necessary.

The literature describing the histological changes involved in such a procedure is limited and so the present study was undertaken in an effort to contribute to the present knowledge on this therapy.

Pulp treatment of teeth with incompletely developed roots in young patients has always presented difficulties and so it was considered to be of interest to evaluate this treatment in such an age group.

The potentiality for repair in older patients has been the subject of ever changing views. Grossman (1960) stated that "With increasing age there is a diminution in reparative powers. On general principles, therefore, root canal work should be undertaken with caution in individuals past middle life ...." Many studies, e.g. Nyborg (1958) have shown healing in aged pulps can occur readily. Nyborg has stated that "The state of the pulp is probably more important than the age of the patient" (Nyborg and Slack, 1960). This potentiality for repair in the pulps of older patients has been included in this study as the method under discussion would seem on theoretical grounds, to be of value in dealing with some of the special problems encountered in this age group. The treatment of fine curved root canals in adult patients can prove extremely difficult, especially in molar teeth. Vital remnants of pulpal tissue sometimes remain after attempted total pulpectomy and their elimination usually causes the patient some discomfort. If these remnants can undergo repair successfully, much time and inconvenience could be avoided and the end result may prove as satisfactory as more conventional therapy.

Thus the aim of the present histological survey of human teeth treated by partial pulpectomy in young and adult patients has been to add to the present limited material on this subject especially in regard to the healing mechanisms involved and also to investigate any differences in healing due to the age factor.

To supplement the study, partial pulpectomy has been performed on rats. It was hoped from this study to gain some useful additional information for the main study, and, as there does not appear to be any reported method of performing partial pulpectomy on rats, the technique may prove useful to other workers in this field.

THE ANATOMY AND PHYSIOLOGY OF THE PULP

This specialised connective tissue structure has been described in detail by many workers. The therapy under investigation deals with the apical 2 - 4 millimetres of the pulp, while other forms of therapy deal with the pulp at higher or lower levels. A description of differences in anatomy or physiology within a dental pulp may be of some help in assessing the reactions which may occur in one special region of the pulp.

A general description of the pulp is that of a loose connective tissue structure of mesemchymal origin. It consists of cells and intercellular substance, the latter being formed of fibres, collagenous and reticular, lying within a ground substance, composed of muco-polysaccharide with a high percentage of hyaluronic acid.

The pulp has a rich vascular supply of thin walled blood vessels which are terminal in type. Other tissue elements include lymphatics, myelinated and unmyelinated nerves, cells of defense e.g. un-differentiated mesenchymal cells, histocytes and amoeboid wandering cells; fibroblasts and certain specialised connective tissue cells - the odontoblasts. The odontoblasts line the dentine surface and their fine processes enter the dentinal tubules, thus giving an intimate association between the dentine and the pulp from an anatomical and physiological viewpoint.

The cells of defense have been described as belonging to three main groups (Orban, 1957). One group is that of the histiocyte; an adventitial cell which is generally located along the capillaries. Undifferentiated mesenchymal cells described by Maximow (1927, 1950) form another group. These cells are thought to be pluripotential and capable of developing into any connective tissue cell. Accordingly these cells are of major importance in any surgical treatment of the pulp. The amoeboid wandering or lymphoid wandering cells constitute the third group and are thought to be elements from the blood stream.

For descriptive reasons the pulp is usually divided into two sections - the coronal pulp, and the radicular pulp.

For the purpose of this thesis only, the pulp has been subdivided into 3 regions which correspond to the position of pulpal wounds encountered in pulp capping, pulpotomy, and partial pulpectomy. These three regions are: 1). Coronal pulp, 2). Mid-pulp, and 3). Apical pulp.

Accessory Canals

The apical one third in an adult tooth is a relatively complex anatomical area due to accessory canals and the nature of the apical foramen. It has been suggested (Orban, 1957) that "a defect in Hertwig's epithelial root sheath during development of the root at the site of a larger supernumerary blood vessel" might be a possible explanation for the development of all side branches of the pulp. In molar teeth accessory canals can be present in the floor of the pulp chamber.

The accessory canal contains pulp tissue and a blood supply, the presence of a blood vessel being thought to be the developmental cause of the canal. Although an additional blood supply does exist via these accessory root canals it is claimed (Gardner, 1963) that "there is no scientific evidence that accessory or aberrant root canals are significant in providing sufficient collateral circulation to maintain the vitality of the pulp after an injury." Blaney (1929) however considered this collateral blood supply to be of importance.

Vascular Architecture

The vascular architecture of the human dental pulp has been demonstrated in vitro by injection in histological methods following the development of a technique by Kramer (1951) using extracted teeth. Micro-radiographic studies on human teeth have also been described (Saunders, 1957). These authors reported that in many root canals, the sub-odontoblastic plexus is present on one aspect only. Full anastomoses between the vessels of each root in multi-rooted teeth were described. Several lateral canals were found connecting the root canal to the periodontal membrane and these generally contained a pair of vessels - one large and one small.

Provenza (1958) found that the major arteries of the dental pulp never exceed the dimensions of an arteriole, but a normal arterial structure can be distinguished. The lumen of the veins is large and the thin walls consist of an endothelial lining, a poorly developed media and adventitia.

The capillary network is composed of various types of vessels. True capillaries, being solely endothelial in nature, contrast with metarterioles which show single or groups of muscle cells surrounding the endothelium at varying intervals. Arterio-venous anastomoses were also demonstrated.

Cheng and Provenza (1959) observed that the diameters of both arteries, and veins were greater in the mid pulp than in the apical or coronal pulps. Generally veins were of greater diameter than the arteries, however in the mid pulp the converse was the case. These authors suggested that this variation in size may indicate a greater demand for arterial circulation in the mid pulp and therefore a higher degree of metabolic activity.

Capillaries in the odontoblasts layer have been demonstrated by Meyer (1951). Orban (1928, 1957) stated "the capillaries form loops close to the odontoblasts, near the surface of the pulp, and may even reach into the odontoblast layer." Many workers, e.g. Thoma (1950) and Stones (1954), maintain that capillaries in the odontoblast layer constitute new formations due to trauma, e.g. due to the pressure of the forceps during extraction. Langeland (1957) however, confirmed Orban's finding in this regard.

In vivo studies mainly dealing with reactions of the pulpal tissues to medicaments applied locally have been done by Taylor (1950) and Pohto and Scheinin (1958) and these workers have added to the knowledge of the vascular physiology of the pulp.

Thus in summary of any regional differences in blood supply, the apical pulp would appear to consist mainly of thick walled arteries which have not undergone much early branching. Additional blood vessels are transmitted through the accessory canals which are found in this region. The capillary loops are present but not in the profusion of the mid and coronal pulp. The mid pulp appears to be the area of maximum vascularity and possibly the area of maximum metabolic activity. Considerable side branches of the main arterial supply are present but the main artery (or arteries) is still present in a central position and is of the same diameter or possibly larger than in the apical pulp. The capillary bed is extremely rich.

The coronal pulp is not as richly supplied with blood vessels as the mid pulp, but is much more profuse than the apical pulp. This region contains the final branching of these main terminal type arteries. The pulpal horns are richly supplied with capillary loops.

Lymphatic Supply

On the basis of investigations by Schweitzer (1909), Noyes (1918, 1927, 1929) and Fish (1932), Orban states that lymph vessels in the pulp, with branches connected to the regional lymph nodes, have been demonstrated.

No regional description of lymph vessels is possible from the literature available. However, the number of lymph vessels may be supposed to be dependent on the blood supply in the various regions.

Nerve Supply

The pulp is richly supplied with nerves which enter the apical foramen as thick nerve bundles and divide into numerous fibre groups

in the coronal pulp. These smaller fibre groups divide into single fibres. Generally nerve bundles and fibres run in close relation to corresponding blood vessels and capillaries. The pulp is supplied with both myelinated and unmyelinated elements, the former supplying the sensory nerve supply and the latter the "sympathetic" nerve supply to the blood vessels. The myelinated fibres after dividing in the coronal pulp form a layer beneath the subodontoblastic zone of Weil. From this zone the individual fibres pass through the sub-odontoblastic layer; and losing their myelin sheath, enter their terminal distribution in the odontoblastic layer.

From a regional point of view the coronal pulp is the site of final branching of the major trunks and would seem to be the region most profusely supplied with myelinated nerves. As the unmyelinated nerve supply would parallel the blood supply, it should be most abundant in the mid pulp. The apical pulp would appear from the available literature to contain large nerve trunks with a moderate supply of small fibres from the final arborisation from the coronal pulp.

Cellular Components

Langeland (1957) states that "it is generally accepted that the number of cellular elements varies in different parts of the pulp". The 'cell rich' layer which lies pulpward to Weil's zone contains many more cells than the central area of the pulp. In the fully developed tooth, the cellular elements decrease in number toward the apical region, while the fibrous elements increase (Orban 1957).

The odontoblasts - the highly differentiated connective tissue cells are grouped along the predentine. They vary in shape and size depending on the location. In the coronal pulp they are described as "high columnar", in the mid pulp as "low columnar" and in the apical pulp as "spindle shaped" (Orban, 1957). Orban (1957) has suggested that the change in shape of odontoblasts towards the apical foramen may be caused by mechanical factors, e.g. movement of the apex when the tooth is in function, or by changes in the blood and lymph stream producing varying pressure in the narrow apical portion of the root canal.

Fibrous Components

The fibres of the pulp are partly pre-collagenous, and partly collagenous. The amorphous ground substance of the pulp forms a continuous mass thus making the pulp the only connective tissue in the human body without interstitial spaces (Maximow and Bloom, 1950).

Zerlotti (1964) studied histochemically the connective tissue of the dental pulp with special reference to reactivity and organisation and also the changes of tissue colloids during ageing. His results indicated that the extracellular matrix of the connective tissue of the dental pulp contains glyco-proteins, sialic acid, acid muco poly-saccharides and proteins bearing reactive a ϵ- amino groups of lysine and hydroxylysine. The fibres and the ground substance were considered to interact forming co-acervates. The extra-cellular matrix is organised into a heterogeneous colloid containing soluble and insoluble fractions. In young pulps the soluble fraction was

often the only site of "diffuse calcifications". These amorphous small structures are considered to be degenerative calcifications following hyaline degeneration of the pulp, associated with age changes. Generally they are deposited in close relation to collagenous fibre bundles or blood vessels. They may occur in mid pulp, but rarely in the coronal pulp.

Age Changes

Age changes in the pulp usually consist of a decrease in the cellular components and an increase in the fibrous content of the pulp due mainly to a decrease in the blood supply. With this general reduction in blood supply, fibrosis, hyalinisation and diffuse calcification, occur especially in the apical region. Deposition of secondary dentine leads to a narrowing of the root canals and deposition of apical cementum and causes a constriction of the apical foramen. The histo-chemical results obtained by Zerlotti (1964) relative to age changes in the pulp have been discussed earlier.

Calxyl has been reported to have a haemolytic effect (Sauerwein, 1951) as well as an antiseptic action. Investigations have demonstrated that calcium hydroxide solution has bactericidal action on members of the oral flora and a number of pathogenic spore forming organisms. (Hermann, 1930, Jansen, 1949, Proell, 1949, Hailer and Heicken, 1941, Conrados (1963)).

Reactions on tissues other than the pulp have revealed many interesting properties of this material. Although an intact mucous membrane is not damaged by calcium hydroxide, the reaction of sub-epithelial tissues to this material is that of superficial tissue necrosis, presumably due to the high alkalinity. This necrotizing effect has been studied on animals by many workers e.g. Hansen (1949), Effinger (1953), Mitchell and Shankwalker (1958), McDonald et al (1959) and Jeffrey (1962). All these workers confirmed the localized caustic effect of calcium hydroxide on sub-epithelial tissues.

The osteogenic potential of calcium hydroxide is of great significance in endodontic therapy. Mitchell and Shankwalker (1958) implanted calcium hydroxide into subcutaneous and bone tissue in rats. Following the subcutaneous implantation they observed that a form of heterotropic calcification occurred 10 - 35 days after implantation. In the early specimens there was a considerable inflammatory reaction which was replaced by fibrosis. Fatty tissue was often seen to be associated with bony deposits. Implantation of calcium hydroxide into bone resulted in delay in healing compared with a control wound.

Yoshiki et al (1960) also implanted calcium hydroxide in bone marrow, muscle, subperiosteally, in subcutaneous tissue and in extraction sockets. They observed early indications of mineralization in these different areas. Similar results were reported by McDonald, Sawawi and Mitchell (1959), but could not be duplicated by Jeffrey (1962) or Laws (1962).

These investigations illustrate the osteogenic effect of calcium hydroxide. The material may accelerate calcification in tissues which are osteogenic, and also produce calcification in tissues which normally do not undergo calcification.

A further study dealing with tissue reactions to calcium hydroxide paste, implanted subcutaneously into the backs of rats and guinea pigs, has been done by Yoshiki and Mori (1961). They have surveyed the distribution of various enzymes in tissue into which calcium hydroxide had been implanted and also described the general tissue reactions.

They observed that after one week the implanted cylinder of calcium hydroxide had lost its original shape and had been penetrated by a coarse fibrillar network. The network appeared to be completely necrotic and structureless, whilst the surrounding tissue was strongly basophilic and partly invaded by proliferating fibroblastic tissue. With longer experimental periods the central area of the network was replaced by cellular fibrous tissue while later the basophilic network lying within the fibroblastic tissue, thickened to some extent and developed a cellular element, producing a bone-like structure.

Their histochemical analysis revealed that the initiation of hard tissue formation due to calcium hydroxide occurred at a very early stage and that acid phosphatase, esterase, glycosidase, succinic dehydrogenase and T.P.N. diaphorase were probably concerned with the mineralisation.

In dental practice, calcium hydroxide as a pulp dressing material, is usually used in the form of a paste with either distilled water or Ringer's solution as a vehicle, although the powder has been applied alone by insufflation. Calxyl is basically calcium hydroxide in Ringer's solution. Methyl Cellulose in combination with calcium hydroxide is also used extensively and the resulting paste is a-granular and of medium viscosity. The American preparation Pulpdent is an example of this combination. Other commercial preparations which have calcium hydroxide as the principal ingredient include Calciform Serocalcium and Biocalc.

The reactions of pulp tissue to calcium hydroxide has been studied extensively and with each new investigation further hypotheses on its action have been advanced. Still further knowledge is necessary however before the exact mode of action of calcium hydroxide can be formulated.

Review of histological and histochemical studies relating to pulp reaction to calcium hydroxide

Flohr (1936) observed that the surface layer of the pulp in contact with calxyl became necrotic and ascribed the change as being due to the alkalinity of calxyl. Zander (1939) reported one histological examination of a clinically successful pulpotomy. His

main study was a clinical and radiographic study of pulpotomy in 150 cases using calxyl or calcium hydroxide. He described a dark amorphous structureless layer on the outside of the dentine bridge, which showed cell inclusion or empty spaces and this layer was thought to be the result of the first chemical reaction between calcium hydroxide on the tissue surface of clotted blood and degenerated injured pulp cells. This layer was thought to form the matrix into which calcium phosphate was precipitated. Zander's hypothesis on the action of calcium hydroxide at this time was the simplest and seemingly most obvious explanation possible, namely that of simple mass action. The alkaline conditions induced by calcium hydroxide were known to increase phosphatase activity. Thus the abundance of calcium ions, high PH and the presence of an organix matrix were thought to be the decisive factors in the rapid laying down of calcium phosphate.

Hori (1943) noted that calcium hydroxide destroyed tissue when directly in contact, but promoted calcification when applied indirectly thus emphasizing the fact that calcium hydroxide can exert an effect through a medium.

Glass and Zander (1949) in the most convincing human comparative study of the actions of calcium hydroxide and zinc oxide and Eugenol, described the tissue changes at observation periods of 24 hours, 2 weeks, 4 weeks and 8 weeks. The result of pulp capping with Zinc Oxide and Eugenol was that of an extending chronic pulp inflammation while the response to calcium hydroxide was one of repair which was complete by 4 weeks.

At 24 hours they demonstrated a pulp showing superficial necrosis, but no haemorrhagic mass. The necrotic area was demarcated from the healthy pulp tissue below by a layer described as the "proteinate zone" as this was thought to be made up of calcium proteinate.

At 2 weeks: Zones seen were (1) necrotic zone (2) proteinate zone (3) fibrillar zone (4) normal pulp tissue. The fibrillar zone was likened to fibrous or primitive bone and was thought to be only partly calcified.

At 4 weeks: The necrotic layer was not visible, but the proteinate and fibrillar zones were present, the latter having new dentine deposited on it. A new layer of odontoblasts had been formed.

At 8 weeks: Further healing resulted in a thicker dentine barrier and newly organised odontoblasts.

Thus at this stage the calcium hydroxide was thought to produce calcium proteinate up to its depth of penetration. The necrotic tissue was not thought to be of any significance in calcification and Zanders original theory of mass action was still considered valid.

Dausch and Sauerwein (1952) studied healing following pulpotomy. They considered that the necrotizing action of calxyl continued for 2 - 3 days. Using the von Kossa staining method they demonstrated the existence of calcium salt bordering the vital pulp and the necrotic tissue due to the influence of calcium hydroxide. This observation was also made by Noguchi (1954).

Thus the calcium ion from the calcium hydroxide was thought to play some part in the calcification process, acting through the necrotic layer.

The theory of action was disproved by Nyborg (1952).

In a differential study he capped human pulps with both calcium hydroxide and calcium sulphate. Both these materials in saturated solution yield approximately the same number of dissociated calcium ions. Histological examination revealed no healing with calcium sulphate, although considerable hard tissue was formed over the chronically inflamed pulp.

Further evidence has been supplied by Berman and Massler (1958), and Koslov and Massler (1960), working with amputated pulps in rats. They found that healing occurred within the pulp by bridging when materials other than calcium hydroxide were used, such as zinc oxide and Eugenol. The conclusion was that the calcium ion was not necessary for bridge formation. It should be noted however that studies on rats may not necessarily be applicable to humans. The pulps in the teeth of rats rapidly undergo connective tissue changes and are less specific in their response. This is emphasised by the fact that no study on human pulps has been able to duplicate the healing with zinc oxide in rats as found by Berman and Massler, and Koslov and Massler.

The most precise method of demonstrating whether or not calcium ions enter into the formation of the calcified bridge has been to use radioactive calcium hydroxide as a pulp capping agent. This method has been used by Yoshida (1958) and Siaky and Pisanty (1960). These workers used radioactive calcium 45 to prove that the calcium ions of the layer were not derived from the calcium hydroxide paste.

They concluded that these ions were derived from the tissue fluid of the pulp.

Nyborg and Slack (1960) suggested that the favourable effect of calcium hydroxide probably depended on its hydroxyl ion. These authors pointed out however, that work by Svejda (1958) had shown that the hydroxyl ion was not the only vital factor; as the results of capping with magnesium hydroxide (PH 9.5) compared unfavourably with calcium hydroxide. These unfavourable results with magnesium hydroxide however have not been confirmed by Eda (1961). In a comparative histochemical study using dogs four pastes were used as pulp dressings following pulpotomy. The materials used were calcium hydroxide paste (PH 12.7), magnesium oxide paste (PH 10.3), triozinc paste (PH 6.3) and calcium fluoride (PH 4.1).

Magnesium oxide dissolved in water forms magnesium hydroxide. Both calcium hydroxide and magnesium hydroxide were found to be equally effective on the formation of new dentine, but the deeper pulpal response in the case of magnesium hydroxide did not appear favourable.

Eda (1961) has presented a good description of the reaction of the pulp tissues to calcium hydroxide. He concluded that the following reactions occurred:

(1) Necrosis of that part of the pulp contacting calcium hydroxide occurred within 30 minutes and reached its maximum penetration by 6 hours, thereafter maintaining the same width until 20 days or more after operation.

(2) Granules consisting mainly of calcium carbonate appear in the necrotic layer within 30 minutes. The calcium was thought to have come from the paste. With increase in number and size of the granules, deposits of magnesium and phosphoric acid occur in the granule.

(3) Extremely fine particles reacting positively to von Kossa's stain appeared in the vital pulp tissue just beneath the necrotic layer. With extension of the superficial necrosis, these particles became deposited in the necrotic layer. The calcium salt, forming the fine particles was derived from tissue fluid and not from the overlying paste. Magnesium appeared in small amounts in this layer after 3 hours, reaching a maximum after 10 days. Phosphoric acid was also deposited in this layer after 24 hours.

(4) Large pear shaped cells appeared in the pulp beneath the layer of fine particles after 5 days, assuming odontoblastic form in 10 days, and producing dentine after 15 days.

(5) Dentine formation commenced not in the necrotic layer, but directly below it.

(6) Magnesium, being observable after 3 hours in the zone of future dentine deposition, perhaps indicates that magnesium plays an important part in calcification.

Although there are still many unproved hypotheses relating to the action of this material, through the thirty four years of its use in vital pulp therapy it has been the greatest contributing factor to the preservation of teeth with pathologically involved pulps.

REPAIR OF THE PULP FOLLOWING VITAL PULP THERAPY

All forms of vital pulp therapy involve trauma to the pulp; or in the case of total pulpectomy, the periapical tissues. A wound with accompanying inflammation results.

It has been shown that if an aseptic wound has the blood clot and all other debris removed, and the surface is protected from any irritation, then healing will not occur. (Carrell and Ebeling (1926)). This applies equally to the pulp, as has been shown by Nyborg (1955). Nyborg found no evidence of repair of the pulp following pulp capping with an inert material. Thus for healing to occur within the dental pulp a wound dressing is necessary.

When considering the changes which occur in the pulp following vital therapy there are two main factors. First there is the inflammatory reaction resulting from the mechanical trauma. Secondly there is the effect of the medicament on the pulp tissue. The effect of calcium hydroxide on pulpal and other tissues has been discussed in the previous section. As both these reactions occur simultaneously it is difficult to distinguish between their individual effects. The pulp is a highly differentiated fibrous connective tissue. The inflammatory response is similar to that which occurs generally in fibrous connective tissue and this may be briefly described as follows:

Mechanical injury to connective tissue results in tissue destruction at the site of the injury. This tissue injury causes the release of tissue metabolites - histamine or a similar substance, termed H-substance (Lewis & Grant 1924) and peptides in the form of serotonin or related compounds (Hilton & Lewis 1955). These metabolites have two main effects on the surrounding blood vessels. The first effect is that of an axon reflex (Lewis 1924) resulting in arteriolar dilatation which is later followed by capillary dilatation. The second main effect of the tissue metabolites is a direct effect on the capillary wall producing capillary dilatation and an increase in capillary permeability.

This increased capillary permeability coupled with an increase in the capillary hydrostatic pressure due to the arteriolar and capillary dilatation, results in the passage of fluid to the interstitial spaces. This exudation of fluid causes an increase in the viscosity of the blood with accompanying slowing of the circulation. Venular hypertension develops which only accentuates the capillary hydrostatic pressure and so increases the exudation.

The exudate of inflamed, fibrous connective tissue consists of fluid and cells. The fluid fraction consists to a large degree of certain proteins, which is of importance, not only because of the antibodies these molecules contain, but also as the fibrinogen fraction may be converted into fibrin. Such deposition of fibrin creates a barrier to the inflammatory process and also blocks the lymphatic capillaries.

The cellular component of the inflammatory reaction consists mainly of polymorphonuclear leucocytes, macrophages, lymphocytes, plasma cells and some mast cells, and eosinophils. The inflammatory cells are derived either from a vascular source (haematogenous) or from tissue cells (histogenous). The cell type which appears is determined by a series of local factors. The cells of vascular origin migrate from their blood vessels following the initial slowing of the blood stream and margination of the cellular components along with rouleaux formation of the red cells. Migration of these cells, is an active process and occurs after leucocytes have stuck to the endothelial lining. The increased stickiness of the leucocytes coupled with their active movements are thought to be the two most important factors in this process.

The power of active movement is a property of most of the cells in an inflammatory exudate. Perez Tamayo (1961) considered that there had not been any satisfactory demonstration of the alleged positive chemotaxis of injured tissues although he considered that some extracts of inflammatory exudates, e.g. leukotaxine, were powerful agents. Most bacteria on the other hand certainly exhibit a positive chemotactic effect.

The inflammatory cells of tissue origin are derived mainly from fixed and wandering tissue cells - derived from undifferentiated mesenchymal cells.

Both the haematogenous and histogenous cells either move

spontaneously, or possibly under a chemotactic influence, migrate into the injured area where their role in tissue defence comes into play - the main defence mechanism being phagocytosis. The cells which usually are the first to appear in the injured area are polymorphonuclear leucocytes. Later they are followed and mainly replaced by lymphocytes, macrophages, and/or plasma cells, etc. The most acceptable hypothesis for this variation in the cells which move into the injured area at a particular time in the inflammatory reaction is that a change in pH. is responsible.

Thus by a combination of the activity of the cells and the fluid exudate of the inflammatory reaction, the injured area is prepared for the next stage, namely repair. Debris, comprising damaged tissue cells and extravasated redblood cells is removed and toxic substances neutralised by antibodies or absorbed into lymphatics.

The process of repair, which usually results in the restitution of form and function in the injured part, overlaps the inflammatory reaction and is initiated by it (Perez Tamayo 1961). A pulpal wound following vital pulp therapy corresponds to a wound which would normally be repaired by secondary intention (by granulation).

On a purely descriptive basis, healing can be divided into three sections: (a) cellular activity (b) vascular proliferation and (c) deposition of intercellular substances.

(a) Cellular Activity

When leucocytes and macrophages have commenced the preparation of the injured area for repair by removing tissue debris, undifferentiated

mesenchymal cells, become activated. These cells are normally present in connective tissue, and following inflammation they move by amoeboid action to the site of the injury. After exhibiting intense mitotic activity, these cells are considered to differentiate into young fibroblasts and can be seen penetrating the injured area (Perez-Tamayo 1961). The fibroblastic cells become star shaped and then produce protoplasmic prolongations which form a relatively dense and fine network around the cell body.

From the fourth day the fibroblasts become elongated and bipolar. Gradually as the fibroblasts become actively engaged in laying down interstitial fibrils, the cells become smaller and less conspicuous and the fibres more abundant. At ten days, fully developed collagen bundles are present with intermingled cells which show a small hyperchromatic nucleus with a much reduced cellular cytoplasm and which may now be termed fibrocytes.

(b) Vascular Proliferation

Solid extensions from existing blood vessels neighbouring the damaged area develop two to three days after injury and are the result of cell division and cytoplasmic elongation of the endothelial cells. These extensions grow at a comparable rate to the proliferating fibroblasts. Thus vascular sproutings are produced which unite together to form arches, become canalised and so produce a circulation. Some capillary buds are reabsorbed but others become converted into arterioles, capillaries and venules. This so-called granulation tissue contains at this stage a vascular system with the number of

blood vessels far exceeding that found in other parts of the same tissue. After eight to ten days there is a reduction in both the size and number of the blood vessels to a degree which is thought to be related to the metabolic activity of the tissue (Williams 1951). When scar tissue is the end result it becomes virtually avascular.

(c) Deposition of Intercellular Substances

Oedema is an early sign after injury to tissues and is due to increased capillary permeability. During the next two to three days there is increasing oedema and deposition of acid mucopolysaccharides which are demonstrated as metachromatic material (Taylor & Saunders 1957). The peak of this deposition occurs 4 - 6 days after injury, after which the concentration falls until normal levels are reached after ten to twelve days. Free amino acids which enter into the formation of collagen also appear between four to six days.

Six days after injury there generally appear fine argyrophylic fibrils which tend to arrange themselves perpendicular to the capillaries. These fibrils develop from fine protoplasmic projections of fibroblasts which have a similar orientation. The free argyrophylic fibrils increase in size due probably to fusion of the thin fibrils (Perez-Tomayo 1961) or due to the precipitation into the fibrillar matrix of soluble precursors synthesized by the cells (Jackson 1958).

These fibre bundles are converted into acidophilic collagen bundles which become mature collagen by ten to twelve days.

Thus at the completion of healing the intercellular substance is made up of abundant collagen fibres with fibrocytes scattered between them supplied by a few narrow blood vessels.

HEALING FOLLOWING PULP CAPPING

The reaction of the pulp to pulp capping with calcium hydroxide has been studied by many workers. While clinical studies are of value, assessment of the therapy histologically is more satisfactory and conclusive. A summary of studies of pulp capping with calcium hydroxide is shown in Table I (after Nyborg 1958).

Three major studies on pulp capping in which histological assessment of the results has been used, in addition to clinical observation provide excellent descriptions of the pulp reactions to capping with calcium hydroxide. Glass and Zander (1949) were the first to give a precise description of the healing process and their results have been discussed in detail in the previous section.

Nyborg (1955) reported an extensive histological study on experimental pulp capping in dogs and humans. The same author (Nyborg 1958) reported on the histological findings of a clinical series of pulp cappings in humans. As the work of Nyborg provides such an excellent description of the healing process involved in pulp capping, it is intended to review solely his two studies with some reference to the earlier work of Glass and Zander (1949).

Nyborg described the reactive processes in the lesion as consisting of various zones, all of which were subject to changes as the processes of inflammation and repair progressed. The zones described were as follows:

1. Superficial Debris. This layer consisted of dressing material,

TABLE I RESULTS OF CAPPING IN PERMANENT TEETH

(After Nyborg, 1958)

Author	Intact tooth or superficial caries	Deep caries; no pulpitis	Caries and pulpitis	Age of patient (yrs)	Method and capping material	Control period	Clinical examination No. of satisfactory cappings	Histologic examination	
								No. of pulps	No. of satisfactory cappings
Flohr 1936	1			Young pat.	Calxyl	6 weeks		1	1
			1	Young pat.	Vitapulp	4 weeks		1	1
Teltzrow 1938			1	15	Vitapulp	4 weeks		1	1
		1		15	Vitapulp	4 weeks		1	1
Kirsten 1038		2		29,36	Calxyl	3 - 4 weeks		2	2
Beerendonk 1939		12			Calxyl	2 - 31/2 mo.	11		
Pajarola 1940	41	18		Children & adults	Calxyl	3 - 9 mo.	59	26	23
			35	Children & adults	Calxyl	3 - 9 mo.	35	11	8
Maraasse 1942		15		12 - 60	Calcium hydroxide	3 - 5 mo.	13		
Fenner 1944		90		Children & adults	Calxyl	3 mo - 5 years	86	4	3
Glass & Zander 1949	20			Children	Calcium hydroxide	1 - 16 wks		16	16
Büchtold 1949	10			Children	Calxyl	3 mo.	10	10	9
Tannenbaum 1951		54		Children & adults	Calcium hydroxide	6 mo - 1 year	49		
Cabrini, Maisto & Manfredi 1953	27			16 - 30	Calcium hydroxide (21 cases) Zno-eugenol (5 cases) dentine spicules (1 case)	1/2hr - 102 d.		27	11
Castagnola 1953		200		Adults	Calcium hydroxide	1 - 9 yrs	178		
Schug-Küsters & Kierse 1953	2	3	3	Adults	Calxyl, Roegan	1 wk - 6 mo.		8	3
Warrer 1954		24		7 - 15	Calcium hydroxide	41/2 - 39 mo.	20		
Nyborg 1955	44			11 - 28	Calcium hydroxide	2 dys - 32 mo.		44	31
Ahlström & Krasse 1956		118		Adults	Calxyl	8 mo - 4 years	85		
Pritz 1957		85		Children & adults	Calxyl	6 wks - 41/2 yrs	80		
			50	"	Calxyl	6 wks - 41/2 yrs	41		

dentine spicules, fragments of necrotic tissue, a fibrin-like mass and blood pigment.

2. The Necrotic Layer. Due to the action of calcium hydroxide alone, the superficial layer of the pulp was necrosed. The extent of the necrotising action was limited, being at its maximum after 2 - 3 days. As this was a gradual process, the deeper tissue, before undergoing necrosis, had undergone inflammation and subsequent organisation. Thus the deeper layers of this zone were described as having a somewhat fibrous structure with numerous argyrophilic fibres present on occasions. After two days the necrotic layer began to separate from the underlying tissue until the seventh day when separation was usually complete.

3. Blood Pigment Layer. Nyborg demonstrated that haemolysis occurred when calcium hydroxide came into contact with blood. The resulting blood pigment was especially evident in the superficial aspects of the dense zone and the deepest parts of the necrotic layer. When separation of the necrotic layer occurred, it was usually located at or within this layer.

4. The Dense Zone. The tissue below the necrotic zone was dense in appearance and histologically was easily differentiated from underlying pulp tissue. This zone represents basically the zone of organisation and repair of the pulp.

The cellular reaction in the dense zone and subjacent tissue after two days was shown to contain cells of connective tissue

origin and, in addition, a small number of leucocytes and plasma cells. The cellular exudate in the zone disappeared between the second and seventh days following capping. A cell-rich zone was described in the deepest parts of the dense zone and subjacent pulp tissue. The connective tissue type cells increased in number first around the blood vessels, as they were thought to be derived from these vessels, or from the undifferentiated mesenchymal cells. These cells were fibroblast-like cells which were thought to produce the argyrophilic fibres and the collagen in the dense zone. In addition to these fibroblast-type cells - derived from the undifferentiated mesenchymal cell, odontoblasts were described. These were only found on the edge of the wound lining the dentinal wall. This contrasted with the odontoblast-type cells described by Dausch and Sauerwein, 1952; and Glass & Zander 1949.

Organisation

Increased vascularity and formation of argyrophilic fibres were observed after two days. The vessels were rapidly surrounded and eventually obliterated, by argyrophilic and later collagenous fibres. The collagen formation seemed to begin near the blood vessel wall and slowly extended throughout the zone, usually being complete by four to five weeks.

The initial formation of collagen seemed to commence from argyrophilic fibres which thickened and merged into homogenized collagen. This homengenized or hyalinized collagen, often with cell inclusions, appeared to be an un-mineralised immature hard tissue - similar to osteoid. Glass and Zander (1949) referred to this tissue as "primitive dentine".

Calcification

Calcification of this newly formed tissue occurred after only two days and was considered to be a result of the action of calcium hydroxide. The extent of subsequent calcification was dependant on the condition of the deeper tissue. Nyborg considered that, as no quantity of mineral salts could be found in the necrotic layer, the greater proportion of the salt content of the dense zone was supplied by the pulp and not by the calcium hydroxide capping agent.

Mineral salts appeared to be precipitated first in the walls of the blood vessels, then both in the argyrophilic and collagenous substance in the dense zone.

The dense zone was considered to be organised finally into a calcified tissue, the surface of which consisted of argyrophilic fibres and bundles while the deeper tissue consisted of homogenised or hyalinised collagen.

Lacunae in this calcified tissue were described and were thought to occur when the process of necrosis involved an area at so early a stage that the maturation of the collagen was arrested. The surrounding region subsequently became strongly calcified. Some cell inclusions were found, while other lacunae spaces were due to calcified vessels.

The New Blastema

After fourteen days a blastema-like arrangement in localised regions of the cell-rich layer was described. This did not become continuous until one month or more, when some of the cells resembled mature odontoblasts. Nyborg observed that with longer periods a

continuous "odontoblastema" was found, but the cells lay with their long axes parallel to the calcific deposit in contrast to the orientation of normal odontoblasts. He observed that undifferentiated connective tissue cells could also be observed in this layer in a 16-month case.

The Dentine-like Tissue

After the initial deposition of a calcific barrier, having no definite structural arrangement, the cells of the new blastema began to lay down dentine. This deposition was observed within a month of capping. As more dentine-like tissue was formed, the number of dentinal tubules tended to increase and the pattern of deposition became more regular.

Pulpal Reactions away from the Lesion Area

The reaction in the pulp away from the capping area consisted of hyperaemia, which lasted for some time. Nyborg considered it possible that this reaction was due to the trauma involved in extracting the tooth prior to histological examination. Other reactions described in the pulp were possible "reticular atrophy" and slight disruption in the odontoblast layer which resulted in slight changes in predentine formation. Internal resorption following pulp capping was not reported in this series.

REPAIR FOLLOWING PULPOTOMY

There have been many clinical and radiographic studies of pulpotomy in humans (e.g. Teuscher and Zander, 1938; Retarski, J.S. 1940; Zander and Law, 1942; Brinsden, 1955; Law, 1956; and Vin, 1955).

These studies indicated a considerable variation in the repair potential of the pulp following pulpotomy. Percentage success varied from 31°/₀ to 99°/₀ and this variation further emphasises the importance of a histological evaluation of such treatment.

Such histological studies have been carried out in humans by Zander (1939), Roth (1940), Dausch & Sauerwein (1952), James Englander & Massler (1957). Further histological studies in rats have been done by Massler, Perrault & Schour (1955), O'Malley (1956), Myamoto (1957), Berman & Massler (1958), and Koslov & Massler (1960).

The above studies in humans show a similar but not identical repair reaction to that occurring after pulp capping.

Reactions in the Lesion Area

(a) Necrotic Layer

Dausch and Sauerwein (1952) demonstrated that the necrotising effect of calxyl lasted for two to three days. 12 to 24 hours after pulpotomy the pulp tissue presented normal cellular and exudative response to injury, but after two days, the cells were no longer visible.

(b) Blood Pigment Layer

Endothelial damage and some haemolysis was observed by Dausch and Sauerwein near the necrotic zone. This would appear to correspond to the blood pigment zone described by Nyborg.

(c) Dense Zone and Calcific Barrier

Within the demarcation zone between the necrotic layer and the vital pulp, diffuse calcification was observable after one to two days; and after seven to ten days a zone of calcific deposits was evident. Zander (1939) described this zone as "a dark amorphous structureless layer showing in some places cell enclosures or empty spaces". He considered that these cell enclosures resulted from the first chemical reaction between calcium hydroxide and the tissue surfaces which consisted of clotted blood and degenerated injured pulp cells. Into this matrix, the first calcium phosphate was thought to be precipitated, thus giving the base for true dentine formation. Roth (1940) described this layer as "fibrillar hard substance".

(d) Dense Zone

Dausch and Sauerwein observed a few weeks after pulpotomy that collagenous fibre bundles were present lying perpendicular to the demarcation zone and were undergoing calcification.

(e) Dentine Formation

Odontoblast-like cells were observed between these bundles and these slowly increased in number until six to eight months when an odontoblastema was formed which deposited dentine against the calcified collagenous bundles.

Roth (1940) considered that the odontoblasts were produced by metaplasia of the pulpal mesenchymal cells. He likened the odontoblastema formed beneath the hard tissue barrier as being similar to epithelium repairing a skin wound and considered that the odontoblasts

formed a cover over the pulpal remnant. He also considered that dentine spicules acted as centres of growth by stimulating the pulp cells.

Reactions in the Pulp Remnant away from the Lesion Area

(a) Increased Vascularity

James, Englander & Massler (1951) reported the increased vascularity in the pulp tissue below the amputation. This would be expected following the trauma inflicted on the pulp tissue during pulpotomy. Following the general principles governing resorption and deposition of bone, laid down by Grieg (1931), the increased vascularity could be expected to produce resorption of dentine.

(b) Internal Resorption

Roth (1940) discussed osteoclastic and osteoblastic actions in the dentinal marrow following pulpotomy. Internal resorption was reported in 20°/₀ of adult teeth treated by pulpotomy and dressed with calcium hydroxide (James, Englander and Massler, 1957). Jeffreys (1963) reported internal resorption in 2.5°/₀ of deciduous teeth treated by the same method.

(c) Calcifications

Calcifications within the pulp remnant were described by Roth as being due to a change in metabolism of the pulp accompanied by an alteration in the colloidal composition. This reaction occurred on devitalised tissue deep within the pulp remnant, and resulted in an encrustation with calcium salts.

Calcification in areas other than the amputation site were observed in 15°/₀ of cases investigated by Jeffrey (1961).

Thus the main comparative points in the reported studies between pulp capping and pulpotomy appear to be -

1. Lesion Area

Although there is considerable variation in the description and interpretation by the various workers, the basic reactions and layers formed appear to be the same.

2. Pulp away from Lesion Area

The pulp beneath a tooth treated by pulp capping appeared to remain in a relatively normal state if the therapy was successful. No internal resorption or pulp calcifications were reported. In contrast, the pulp remnant following pulpotomy appeared to exhibit increased vascularity leading to internal resorption in a relatively high proportion of treatments. Irregular calcifications also seemed more evident in teeth treated by pulpotomy.

REPAIR FOLLOWING TOTAL PULPECTOMY

Removal of the contents of the root canal produces a wound usually located at the dentino-cemental junction within the root canal. This region represents a transition from pulp to periodontal tissue. The amputation wound thus transects the major blood vessels, nerves and lymphatics which enter the canal in this region.

Grove (1921) on the basis of histological studies, reported two main changes which occurred at the apex following vital pulpectomy and root filling. Either the apical periodontal membrane had a tendency to invaginate itself into any unfilled region at the apex, or the remaining soft tissues deposited new layers of a cementum-like substance, both within and outside the canal possibly causing its complete obliteration.

Although the contents of the major canal are removed, the accessory canals generally retain vital tissue. Blayney (1929) described the reactions which occur within the apical delta of canals following the removal of a vital pulp. Resorption and rebuilding were features of repair following pulp removal and this was considered to be due to the contents of the accessory canals adapting themselves to the new conditions.

Blayney considered that the blood vessels entering through the accessory canals constituted an important part of the normal supply to the pulp, and therefore pulpectomy resulted in a great reduction in the blood supply required for these regions. Because the pulp remnants

required reduced blood supply, the size of the vessels decreased, and so the lumen of the canals became correspondingly narrowed by deposition of a hard scar tissue.

Some remnants were found to undergo marked atrophic degeneration, thus requiring even less blood supply leading to even greater obliteration. Blayney considered healing to be complete when the apical periodontal membrane and soft tissue within the canal from the root filling to the root end were entirely free of all evidence of inflammation.

Hatton (1931) considered the healing processes following pulpectomy and root filling to be essentially similar to those following a bone fracture. He described three stages in this process, viz:

1. A destructive stage in which all tissue either within the root canal or in the apical space which had seemed damaged to the extent that it could no longer function, was removed by resorption and dissolution.
2. A constructive stage in which lost hard tissues of the tooth were replaced by osteoid cementum and resorbed bone by new bone. This process was thought to be extended so that the space formerly occupied by soft tissues became calcified, and in some instances this led to almost complete obliteration of the unfilled portions of the pulp canals.
3. A resting stage in which the calcifying process eventually ceased and the tissues in general remained in a constant state except for a tendency for the soft tissues to undergo fibrous atrophy.

Fukunaga (1959) made a comparative study of the effect of

different root filling materials on healing following pulpectomy, sterilisation and sealing of the canal. The materials used were calcium hydroxide paste, paraformaldehyde, zinc oxide and eugenol and gutta-percha.

The healing process appeared to occur in three general ways:

1. Gradual healing after the incidental acute inflammation resulting from treatment.
2. Repair of the periapical tissue destroyed by the trauma during treatment.
3. Closure of the apical foramen by hard or soft scar tissue.

Sekine, Machida & Imanishi (1963) in a comparative clinical and histological study have described healing following vital pulpectomy. The canals were filled with a material named "New Paste" which consisted of 98.5°/₀ calcium hydroxide plus various antibacterial substances. Healing occurred in two main forms:

1. Granulation tissue from the periodontal membrane proliferated into the apex of the root canal. This tissue subsequently became cicatrized and a cementum bridge was formed sealing the canal completely. This extension of the periodontal tissues into the root canal has been reported by many investigators e.g. (Grove 1921, Tollardo, 1931; Biolcati et al 1942; Nygaard Ostby, 1944; Engel, 1950; Kukidome, 1957; Hyaksisoski, 1959; Kurovica, 1960; Matsumiya & Kitamuva, 1960) quoted by Nygaard Ostby, 1961. This has occurred in cases where the canal has been filled with a resorbable material or not filled completely to the apex.

2. Cicatrization of the periodontal membrane occurred outside the apical foramen. Cementum was subsequently deposited to close the apical foramen completely.

In both these forms of healing the trauma involved in pulpectomy, with the accompanying inflammatory response in the periodontal membrane resulted in resorption of the dentine and cementum of the apex.

HEALING FOLLOWING PARTIAL PULPECTOMY

Previous Studies

The ability of apical pulp tissues to undergo repair following partial extirpation or partial pulpectomy was demonstrated in detail in the 1920's by histological studies of teeth apparently treated by total extirpation. Nyborg (1965) however, reported that Preiswerk (1901) was probably the first to demonstrate healing of a vital pulp stump following partial removal.

Davis (1921) in a series of articles, advocated partial removal of the pulp rather than total pulpectomy. He considered that any amputation positioned between the bulbous portion of the pulp and the dento-cemental junction was desirable and would result in healing of the pulp by calcific tissue. His concept was courageously presented at a time when endodontic treatment was viewed with the utmost suspicion. His published work showed photomicrographs of two treated teeth, although the total number of teeth examined is not mentioned.

Davis compared the repair of a vital pulp remnant to the closure of the medullary canal following amputation of long bones. The essential repair reactions described were:

(1) a "pulp callus" formed over the vital pulp tissue;

(2) osteoid tissue was laid down in the pulp canal until

(3) partial or complete obliteration of the root canal was produced.

Davis also came to the conclusion that youth seemed more favourable for

this repair, although with advancing years repair did occur, but at a reduced rate. The larger the pulp canal the more quickly repair was found to begin and the more complete was the closure.

Skillen (1924, 1926) reported on a histological assessment of 500 root treated teeth, approximately half of which were without radiographic evidence of periapical pathology. He presented photomicrographs of four teeth showing remnants of pulp tissue - two inflammation-free under a calcific bridge and surrounded by a considerable internal deposit of an osteoid material. The other two showed total necrosis. Skillen noted that resorption of the root canal frequently preceded calcification and he considered the resorption to be due to the acute inflammation produced by the treatment.

Coolidge (1933) showed healing of an apical pulp stump following partial pulpectomy. He published photomicrographs of three cases in which vitality of a pulp stump had been maintained under a gutta percha root filling. No bridging occurred within the pulp tissue in contact with the gutta-percha but deposition of secondary cementum was extensive, reducing the pulp to a fine remnant.

In 1939 and 1944 Nygaard Østby described the therapy of partial pulp extirpation in some detail. He recommended the use of a Hedström file with a flattened tip to facilitate the amputation of the pulp a few millimetres from the apex. Healing of the pulp stump was reported in 7 out of 9 cases treated by partial pulpectomy and root filled with gutta percha and either Hartz chloroform or chloropercha. The repair reactions described were basically the same as those observed by the previously mentioned workers.

Table II lists the histological studies of partial pulpectomy from 1939 and gives the number of successful cases. The treatment methods vary considerably and so direct comparison is only possible between a few of the studies.

Calcium hydroxide in varying forms has been used in four of the studies. Rohner (1940) experimented on 20 teeth; of these 10 were devitalised with cobalt and of the remainder, two had established pulp pathology at the time of treatment. Thus the total number of cases treated by vital partial pulpectomy could, for the purpose of comparison, be considered to be 8. Calxyl was used as a dressing material but this was followed by a root filling of gutta percha after varying periods.

Machida (1960) used a preparation termed "New Paste" which consisted of powder:

	calcium hydroxide	98.5	)
	sulfathiazole	0.8	)
	homosulfamin	0.3	)
	guanofuracin	0.1	)
	sulfasaraizin	0.3	)
	asbestos powder		30 gr.
liquid	propyleneglycol		30°/₀ solution

25 teeth were treated by partial pulpectomy using especially prepared long shank burrs to amputate the pulp. Of these 21 were considered to have produced successful healing.

Laws(1962) used a propylene glycol base in his experimental study in which 8 teeth were treated by partial pulpectomy. Amputation was by means of blunted root canal files. One tooth from this study

failed clinically and histologically was the only tooth to exhibit inflammatory changes.

Nyborg and Tullin (1965) used calcium hydroxide mixed with Ringers solution as a dressing material in 11 of the 15 teeth treated by partial pulpectomy. In 4 of these the calcium hydroxide was allowed to remain as the only root filling material for the entire experimental period. In the remaining 7, a root filling of gutta percha and chloropercha was placed after periods varying from 1 1/2 to 3 months following amputation. The pulp remnants in the remaining 4 teeth were dressed with Lugol's iodine solution for periods between 3 and 7 days priorto root filling with gutta percha and chloropercha. A flattened Hedström file was the amputation instrument of choice following the experimental studies of one of the authors (Nyborg 1960, Nyborg and Hålling 1963).

Although Ketterl (1963) used Diaket as a root filling material, his method of pulp amputation was that of the Stafen-Mess technique, which is claimed to result in a layer of dentine spicules being formed over the amputated pulp. This technique utilises a special burr (Q Bohrer) which amputates the pulp and is stated to pack dentine spicules from the surrounding dentine walls over the pulp stump at an optimum pressure.

The repair reactions in the studies mentioned above will be discussed later in relation to the present author's investigation.

TABLE II

AUTHOR	NO. TEETH	ROOT FILLING OR DRESSING MATERIAL	EXPERIMENTAL PERIOD	NO. SUCCESSFUL
NYGAARD-OSTBY 1939 - 1944	9	Gutta Percha- and Harts Chloroform or Chloropercha	28 days -6yrs 10mths	7
ROHNER 1940	8	Calxyl and Gutta percha	4 1/2 - 10 months	6
MACHIDA 1960	25	"New Paste" (Calcium Hydroxide + Anti-Bacterial Substances) + Propylene Glycol	2 - 439 days	21
LAWS 1962	8	Calcium Hydroxide with Propylene-Glycol	19 days - 4 months	7
KETTERL 1963	38	Dentine Spicules Diaket	1 month - 9 years	28
WAECHTER & STOCKINGER 1964	20 Roots			9
NYBORG & TULLIN 1965	15	Chloro Percha & Gutta Percha or Calcium Hydroxide	5 1/2 months - 4 years 8 months	10

PRESENT INVESTIGATION

MATERIALS AND METHODS

The present investigation consisted of two main sections - the first using the hooded mutant strain of the rattus Norwegicus and the second using human material. A pilot study was also completed as a check on the method employed in the human subjects.

A. Rat Study

A study of the rats dentition suggested that the most suitable tooth for endodontic treatment was the upper first molar. A view of the skull of the rat is shown in Appendix I (Figs 124, 125). The mesio-buccal root was chosen as that most suitable for endodontic instrumentation. The opening into the mesio-buccal root canal was found to lie directly below the mesio-buccal cusp and this landmark made the localisation of this small canal somewhat easier. The root curves forwards and upwards which meant that the file had to be inserted with a distal inclination of the handle if the canal was to be satisfactorily entered. The curvature of this root made it extremely difficult to ensure that the apical foramen and the pulp remnant were in the same plane of section, when prepared histologically.

15 of the hooded mutant strain of the rattus Norwegicus were used for this study. Each rat was fed on a stock diet. The average weight was 300 gms.

The procedure followed in each treatment was as follows:

a. The rats were weighed.

b. Open ether was used to induce anaesthesia, followed by

an intraperitoneal injection of Sagatal. The amount of Sagatal was calculated in relation to the body weight (3mgms Sagatal required for each 100 gms body weight).

c. The rat was then held in a special stand (Appendix I Figs 126, 127) which prevented movement. The tongue was controlled with sutures.

d. Application of rubber dam was found extremely difficult and so the field was isolated solely by means of retractors and cotton rolls. The field was disinfected with hydrogen peroxide then isopropyl alcohol, followed by isopropyl alcohol and iodine.

e. A coronal opening was made in the upper first molar in the region of the mesio-buccal cusp, which gave access to the mesio-buccal root canal.

f. The pulp was amputated with a flattened Hedström file (No. 2) set to a depth of 4mm (calculated to leave 2mm pulp tissue at the apical region).

g. The canal was irrigated with sterile normal saline, haemorrhage controlled with paper points, and a dressing of calxyl, introduced with a Lentulo spiral, was packed gently down with the blunt end of a fine paper point. The coronal opening was then sealed with cavit. (refer Appendix V for composition)

h. Wherever possible the procedure was repeated on the opposite side.

i. The rats were sacrificed at varying times from immediately after treatment up to 8 weeks post-operative.

j. Radiographs were taken of all cases.

k. The relevant regions were fixed in buffered formo-saline and then decalcified, after which they were sectioned and stained with the following - H & E, Silver (Lillie) P.A.S., Van Gieson and Mallory stains wherever sufficient sections of the treated area were available.

Details of treatment and histological findings were recorded on charts as shown in Appendix II. (Page 183)

B. Human Study

Patients were obtained for this study with some difficulty, due to the nature of, and the inconvenience which the treatment involved. The majority of volunteers were from patients of the Department of Orthodontics and the Department of Prosthetics, Dental Department, Royal Adelaide Hospital. Other patients were from the author's practice. The proposed treatment was fully explained to the patients and parents and a consent form was signed.

The human subjects were divided into a "younger age" group varying from 11 years to 15 years, and an "older" age group of 40 to 50 years.

The patients of the younger age group required extractions of premolar teeth for orthodontic reasons, while the members of the older group generally exhibited caries and periodontal breakdown and were to receive either partial or full dentures.

If multi-rooted teeth were treated in this study, each individual root was considered separately in the histological analysis. A total of 69 roots were treated in the major study and 6 in the pilot.

43 roots were from 9 patients of the younger group, while 26 were from the 4 patients who constituted the older age group. The individual teeth treated are shown in Tables V & VI.(Pages 90 & 137a)

Each tooth was treated in the following manner.

a. A pre-operative radiograph using Kodak extra fast film was taken to allow an estimate of the root length.

b. Local anaesthesia was administered usually by infiltration (Carbocaine - 2%+Epinephrine, approximately 0.5cc).

c. Caries if present was removed and the cavity dressed. The crown of the tooth previously scaled where necessary, was then polished with pumice.

d. Rubber dam was applied and the field disinfected with hydrogen peroxide, then isopropyl alcohol, followed by isopropyl alcohol and iodine.

e. A coronal opening was made with a high speed drill under water spray. Access was improved using a three quarter length round burr.

f. The canal was then explored with a smooth broach to a depth just short of the proposed amputation.

g. Using a flattened Hedström file with a rubber dam marker attached, the pulp was amputated to the predetermined depth so that a remnant of vital pulpal tissue remained in the apical 2 - 4 mm of the canal. The pulp was amputated using the largest file which could pass to the predetermined apical position. This was necessary to allow the file to bind with

the canal walls in an attempt to make a clean amputation. In addition, dentine spicules may be incorporated in the wound using this method. The file was twisted firmly clockwise through 90° and then anti-clockwise to the same degree. The position of the file was checked radiographically after which the file was removed.

h. Irrigation of the canal with sterile normal saline was then undertaken and haemorrhage controlled by means of paper points.

i. The pulp stump was dressed with calxyl introduced into the canal with a Lentulo spiral root canal filler and tapped into position using the blunt end of a paper point.

j. The coronal opening was sealed with Cavit and amalgam after which a radiograph was taken.

k. The teeth were extracted at intervals from immediate post-operative to a period of one year. A radiograph was taken prior to extraction to record any observable pulp and periapical changes. Tables V and VI show the distribution of experimental periods.

Many of the experimental periods were dictated by other treatment the patient was to receive and so an even distribution was difficult to achieve.

l. After extraction the teeth were fixed in a buffered formo-saline solution (Appendix III). The teeth were then decalcified, sectioned and stained in accordance with the following staining sequence, if sufficient sections were available.

Staining sequence: (Refer Appendix III for details of staining methods employed)

1. Haemotoxylin and eosin.
2. Gomori stain.
3. P.A.S.
4. Van Gieson.
5. Mallory.

The sections were examined microscopically and where possible photomicrographs were taken.

Recording Charts: The patient's dental examination and medical history were recorded on charts as shown in (Appendix IV). Another chart (Appendix IV) recorded the clinical details of each treated tooth, follow-up examinations and histological findings.

Controls: The only controls possible without subjecting the volunteer patients to considerable surgical trauma, was to extract untreated teeth from the same mouth. This was sometimes possible on those patients in the older group who were to have full prostheses. In the younger age group as the patients available for experimental treatment were limited, and as the 'normal' histological appearance of the pulps within this age group has been well described previously only two such teeth were extracted without prior treatment.

Pilot Study

A pilot study was necessary to check on the method of amputation and to determine whether the amputated remnants might have been lost during the extraction of the tooth. As many of the teeth treated

within the young group had incompletely developed apices, it was considered possible that with the inflammatory changes following the amputation, the attachment of the pulpal tissue to dentine via the odontoblastic processes may have been weakened or lost.

This added to the lack of any anatomical barrier in the incompletely developed tooth, may result in the loss of the remnants at the time of extraction - the remnant remaining attached to the periapical tissues. Thus an empty root canal would result and the treatment as judged histologically would be presumed to be unsuccessful.

Method

The pilot study was carried out on 6 premolar roots in a boy aged 10 years. The same treatment procedure as outlined in the main study was followed except that a viscous radio-opaque medium - Neohydriol (iodised ethyl esters of the fatty acids of poppy seed oil containing 4 per cent iodine) was syringed into the canal after stage (h). Considerable care was necessary to avoid air bubbles within this highly viscous material. A radiograph was then taken and the canal sealed with cavit. The teeth were immediately extracted and fixed in formo-saline. Further radiographs were taken, then the teeth were prepared histologically as previously described.

RAT MATERIAL

RESULTS

A summary of the results of histological examination of the rats whose upper first molars were treated by partial pulpectomy is shown in Table III. Difficulty was experienced in sectioning the treated root so that the treated areas could be demonstrated. As a result it was impossible to make any histological assessment in 12 of the teeth. Cases were considered successful if the pulp remnant was present in a inflammation-free state. "Doubtful" prognosis was accorded when some degree of inflammation was present, but the experimental time was inadequate to determine a definite failure or success.

Histological Observations at varying Experimental Periods

Untreated case: (R5)

The histological appearance of the mesio-buccal root and its surrounding structures is shown in Figs 1 & 2. The pulp was cellular in nature and contained numerous relatively large blood vessels. The odontoblast layer consisted of densely packed cuboidal cells.

Immediate reaction Case Nos R8 (left & right) (Figs3 - 6).

Lesion Area

Nature of wound - both wounds were of the lacerated type. Although R8(L) had minimal tearing. Twisting of the pulp stumps had occurred to some degree. Superficial debris consisting of dressing material, dentine spicules and blood clot was present.

Deeper Pulpal tissue: Generalised hyperaemia

Periapical tissue - Normal.

TABLE III

RAT STUDY

HISTOLOGICAL RESULTS

No.	Experimental Period (days)	Inflammation					Calcific Bridging	Internal Resorption	Cellular Secondary Dentine on (a) walls (b) bridge		Histological Result		
		Nil	Acute Local	Acute General	Chronic Local	Chronic General					Fail	Doubtful	Success
1^{L}	1		-	-	-	-	-	-	-	-	-	-	-
2^{L}_{R}	3		- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -
3^{L}_{R}	3		- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -
4^{L}_{R}	3		-	-	x -	-	-	-	-	-	-	-	- -
5L	7		x									x	
6^{L}_{R}	14	x x							x x				x x
7^{L}_{R}	14	-	-	-	-	- Total Nec.	- x	- x	-	-	- x	-	-
8 L R	Immed Immed		x x										- -
9 L Re	21	-	-	-	-	- x	-	-	-	-	- x	-	-
10 L R	21	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -
11 L R	21	x				x					x		x
12 R	21	x					-						x
13 L R	42	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -	- -
14 L R	56	x x					- -						x x
15 R	28					x		x			x		

— Signifies no assessment possible

Fig. 1 R.5. H & E x 40

MESIOBUCCAL ROOT AND RELATED STRUCTURES

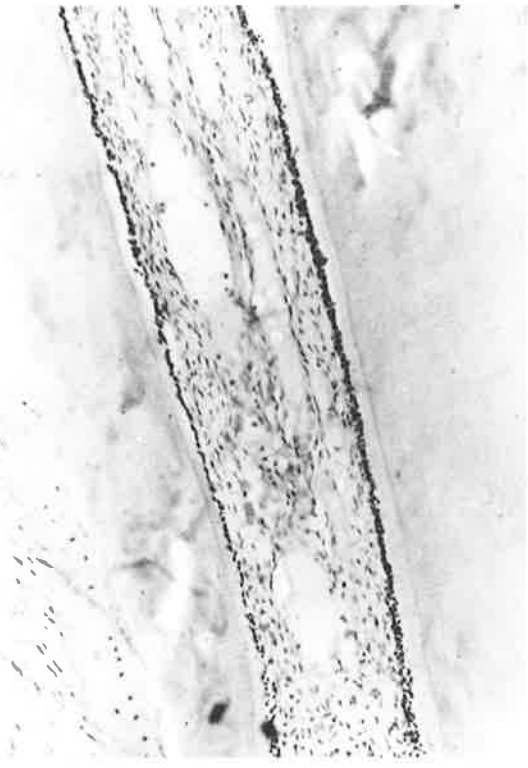


Fig. 2 R.5. H & E x 100

SHOWING CELL RICH PULP IN THE MIDDLE SECTION OF THE ROOT CANAL

Fig. 3 R.8. (Left) H & E x 40

E.P. IMMEDIATE.
PULP REMNANT WITH OVERLYING DEBRIS AND DRESSING MATERIAL

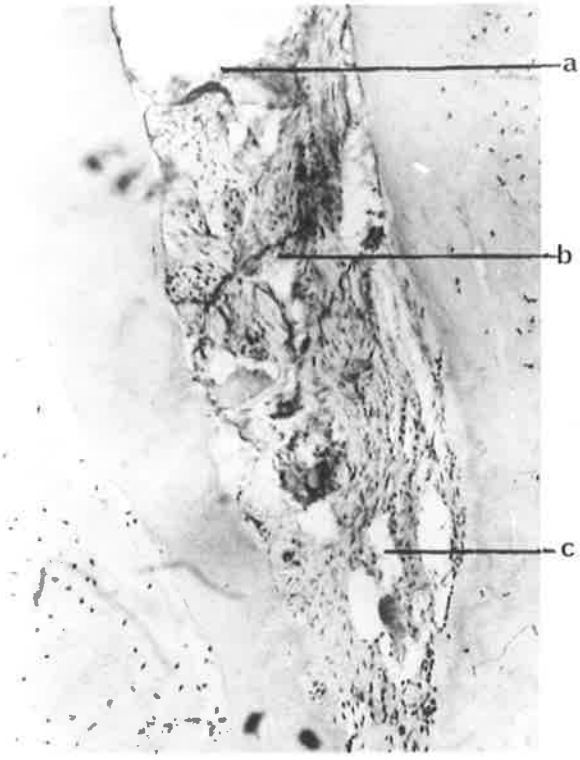


Fig. 4 R.8 (L +) H & E x 100

AMPUTATION WOUND (a) SHOWING A SLIGHT DEGREE OF LACERATION AND TWISTING OF THE REMNANT OF THE PULP eg. AT (b). APPARENT VASCULAR DILATATION HAS OCCURRED AT (c)

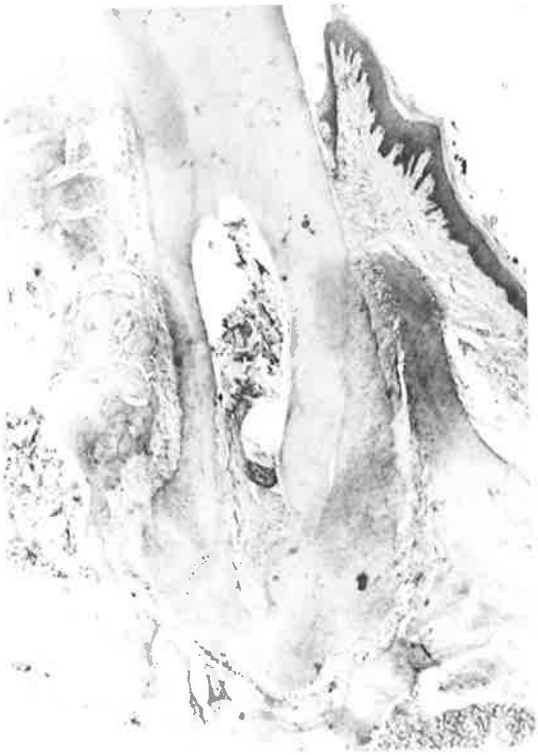


Fig.5 R.8. (Right) H & E x 40

E.P. IMMEDIATE.
"LACERATED" PULP REMNANT WITH OVERLYING SUPERFICIAL DEBRIS

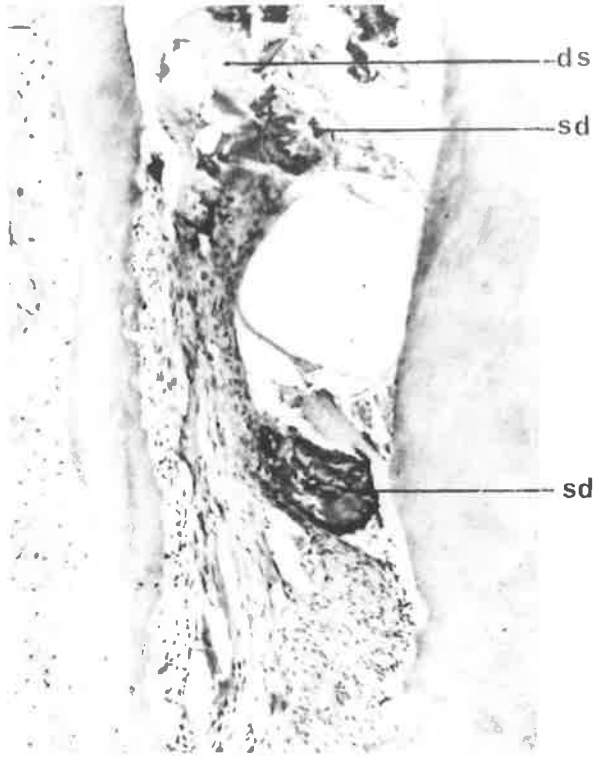


Fig.6 R.8 (Rt) H & E x 100

PULP WOUND WITH SUPERFICIAL DEBRIS (.s.d) CONTAINING DENTINE SPICULES (d.s). SURFACE OF WOUND IS RAGGED AND COVERED IN PLACES BY BLOOD CLOT AND DRESSING MATERIAL. VASCULAR DILATATION OF THE PULP REMNANT HAS OCCURRED

3 days: Case Nos R2 (L&R) R3 (L&R) R4 (L&R)

Histological assessment was only possible in R4 (left) Fig. 7.

Lesion Area: Considerable connective tissue changes had occurred in the pulp in the region of the amputation. Fibrosis and hyalinisation were evident (Fig. 7).

Deeper Pulp tissue - Normal cellular nature of pulp with a localised area of extravasated red blood cells.

Periapical tissue - Normal

7 days: Case No R5(L) (Figs. 8, 9).

Lesion area: The surface of the wound was heavily infiltrated with chronic inflammatory cells. There was some reduction in the number of pulp cells in the region, the tissue showing signs of necrosis.

Deeper pulp tissue - Some slight cellular infiltration was evident but generally the deeper pulp was normal. Some extravasation of R.B.cs had occurred.

Periapical tissue - Normal

14 days: Case Nos R6 (L&R) R7 (L&R)

No assessment was possible in R7 (L).

Lesion area: R6 (L) (Fig. 10). Well defined layers were present. Beneath the medicament or superficial debris layer was a necrotic zone which showed some separation from the underlying pulp. Fibrosis and hyalinisation of the superficial pulp tissue had occurred. Early calcific changes within the hyalinised layer were seen. R6 (R) showed apical calcific areas but sections did not allow sound assessment of re-active zones. R7 (R) (Figs 11 & 12) showed a densely packed plug of degenerated cells, dentine

spicules and amorphosis material overlying the lesion area, but no repair response.

Deeper Pulp tissue: R6. Definite changes were evident resembling diffuse calcification. R7 - Necrotic Pulp.

Internal resorption - R7 - small degree. Cellular secondary dentine - Very small deposits evident in R6 (L&R).

Periapical tissue - Normal.

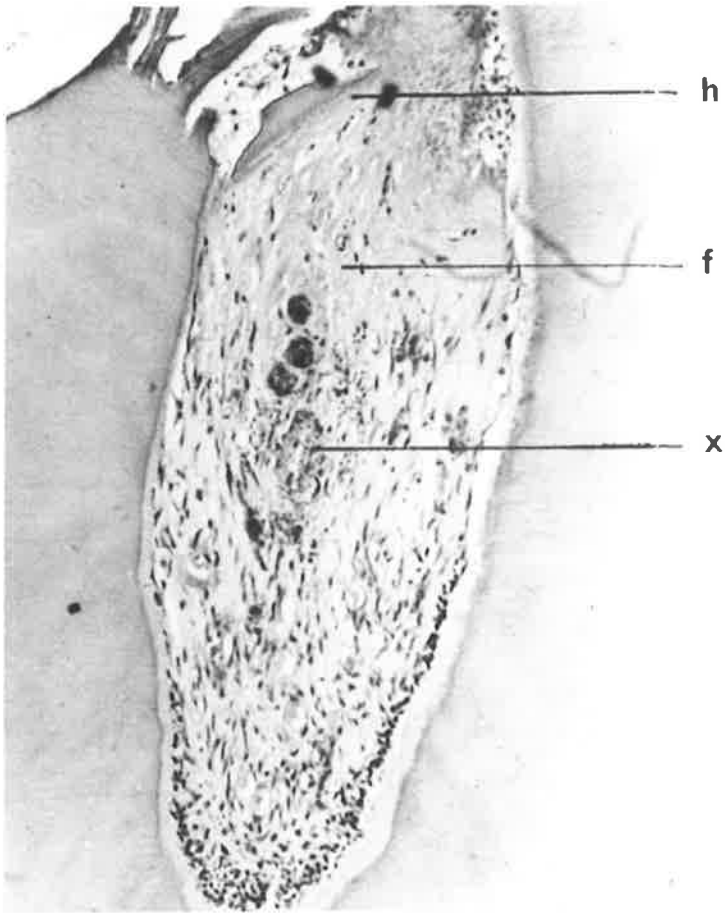


Fig.7 R.4 (Left) H & E x 100

E.P. 3 days.
FIBROSIS (f) AND HYALINISATION (h)
OF THE PULP IN THE SUPERFICIAL REGION
OF THE PULP REMNANT. AT (x)
EXTRAVASATION OF R.b.cs. HAS OCCURRED.

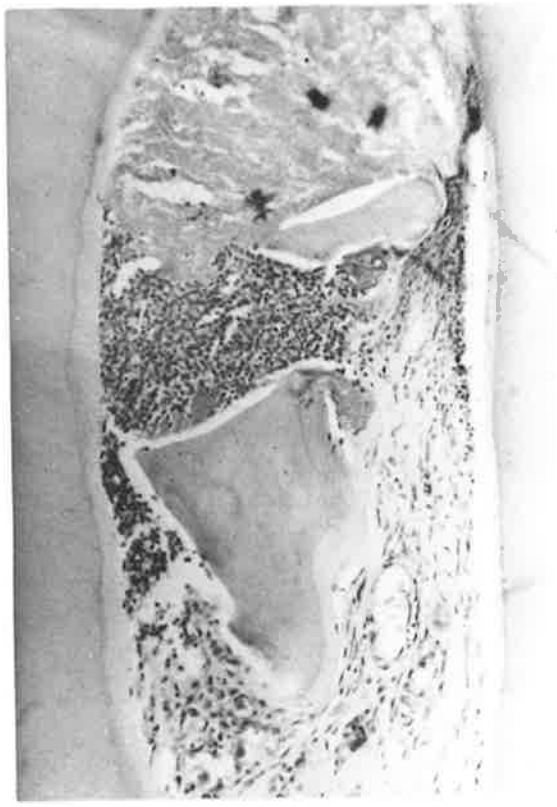


Fig.8 R.5. (left) H & E x 100

E.P. 71 DAYS:
SURFACE OF AMPUTATION WOUND
SHOWING CELLULAR INFILTRATION

Fig.9 R.5 (Lt) H & E x 100

PULP REMNANT WITH LOCALISED
CHRONIC INFLAMMATION.

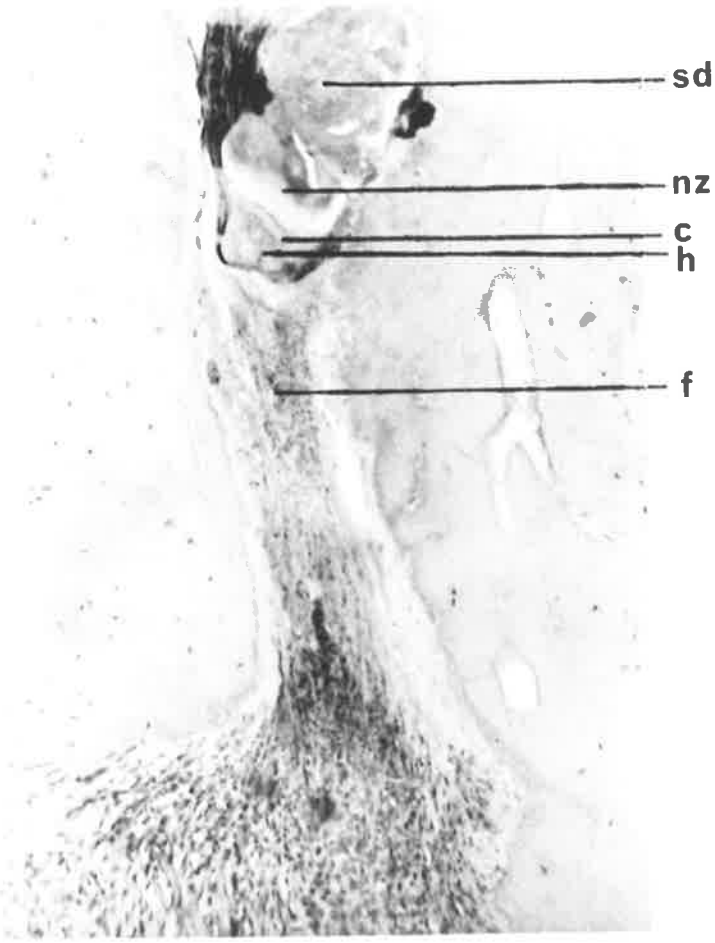


Fig.10 R.6 (Left) H & E x 100

E.P. 14 days

SUPERFICIAL DEBRIS (s.d)
NECROTIC ZONE (N.Z.)
REGION OF FIBROSIS (f)
HYALINISATION (h) AND EARLY
CALCIFICATION (c)

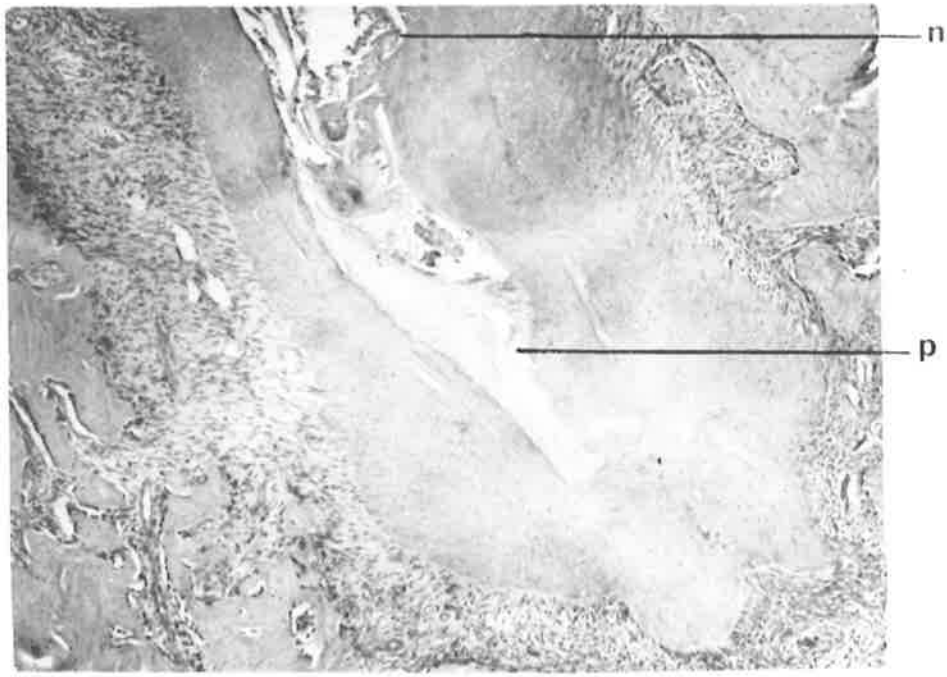


Fig.11 R.7 (RIGHT) H & E x 40;

E.P. 14 days.
THE AMPUTATION NOTCH (N) IS SHOWN ABOVE A SUPERFICIAL DEBRIS ZONE. PULP REMNANT (P) HAS UNDERGONE NECROSIS.

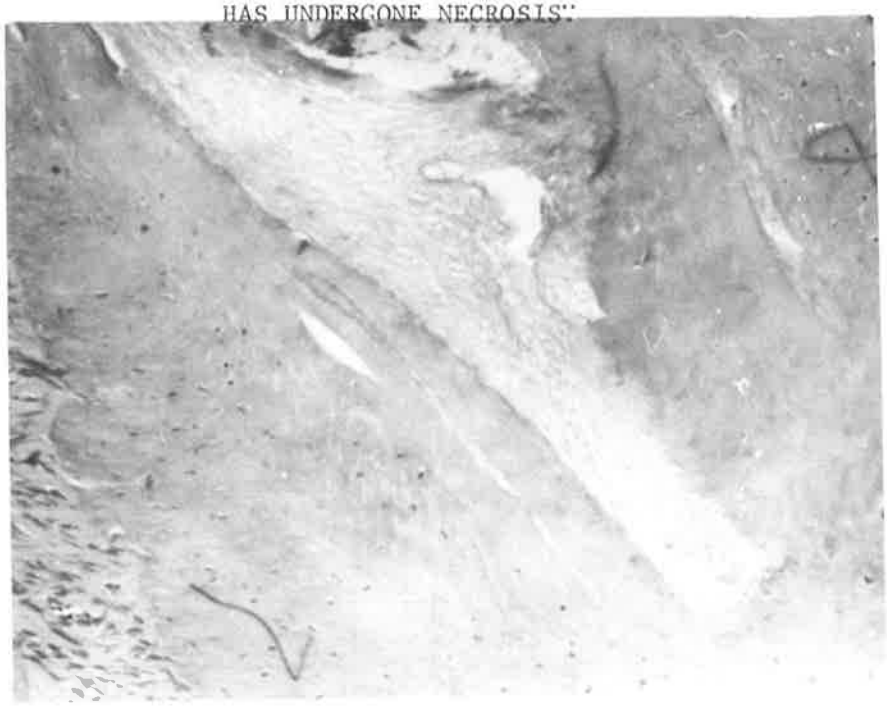


Fig.12 R.7 (Rt) H & E x 100

PULP REMNANT IS NECROTIC.

21 days: Case Nos R9 (L&R) R10 (L&R) R11 (L&R) R12 (R) No assessment possible in R9 (L) R10 (L&R).

Lesion area: R11 (R) showed possible bridging which appeared incomplete. The underlying pulp was infiltrated with chronic inflammatory cells. No definite zones were visible in other cases.

Deeper pulp tissue - Normal pulp was present in R11 (L) R12 (R). Fibrosis of the pulp occurred in R9 (R).

Generalised chronic inflammation was observed in R9(R) and R11 (R).

Periapical tissue. This was normal in all cases except R 11(R) in which there was acute inflammation as indicated by infiltration of neutrophils.

28 days: Case No R15 (R) (Figs 13 & 14)

Lesion Area: The superficial debris contained many dentine spicules. An initial necrotic zone appeared to have been formed but no further reactive zones had been produced as the underlying pulp became necrotic.

Deeper pulp tissue: This was necrotic. Limited internal resorption was present. The periapical tissues were normal.

42 days: Case No R13 (L&R)

No assessment was possible.

56 days: Case No R14 (L&R)

Lesion area - not shown.

Deeper pulp tissue - normal pulp.

Periapical tissue - Normal.

ASSESSMENT OF SUCCESS

Of the cases which could be assessed partially or completely, 9 were judged to be successful, 1 doubtful and 4 were failures.

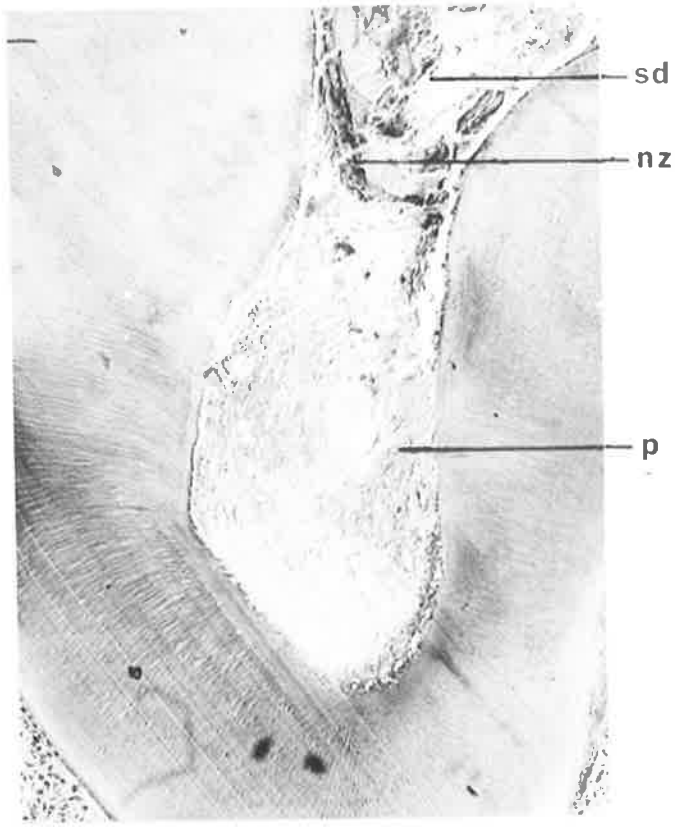


Fig.13 R15 (Right) H & E x 100 E.P. 28 days.
NECROTIC PULP REMNANT (p) SUPERFICIAL DEBRIS (s.d) NECROTIC ZONE (n.z)

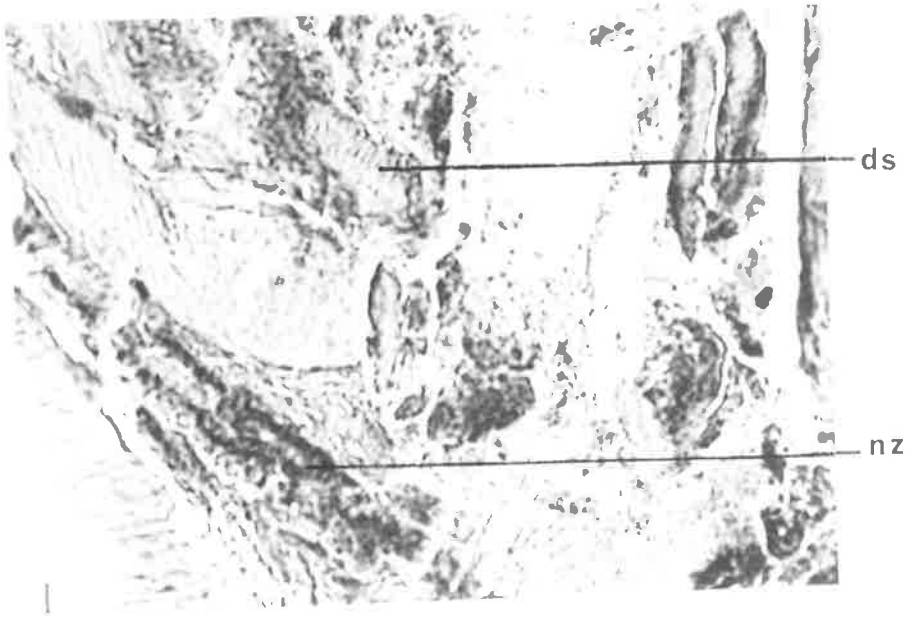


Fig.14 R.15 (Rt) H & E x 100

DENTINE SPICULES IN SUPERFICIAL DEBRIS LAYER. ILL DEFINED NECROTIC ZONE (n.z) UNDERLYING PULP IS NECROTIC

DISCUSSION - RAT MATERIAL

The material available from the above study did not allow any definite pattern of healing to be described as has been possible in other rat studies on pulp capping and pulpotomy (eg. Berman and Massler 1958). Although some cases produced reactive zones, the experimental and histological problems mentioned later have made it difficult to give a progressive description of the healing process. The problems involved in performing an amputation in the mesio-buccal root canal of the upper first molar tooth were not unsurmountable, and after initial experimentation it was felt that this procedure was effective. This was confirmed by histological examination. The difficulties associated with isolating the tooth by rubber dam made the success of the procedure hazardous due to possible contamination by saliva.

Considerable material was lost due to difficulties in histological preparation. The curved and extremely small size of the mesio-buccal root of the upper first molar seemed to cause difficulty in orientation of the block and the relevant regions containing the pulpal remnant were often in a direction which did not allow any assessment of the lesion area.

With an improved mounting and sectioning technique it should be possible to use rats in other studies on partial pulpectomy if human or other suitable animals (e.g. miniature pig) are not available.

ANALYSIS OF REACTIONS OBSERVED

The immediate reaction to pulp amputation was that of hyperaemia due to the trauma involved. The nature of the wound would be best classified as a "lacerated type". Overlying the wound was a mass of debris, dressing material and dentine spicules. The latter often seemed to become intimately associated with the wound and sometimes impacted into it. These spicules may act as centres of calcification.

Surface necrosis due to the action of the calcium hydroxide dressing was observable only in some of the longer term cases (e.g. R6 (R)).

In the earlier experimental periods this layer had apparently separated from the underlying pulp and had been lost during histological preparation.

Connective tissue changes in the surface layers of the pulp wound were evident at 3 days (R4 (L)). Fibrosis of the deeper and hyalinisation of the superficial layers occurred producing a so called "dense zone" as described by Nyborg (1955). Further changes in these areas resulted in definable zones by 14 days (R6 (L)). Early calcification in the dense zone occurred, leading to bridge formation. Minimal internal resorption was observed at 14 days along with deposition of small amounts of cellular secondary dentine.

Bridge formation was not a sign of a healthy pulp remnant as the only case showing a calcific bridge (which was not quite complete) had, after showing this initial response, undergone necrosis.

Although the pulp in 4 cases was necrotic, in only one case was this associated with a periapical reaction. This would seem to indicate that death of the pulp was not due to superimposed infection, but to trauma involved during the amputation of the pulp. One could speculate that with longer experimental periods in these cases, ingrowth of the healthy periapical tissues within the canal or closure of the apex by cementoblasts could have occurred.

The reactions described did not differ to any degree from those observed following pulpotomy by Berman & Massler (1958).

CONCLUSIONS

1. Partial pulpectomy in the mesio-buccal root canal of the upper first molar tooth in the rat is possible technically and the method, although presenting considerable operative and histological problems, could be used to study reactions following this therapy.
2. Repair of the pulp following partial pulpectomy and dressing with calcium hydroxide has been shown to occur usually by organisation of the superficial pulp tissue leading to fibrosis, hyalinisation and finally calcification of the amputation zone.

HUMAN MATERIAL

RESULTS - PILOT STUDY

The method of amputation used for this study produced variable surgical wounds. These are summarised in Table IV.

A. Nature of Amputation Wound

(1) "Incised wounds" which may be defined as clean cut linear wounds, were found in 2 of the 6 roots treated (Figs 15-18).

(2) "Lacerated wounds" or irregular ragged wounds occurred in 2 roots, an example of which is shown in Figs. 19-21.

(3) Twisting of the pulpal remnant occurred in two of the above wounds (Figs. 16, 20).

Of the two remaining roots treated, the amputation site was not demonstrated in one and the pulpal remnant was not present in the other. Dentine spicules of varying size were present over each amputation wound. Very large dentine fragments observed in some sections may have been preparation artefacts, e.g. Fig. 15.

B. Inflammatory Reactions in the Pulp Remnant

Extravasation of red blood cells occurred in all the wounds and the remaining pulp tissue showed generalised severe hyperaemia. Some degree of tearing of the deeper pulp tissue occurred in most cases.

TABLE. IV.

PILOT STUDY

No.	Age	Root	Pulpal Remnant Demonstrated Prior to Extraction	Histological Examination Pulpal Remnant Present	Pulpal Remnant Absent	Nature of Wound Incised	Lacerated	Twisted
P1	11	$\overline{4/}$	X	X			X	
P2	11	$/\overline{4}$	X	X		X		
P3	11	4/B	X	X				
P4	11	4/P	X		X			
P5	11	/4 B	X	X			X	X
P6	11	/4 P	X	X		X		X

Fig.15 P.6. /4 Pal. H & E x 100.
SHOWING INCISED WOUND WITH CONSIDERABLE VASCULAR EXTRAVASATION SUPERIORLY

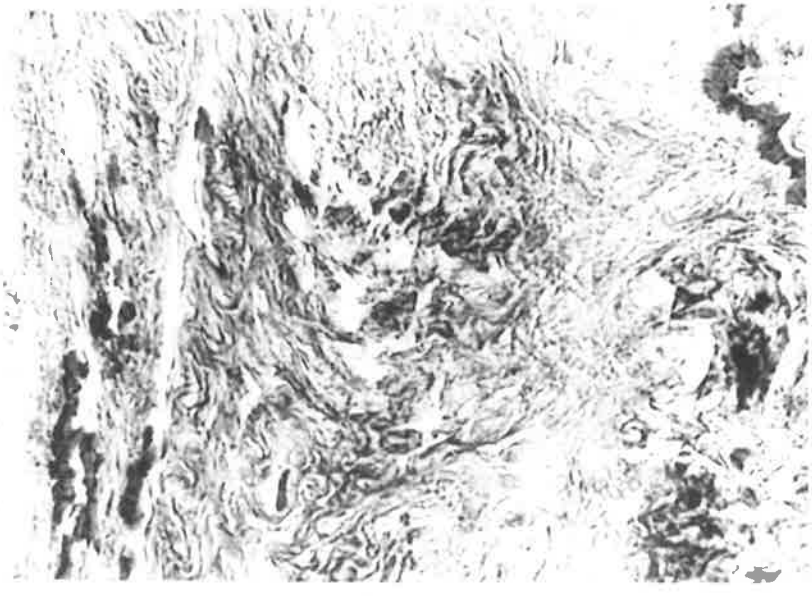


Fig.16 P.6. /4 Pal. MALLORY x 400
SHOWING THE TWISTED NATURE OF THE FIBRES OF THE PULP REMNANT.

Fig.17. P.2. /4 H & E x 100

A RELATIVELY CLEAN AMPUTATION WOUND IS SHOWN. TWISTING OF THE SUPERFICIAL PULP TISSUE IS MINIMAL. MANY DENTINE FRAGMENTS OR SPLINTERS CAN BE SEEN IN THE DEBRIS OVERLYING THE AMPUTATED PULP. ENGORGEMENT OF BLOOD VESSELS AND SOME TEARING OF PULP TISSUE IS EVIDENT.

Fig.18 P.2. / 4 H & E x 100

PULP TISSUE AWAY FROM THE AMPUTATION SITE, SHOWING DILATED BLOOD VESSELS AND INITIAL ACUTE INFLAMMATORY CHANGES.

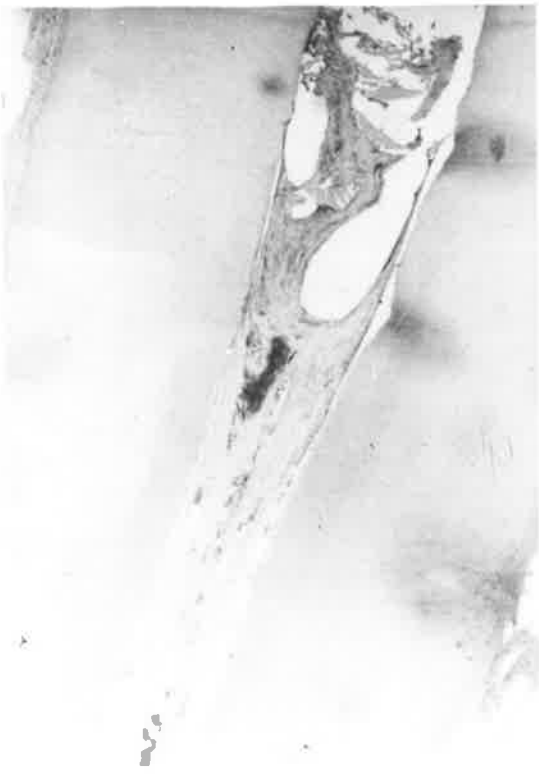


Fig.19 P.5 /4 Buc. H & E x 40.
THE AMPUTATION HAS PRODUCED A LACERATED WOUND

Fig.20 P.5 /4 Buc. H & E x 100
SHOWING TWISTED NATURE OF THE PULPAL WOUND. CONSIDERABLE R.b.c. EXTRAVASATION IS EVIDENT IN THE DEEPER PULP TISSUE

Fig. 21. P.5 /4 Buc. H & E x 100.
SUPERFICIAL SURFACE OF PULPAL WOUND SHOWING PRESENCE OF DENTINE SPICULES.

C. Loss of Pulp Remnant during Extraction

Histological examination revealed only one tooth (P4, Figs 22-29), in which a remnant had been lost. This had been shown by means of the radiopaque Neohydriol, to be present prior to extraction. Debris and the dressing material could be seen up to a point 3mm from the apex of the tooth, while the remainder of the canal was empty except for a retained layer of odontoblasts lining the walls in mostplaces (Figs 26, 28, 29). This layer of odontoblasts did not extend above a level 3mm from the apex.

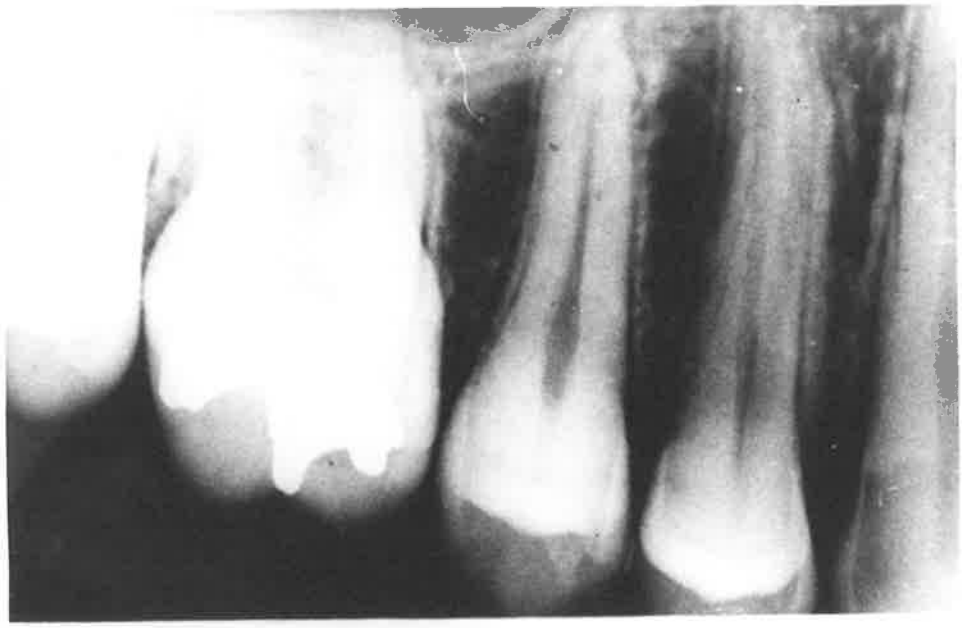


Fig.22. P.4.
PRE-OPERATIVE RADIOGRAPH

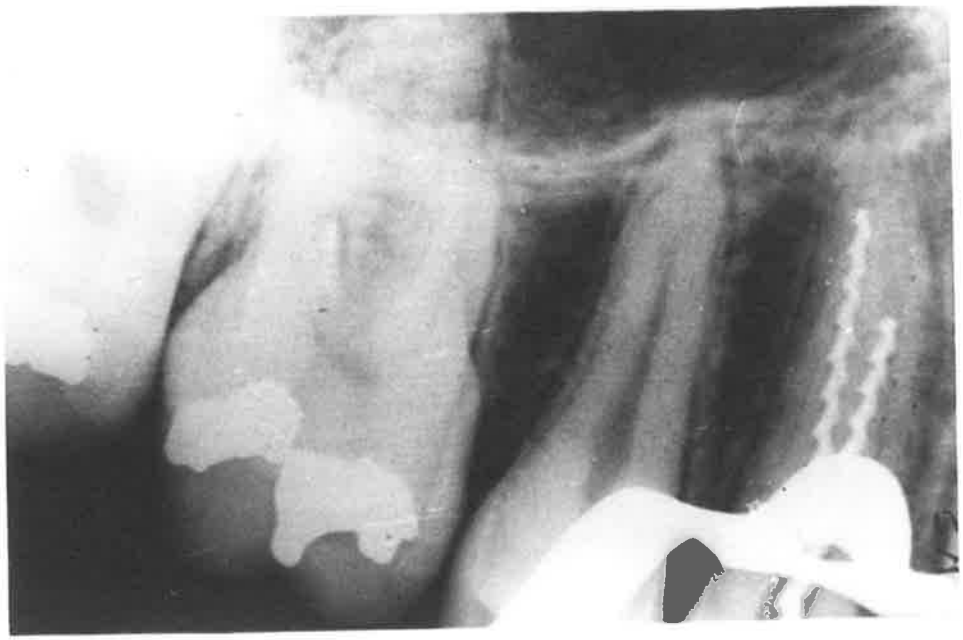


Fig. 23. P.4.
AMPUTATION BY MEANS OF FLATTENED HEDSTROM FILES.

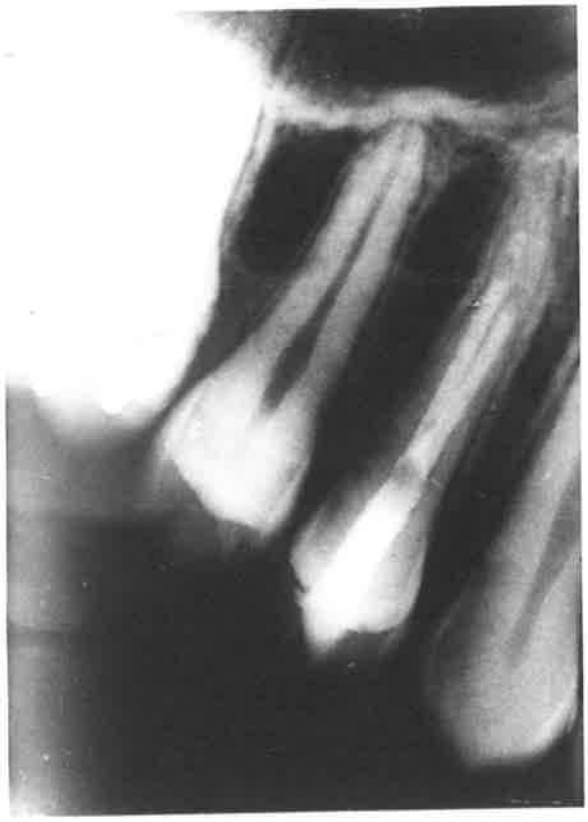


Fig.24. P.4. NEOHYDRIOL SYRINGED INTO THE ROOT CANAL TO THE LEVEL OF THE PULP REMNANTS

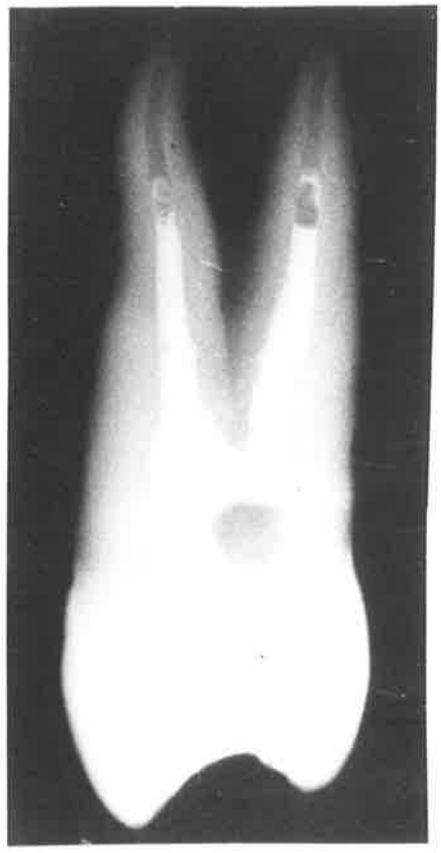


Fig. 25. P.4. POST EXTRACTION RADIOGRAPH SHOWING LEVEL OF NEOHYDRIOL WITHIN THE CANALS.

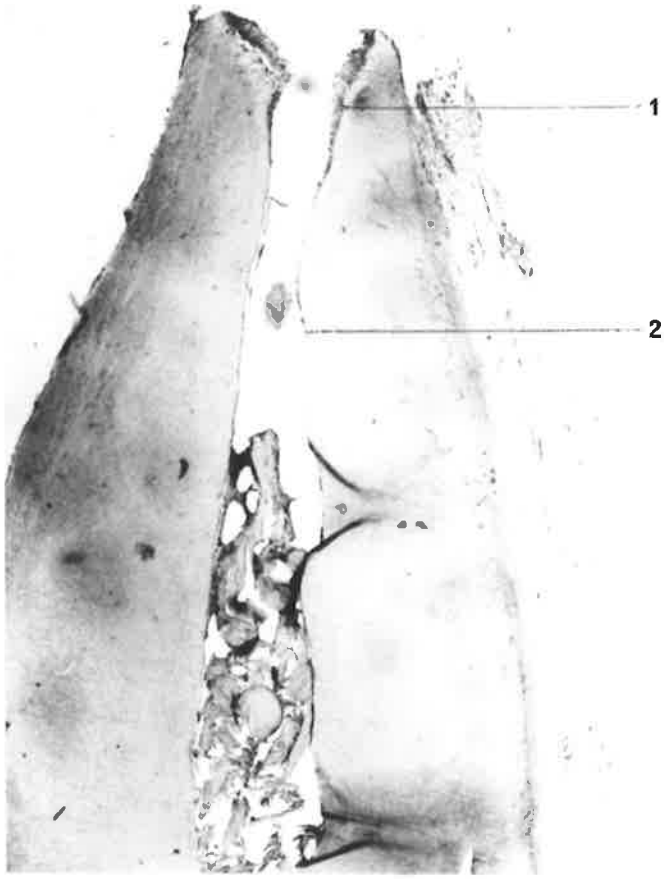


Fig 26. P.4 4/ Buc. H&E x 40
CANAL EMPTY BENEATH MASS OF DEBRIS AND DRESSING MATERIAL, EXCEPT FOR A THIN LAYER OF ODONTOBLASTS (REFER Fig. 28)

Fig 27. P4 4/ H&E x 100.
SUPERFICIAL DEBRIS WITH MANY DENTINE FRAGMENTS.

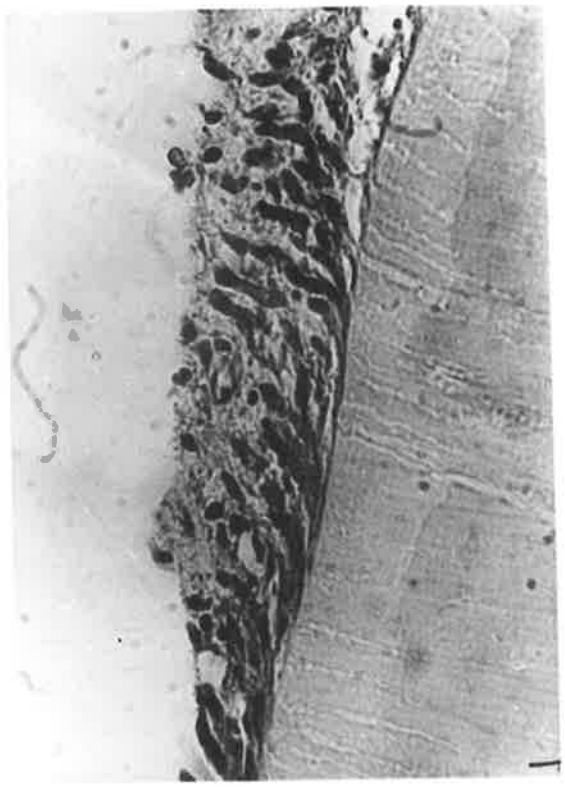


Fig. 28. P.4 4/ buc. H & E x 400
SHOWING RETAINED LAYER OF ODONTOBLASTS WITH SOME ATTACHED PULP TISSUE
(FROM REGION (1) FIG. 26).

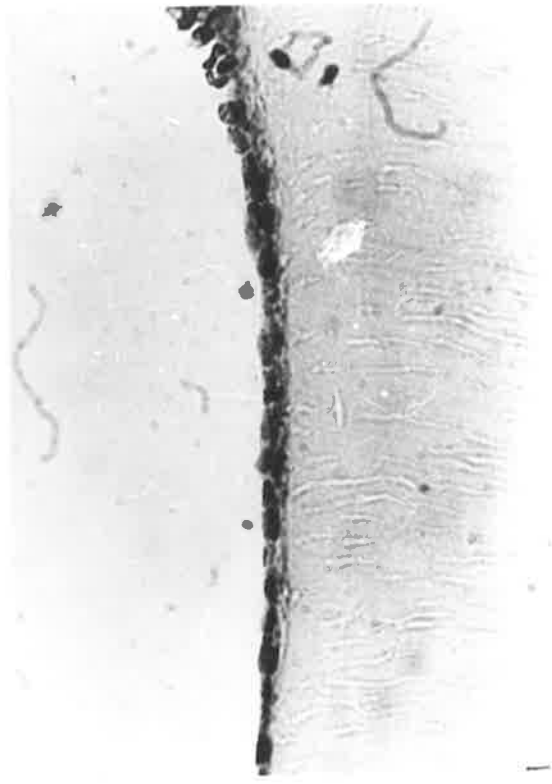


Fig. 29. P.4 4/ buc. H & E x 400
RETAINED LAYER OF ODONTOBLASTS WHICH HAVE BEEN SLIGHTLY DISPLACED DURING PREPARATION (FROM REGION (2) FIG. 26).

DISCUSSION

The rate of healing of uninfected wounds in the body is dependant to a large extent on the type and severity of the injury. An incised wound will heal much more quickly and effectively than a lacerated, contused or punctured wound. In this preliminary study the nature of the wound varied although a standardised technique was employed. As twisting of the pulp remnant occurred to some degree with most remnants, the method of pulp removal is certainly more traumatic than the ideal amputation, which would produce an "incised wound". One could anticipate on general principles that the healing of the above wound would be somewhat slow. Nyborg and Hålling (1963) in a comparative study on amputation instruments also found some twisting and irregularity in the surface wound when using a Hedström file with a flattened tip. The Antheos root canal reamer which was also tested by these workers produced a similar surface wound but with greater twisting of the pulp remnants.

Thus although the flattened Hedström file has been shown to be effective in partially removing the pulp, the nature of the wound is evidence that the ideal instrument for the procedure has still to be developed.

The possibility that the pulp remnant can be lost during extraction has been demonstrated. Presumably the attachment of the remnant to the periapical tissues was sufficiently strong to cause its withdrawal from the canal by those tissues. In such cases, histological examination would reveal an empty canal, and the result would be

assessed as a failure from the point of view of the partial pulpectomy, due to an apparent total pulpectomy.

Teeth with partially developed roots have, by virtue of their anatomical form, no barrier, other than the odontoblastic projections into the dentinal tubules, to help retain the remnant within the canal during extraction. The pulp remnants of teeth under examination at this stage exhibit the first stage of acute inflammation - which is severe hyperaemia. The later inflammatory changes with associated oedema would doubtless further reduce the tissue attachment to the dentinal tubules and so if no other anatomical barriers existed, the chance of losing the remnant during extraction would be increased.

The specimen (P4) which demonstrated the loss of the remnant also showed that a layer of odontoblasts was retained which was confined to the apical 3mm of the canal and attached by the fibrils to the dentinal tubules. The retention of a layer of odontoblasts following the removal of a pulp from a tooth which has been split, has been established by Gotjamanos (1965) and has been used as a method of isolating these cells. Thus the presence of these cells in the region of the remnant may be considered as additional evidence of their loss during extraction.

It seems significant that few reports of partial pulpectomy have been made with treatment times less than 10 days. Those which have been reported (Nygaard Østby, 1939) (Machida 1960, Nyborg 1960, Nyborg & Halling, 1963) have been with adult material with completed apices, thus presenting an anatomical barrier and so preventing the loss of the remnant even in cases of advanced necrosis.

CONCLUSIONS

1. The pulp wound produced with a Hedström file is not consistently a cleanly incised, but is often a lacerated type.
2. Twisting of the pulp remnant occurs to some degree.
3. The pulp remnant, in experimental teeth with partially developed apices, may be retained by the periapical tissues at the time of extraction.
4. If the pulp remnant is lost during extraction a layer of odontoblasts is retained. Their presence in histological specimens may be used as evidence of this loss, however this requires confirmation. In the present investigation this has been provided by the introduction of neohydriol prior to extraction.

RESULTS

1. YOUNG AGE GROUP

A summary of the results of the treatment by partial pulpectomy of 43 roots in patients aged 11-16, is given in Table V. An assessment of the results judged both by Clinico-Radiographic means and histological evidence, has been made.

Cases were considered successful histologically if the pulp remnant was present in a vital inflammation free state. "Doubtful" histological evaluation was accorded to those cases in which some degree of inflammation was present, but the experimental period was inadequate to assess a definite failure or success. No histological or clinical assessment of success was possible in cases where treatment was less than of 7 days duration. Histological assessment was not possible in 12 additional roots, with longer experimental times due either to loss of the remnant at the time of extraction or to technical difficulty in obtaining sections of the relevant area.

Clinico-Radiographic evaluation of success was based on absence of clinical symptoms and radiographic evidence of a normal periapical area. Radiographic evidence of bridge formation, internal resorption, narrowing or obliteration of the canal was also taken into consideration in this evaluation.

Cases were classified as doubtful if radiographic evidence of pathology was negative although clinical symptoms were present.

These clinical and radiographic results are shown in Table V.

TABLE V SURVEY OF EXPERIMENTAL PARTIAL PULPECTOMY IN THE YOUNG AGE GROUP

TABLE V

No.	Age	Root	Clinical State Caries* (0,1,2,3)	Perio** (0,1,2)	Experimental Period	Post Operative Clinical Symptoms Neg.	Thermal	Spontaneous Pain	Percussion	Radiographic Evidence of Periapical Pathology Neg.	Pos.	PULPAL STATE Inflammation Bil	Acute Local.	Acute Gen.	Chronic Local	Chronic Gen.	Calcific Bridging Radiog.	Internal Resorps. Hist.	Internal Resorps. Rad.	Internal Resorps. Hist.	Cellular secondary Dentine Deposition on (a)walls	(b)bridge	Histological Result Fail.	Doubtful	success	Clinico-Radiog. Result Fail.	Doubtful	success
Y23	15	/4B	0	0	Immed									x											-			-
Y24	15	/4P	0	0	"									x											-			-
Y26	13	4/B	0	0	"									-											-			-
Y27	13	4/P	0	0	"									-											-			-
Y28	13	/4 B	0	0	"									-											-			-
Y29	13	/4 P	0	0	"																				-			-
Y 9	14	4/ B	0	0	2	x				x			x												-			-
Y10	14	4/ P	0	0	2	x				x			x												-			-
Y 7	14	4/	0	0	2	x				x					x										-			-
Y 8	14	/4	0	0	7	x				x															-			-
Y11	14	/4 B	0	0	7	x				x						x							x					x
Y12	14	/4 P	0	0	7	x				x						x							x					x
Y16	14	/4 B	0	0	14	x				x						x							x					x
Y17	14	/4 P	0	0	14	x				x																		x
Y18	14	/4	0	0	14	x				x						x				x			x					x
Y38	11	/4	0	0	16	x				x					x								x			x		
Y39	12	4/	0	0	16		x			x						x							x			x		
Y40	11	/4 B	0	0	19		x			x																		x
Y41	11	/4 P	0	0	19		x			x																		x
Y42	11	4/B	0	0	19		x			x																		x
Y43	11	4/ P	0	0	19		x			x																		x
Y13	14	4/	0	0	21	x				x					x									x				x
Y14	14	4/ B	0	0	22	x				x						x				x			x					x
Y15	14	4/ P	0	0	22	x				x																		x
Y21	15	4/ B	0	0	28	x				x		x						x		x		x			x			x
Y22	15	4/ P	0	0	28	x				x					x									x				x
Y25	15	/4	0	0	28	x				x						x								x				x
Y30	16	/4	0	0	46	x				x																		x
Y31	16	4/	0	0	46	x				x																		x
Y19	15	4/ B	0	0	48	x				x																		x
Y20	15	4/ P	0	0	48	x				x		x						x							x			x
Y 6	12	/4	0	0	97	x				x		x						x		x		x			x			x
Y 5	12	4/	0	0	97	x				x									x									x
Y 3	12	/4 B	0	0	102	x				x																		x
Y 4	12	/4 P	0	0	102	x				x																		x
Y 1	12	4/ B	0	0	108	x				x		x													x			x
Y 2	12	4/ P	0	0	108	x				x		x						x				x	x		x			x
Y32	12	4/	0	0	169				x	x						x						x			x		x	
Y33	12	4/ B	0	0	169	x				x		x						x	x	x					x			x
Y34	12	4/ P	0	0	169	x				x					x			x	x									x
Y35	12	/4	0	0	182	x				x		x										x			x			x
Y36	12	/4 B	0	0	182	x				x		x						x							x			x
Y37	12	/4 P	0	0	182	x				x					x			x	x						x			x
TOTAL																							6	7	8	1	1	31

* Caries - 1 = incipient 2 = caries of dentine 3 = caries involving pulp

** Perio - 1 = Gingivitis 2 = periodontitis simplex.

(-) Signifies no assessment possible.

HISTOLOGICAL OBSERVATIONS

Untreated teeth (YC1, YC2 - ♂ 12 years) Figs. 30 - 34.

Both pulps were cellular and contained large and numerous blood vessels. The vascular nature of YC1 was evident, due to some degree of vascular dilatation. The apical pulp showed some relatively thick muscular walled blood vessels (arteries). In the "mid-pulp" these vessels showed considerable branching into arterioles and capillaries. The coronal pulp showed considerable numbers of small blood vessels and the sub-odontoblastic plexus of vessels was evident. The odontoblasts were typically tall columnar cells in the coronal pulp and "mid-pulp" becoming more cuboidal towards the apex (Figs 31 - 34).

Fig. 30. Y.C.I. /4 H & E x 100
UNTREATED TOOTH SHOWING VASCULAR HEALTHY PULP

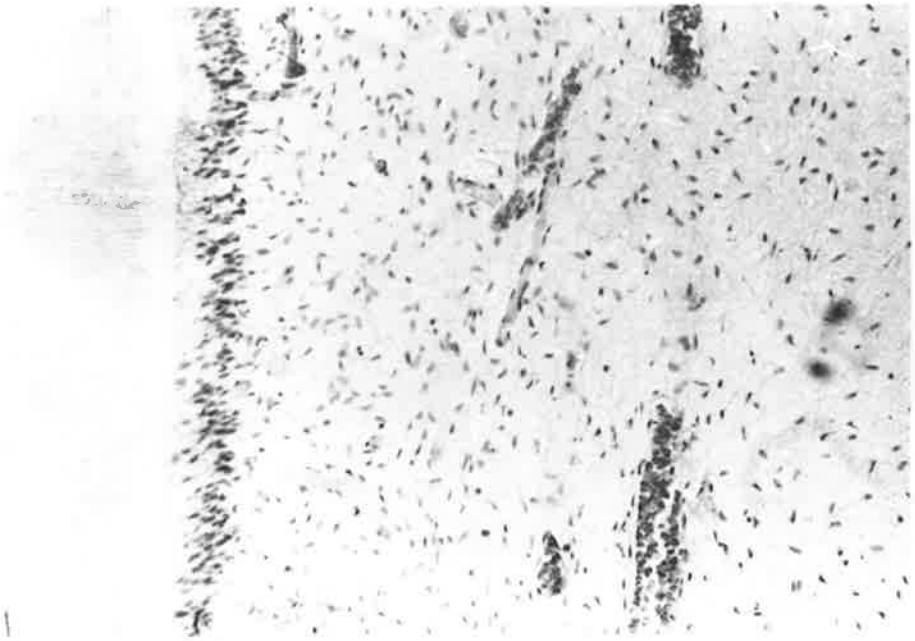


Fig. 31. YC2 $\overline{47}$ H & E x 100
THE PULP TISSUE IN THE "MID PULP". THE ODONTOBLASTS ARE TALL COLUMNAR CELLS IN THIS REGION.

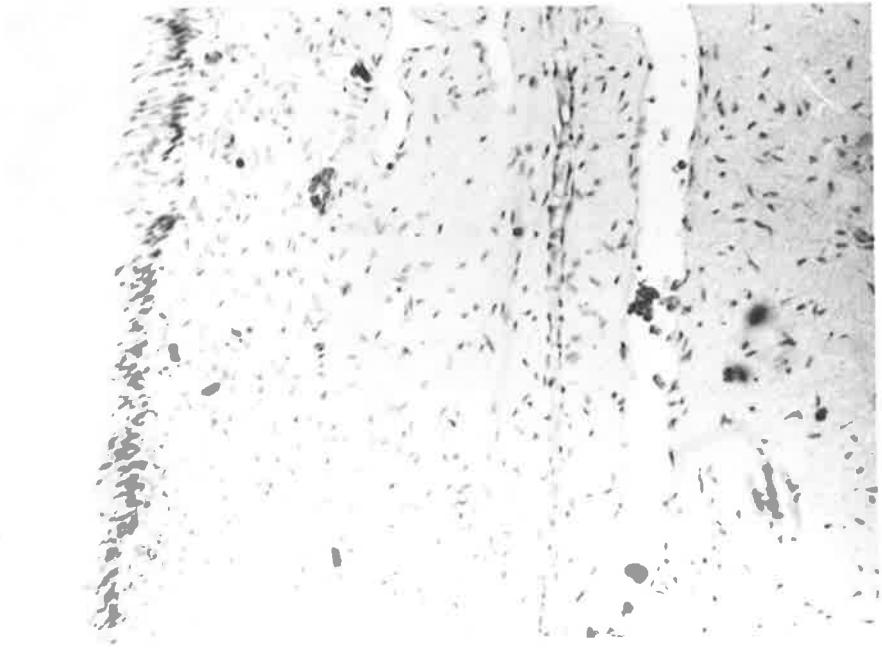


Fig. 32. YC2 $\overline{47}$ H & E x 100
PULP TISSUE TOWARDS THE APICAL REGION. THE ODONTOBLASTS ARE BECOMING MORE CUBOIDAL IN NATURE AND THE PULP LESS CELLULAR.

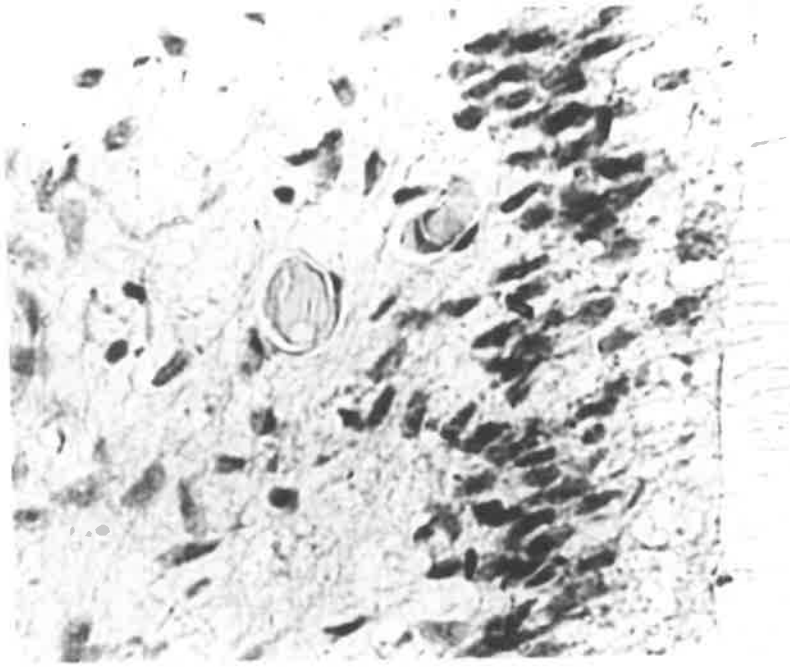


Fig. 33. YC2 $\overline{4/}$ H & E x 400
THE ODONTOBLAST LAYER IN THE "CORONAL PULP".

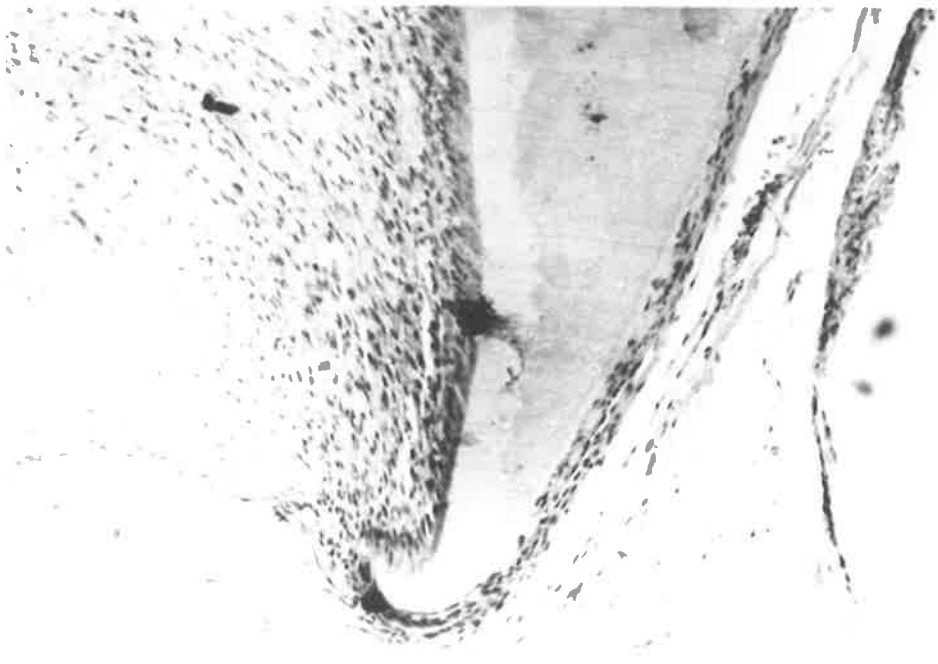


Fig. 34. YC2 $\overline{4/}$ H & E x 100
APICAL PULP TISSUE - THE TRANSITION ZONE
BETWEEN PULP AND PERIODONTAL MEMBRANE.

REACTIONS OBSERVED AT VARYING EXPERIMENTAL TIMES

Immediate reaction: Case Nos Y23, 24 (♀ 15 yrs), 26, 27 (♀ 13 yrs), 28, 29 (♂ 13 yrs). Severe hyperaemia of the entire pulpal remnant was evident in Y23, 24. The nature of the amputation wound was often obscured by a blood clot and what probably was excess dressing material (Fig. 35).

The deeper pulp showed engorged blood vessels and considerable extravasation of red blood cells (e.g. Y24 Fig 36).

Y26, Y27, Y28, Y29 all showed empty canals in the apical 2-3 mms except for a retained layer of odontoblasts (Figs 37-39).

2 Days: Case Nos Y7, 9, 10 (♂ 14 yrs).

Lesion Area: Superficial necrosis of the pulp was evident in Y7 (Fig 40) and Y10. Y9 did not show the amputation site in the prepared sections. The amputation wound was relatively clean in Y7, but in Y10 it was severely lacerated and dentine spicules could be seen deeply impacted into the pulp remnant.

Deeper Pulp Tissue: All cases showed normal inflammatory changes, the vascular response being one of dilatation with an exudate consisting of neutraphils, mononuclear cells and macrophages.

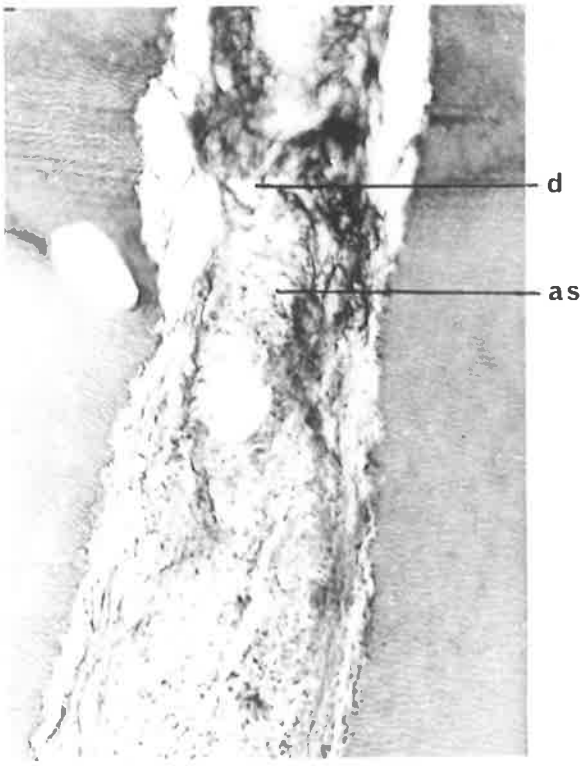


Fig.35 Y24 /4 Pal. V.G.x 100 E.P. IMMED.

THE AMPUTATION SITE (A.S.) IS OBSCURED TO A LARGE DEGREE BY EXTRAVASATED R.B.Cs. THE DRESSING MATERIAL AND DEBRIS IS MORE CLEARLY SHOWN (D).

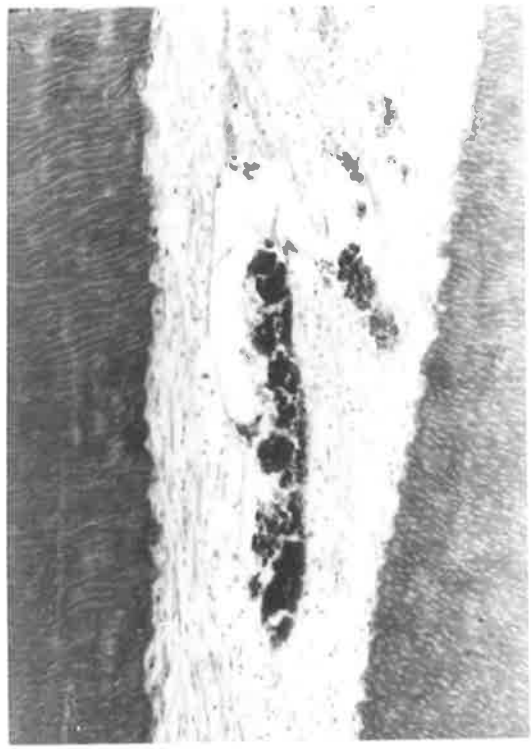


Fig.36 Y24 /4 Pal. V.G x 100 E.P. IMMED.

CONSIDERABLE DILATATION OF THE DEEPER BLOOD VESSELS HAS OCCURRED. ALONG WITH EXTRAVASATION OF MANY R.B.Cs.

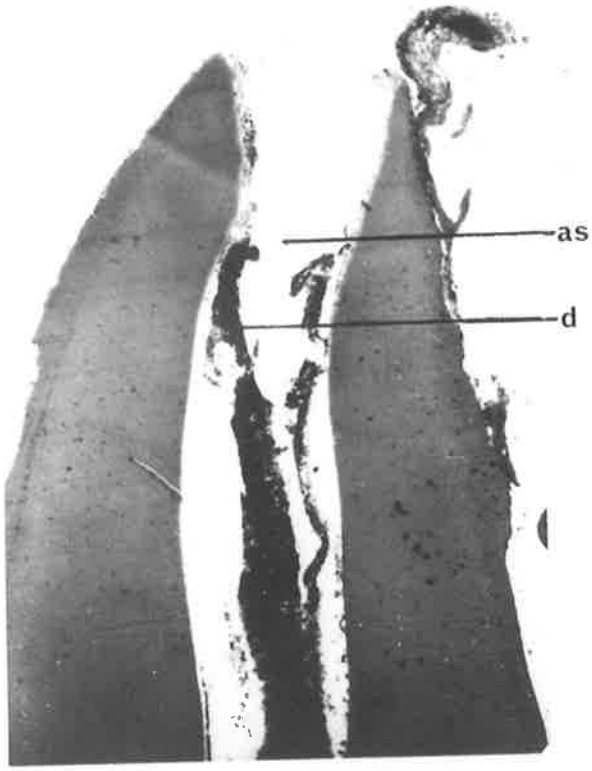


Fig.37 Y29. /4 Pal. H & E x 40
E.P. IMMED.

PULP REMNANT LOST AT TIME OF EXTRACTION. CANAL IS EMPTY BELOW DRESSING MATERIAL (d) EXCEPT FOR AN INTACT LAYER OF ODONTOBLASTS. AMPUTATION SITE SHOWN (a.s.)

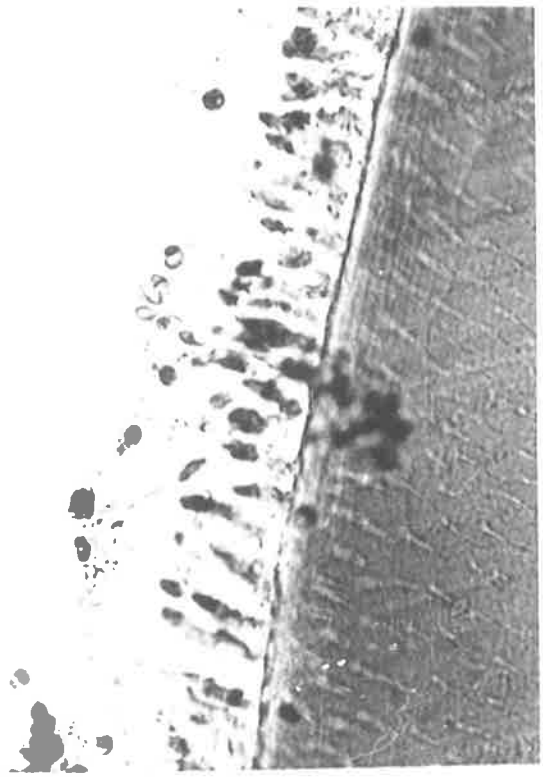


Fig. 38. Y29 /4 Pal.
V.G. x 400 E.P. IMMED.

THE INTACT LAYER OF ODONTOBLASTS

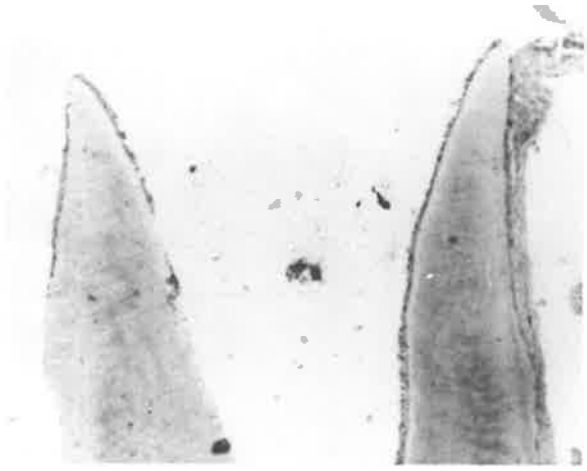


Fig. 38. Y26 4 / buc H & E x 40

E.P. IMMED.
THE CANAL CONTAINS ONLY A LAYER OF ODONTOBLASTS

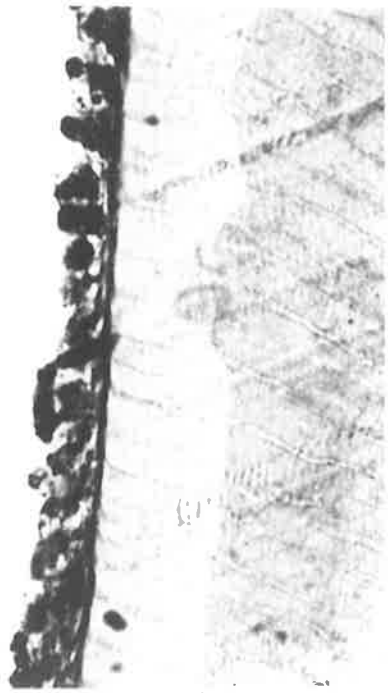


Fig. 39. Y26 4/ buc H & E x 400

A LAYER OF RETAINED ODONTOBLASTS - TYPICALLY CUBOIDAL IN SHAPE.

Fig.40 Y7 4/ Pal. H & E x 100 E.P. 2 DAYS

THE AMPUTATION SITE IS RELATIVELY WELL DEFINED. SUPERFICIAL NECROSIS (S.N.) HAS OCCURRED WHILE THE DEEPER PULP TISSUE SHOWS SOME VASCULAR DILATATION AND A FEW EXTRAVASATED R.b.cs.

7 Days: Case Nos Y8, Y11, Y12 (♂ 14 yrs).

All specimens showed an unfavourable response, with the pulp remnant either in a state of acute generalised inflammation (Y12) or totally necrotic with little pulp tissue remaining in the canal (Y8, Y12).

14-16 Days: Case Nos Y16, Y17, Y18 (♂ 14 yrs) Y38, Y39 (♀ 11 yrs).

Y38 (Fig 41).

Lesion Area: A broad necrotic zone was produced which showed some demarcation from the underlying pulp. The necrotic zone and overlying debris and dentine spicules could not be differentiated. Some separation of the necrotic zone from the underlying tissue was evident. (Possible preparation antefact).

Deeper Pulp Tissue: The underlying pulp appeared fibrotic. A very mild localised plasma cell infiltration was evident in the deeper pulp tissue. The prognosis was judged as doubtful. Y16, Y18, Y39 showed chronic inflammation - total in the case of Y39 and localised in Y16, Y18 (Fig 42). Y17 was apoilt during laboratory preparation.

19 - 22 Days: Case No Y40, Y41, Y42, Y43, Y13, Y14, Y15 (♀ 13 years).

Lesion Area: A well defined zone of necrosis was present in Y13, (Figs 43) and Y14 (Fig 45). Islands of dystrophic calcification were also observed in the amputation zone Y14, but in addition the underlying pulp in this case had undergone necrosis with an associated chronic inflammation in the periapical tissues (Figs 46, 47). Beneath the necrotic zone in Y13, the pulp tissue showed

hyalinisation. Chronic inflammatory cells were also present in a localised area.

Deeper pulp tissue: The deeper pulp tissue showed considerable fibrosis (Fig 44). Y13 was classified as of doubtful prognosis; Y14 as a failure. The sections produced from Y15, Y40, Y41, Y42, Y43 were such that no reasonable histological assessment was possible.

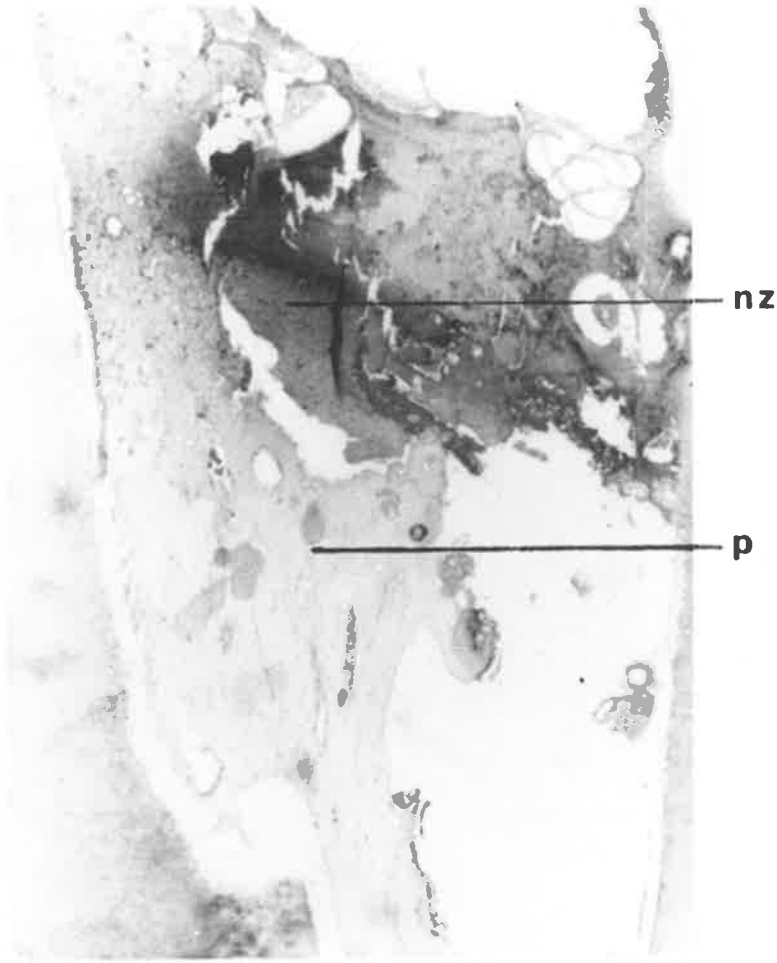


Fig. 41. Y38 /4 H & E x 100 E.P. 16 days

THE AMPUTATION WOUND IS BROAD AND SHOWS A WIDE NECROTIC ZONE (nz) WHICH HAS SEPARATED FROM THE UNDERLYING FIBROTIC PULP (P) CONSIDERABLE TISSUE DISPLACEMENT HAS OCCURRED FROM THE CANAL WALL.

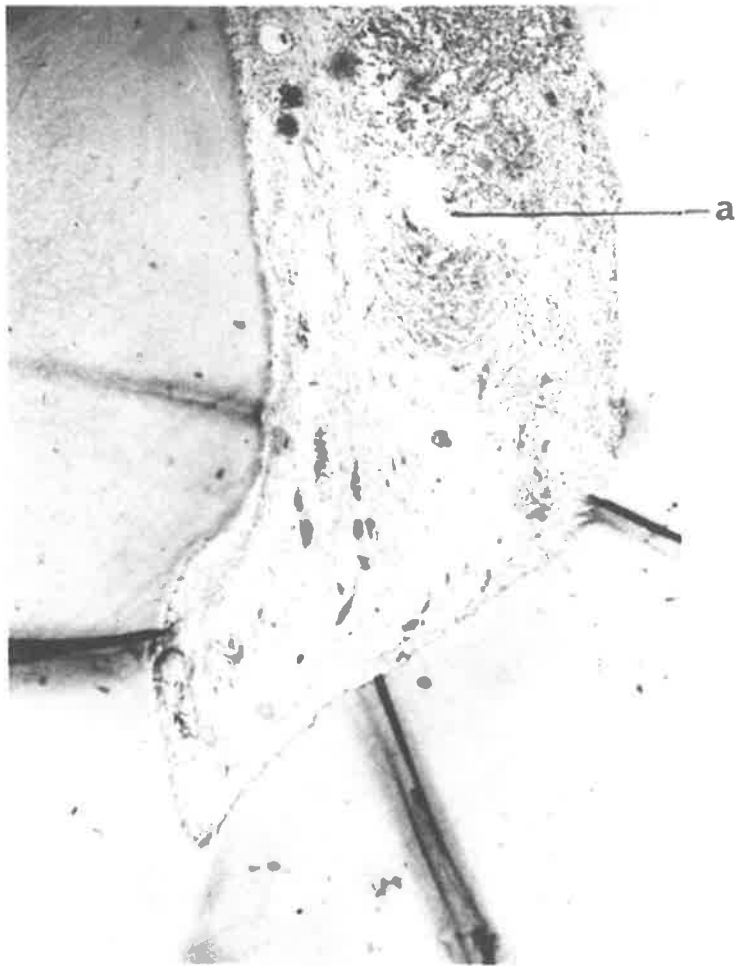


Fig 42. Y 18 / 4 H & E x 100

E.T. 14 days
ABSCESS FORMATION WAS DEVELOPED
INTRA-PULPALLY WHILE THE
SURROUNDING TISSUES SHOW
CONSIDERABLE CELLULAR INFILTRATION.
DEEPER PULP TISSUE IS NORMAL.

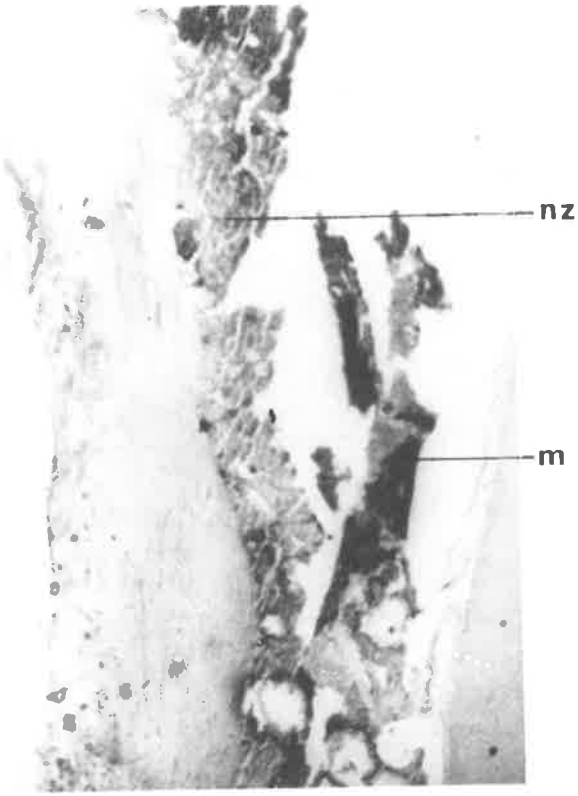


Fig 43. Y13 $\overline{47}$ H & E x 100

E.P. 21 days
AN ACUTELY ANGLED AMPUTATION WOUND WITH OVERLYING MEDICAMENT LAYER (M) NECROTIC ZONE (N.3) AND HYALINISED PULP TISSUE.

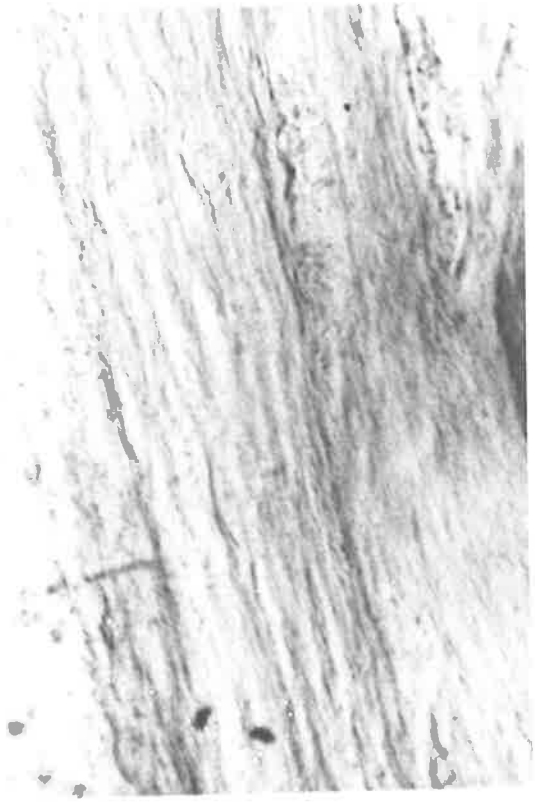


Fig. 44. Y13 $\overline{47}$ V.G x 400

E.P. 21 days
APICAL FIBROSIS

Fig. 45 Y14 4/ Buc. H&E x 100. E.P. 22 days.
LESION AREA SHOWING MEDICAMENT LAYER, NECROTIC LAYER WITH ENCLOSED DENTINE SPICULES, ISLANDS OF DYSTROPHIC CALCIFICATION AND UNDERLYING NECROTIC PULP

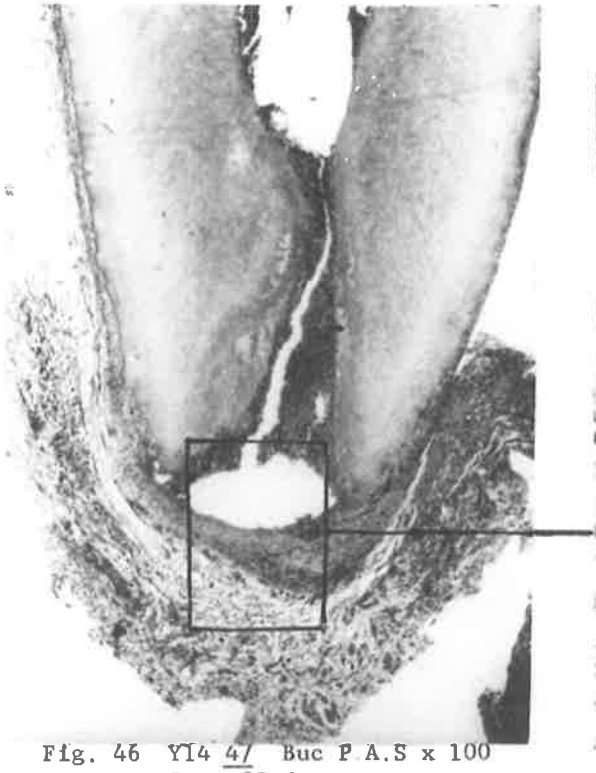


Fig. 46 Y14 4/ Buc P A.S x 100
E P. 22 days
PULP REMNANT TOTALLY NECROTIC
PERIAPICAL TISSUES SHOW
FIBROUS ENCAPSULATION OF
NECROTIC AREA

Fig 47. Y14 4/ Buc. P.A.S. x 400
FRAMED AREA SHOWING FIBROUS NATURE OF PERIAPICAL TISSUE CAPSULE ADJOINING ABSCESS CAVITY.

28 days: (Case No. Y21, Y22, Y25 (♀ 15 years))

Lesion Area: A moderately well formed calcific bridge which seemed complete was present in Y21 (fig 48. 49). Y25 (fig 50) also showed calcific material in the superficial portion of the pulp remnant, but it could not be demonstrated as a definitely intact layer. The calcific material in both cases did not show any signs of tubule formation. Dentine spicules were found in the medicament and superficial debris layers in Y21.

The pulp beneath the bridge showed hyalinisation, and appeared less cellular than normal.

Deeper pulp tissue: The deeper pulp in both Y21 and Y25 appeared normal although there had been some displacement of tissue during preparation of Y21. The pulp remnant in Y22 was chronically inflammed and showed no signs of repair (Figs 52, 53).

46 - 48 days: Case Nos Y30, Y31 (♂16 yrs), Y19, Y20 (♀ 15 yrs).

Lesion Area: Y20 and Y30 showed evidence of calcific material in the superficial layer.

In Y20 (Figs 54-56), dentine spicules had been packed over the pulp stump, beneath the well defined amputation notch. Some dentine spicules had become deeply impacted into the pulp.

The layer of superficial necrosis was ill-defined.

Deeper Pulp Tissue: The underlying pulp tissue appeared somewhat degenerative. There was however, no sign of inflammatory cells. Y31 and Y19 were spoilt during processing.

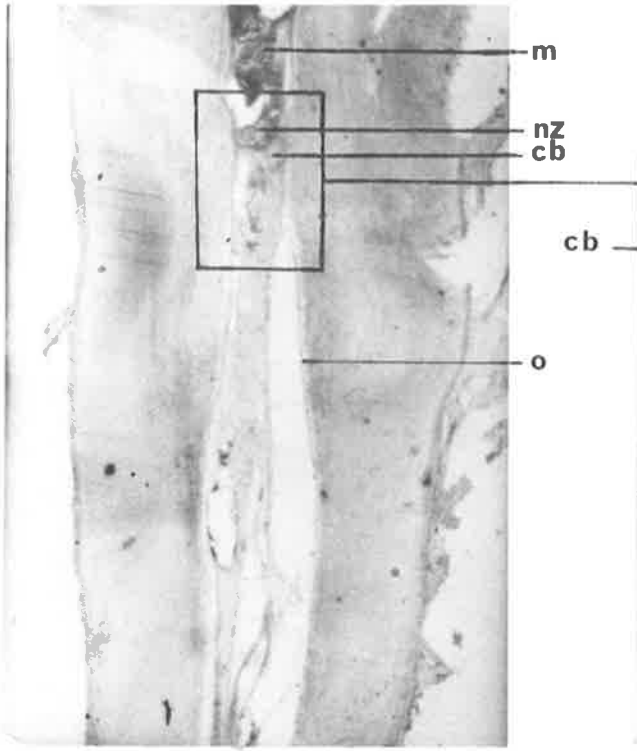


Fig. 48. Y21 4/ Buc. H&E x 40 E.P. 28 days

PULP REMNANT HAS BEEN COVERED BY A CALCIFIC BRIDGE (c.b) MEDICAMENT (m) AND NECROTIC LAYER (n.z) SHOWN. PULP TISSUE DISPLACED DURING PREPARATION AS INTACT LAYER OF ODONTOBLASTS (o) STILL LINE THE CANAL.

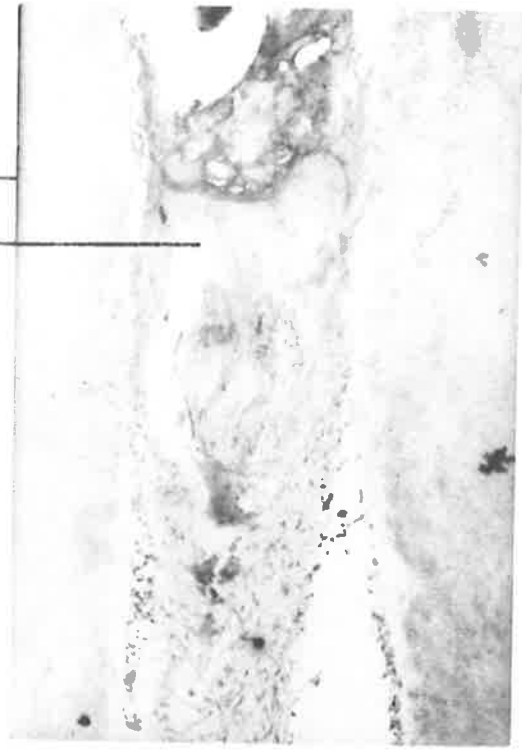


Fig 49. Y21 4/ Buc. H&E x 100

FROM FRAMED SECTION (Fig 48) THE IRREGULAR BAND OF CALCIFIC MATERIAL (c.b) EXTENDS THE WIDTH OF THE ROOT CANAL AND DEEPLY INTO THE PULP.

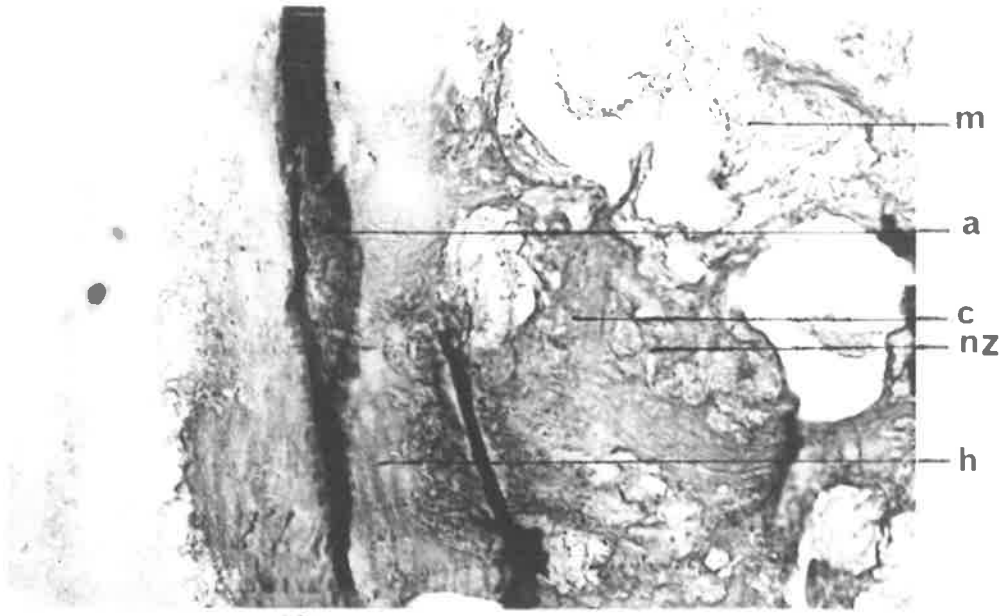


Fig 50. Y25. $\overline{/4}$ H & E x 100 E.P. 28 days.
AN IRREGULAR INCOMPLETE CALCIFIC LAYER IS PRESENT (c) UNDER THE MEDICAMENT LAYER (m) NECROTIC ZONE (n3) HYALINISED PULP (h), ARTEFACT (a).

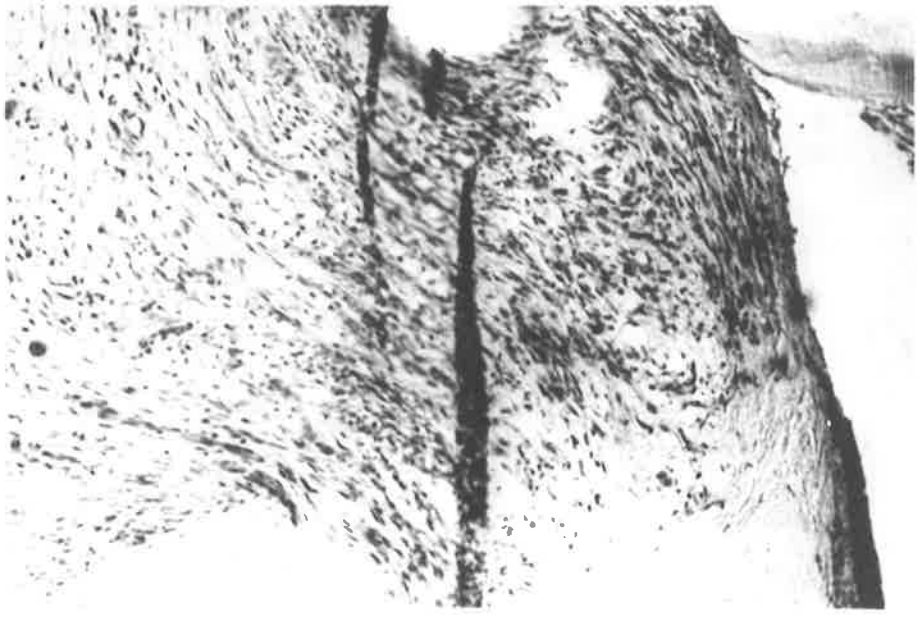


Fig 51. Y25 $\overline{/4}$ H & E x 400 SHOWING THE PULP AT THE APICAL FORAMEN. EXTRAVASATION OF R.b.c's HAS PROBABLY OCCURRED DURING EXTRACTION.

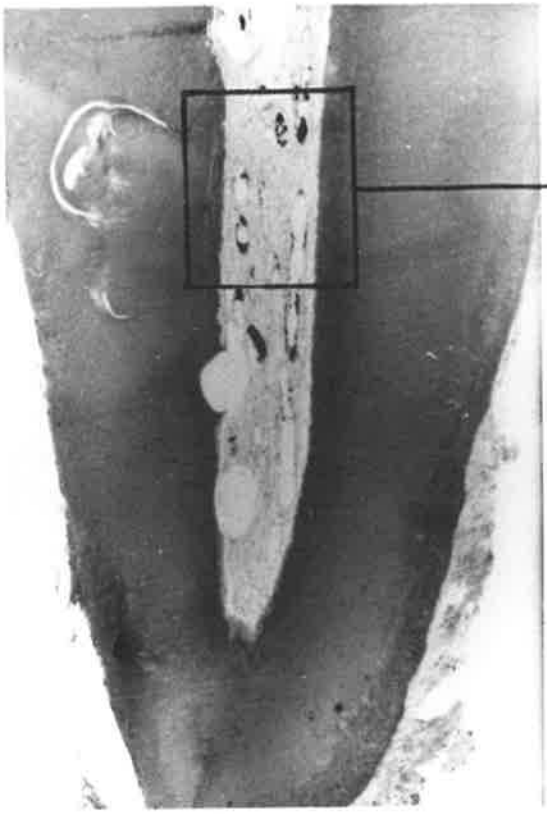


Fig. 52.Y22 4/ Pal H & E x 40
E.P. 28 days.

VITAL PULPAL REMNANT
PRESENT SHOWING MANY
DILATED BLOOD VESSELS

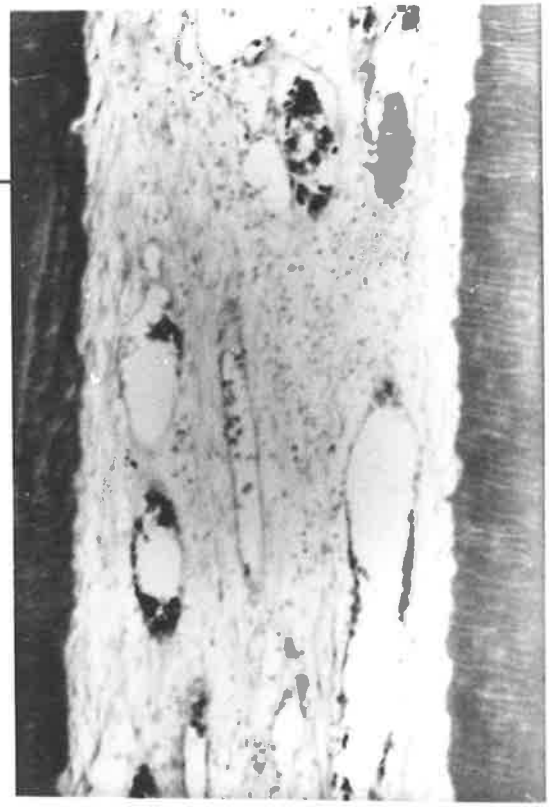


Fig 53. Y22 4/ Pal H & E x 100
SHOWING THE FRAMED AREA
IN FIG 52. PULP CONTAINS
DILATED BLOOD VESSELS AND
EXTRAVASATED R.b.c's.
IN ADDITION MANY NEUTRAPHILS
ARE PRESENT.

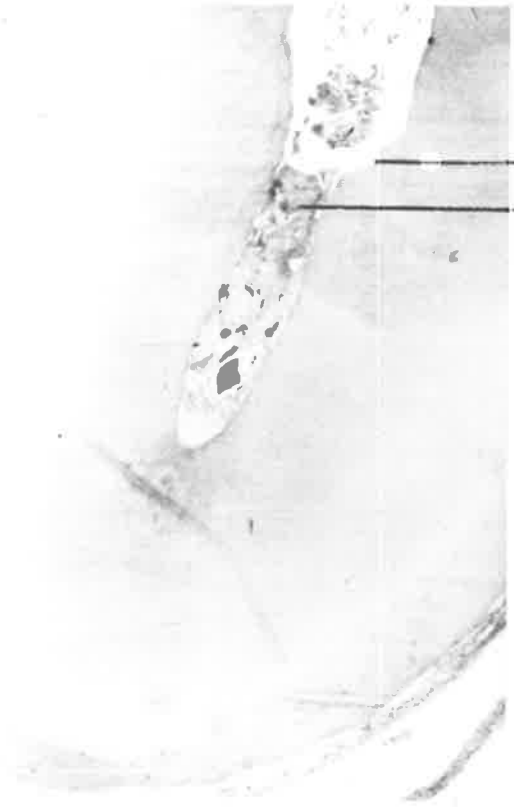


Fig 54. Y20 4/ Pal V.G.x 40
E.P. 48 days

AMPUTATION NOTCH WELL SHOWN (n) CONSIDERABLE PACKING OF DENTINE SPICULES (d.s) HAS OCCURRED

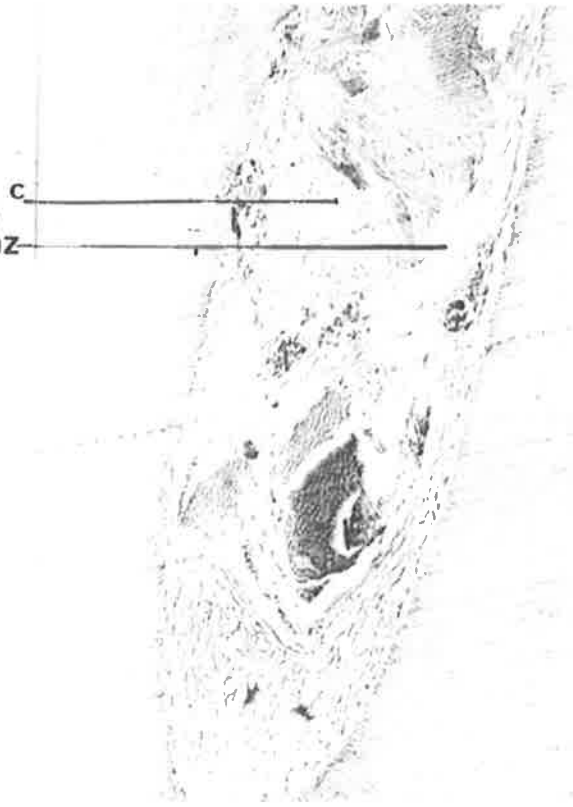


Fig. 55. Y20 4/ Pal. V.G. x 100.

DENTINE SPICULES CAN BE SEEN DEEPLY IMPACTED INTO THE PULP TISSUE. ILL DEFINED NECROTIC ZONE (n.3) IS PRESENT, AND SOME IRREGULAR ISLANDS OF CALCIFICATION (c).

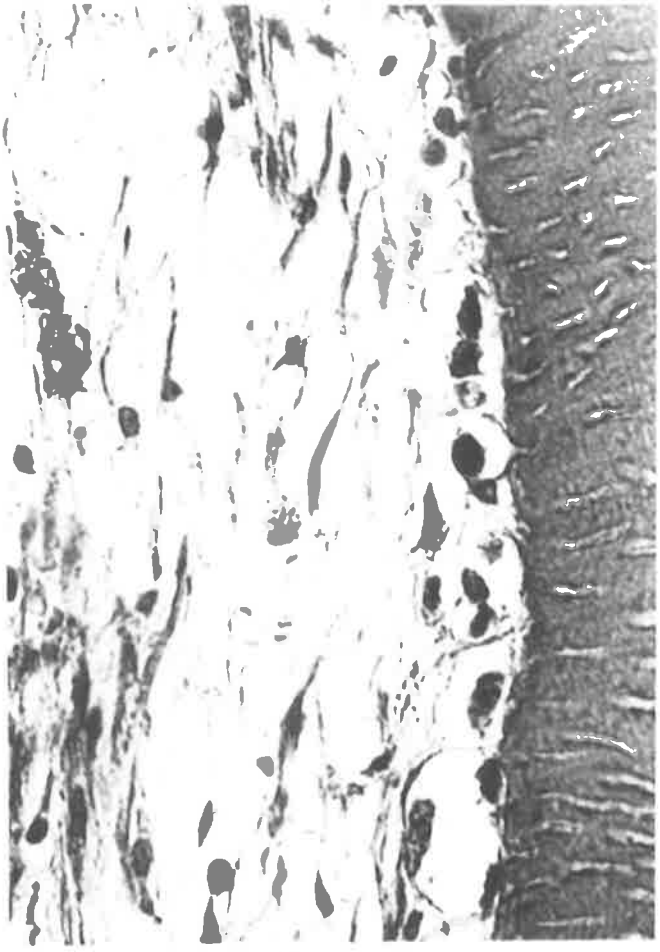


Fig 56. Y20 4/ Pal. H & E x 100 E.P. 48 days

DEEPER PULP TISSUE. CUBOIDAL SHAPED ODONTOBLASTS AND PULP SHOWING VACUOLISATION.

97 - 108 days: Case Nos Y1 - 6 (♀ 12 years)

Lesion area: Calcific bridging - although not conclusively proven (Figs 57, 58) appeared to have occurred in Y2. Incomplete bridging occurred in Y1 and small calcific areas were present in Y6. (Fig. 60).

The apparent bridge shown in Y2 (Figs 57, 58) may have been due to the angulation of cutting and so cellular secondary dentine from the walls may appear to be included in the bridge. However, above this material there appeared to be a wide calcific layer which did not resemble dentine or cellular secondary dentine, and above this layer was a layer of superficial necrosis and debris. Some bridging whether complete or not thus appeared to have occurred. The lesion area in Y6 (Figs 59,60,63, 64) was characterised by a broad necrotic zone, well demarcated from the underlying healthy but somewhat fibrous pulp. Haematoxylin and Eosin staining (Figs 60, 61, 62) revealed that this broad necrotic zone possibly contained a few islands of calcific material.

Deeper Pulp Tissue: The underlying pulp in Y6 was free of inflammatory cells. Resorption of the walls was pronounced. No multinucleated giant cells were present in these areas. Some isolated dentine spicules were present in the pulp tissue. Van Gieson's stain demonstrated generalised fibrosis of the pulp, but there was no sign of any connective tissue changes, immediately below the necrotic zone, which could indicate organisation leading to hyalinisation and calcification. There was an increase in the amount of fibrous tissue on the peripheral aspects of the pulp. Relatively loose connective tissue was present centrally (Fig 64).

Using Mallory stain, the blue staining fibrous section of the pulp was well defined from the necrotic layer. In one central area below the necrotic zone the tissue was less fibrous than the rest of the pulp. Dense fibrous tissue possibly undergoing calcification was present near the root canal walls. P.A.S. staining however revealed little activity in respect to mucopolysaccharide formation, but silver stain (Lillie's) showed reticulin formation in the region of the newly formed cellular secondary dentine (Figs 65 , 66). The reticulin fibres could be seen extending in a fashion similar to Korff's fibres.

Internal resorption: was present in Y6 (Fig 60) and had occurred in Y2 before repair with cellular secondary dentine. Cellular secondary dentine deposition, both on previously resorbed and on normal dentine surface had occurred in Y2, Y6 (Figs 57, 58, 60, 62, 65, 66). Y3, Y4 have been removed from the study due to contamination of the root canals following the loss of the temporary filling material.

Y5 - At time of amputation it was considered that a total pulpectomy had been produced. Histological examination confirmed this. The adherent periapical tissues were free of inflammation.

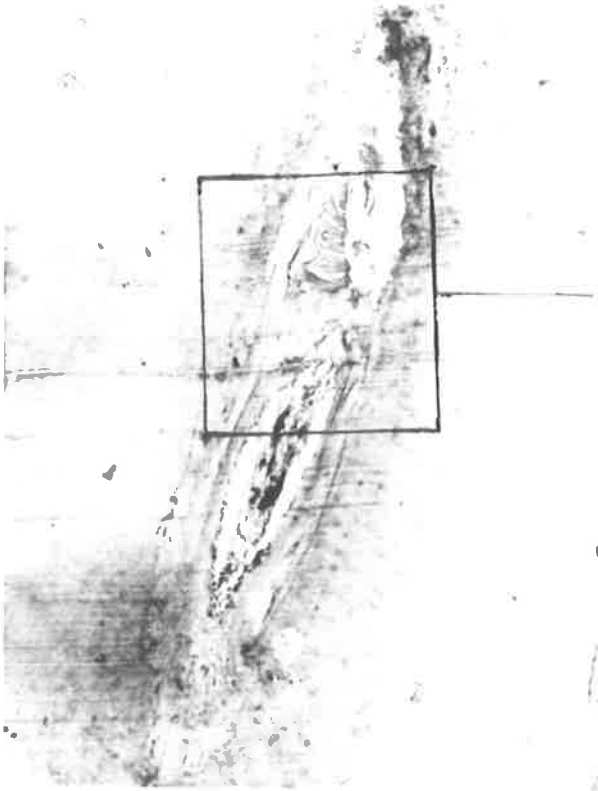


Fig 57 Y2 4/ Pal H & E x 40
E.P. 108 days

THE RETAINED PULP APPEARS TO BE COVERED SUPERIORLY BY A CALCIFIC BRIDGE CONSISTING OF A LAMINATED CALCIFIC LAYER AND A LAYER OF SECONDARY CELLULAR DENTINE. BRIDGE MAY NOT BE COMPLETE (REFER TEXT)

Fig 57(a). Y2 4/ Pal. H&E x 100

FRAMED AREA. PULP HAS BEEN DISPLACED FROM NORMAL POSITION REFER FIG 58 FOR FURTHER SECTION OF SAME AREA

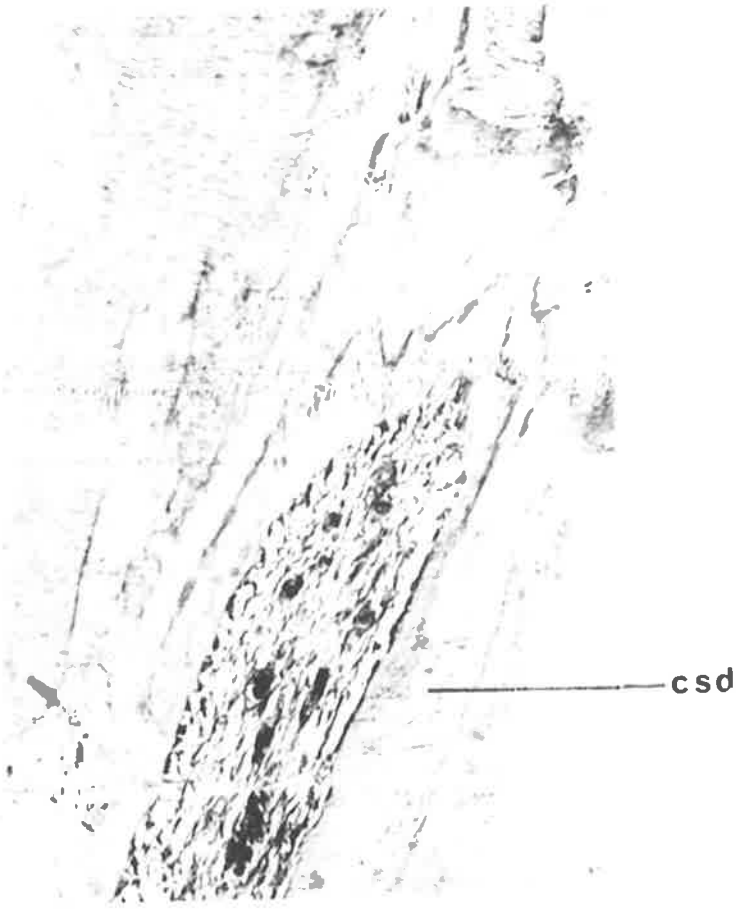


Fig 58. Y2 4/ Pal. H & E x 100

FRAMED AREA FIG 57 (FURTHER SECTION) SHOWING RELATIVELY FIBROUS NATURE OF THE PULP. THE ROOT CANAL HAS CELLULAR SECONDARY DENTINE (c.s.d) DEPOSITION ON ITS WALLS AND APPEARING TO LINE THE CALCIFIC BRIDGE SUPERIORLY.

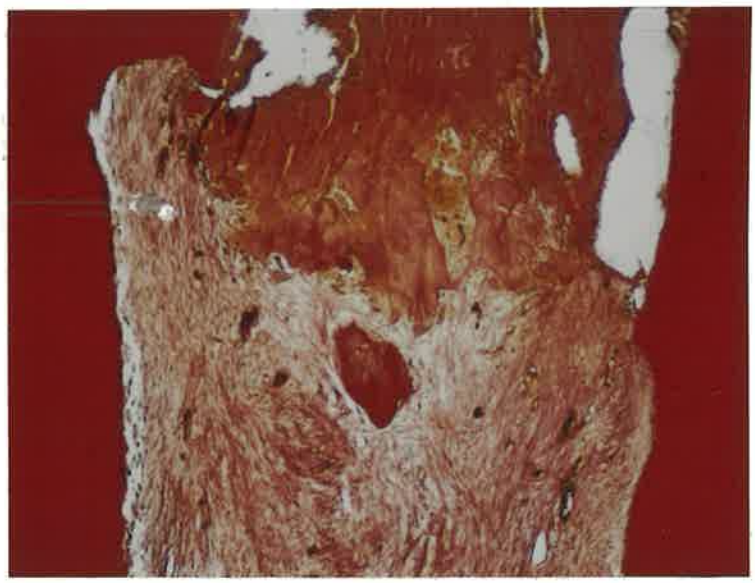


Fig 59. Y6 $\overline{/4}$ V.G. x 100 E.P. 97 days.

BENEATH THE WELL DEFINED NECROTIC LAYER, THE PULP SHOWS VARYING DEGREES OF FIBROSIS, BEING MOST MARKED PERIPHERALLY AND LEAST MARKED CENTRALLY. (REFER FIG 64 FOR HIGHER MAGNIFICATION) RESORPTION OF THE CANAL WALL IS EVIDENT.

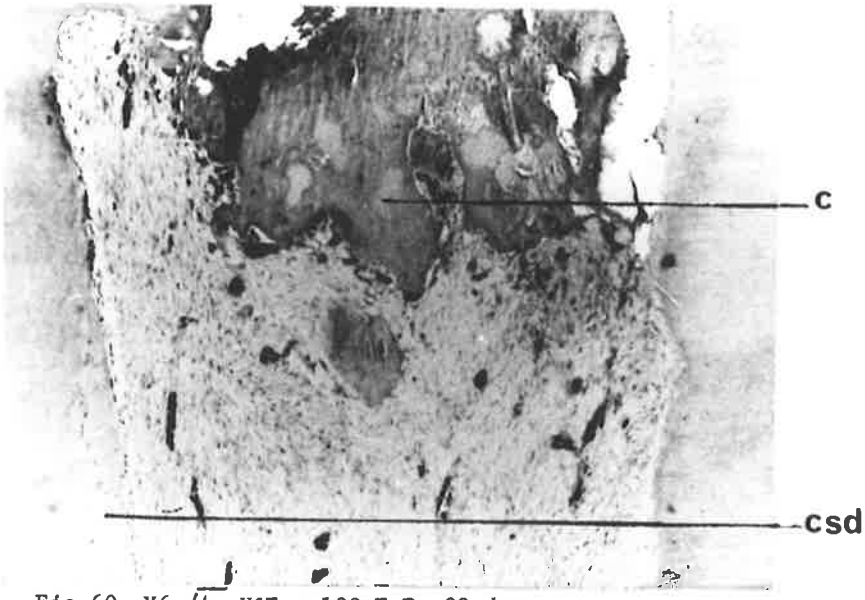


Fig 60. Y6 /4 H&E x 100 E.P. 99 days.
THE LESION AREA RESORPTION OF THE CANAL WALLS HAS OCCURRED GENERALLY. REPAIR BY CELLULAR SECONDARY DENTINE IS SEEN IN SOME AREAS (csd) THE HEALTHY PULP TISSUE IS CLEARLY DEMARCATED FROM THE OVERLYING NECROTIC ZONE BUT NO BRIDGE HAS BEEN FORMED. SMALL ISLANDS OF POSSIBLE CALCIFIC MATERIAL ARE SHOWN (c)

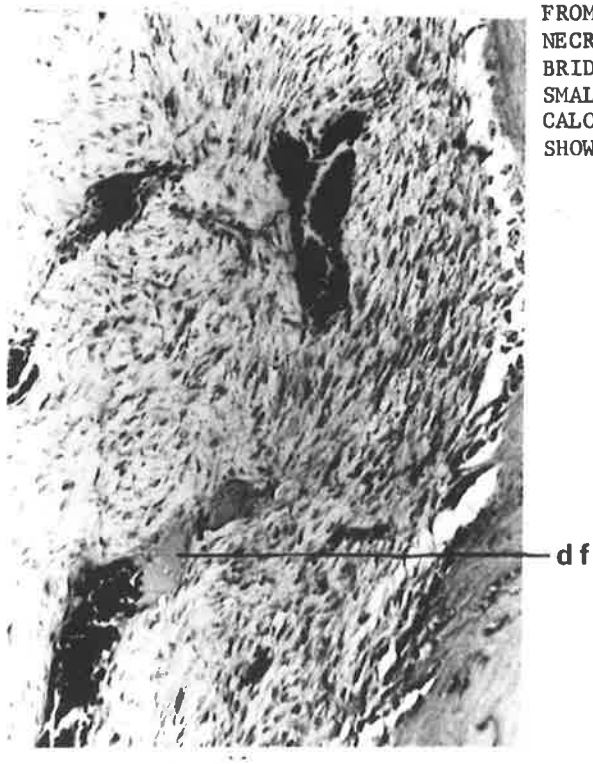


Fig 61 Y6 /4 H&E x 400.
FRAMED AREA PULP SHOWS HEALTHY FIBROUS TISSUE IN ADDITION TO NORMAL PULP CELLS. TWO DENTINE FRAGMENTS (d f) ARE SEEN WITHIN THE PULP

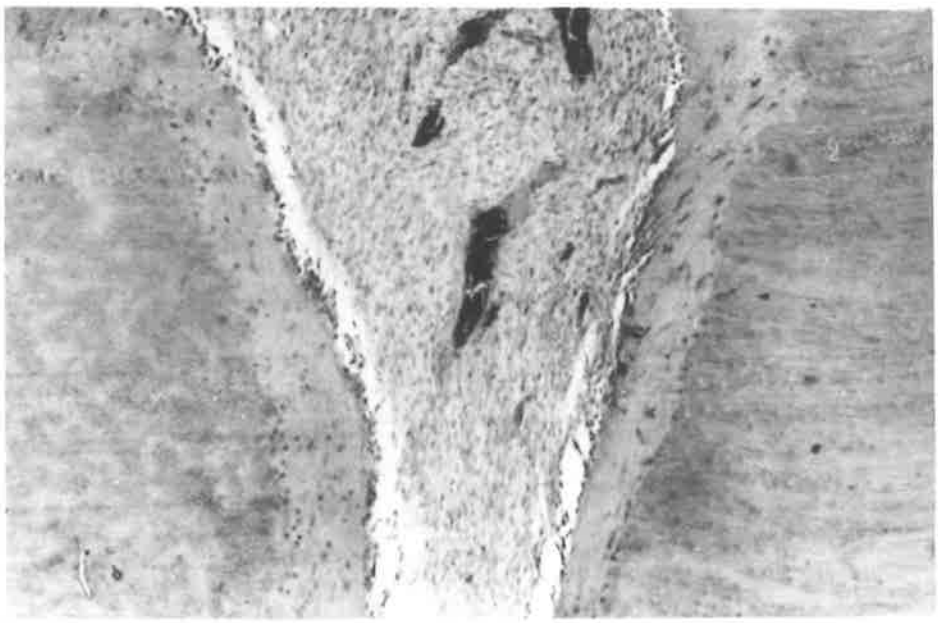


Fig 62. Y6 /4 H & E x 100 E.P. 97 days.

THE PULP IN THE REGION OF THE APEX IS FREE OF INFLAMMATORY CELLS. A WIDE BAND OF CELLULAR SECONDARY DENTINE IS PRESENT ON THE CANAL WALLS REPLACING PREVIOUSLY RESORBED DENTINE.

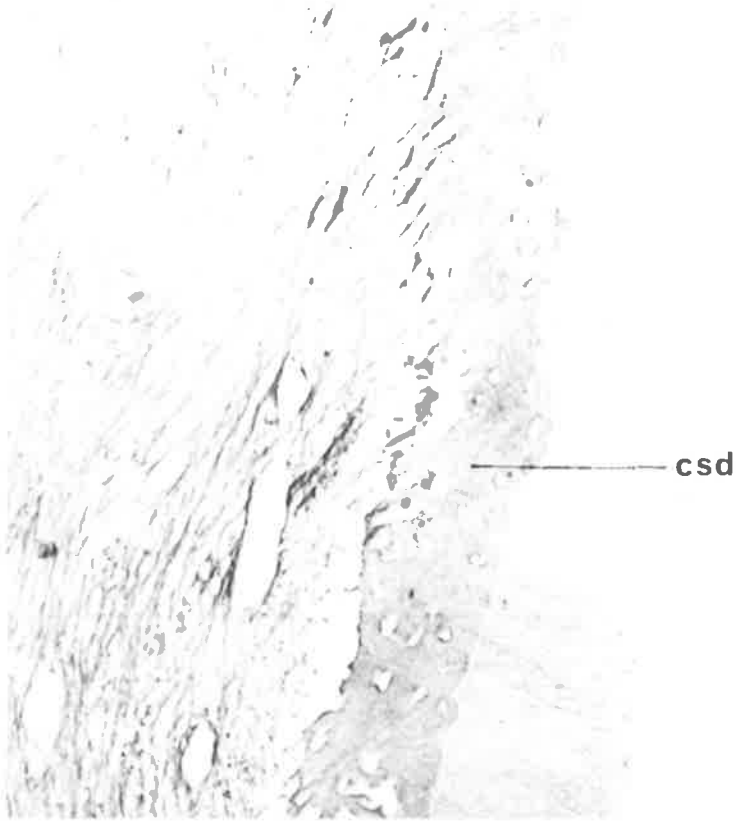


Fig 65. Y6 /4 Silver (Lillie) x 100
E.P. 97 days.
RETICULIN FIBRES EXTEND FROM THE (c.s.d) LAYER OF CELLULAR SECONDARY DENTINE

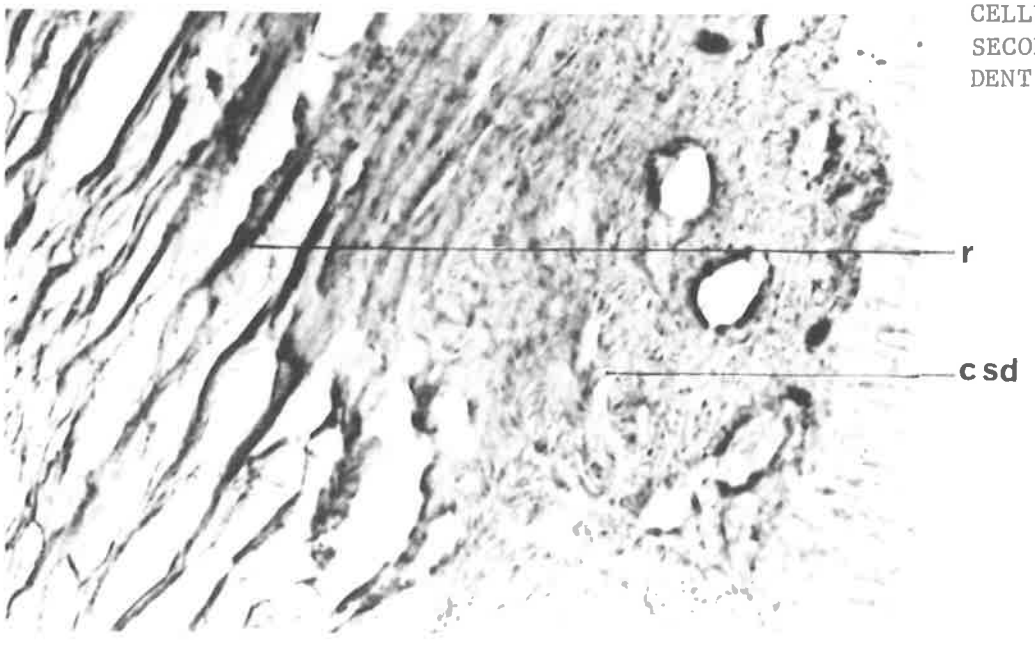


Fig 66. Y6 /4 Silver (Lillie) x 400 E.P. 97 days.
RETICULIN FIBRES (↑) CELLULAR SECONDARY DENTINE (c.s.d)

169 days: Case Nos Y32, Y33, Y34 (♂ 12 yrs).

Localised chronic inflammation was present in Y32 (Figs 67, 68) Y34 (Figs 72, 73) while Y33 (Figs 69 - 71) presented a healthy pulp remnant.

Lesion Area:

Calcific Bridging - Although the angulation of the relevant section made interpretation difficult (Figs 69 - 71) it would appear that a calcific bridge has been formed in Y33. Dentine of the walls of the root canal has been superimposed in some regions (refer Fig 70). However, other areas present the appearance of a dystrophic calcific bridge.

Partial bridging occurred in Y34 (Figs 73, 72). The partial bridge had no definite structural pattern and contained no dentinal tubules. Diffuse islands of calcification were present in the broad surface wound in Y32 (Fig 67). There was no definite line of demarcation between the necrotic zone, calcific areas, and underlying pulp. The polychromatic area thus represented a conglomerate of reactions - superficial necrosis, hyalinisation of underlying tissue and calcification.

In Y33 the pulp beneath the lesion area showed tissue free of inflammation. There was no sign of odontoblasts or odontoblast-like cells beneath the bridge. The same applied to the tissue immediately below the partial bridge in Y34.

Deeper Pulp Tissue: The break in the bridge in Y34 (Figs 72, 73) communicated with a region of pulp showing considerable infiltration

with chronic inflammatory cells. Cellular secondary dentine deposition on the root canal walls was present in the deeper aspects of Y32 and also in Y33. The deposition in Y32 appeared to be in that region of the pulp not showing the presence of inflammatory cells (Fig 68). Small resorption lacunae were present in Y32.

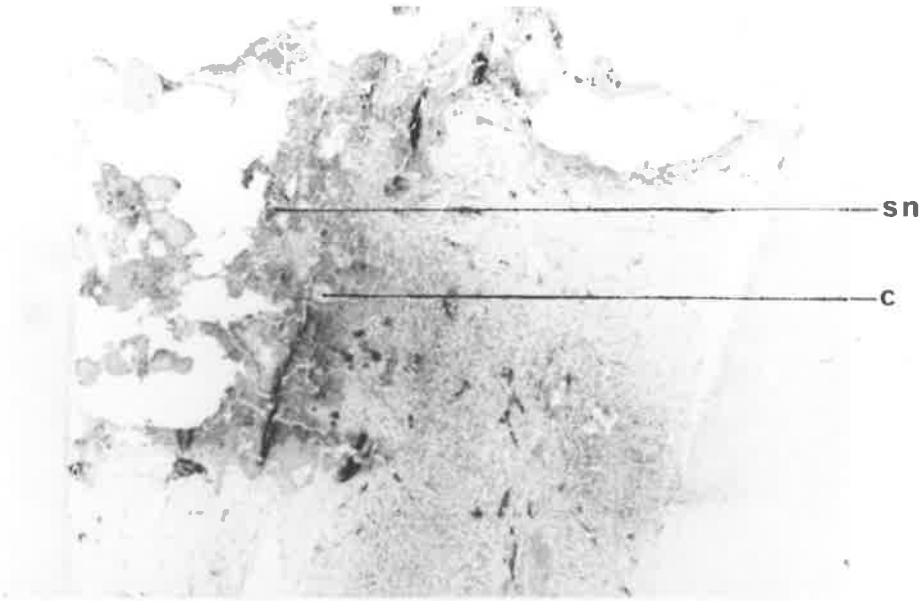


Fig 67. Y32 $\overline{47}$ H&E x 100 E.P. 169 days. THE BROAD AND IRREGULAR WOUND SHOWS SUPERFICIAL NECROSIS. REACTIONS OF UNDERLYING TISSUES HAVE PRODUCED AREAS OF HYALINISATION AND ISLANDS OF CALCIFIC MATERIAL(c). SOME OF THE PULP TISSUE SHOWS NEUTRAPHILIC INFILTRATION.

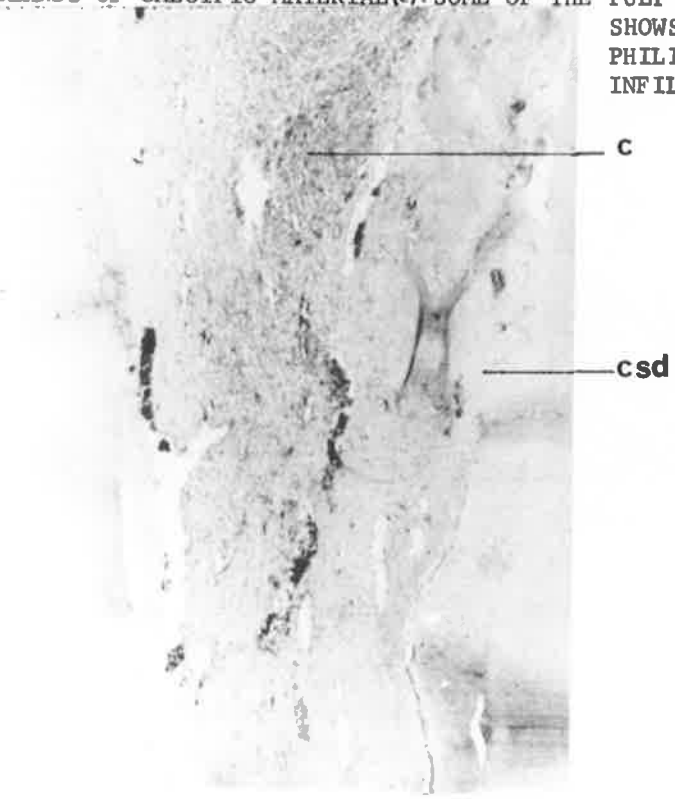


Fig 68. Y32 H&E x 100 E.P. 169 days.
DEEPER PULP TISSUE SHOWING CELLULAR INFILTRATION (c) LIMITED TO SUPERIOR ASPECT. DEEPER PULP TISSUE NORMAL CELLULAR SECONDARY DENTINE DEPOSITION ON CANAL WALLS (c.s.d)

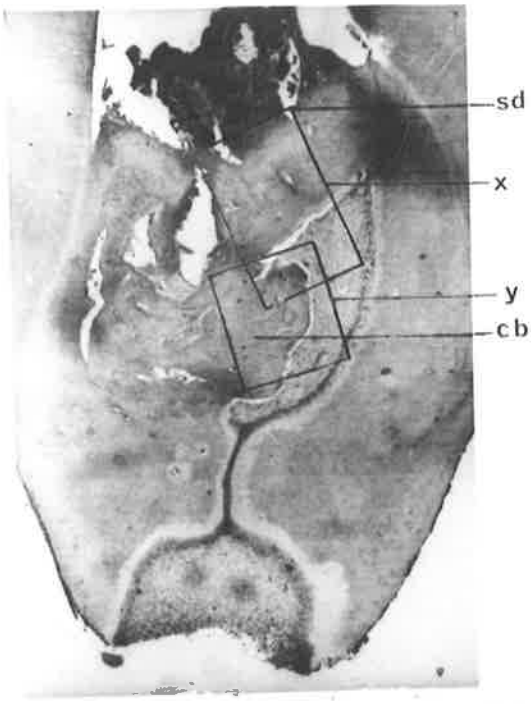


Fig 69. Y334 / Buc. H&E x 40 E.P. 169 days CALCIFIC BRIDGE (cb) IS PRESENT IN AN IRREGULAR POSITION DUE TO THE ANGULATION OF SECTION. SUPERFICIAL DEBRIS (s.d)

Fig. 70. Y33 4/ Buc. H&E x 100. SUPERIORLY FRAMED AREA (x) LARGELY FALSE BRIDGE. DENTINE (d) FROM THE WALLS HAS BEEN SUPERIMPOSED OVER OTHER CALCIFIC MATERIAL.

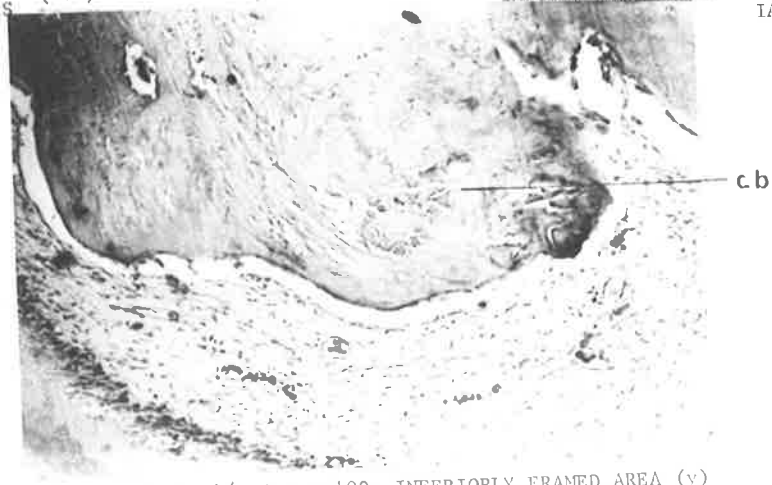


Fig. 71 Y33 4/ Buc x 100 INFERIORLY FRAMED AREA (y) TRUE BRIDGE OF CALCIFIC LATERAL DYSTROPHIC IN NATURE (c.b). UNDERLYING PULP SHOWS NO EVIDENCE OF A NEW ODONTOBLAST LAYER.

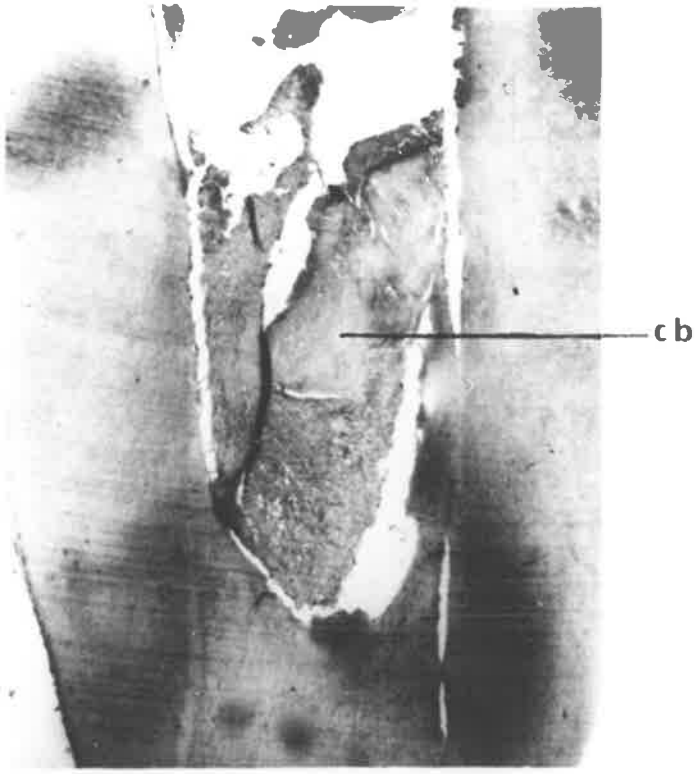


Fig 72. Y34 4/ Pal. H&E x 40 E.P. 169 days
A PARTIAL CALCIFIC BRIDGE HAS BEEN FORMED (cb)
PULP REMNANT SHOWS CHRONIC INFLAMMATION

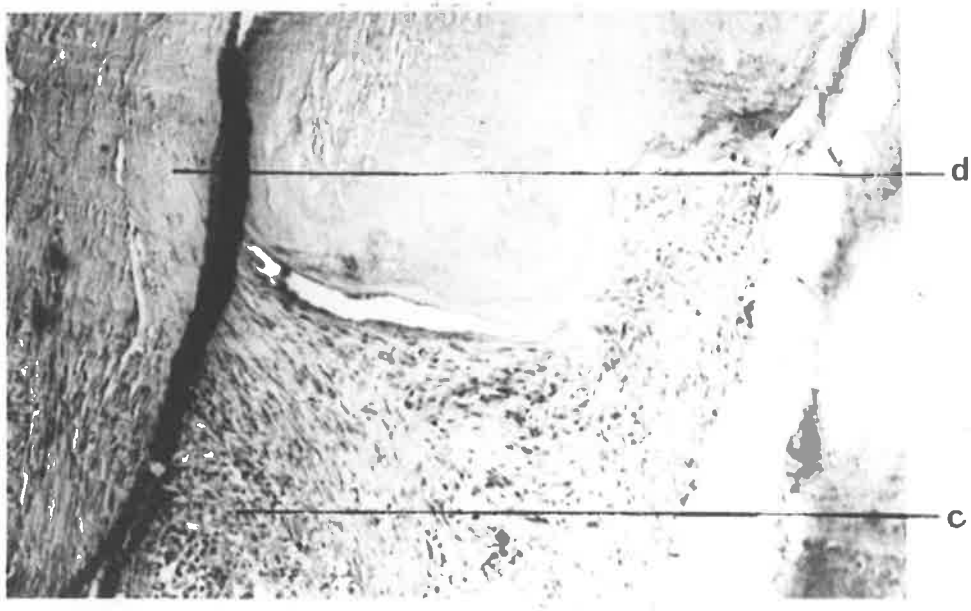


Fig 73. Y34 4/ Pal H&E x 100 E.P. 169 days.
THE DEFICIENCY (d) IN THE CALCIFIC BRIDGE IS SHOWN WITH DIRECT CONNECTION WITH AN AREA OF CHRONIC INFLAMMATORY CELLS (c)

182 days: Case Nos Y35, Y36, Y37 (♂ 12 yrs)

Lesion Area: Y35 (Figs 74 - 78) and Y36 (Figs 79, 80) showed an inflammation free pulp remnant with no evidence of fibrous or calcific repair. Y37 (Figs 81 - 85) showed a positive response in relation to the organisation, and eventual calcification of the lesion area. This response however did not result in complete bridging (Figs 81, 83) but a deficiency existed at one border. Tissue which was fibrous in nature passed through this deficiency and connected with a large but well defined area of chronic inflammatory cells. This area of cells was surrounded by a thick capsule of fibrous tissue (Fig 82). Beneath the bridged area the pulp was not infiltrated with inflammatory cells.

Nature of bridge in Y37 - (Figs 83, 84). This incomplete calcific barrier showed some small rounded areas of possible cell inclusions. There was no clear line of demarkation between the necrotic zone and the calcific area. A clear line of demarcation could be seen between the bridge and the cells of the pulp. The bridge seemed uniform in structure and appeared non-tubular. It resembled bony callus.

Deeper Pulp Tissue: The underlying pulp in Y37 showed a cell rich area beneath the bridge adjoining normal pulp. The cell rich layer (Fig 84) contained cells which were arranged in a direction parallel to the bridge. Their appearance was that of mature fibroblasts.

Y36 in contrast to Y37 and Y35 showed a well-defined necrotic zone with underlying normal pulp. Y35 showed an unusual area within the

the pulp remnant. This may have been an antefact, but its appearance (Fig 77) was that of an island of cellular calcific material. The deeper pulp tissue was normal.

Internal resorption - was not evident in any of the above specimens.

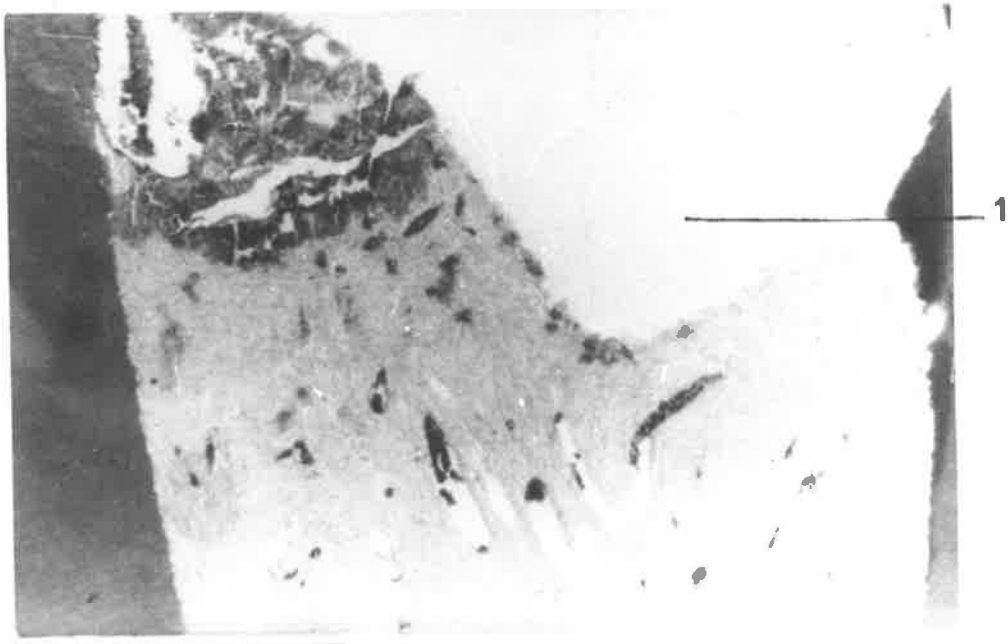


Fig 74. Y35 /4 H&E x 40 E.P. 182 days.

THE BROAD AMPUTATION WOUND SHOWS DEMARCATION OF THE NECROTIC LAYER FROM THE UNDERLYING PULP. REMAINDER OF PULPAL WOUND HAS BEEN LOST DURING PREPARATION (1) (REFER FIG 76). UNDERLYING PULP IS NORMAL.

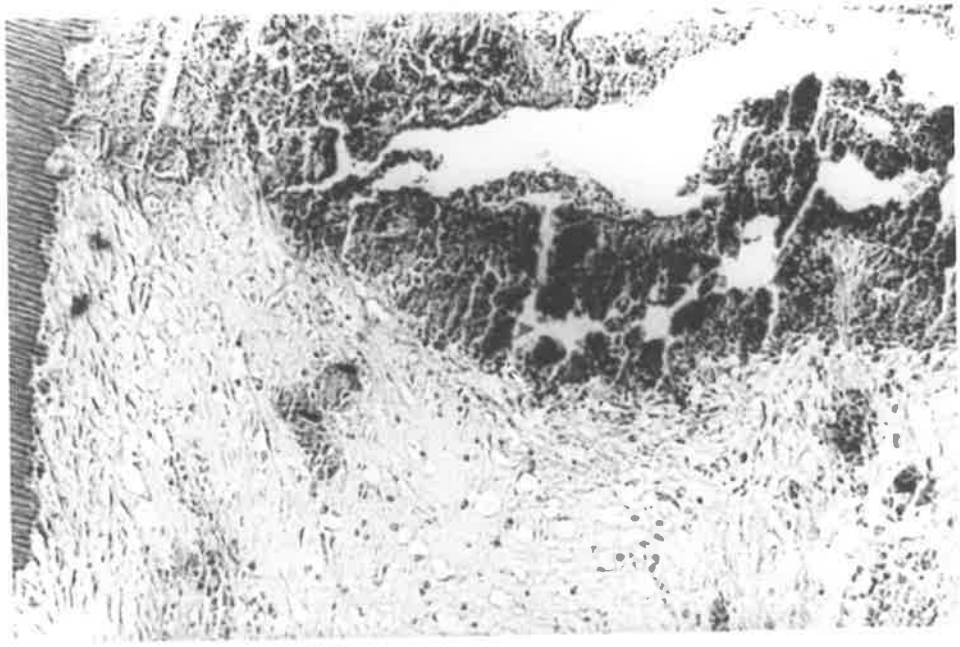


Fig 75. Y35 /4 V.G. x 100 E.P. 182 days.
LESION AREA: THE UNDERLYING PULP TISSUE SHOWS NO SIGN OF ORGANISATION LEADING TO CALCIFICATION. CONNECTIVE TISSUE APPEARS THAT OF NORMAL PULP.

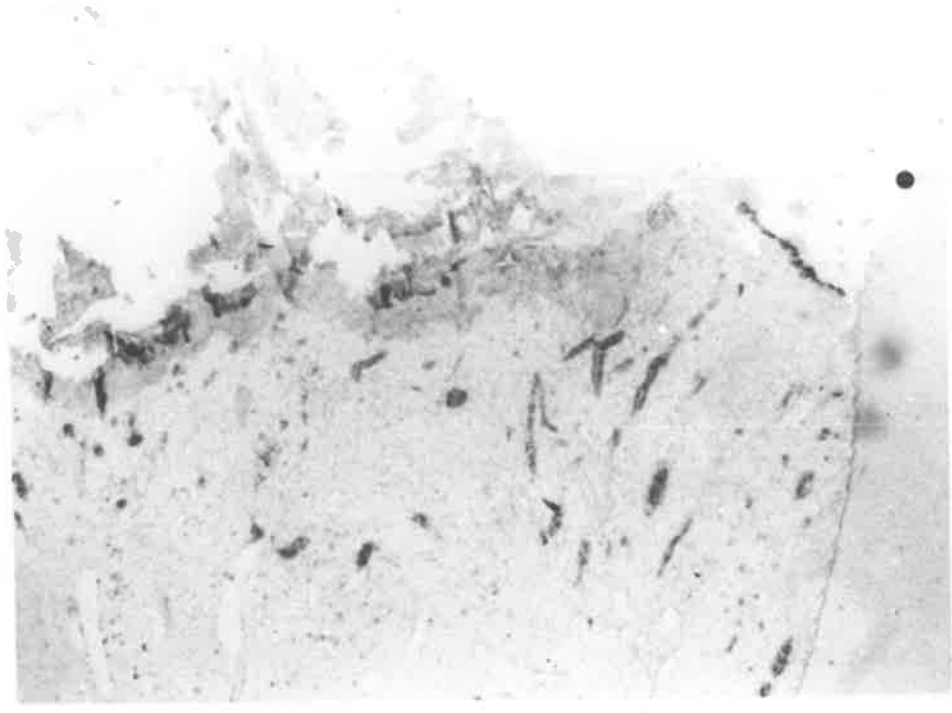


Fig 76. Y35 /4 H&E x 100 E.P. 182 days.
SECTION OF PULP WOUND NOT SHOWN IN FIG 74.

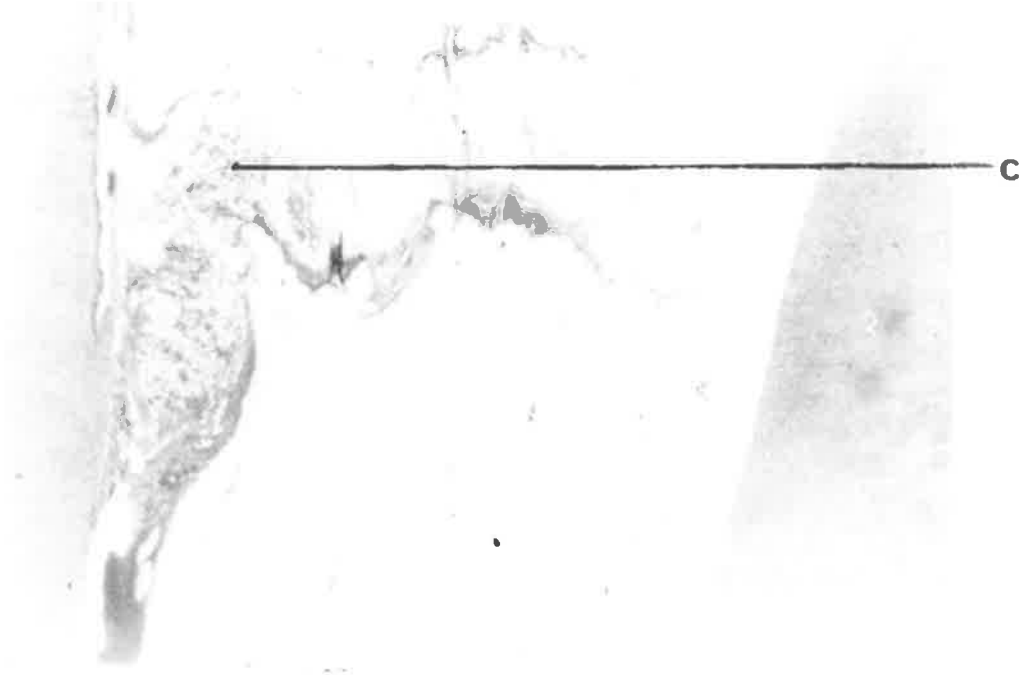


Fig 77. Y35 $\overline{/4}$ V.G. x 100 E.P. 182 days:
AN ISLAND OF CELLULAR CALCIFIC MATERIAL (c) IS PRESENT WITHIN THE PULP REMNANT

Fig 78. Y35 $\overline{/4}$ V.G. x 40 E.P. 182 days.
NORMAL PULP TISSUE AT APICAL FORAMEN.

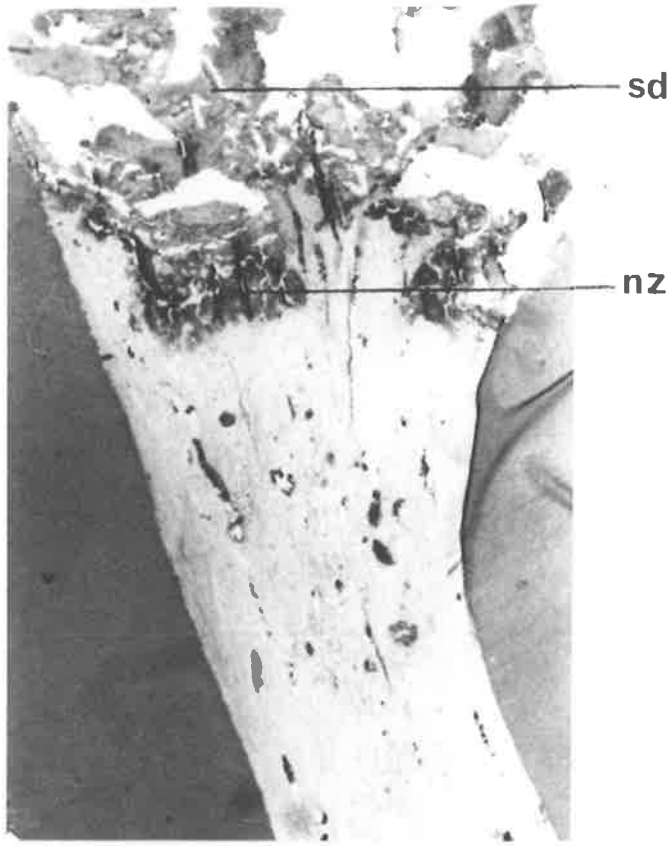


Fig 79. Y36 /4 Buc. H&E x 40. E.P. 182 days:
BROAD WOUND SHOWING SUPERFICIAL DEBRIS AND MEDICAMENT (s.d) NECROTIC ZONE (n.z) HEALTHY PULP

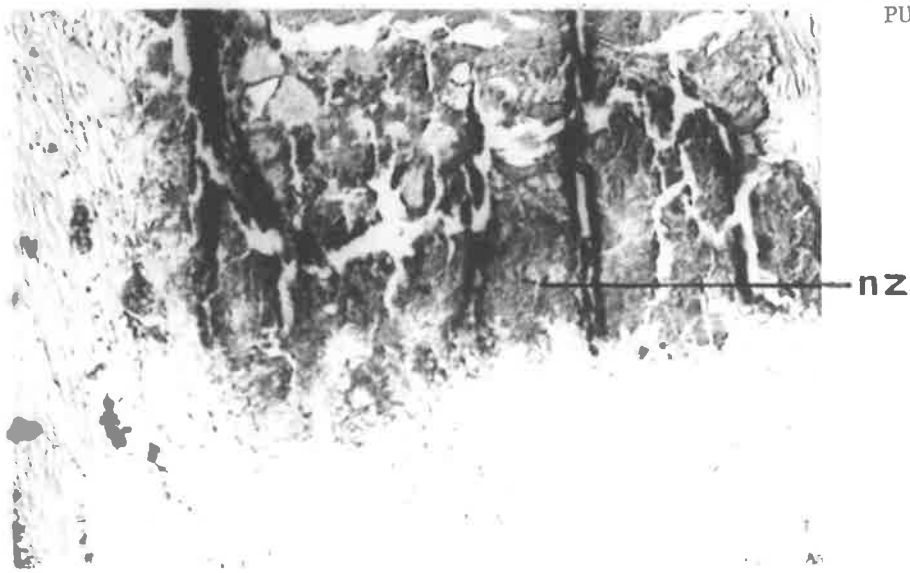


Fig 80. Y36 /4 Buc. V.G. x 100 E.P. 182 days.
THE PULP BELOW THE NECROTIC ZONE (n.z) SHOWS NO COLLAGEN ACTIVITY AND THE PULP APPEARS NORMAL.

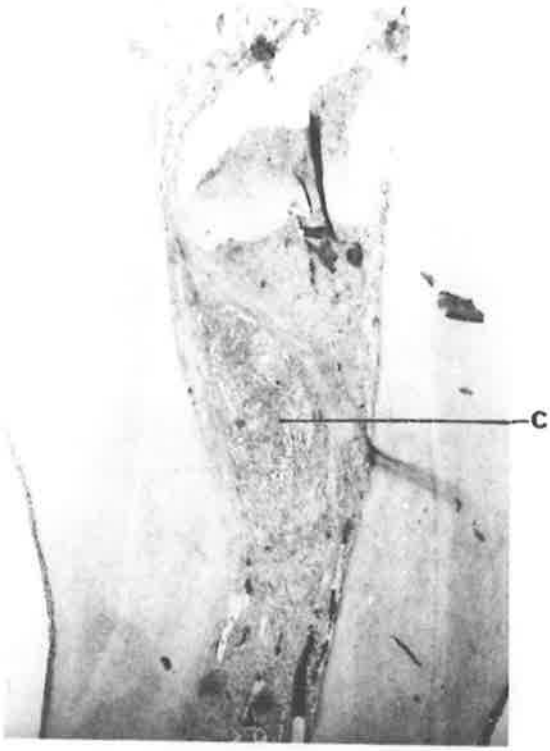


Fig 81. Y37 /4 Pal H&E x 40.
E.P. 182 days.

INCOMPLETE BRIDGE FORMATION. TISSUE PASSING THROUGH DEFICIENCY IN BRIDGE CONNECTS WITH A LOCALISED AREA OF CHRONIC INFLAMMATION (c)

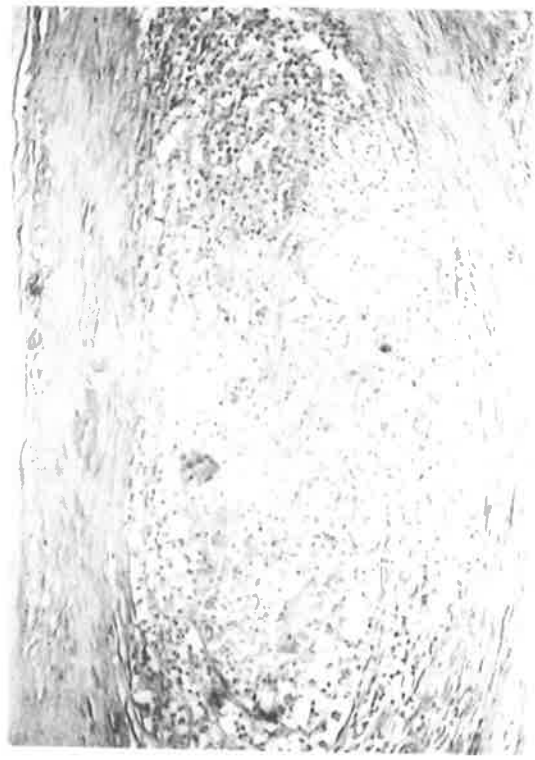


Fig 82. Y37 /4 Pal. H&E x 100
E.P. 182 days.

AREA OF CELLULAR INFILTRATION SURROUNDED BY A THICK FIBROUS CAPSULE.

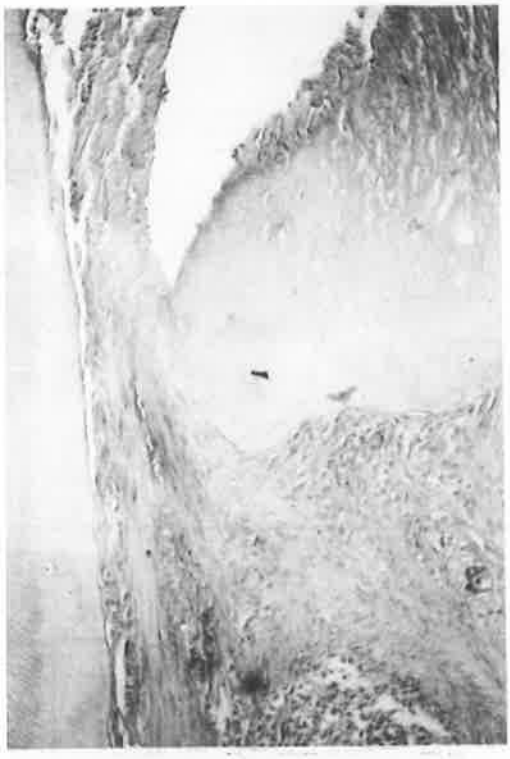


Fig 83. Y37 /4 Pal H&E x 100 E.P. 182 days.
DEFICIENCY IN BRIDGE FORMATION

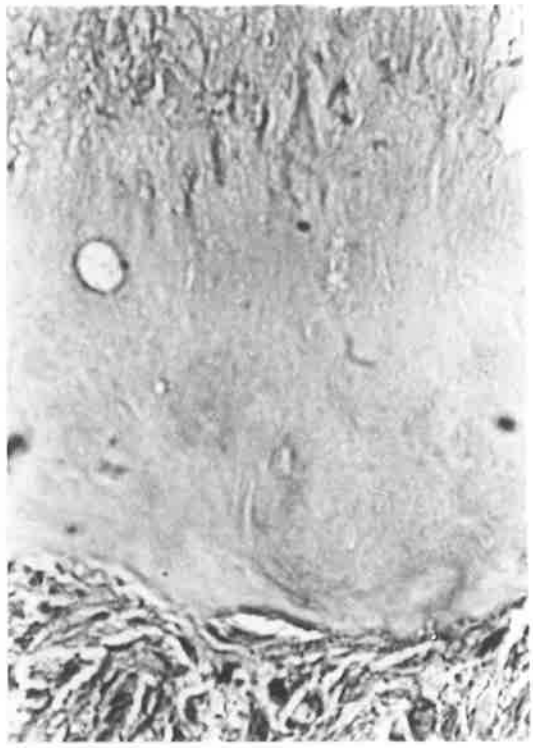


Fig 84. Y37 /4 Pal H&E x 400:
CALCIFIC BRIDGE WITH CELL RICH LAYER BELOW: AXIS OF CELLS IS PARALLEL TO BRIDGE

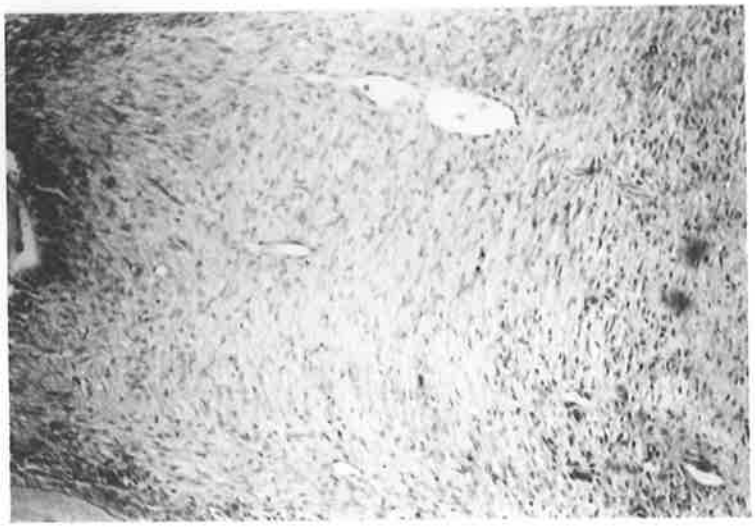


Fig 85. Y37 /4 Pal H&E x 100.
NORMAL PULP AT THE APICAL FORAMEN.

DISCUSSION

Analysis of reactions in young group

The wound usually produced by the amputation with the flattened Hedström file was a lacerated type similar to those produced in the pilot study.

Following traumatic injury an acute inflammatory reaction followed with considerable dilation of the apical blood vessels. The surface of the wound was covered with medicament (calcium hydroxide) tissue debris, blood clots and dentine spicules which had been produced from the neighbouring walls during the filing action.

Superficial necrosis due to the action of calcium hydroxide was a constant finding from the two day experimental period. It was impossible to assess the period during which superficial necrosis occurred. Dausch and Sauerwein (1952) considered that the necrotizing effect continued for 2 - 3 days. The superficial necrotic zone was generally relatively broad. By 14-16 days there was a clear line of demarcation between this layer and the underlying pulp tissue, which showed in some instances, a tendency to fibrosis (Y38).

An increase in the blood supply due to inflammatory changes initiated mild internal resorption at this stage in one case (Y18).

It would seem that after this period two different types of reaction were initiated.

(1) Repair of the pulp by collagen formation leading to fibrosis, hyalinisation followed by calcification of the superficial layers.

(2) Resolution of the pulp under the necrotic zone with no attempt at fibrous or calcific repair.

The above investigation showed in the short experimental cases a predominance of the first type of response, but many of the longer term cases showed the second reaction. The second reaction appeared to occur in broad wounds (e.g. Y6, Y35 and Y36).

The reactions from 14 days will be discussed separately under the two main reactions described above.

(1) Repair: By 28 days definite zones had been produced in those pulps which did not exhibit chronic inflammatory changes (e.g. Y22). The zones differentiated beneath the superficial debris or medicament zone were: (a) Superficial necrotic layer, (b) calcific bridging - usually incomplete (c) Dense zone showing hyalinisation (d) Normal pulp.

Further evidence of calcification in the dense zone was observed at 46 - 48 days, but complete bridge formation was not evident. Calcific bridging was observed in some cases by 97 - 108 days (Y2) but others (e.g. Y1) still showed only small calcific areas. Repair of the resorbed dentine surface by cellular secondary dentine at this stage indicated that there had been some change in the blood supply to the pulp. Resorption of calcified tissue has been shown by Grieg (1931) to be due to the increased vascularity associated with inflammation while deposition of calcific material was conversely shown to be related to a diminution in the normal blood supply.

Although complete bridging was observed after 169 days (Y33) other roots showing this repair reaction did not necessarily have any definite bridge formation at this stage. The nature of the bridge was that of a

dystrophic calcification with no signs of tubule formation. The cells beneath the bridge area resembled fibroblasts and were orientated parallel to the axis of the bridge.

The positive response of complete bridging may represent a repair reaction capable of taking place in a healthy pulp. Some cases in the study were observed with incomplete bridging (Y32, Y34). The regions of deficiency in bridge formation corresponded to areas of localised chronic inflammation within the pulp.

(2) Resolution. At 97 - 108 days one of the pulp remnants (Y6) showed a healthy state with little or no sign of definite areas of fibrosis or calcification within the superficial layers of the pulp. There was a very definite line of demarcation between the superficial debris layer and the underlying pulp, but Mallory and Van-Gieson stains revealed no connective tissue activity in the lesion area. Connective tissue activity was observed however in the peripheral regions where considerable internal resorption, which had previously taken place, was showing signs of repair by cellular secondary dentine. Cellular secondary dentine deposition in the deeper regions of the canal was extensive.

As silver staining (Lillie), Van Gieson, and Mallory stains indicated continuous connective tissue activity in the region of the cellular secondary dentine deposits, one might expect the process to continue to partial or complete obliteration of the root canal.

An example of minimal response was observed as late as 182 days (Y36). Beneath the superficial necrotic layer the pulp was normal in

appearance. Differential stains did not reveal any connective tissue activity either at the surface of the lesion or periphically.

No internal resorption or deposition of cellular secondary dentine had occurred indicating that the initial inflammatory reaction must have been mild and rapid resolution had been achieved without structural changes especially in relation to the blood supply.

RESULTS

ADULT MATERIAL

A summary of the roots treated, experimental periods, post-operative clinical, radiographic and histological results is given in Table VI. The same criteria as described for the corresponding young age group, have been used in the assessment of these results.

HISTOLOGICAL OBSERVATIONS

Untreated teeth

Case Nos ACI, AC2 (♀ 46 years /3, /2 (refer Case Nos A1 - 9). The pulps of this patient (Figs 86-90) showed a degree of fibrosis but the most striking features were the large denticles. Slight vascular dilation was evident. The odontoblast layer lining a marked predentine layer, showed vacuolisation.

AC3 (♀ 42 years /6 (refer Case Nos A14-18). Marked fibrosis and diffuse calcification were observed (Figs 91-93). The diffuse calcification was mainly confined to the apical region. Vacuolisation was evident in the odontoblast layer while the cell content of the pulp in general was sparse.

TABLE VI SURVEY OF EXPERIMENTAL PARTIAL PULPECTOMY IN THE OLD AGE GROUP

TABLE VI

No	Age	Root	Clinical Caries* (0,1,2,3)	State Perio* * (0,1,2)	Experimental Period	Post Operative Clinical Symptoms: Neg.	Thermal	Pos. Spontaneous pain	Percussion	Radiographic Evidence of Periapical Pathology: neg.	pos.	PULPAL STATE: Inflammation: nil	Acute Local	Acute Gen	Chronic Local	Chronic Gen	Calcific Bridging Radiog.	Calcific Bridging Hist.	Internal Resorption Radiog.	Internal Resorption Hist.	Cellular Secondary Dentine Deposition (a) walls	(b) bridge	Histological Result: fail.	doubt.	success	Clinico-radiol. result: fail.	doubt	success
A19	46	/3	0	2	24	x				x		x													x				x
A20	46	1/	0	2	35	x					x					x								x			x		
A21	46	4/B	0	2	35	x				x						x								x					x
A22	46	4/P	0	2	35	x				x		-	-	-	-	-								-					x
A23	46	3/	3	2	35			x	x		x					x								x			x		
A24	46	2/	2	2	35			x	x		x					x								x			x		
A18	42	5/	0	2	38	x				x		x						x		x					x				x
A17	42	4/	0	2	51	x				x		x													x				x
A16	42	3/	0	2	51	x				x						x										x			x
A14	42	/3	0	2	58	x				x		x						x							x				x
A15	42	/4	0	2	58	x					x					x								x			x		
A3	46	6/B M	1	2	105	x					x	x								x		x			x			x	
A4	46	6/B D	1	2	105	x				x		x						x							x				x
A5	46	6/P	1	2	105	x				x		x								x		x			x				x
A6	46	7/B M	1	2	105	x				x						x								x					x
A7	46	7/B D	1	2	105	x				x						x								x					x
A8	46	7/P	1	2	105	x				x						x								x					x
A1	46	/1	2	2	168	x				x		x					x	x	x	x					x				x
A2	46	3/	0	2	172			x (1 day P.O)		x		x					x	x		x					x				x
A9	46	2/	2	2	172	x				x						x			x	x		x				x			x
A13	45	/4	0	2	325	x				x		x						x							x				x
A10	45	8/	1	1	365	x				x		x								x		x			x				x
A11	45	/4	0	1	365	x					x					x								x			x		
A12	45	2/	0	1	365	x				x		x														x			
TOTAL:																								9	3	11	5	1	18

* caries - 1= incipient 2 = caries of dentine 3= caries involving pulp.

** perio - 1 = gingivitis 2 = periodontitis simplex.

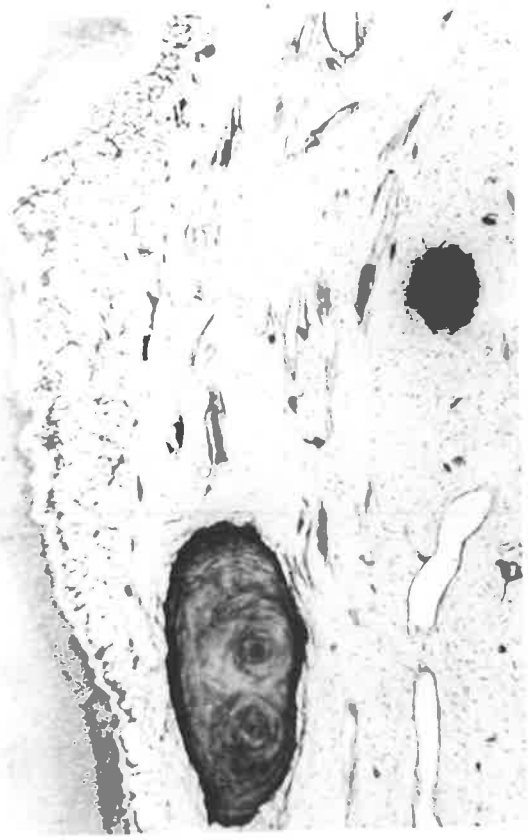


Fig. 86 A.C.I. /3 H&E x 100

PULP IN CORONAL REGION
SHOWING A LARGE CALCIFICATION

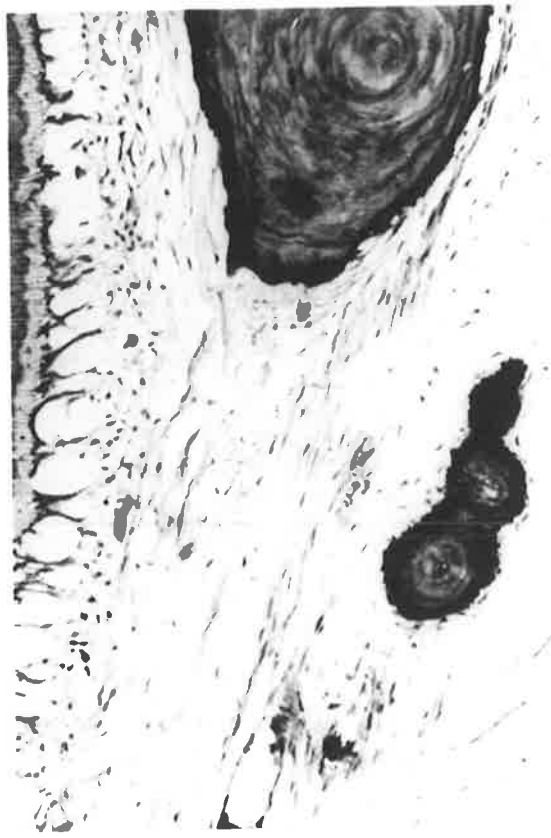


Fig. 87. A.C.I. /3 H&E x 400

FIBROTIC PULP WITH
CALCIFICATIONS

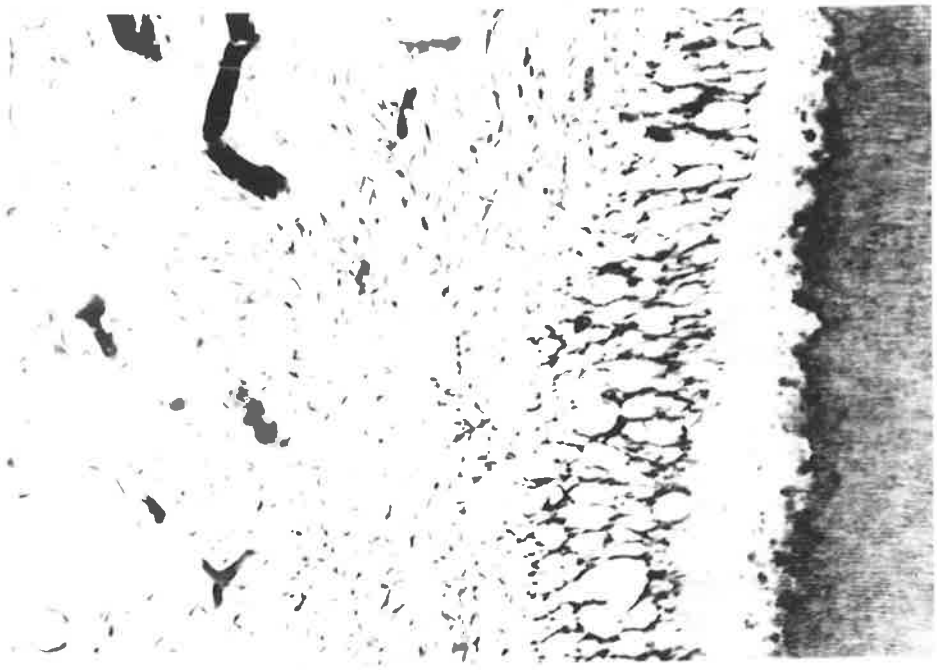


Fig. 88. A.C.I. /3 ♀ 46 yrs H&E x 100
"MID-PULP" REGION

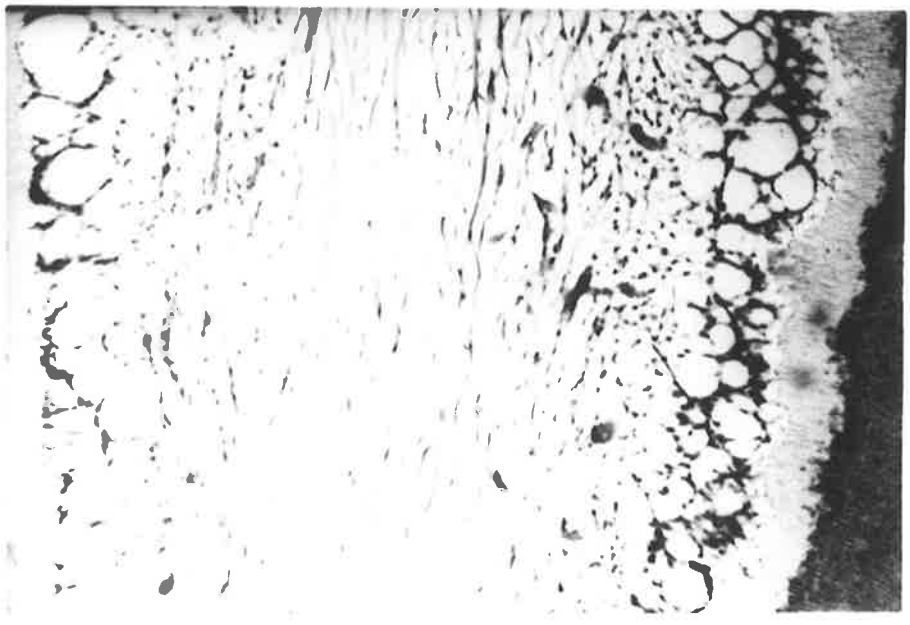


Fig 89. A.C.I. /3 H&E x 100
APICAL PULP REGION

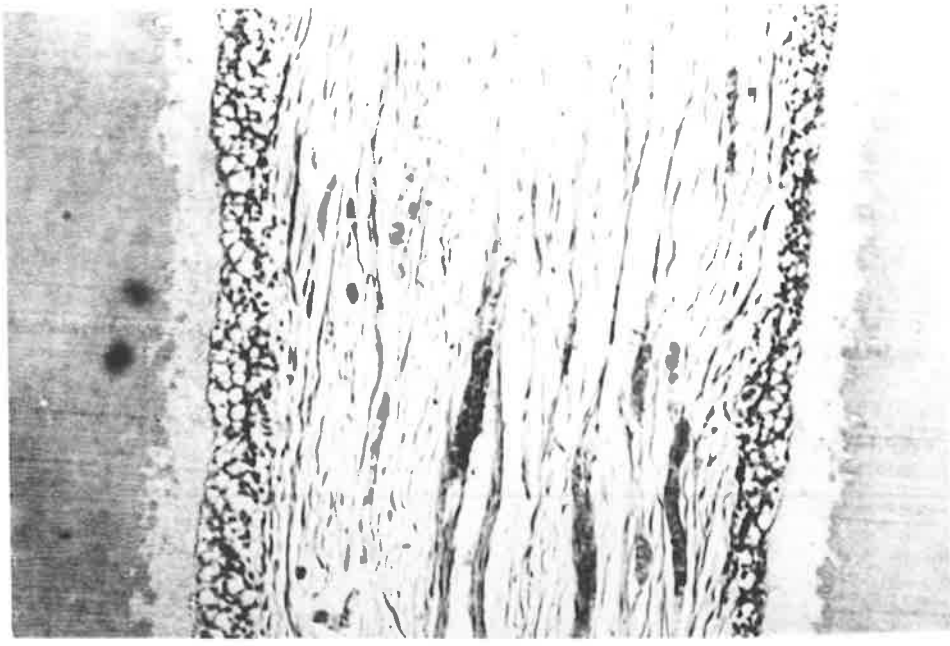


Fig. 90. A.C.2. /2 ♀ 46 years. H&E x 100

APICAL PULP TISSUE SHOWING RELATIVELY DILATED BLOOD VESSELS

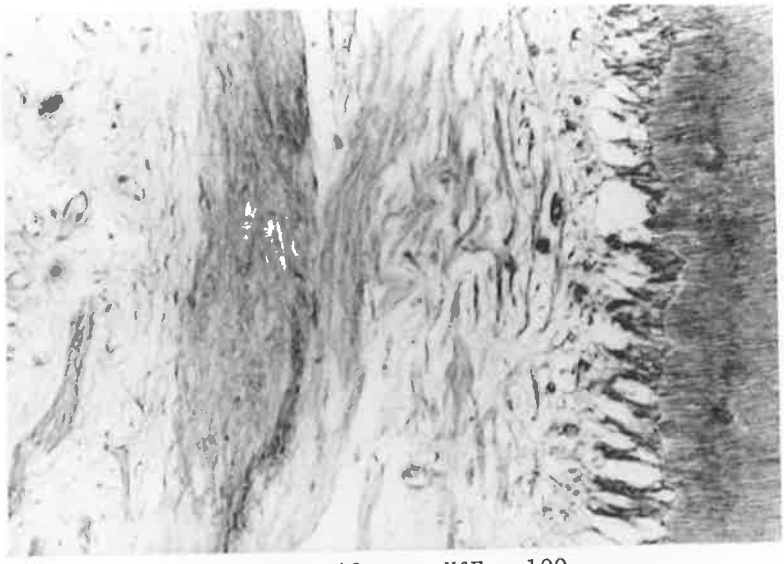


Fig 91. A.C.3. /3 ♂ 42 yrs. H&E x 100
FIBROUS NATURE OF PULP "VACUOLISATION" OF ODONTOBLAST LAYER.

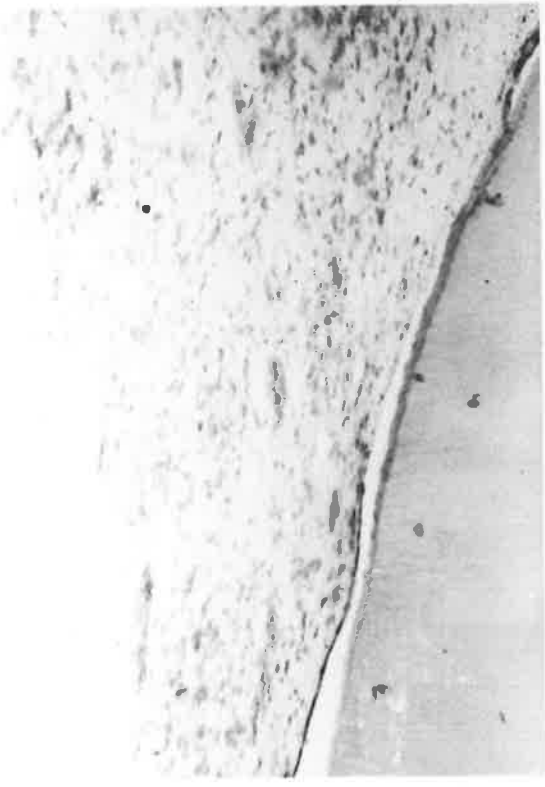


Fig. 92 A.C.3 /6 ♂ 42 yrs H&E x 100
DIFFUSE CALCIFICATION OF APICAL PULP

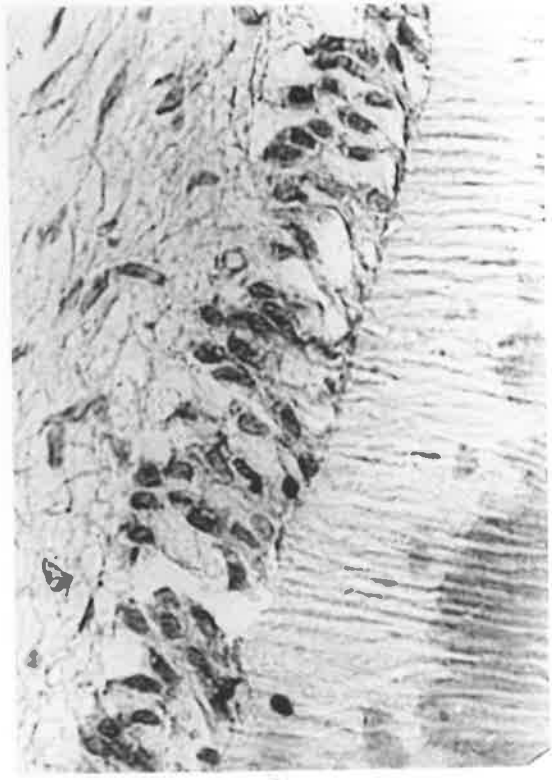


Fig 93. A.C.3. /6 ♂ 42 yrs H&E x 40(
ODONTOBLAST LAYER IN CORONAL PULP.

Reactions observed at varying experimental times

24 days: Case No. A19 (♂ 46 yrs) (Figs 94-96).

Lesion area: The superficial debris contained in addition to dressing material (calcium hydroxide) many dentine spicules. A fragmented but apparently narrow necrotic zone was clearly defined from an underlying "dense zone", in which hyalinisation was evident. There was some evidence of early calcification in these areas.

Deeper Pulp tissue: This showed fibrosis and areas of diffuse calcification similar to that found in pulps of the same age group (Fig 95). No inflammatory cells were evident:

No internal resorption had occurred.

35 - 38 days: Case No A20 - A24 (♂ 46 years) A18 (♀ 42 years).

A22 was spoilt during sectioning. A20, A23, A24 all showed degrees of severe general chronic inflammation to necrosis. There was no evidence of an early repair reaction.

A21

Lesion Area: A21 showed a definite necrotic zone, below which there was a "dense zone" with ill-defined areas of calcific material, especially in relation to impacted dentine spicules. Hyalinisation was evident in the uncalcified areas of this zone.

The Deeper Pulp tissue was heavily infiltrated with chronic inflammatory cells.

A18 - (Figs 97 - 105)

Lesion area: The superficial debris contained many dentine spicules and a necrotic zone was present in one limited area. The dense zone

showed active repair with extensive collagen formation, hyalinisation and early calcification. Fig 97 (Mallory Stain) shows some of these connective tissue changes.

Deeper Pulp tissue: No cell rich zone was present but the pulp beneath the lesion showed early signs of fibrosis. The pulp in the more apical region was similar to that of the control tooth (AC3 Figs 91-93).

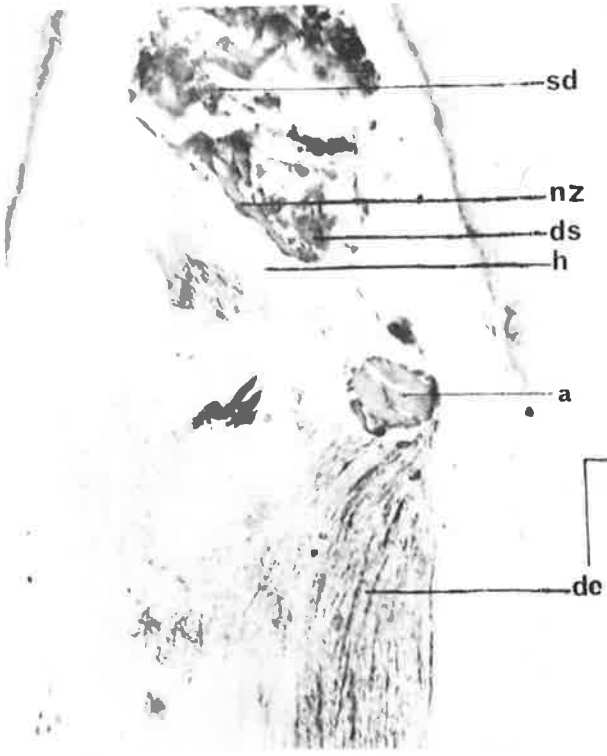


Fig 94. A19 /3 H&E x 40. E.P 24 days

TISSUE DISPLACEMENT HAS OCCURRED DURING PREPARATION. MEDICAMENT AND SUPERFICIAL DEBRIS (s.d).

DENTINE SPICULES (d.s) NECROTIC ZONE (n.z) AREAS OF HYALINISATION (h) DIFFUSE CALCIFICATION OF PULP (d.c) ARTEFACT (a)

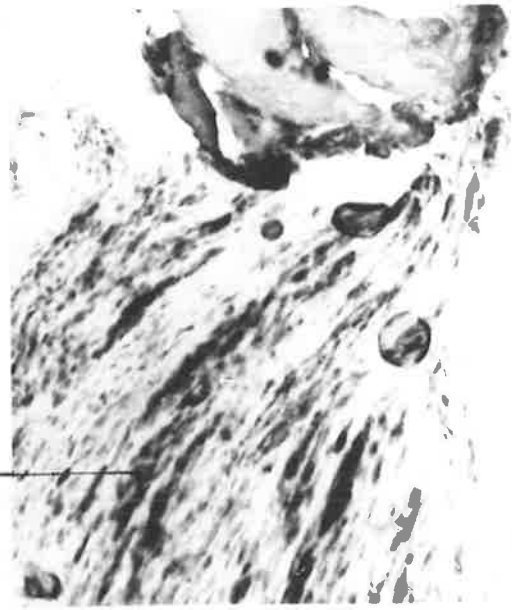


Fig 95. A19 /3 H&E x 100

PULP SHOWING DIFFUSE CALCIFICATION (d.c)

Fig 96. A19 /3 V.G. x 40

SHOWING FIBROSIS OF THE MAJORITY OF THE PULP REMNANT

Fig 97. A18 _5/ Mallory x 100 E.P. 38 days

THE LESION AREA SHOWING THE CONNECTIVE TISSUE CHANGES OCCURRING WITHIN THE "DENSE ZONE" AND THE UNDERLYING PULP "REFER ALSO FIG 99)

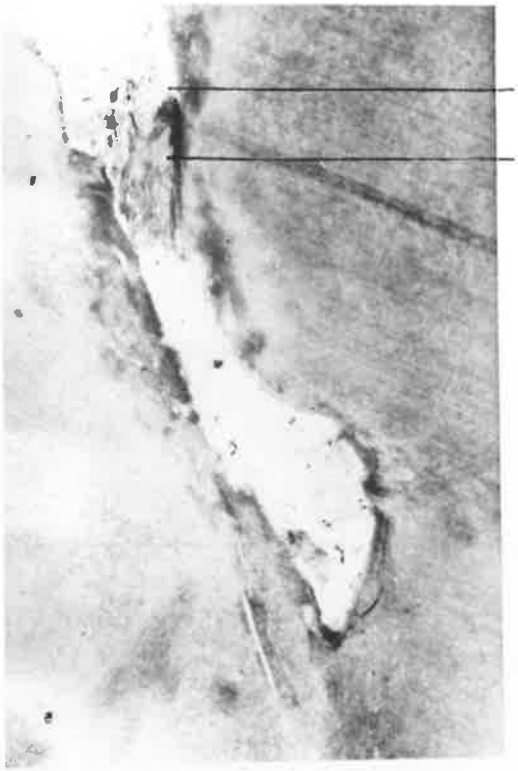


Fig 98. A18 5/ Mallory x 40 E.P. 38 days
AMPUTATION NOTCH (a.n) SUPERFICIAL DEBRIS (s.d) LESION AREA (l.a)

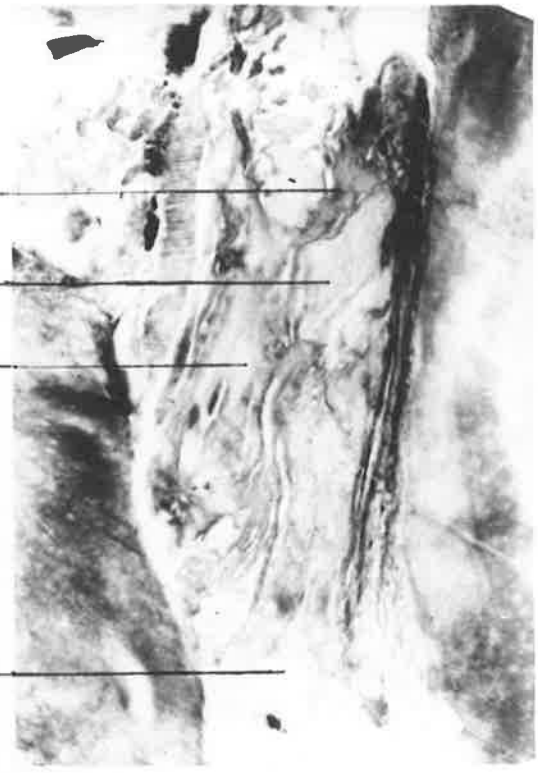


Fig. 99 A18 5/ Mallory x 100
THE LESION AREA SHOWING SUPERFICIAL DEBRIS, SMALL NECROTIC ZONE (n.z) AREAS OF HYALINISATION (h) AND CALCIFICATION (c) WITHIN THE DENSE ZONE - TRANSITION AREA (t)

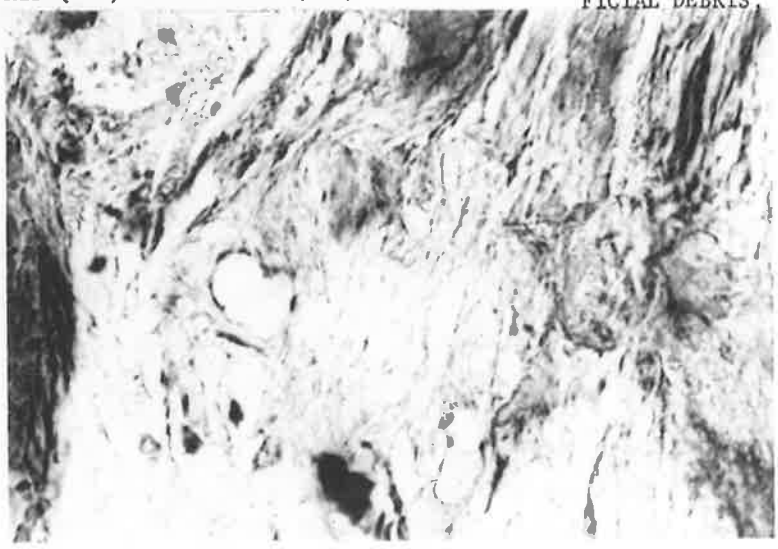


Fig. 100. A18 5/ Mallory x 400
TRANSITION ZONE. PULP BENEATH THE DENSE ZONE SHOWS COMMENCEMENT OF COLLAGEN FORMATION.

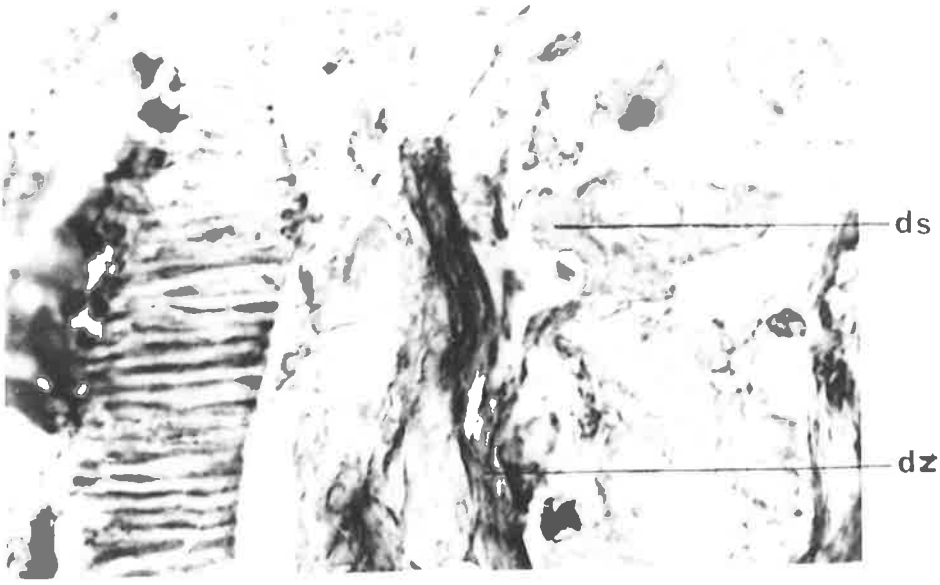


Fig 101. A18 5/ Mallory x 400 EP. 38 days.
SUPERFICIAL DEBRIS WITH MANY DENTINE SPICULES (d.s) DENSE ZONE (d.z)

Fig. 102. A18 5/ Mallory x 400
DENSE ZONE, POLYCHROMATIC, INDICATING VARYING STAGES OF CONNECTIVE TISSUE REPAIR.

Fig 103. A18 5/ H&E x 100. E.P. 38 days
LESION AREA

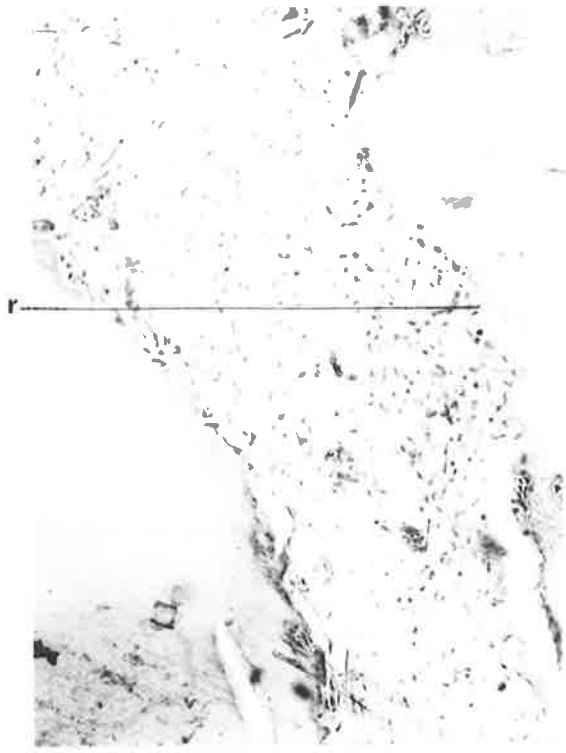


Fig. 104 A18 5/ H&E x 100
DEEPER PULP TISSUE.
RESORPTION OF CANAL WALLS IS ACTIVE e.g. IN REGION (r)

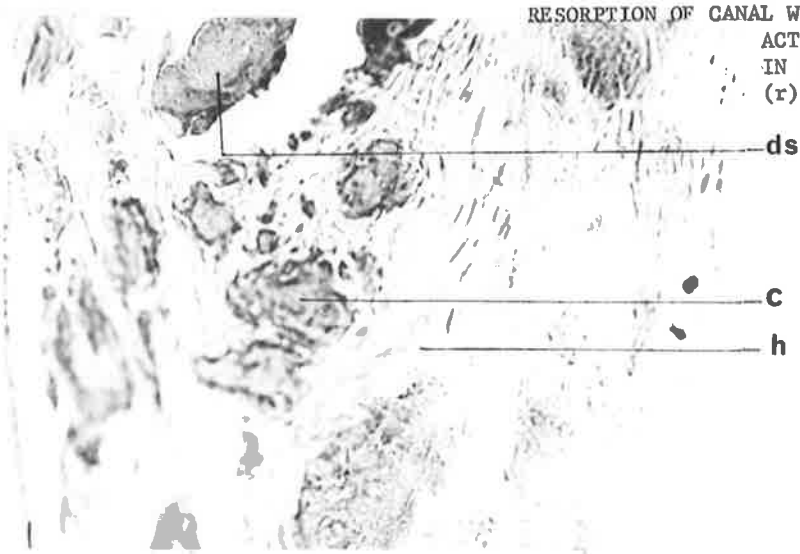


Fig. 105. A18 5/ H&E x 400. E.P. 28 days:
THE DENSE ZONE. AREAS OF HYALINISATION (h) AND ISLANDS OF CALCIFIC MATERIAL ARE PRESENT. DENTINE SPICULES (d.s) LIE BOTH ABOVE THE LESION AND ENCLOSED WITHIN DENSE ZONE.

51 - 58 days: Case No A14-17 (♀ 42 yrs).

The lesion area was not shown on available sections in A14 and A17 but deeper pulp tissue was observable.

Lesion Area: A15 and A16 - showed superficial debris, with dentine spicules. The pulp in the lesion area was heavily infiltrated with chronic inflammatory cells. No repair was evident.

Deeper pulp tissue: A16 - Fibrotic pulp with cellular infiltration. A14, A17 showed pulp remnants exhibiting fibrosis. A14 showed calcific degeneration in relation to apical blood vessels, similar to the control tooth AC3.

105 Days: Case Nos A3 - A8 (♀ 46 years).

Lesion Area: Sections showing the region of the amputation wound were available only in A3, A5 (Figs 106-108). In both cases the lesion area showed little repair. There was no clearly defined necrotic layer and no definite repair of the pulp remnant. In addition there was a very mild infiltration with chronic inflammatory cells below the amputation site (Fig 106).

Deeper Pulp tissue: A3, 4, 5 showed healthy pulp with absence of chronic inflammatory cells. A6, 8 showed generalised round cell infiltration. Internal resorption was present in A5 (Fig 106), many multinuclear giant cells being present.

Cellular Secondary Denture: Some areas in A5 showed repair with secondary cellular dentine while A3 showed generalised deposition of this material within the canal wall.

No assessable sections were available from A7.

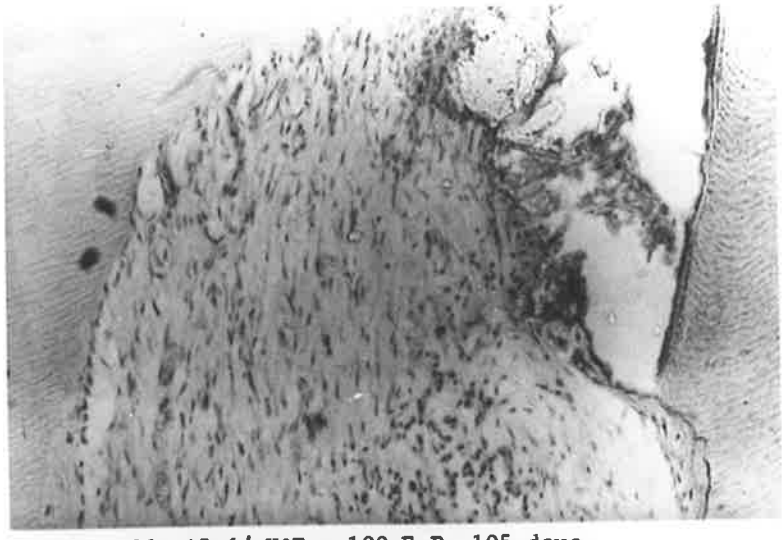


Fig. 106. A5 6/ H&E x 100 E.P. 105 days.
THE LESION AREA. NO SIGNIFICANT REPAIR HAS OCCURRED. A SMALL NUMBER OF NEUTRAPHILS ARE PRESENT IN THE PULP BENEATH THE AMPUTATION SITE. RESORPTION OF THE WALLS IS ACTIVE

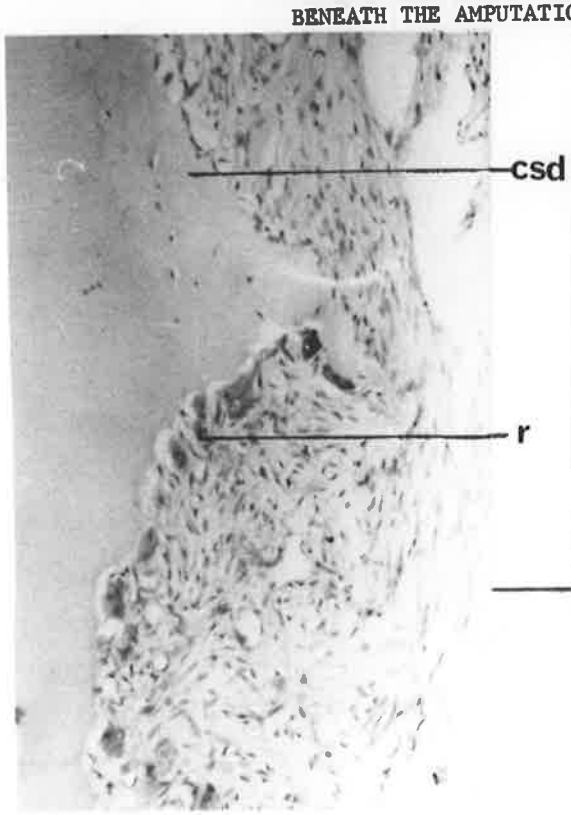


Fig 107. A5 6/ Pal H&E x 400.
FRAMED AREA SHOWING RESORPTION (r) REPAIR BY SECONDARY CELLULAR DENTINE (c.s.d) OCCURING IN NEIGHBOURING REGIONS.

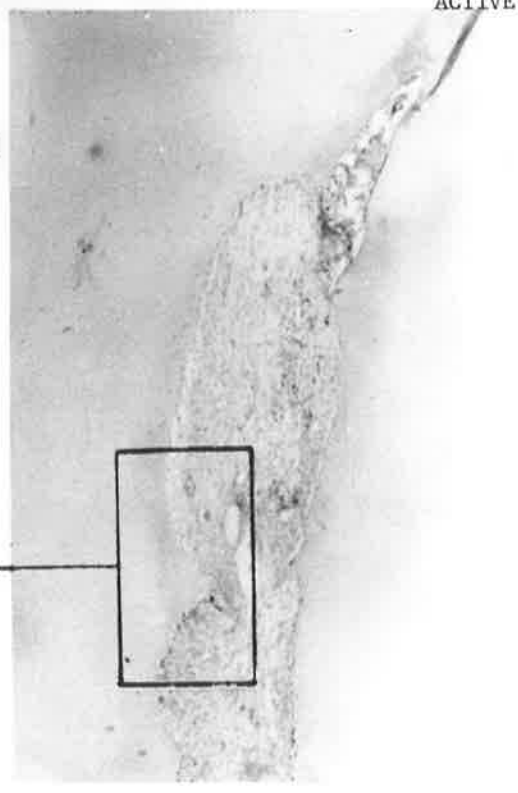


Fig. 108 A5 6/ Pal. H&E x 40
E.P. 105 days.
AMPUTATION SITE & PULPAL REMNANT.

168 - 172 Days: Case Nos A1, A2, A9 (♀ 46 yrs).

Lesion Area: The superficial debris in A1 and A2 had considerable numbers of dentine spicules and many were in intimate contact with calcific areas. A calcific barrier had been formed in A1 & A2 (Figs 109-115) but no attempt at repair was evident in A9 (Fig 116-119), a localised chronic inflammatory lesion being present. The barrier was thought to be complete in both A1 and A2 although in A2, at a position of maximum width of the canal (Fig 109) the bridge was very thin and perhaps deficient in its lateral aspects. Although the section shown in Fig 109 has some superimposition of debris (mainly dentine produced during preparation) over the site of primary bridging, it is possible to trace a seemingly intact calcific barrier across the pulp remnant. Sections of A2 taken in narrower parts of the canal showed that cellular secondary dentine deposition from the walls had lined the bridged area (Figs 111, 112). The cells beneath this area were cuboidal in nature and regularly aligned along the walls. The cells beneath the calcific area in the wider portion of the canal (Fig 109) appeared to be mainly fibroblasts and did not have any definite pattern or increase in number when compared with the cellular nature of the rest of the pulp.

Deeper Pulp Tissue: The entire pulp in A1 and A2 was inflammation free. The same was true of the deeper pulp tissue in A9, although a small localised chronic inflammatory area was present in the lesion zone. All pulps were fibrous in nature.

Internal resorption: had occurred in A1 and A2 but had subsequently been repaired with cellular secondary dentine. Active resorption was progressing in A9 near the lesion area (Figs 118, 119). Nearer the apex of this tooth, repair by cellular secondary dentine had occurred.

Cellular secondary dentine - considerable deposits of this calcific scar tissue were present on the entire walls in A1 and A2 and partially on the bridge in A2 (Fig 111, 112). Deposition in Y9 (Figs 120, 121) was confined to the region near the apical foramen.

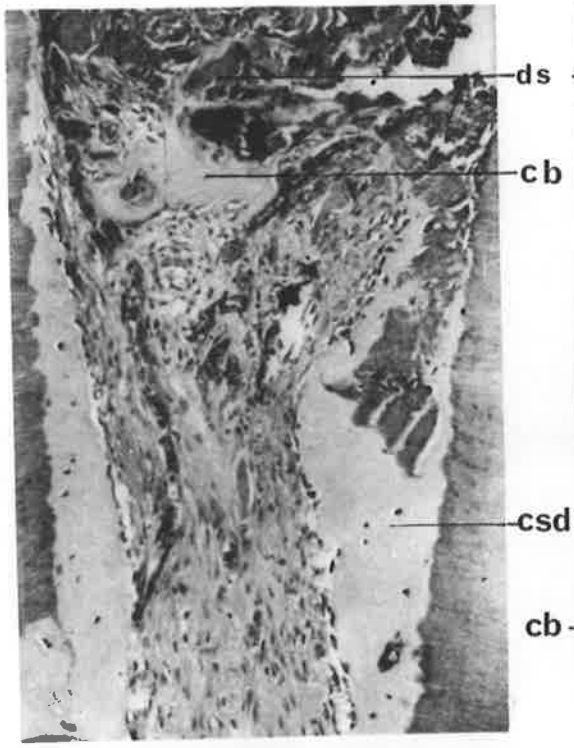


Fig 109 A2 3/ H&E x 100 E.P. 172 days

LESION AREA. SUPERFICIAL DEBRIS CONTAINS NUMEROUS DENTINE SPICULES (d.s) CALCIFIC BRIDGE FORMATION - THICK IN CENTRE, EXTREMELY THIN LATERALLY, THICK LAYER OF CELLULAR SECONDARY DENTINE (c.s.d) IS PRESENT ON THE CANAL WALL.

Fig. 110. A2 3/ H&E x 400

DENTINE SPICULES (d.s) EVIDENT IN SUPERFICIAL DEBRIS LAYER. ALONG WITH DE-GENERATED CELLS AND DRESSING MATERIAL. CALCIFIC BRIDGE (c.b) IS PRESENT.

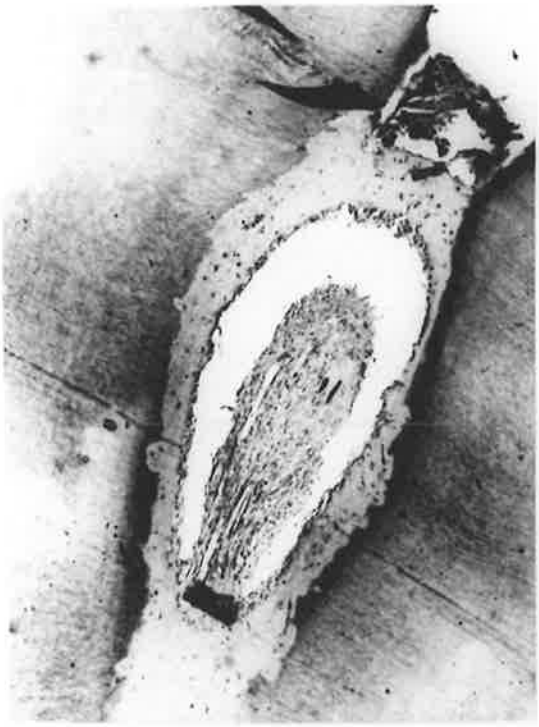


Fig. 111 A2 3/ H&E x 40 E.P. 172 days

SECTION SLIGHTLY LATERAL TO FIG 109 CELLULAR SECONDARY DENTINE DEPOSITION HAS EXTENDED TO LINE THE BRIDGE. PULP RETRACTION IS AN ARTEFACT

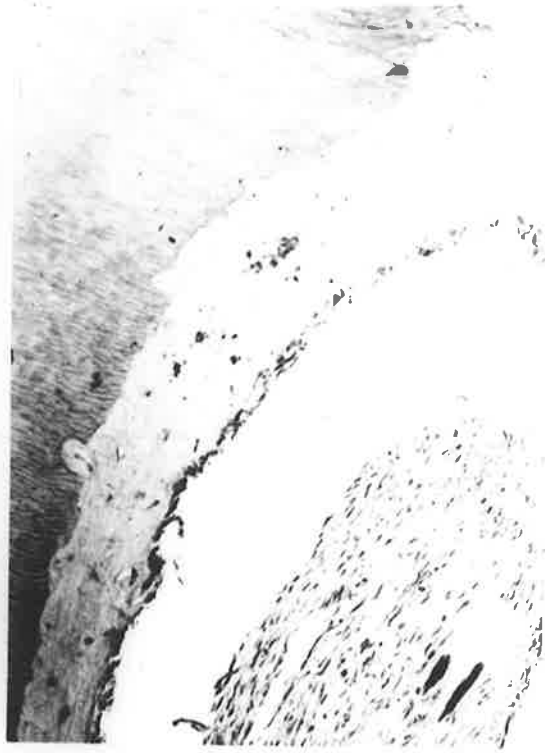


Fig. 112 A2 3/ H&E x 100

CELLULAR SECONDARY DENTINE FILLING THE PREVIOUS RESORPTION LACUNAE.

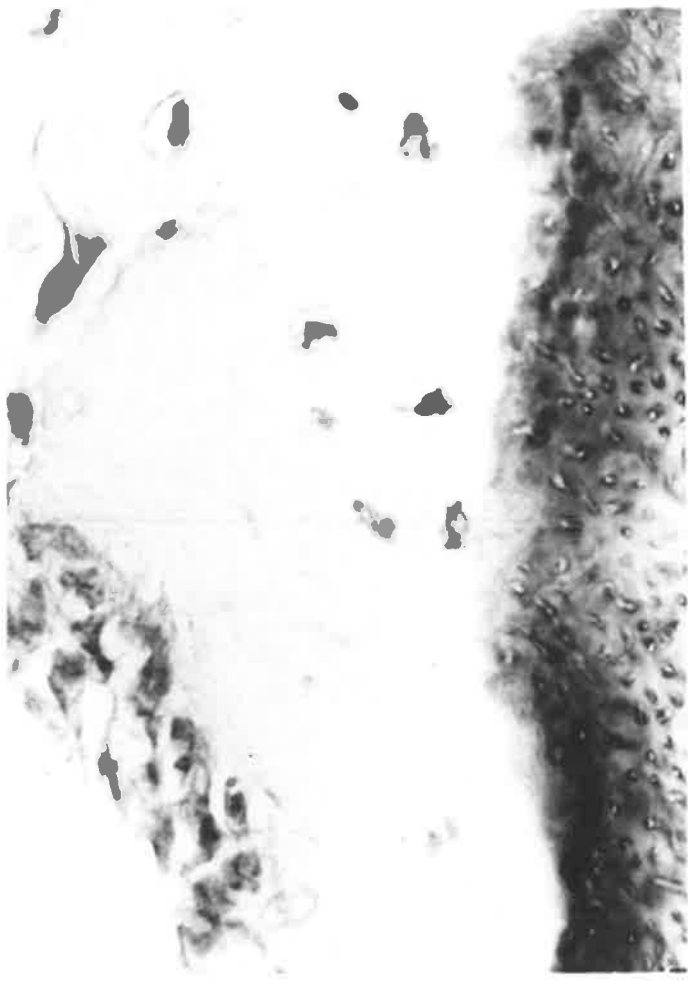


Fig 113. A2 3/ H&E x 400 E.P. 182 days

CELLULAR SECONDARY DENTINE. CELL INCLUSIONS DO NOT HAVE A CANALICULAR STRUCTURE. LINING LAYER OF CELLS ARE CUBOIDAL.

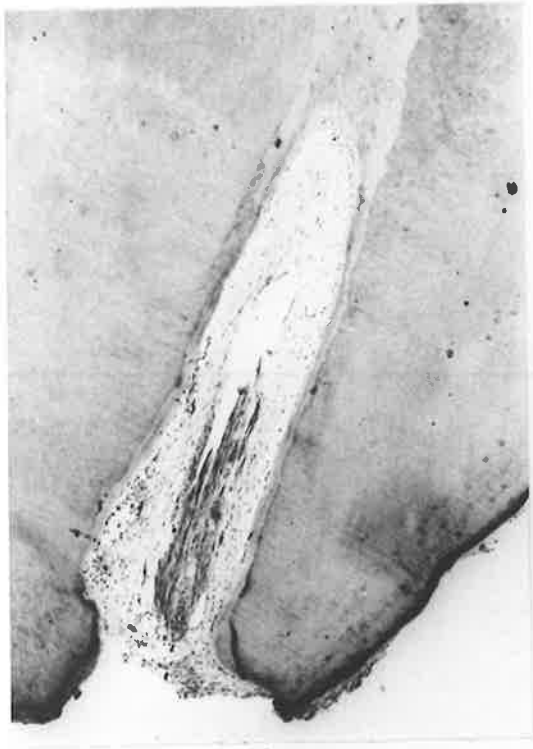


Fig 114. A2 3/ H&E x 40 E.P. 182 days

PULP TISSUE - FIBROUS IN CHARACTER AT THE APICAL FORAMEN

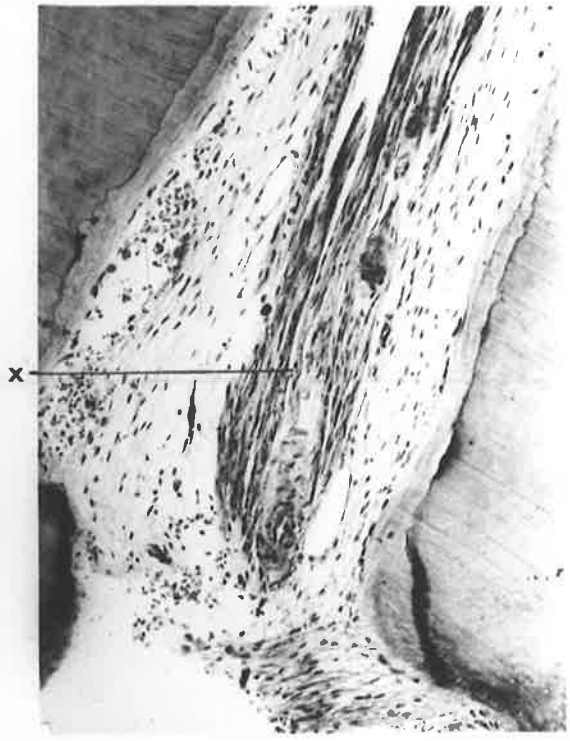


Fig 115 A2 3/ H&E x 100

APICAL PULP TISSUE SHOWING ABSENCE OF INFLAMMATION. BLOOD VESSELS AND NERVES ARE SHOWN AT (x)

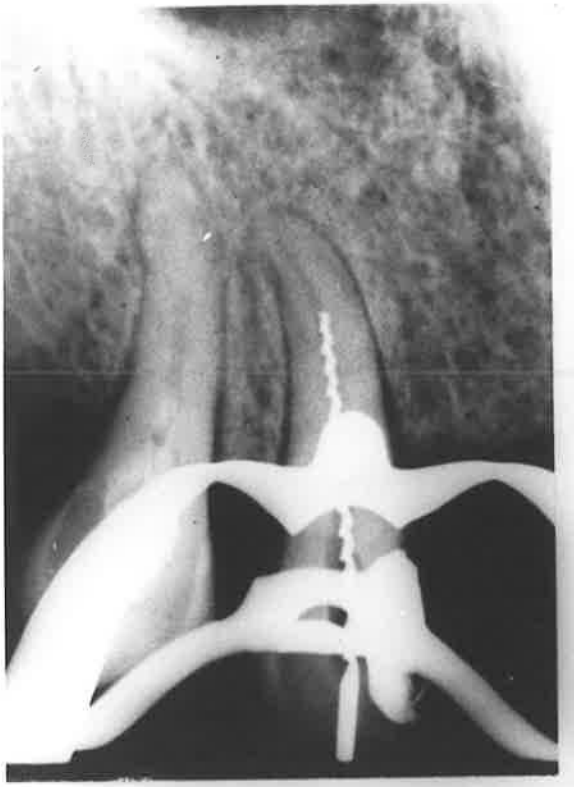


Fig 116. A9 2/ RADIOGRAPH AT TIME OF OPERATION SHOWING AMPUTATION SITE.

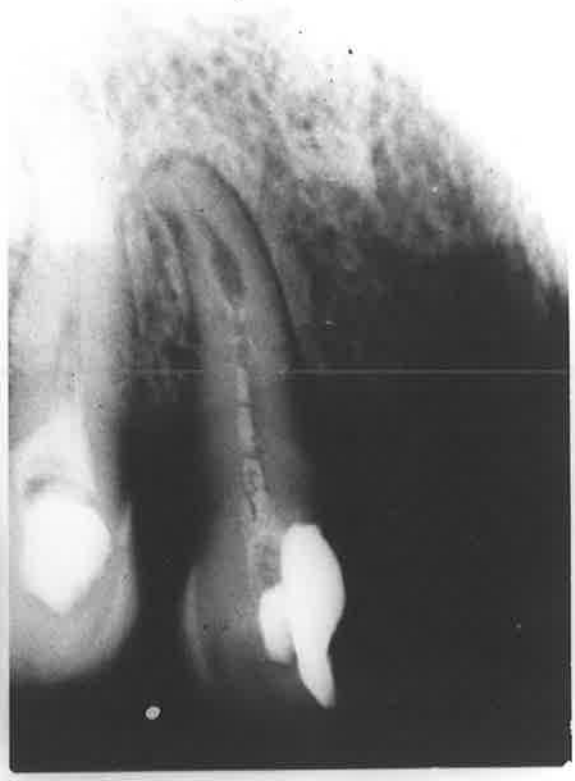


Fig 117. A9 2/ E.P. 172 days. INTERNAL RESORPTION HAS OCCURRED BELOW AMPUTATION SITE

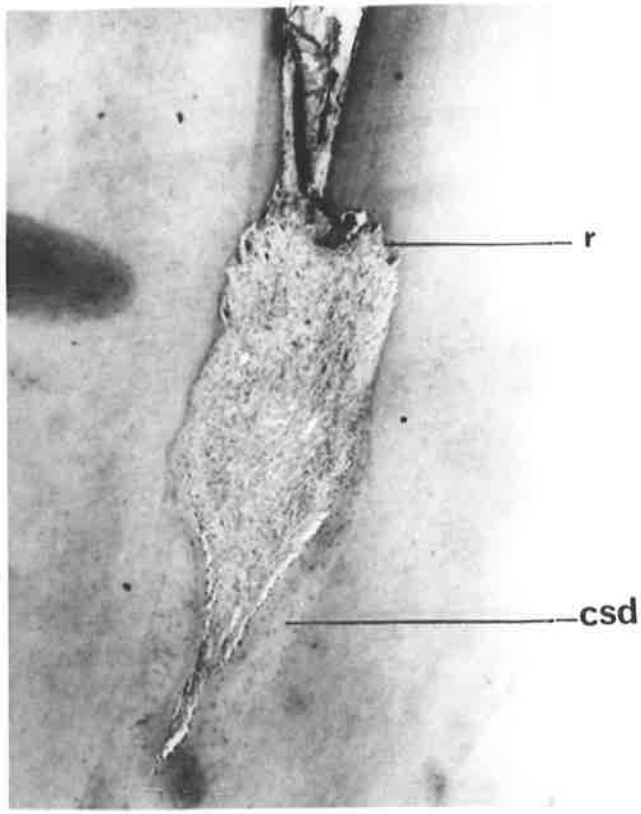


Fig 118. A9 2/ H&E x 40. E.P. 172 days.

SHOWING AMPUTATION SITE INTERNAL RESORPTION (r) AND CELLULAR SECONDARY DENTINE FORMATION (c.s.d)

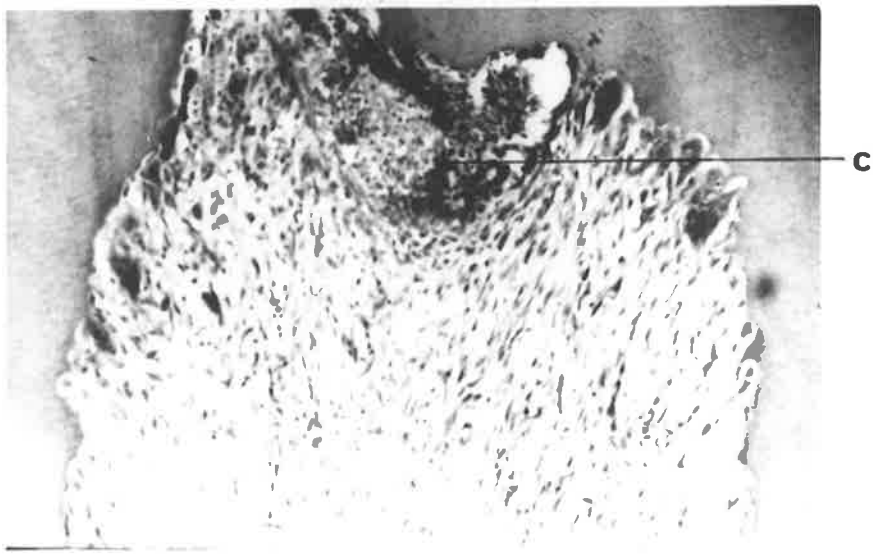


Fig 119. A9 2/ H&E x 100.

ACTIVE RESORPTION BY MULTINUCLEATED GIANT CELLS. LOCALISED AREA OF CHRONIC INFLAMMATORY CELLS (c) NO ATTEMPT AT REPAIR OF AMPUTATION WOUND.

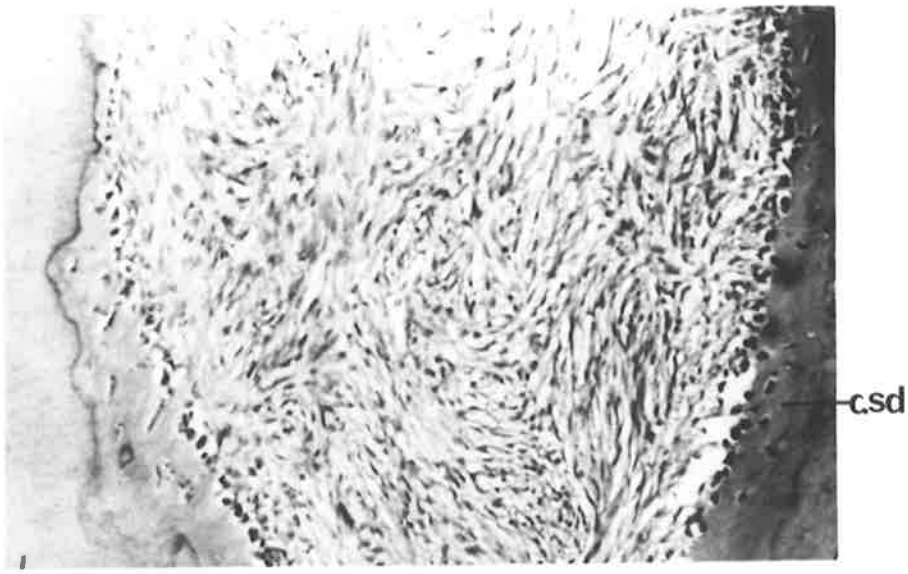


Fig 120. A9 2/ H&E x 100 E.P. 172 days.
DEEPER PULP TISSUE. CELLULAR SECONDARY DENTINE DEPOSITION (c.s.d)

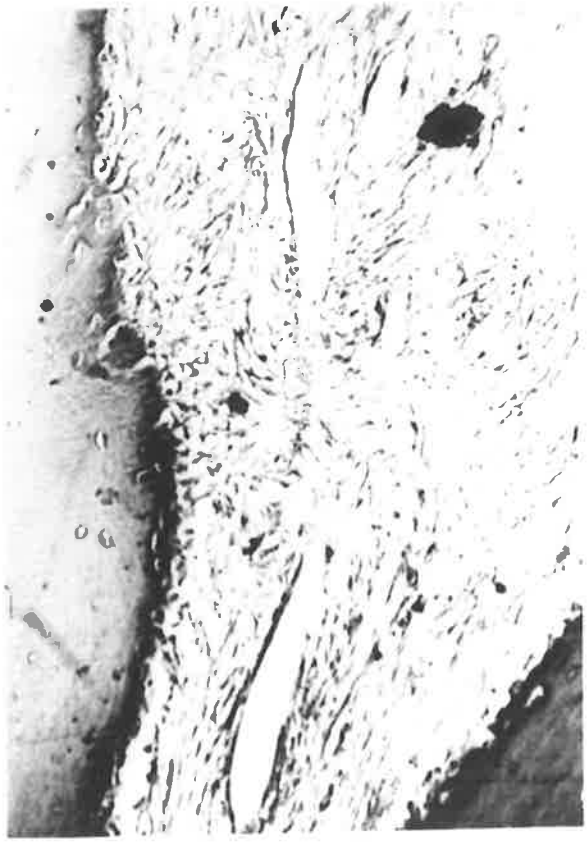


Fig 121. A9 2/ H&E x 100
APICAL PULP TISSUE WITH CELLULAR SECONDARY DENTINE.

325 days: Case No A13 (♂ 46 yrs).

No sections through the amputation area were available.

Deeper pulp tissue: The pulp showed a degree of fibrosis which could be normal for the age group. No inflammation was present.

365 days: Case Nos A10, A11, A12 (♂ 46 yrs).

Lesion Area: A10 (Fig 122). No clearly outlined amputation zone was evident. The superficial area showed many impacted dentine spicules (Fig 123) lying within pulp which was undergoing hyalinisation and calcification. No lesion area was seen in sections of A11, A12.

Deeper Pulp: The entire pulp in A10 was undergoing hyalinisation and calcification (Figs 122, 124).

The pulp in A11 was in a state of acute inflammation. Fibrous tissue was observed in A12 with signs of ingrowth from the periapical tissues.

Fig. 122. A10 8/ H&E x 40 E.P. 365 days.
PULP REMNANT SHOWING GENERALISED HYALINISATION AND CALCIFICATION

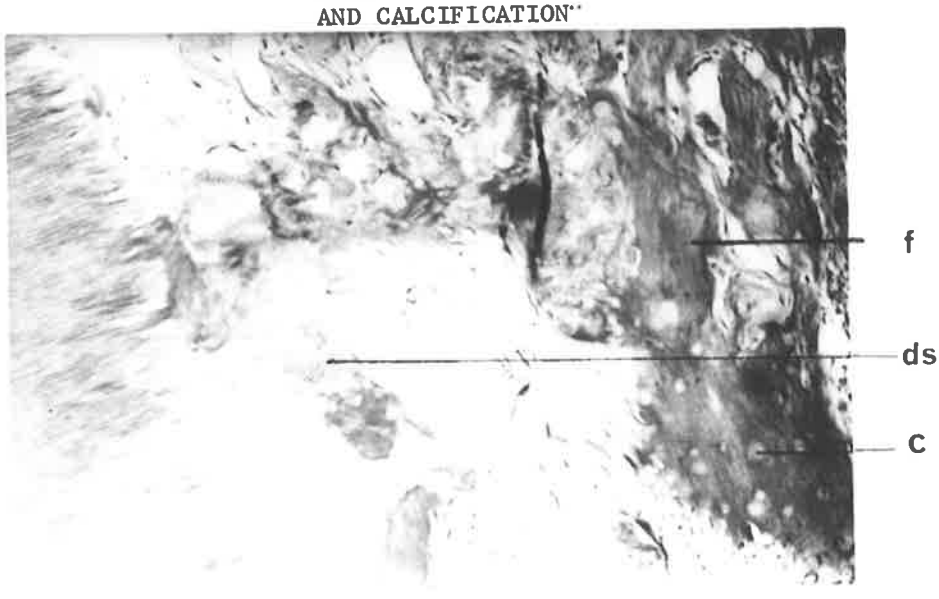


Fig 123. A10 8/ H&E x 100 E.P. 365 days.
LESION AREA SHOWING SUPERFICIAL LAYER CONSISTING OF EXTENSIVE AREAS OF DEGENERATING CELLS, DENTINE SPICULES (d.s) ISOLATED FIBRES (f) AND CALCIFIC AREAS (c).

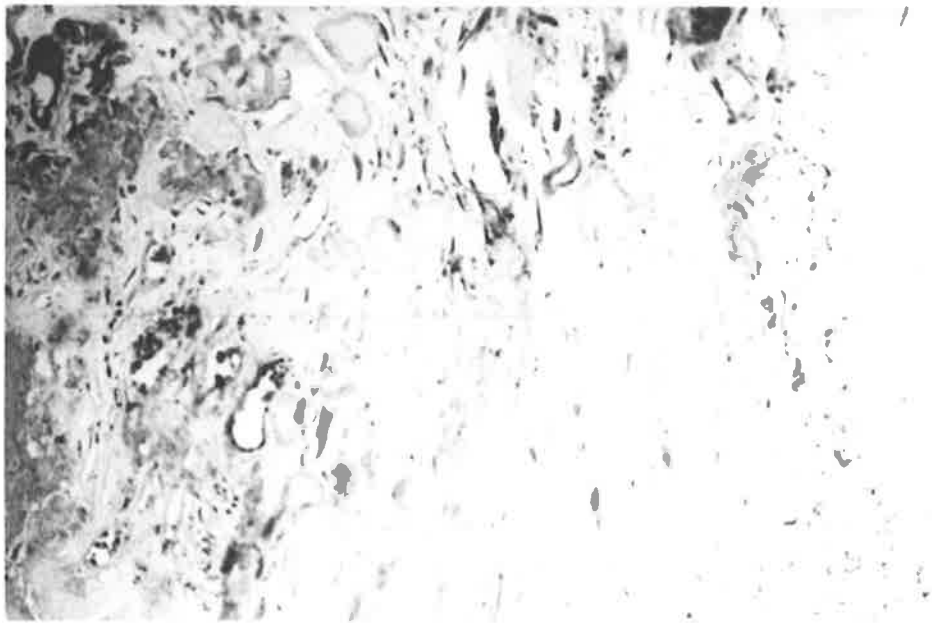


Fig 124. A10 8/ H&E x 100. E.P. 365 days.
DEEPER PULP TISSUE SHOWING HYALINISATION AND AREAS OF DIFFUSE CALCIFICATION.

ANALYSIS OF REACTIONS IN ADULT GROUP

Untreated teeth: The pulps of the untreated teeth showed interesting age changes.

Vacuolisation of the odontoblasts has been considered to be a preparation antefact due to slow fixation of the cells (Nyborg 1955). Similarly the vascular dilatation observed in these teeth is considered to be due to the trauma involved in the extraction. The fibrous nature of the pulp is significant - also the pulp stones in AC1 and AC2 and the diffuse calcifications in AC3.

Treated teeth: The initial reactions to amputation and dressing with calcium hydroxide were not observed due to difficulty in obtaining adult material for these experimental periods.

24 days: Early repair by calcification was evident (A18) and definite reactive zones were present. There was superficial debris (containing many dentine spicules); a necrotic zone and below this a dense zone consisting of fibrous and hyalinised tissue and some early calcific deposits; the underlying pulp had undergone fibrosis. Internal resorption was first observed at 38 days post-operatively (A18)

Some healing pulp remnants observed at 35 - 38 days showed the same definite layers described above but there was no establishment of a continuous calcific bridge. Repair of the resorbed dentine walls by cellular secondary dentine was seen at 105 days. A continuous calcific bridge was present at 172 days (A2), its nature being that of dystrophic calcification in which some cells had become surrounded by a mass of calcific material. Dentine spicules were constantly present in relation

to the lesion areas and were found in intimate contact with the newly formed bridges. Their presence in this region may have been to provide a stimulus to organisation and calcification.

A striking contrast with the positive repair response observed in A2 was provided by A9, both teeth being from same patient and of the same experimental period. There had been no attempt at repair, in the amputation site in A9. Resorption of the dentine in the superficial region of the pulp was active. A small localised region of chronic inflammation was present adjoining the lesion area and no doubt accounted for the complete absence of repair. The rest of the pulp surprisingly appeared extremely healthy and active cellular secondary dentine deposition had occurred. After this relatively long experimental period, although the adverse reaction had been minimal, it would be difficult to make a long term prognosis.

After one year, case (A10) showed generalised hyalinisation and calcification. Although the trauma involved in the amputation may have produced degeneration, it is probable that the pulp exhibited some degree of diffuse calcification before operative intervention.

COMPARISON BETWEEN YOUNG AND OLDER GROUPS

Little overall difference in the success of the treatment in both groups was revealed. Of the 21 roots in young subjects which were assessable, 8 (38°/₀) were judged to be successful; 7 (33°/₀) doubtful; and 6 (29°/₀) failures. In the older group, of the 23 roots which could be evaluated, 11 (48°/₀) were successful; 3 (13°/₀) doubtful; and 9 (39°/₀) failures.

Differentiation of the pulp in the lesion area into reactive zones occurred at similar periods, but the repair response in relation to deposition of calcific material seemed more prolific in the older age group. The repair of resorbed dentine by cellular secondary dentine was more pronounced in the adult group.

One reason for the increased calcific deposition in the older pulps may be due to the decreased blood supply. As has been shown in the untreated cases (AC1, 2, 3) calcific changes often occur in the pulps of the older age group. Thus the older pulps may have had a "calcific potential" due to their physiological state, which could explain the reaction following partial pulpectomy.

The second response which was observed in the young group, namely that of resolution with no other changes, was not seen in the older group. The only case in the older group (A9) which showed a somewhat similar response had a region of chronic inflammation at the surface of the wound which no doubt prevented repair.

Thus the pulps of the older patients in this study showed a capacity to undergo repair following partial pulpectomy, which

compared favourably with the younger pulps. The pulps in the older group predisposed to a rapid calcific response while the more profuse blood supply of the pulps in the younger group favoured a less calcific type of repair or solely resolution.

GENERAL FEATURES

DENTINE SPICULES

Dentine spicules, lying in close relationship to or impacted in, the pulp wound have been a constant observation in this study. Similar observations have been made in most studies on pulpotomy and partial pulpectomy as discussed earlier.

These fragments may, acting as physical or chemical agents provide a centre for repair and finally, calcification. Dentine spicules have been used by many workers (e.g. Castagnola 1953) as a pulp dressing material alone or in combination with other agents with relatively satisfactory results. Hoffman (1937) claimed 82°/. success, judged histologically, following pulp capping with autogenous dentine splinters and alien sterilised dentine powder.

CALCIFIC BARRIER

Calcific barrier formation, either partial or complete was present in 10 out of 35 roots of both young and older pulps whose experimental periods were longer than 28 days. Machida (1960) reported 17 out of his 25 cases with partial or complete dentine bridge formation while Nyborg (1965) found this hard tissue formation in 7 out of 15 cases.

The incidence of healing by resolution rather than by fibrous and calcific repair in the young groups means that the percentage of cases showing calcific bridging in any study will be dependant on the age of the subjects under treatment.

No case was seen in this study in which tubules were present in the calcific bridge or "dentine layer". Nyborg (1965) reported one case of "canalised hard tissue" and Machida showed a photomicrograph of another supposedly tubular dentine bridge. The latter photomicrograph was not convincing evidence of tubular dentine formation or of a newly formed odontoblast layer. The appearance could and probably was, due to the position of sectioning - dentine walls giving the appearance of a complete bridge composed of tubular dentine while the underlying pulp showed a normal odontoblast layer.

The bridge or barrier in this study generally consisted of so-called dystrophic calcification as described by Berman and Massler (1958). This presented a somewhat faintly laminated appearance with areas of cell inclusions. These cells had apparently become enclosed during calcification of the surrounding tissue, and had undergone necrosis.

The pulp cells under an established bridge had the appearance of normal fibroblasts and were orientated parallel to the axis of the bridge. Rohner (1940) claimed that a new odontoblast layer was formed under a newly formed tubular dentine bridge, but his published photomicrographs, in common with the previously discussed case presented by Machida, did not substantiate these claims, and in the present author's opinion the appearance of an apparent dentine covering, in Rohner's study, was due to faulty sectioning technique and interpretation. The other published photomicrographs by Machida showed a similar appearance to those seen in the present investigation - in that the cells bordering the bridge were apparently fibroblasts and were orientated parallel to the bridge.

While complete bridging was only observed in cases with healthy pulps, partial bridging did not necessarily signify a healthy underlying pulp.

Several cases showed chronic inflammation below deficiencies in the bridge. This same observation was made by Nyborg (1955) on pulps treated by capping.

INTERNAL RESORPTION

Internal resorption was frequently observed in the present study from 14 days in the younger and 38 days in the older group. This resorptive process was no doubt the result of an increased blood supply due to the inflammation accompanying the pulp trauma. This has been a common observation in all studies and has usually been ascribed to circulatory changes. Prior to resorption the odontoblast layer is destroyed.

CELLULAR SECONDARY DENTINE

The repair of the resorbed dentine surface, which was related to reduced circulation, was by a cellular calcific material. Lining this layer were small cuboidal cells which most probably were derived from the multipotential undifferential mesenchymal cell of pulp. These cuboidal cells appeared to be incorporated in the layers of calcific material which were laid down in a matrix of collagen formed in a regular pattern as shown in Y6. It would seem that these cells were actively involved in the process of deposition.

The calcific material has been described in this text as cellular secondary dentine. This was considered a logical terminology because of the nature of the material; the cells considered to be responsible; and the site of deposition. The cells which initiated the response and replaced the odontoblasts were thought to have been derived from the undifferentiated mesenchymal cells of the pulp. The calcific material deposited was on resorbed dentine and so may be considered as a type of secondary dentine. The term cellular secondary dentine was thought to be apt and specific to describe this layer of calcific material containing the cell inclusions. Calcific scar tissue or possibly osteoid were also considered to be suitable terms.

This material has also been described as cementum (Kronfeld 1953) (Stones 1954) (Machida 1960) and "cementum like" tissue (Laws 1962). The latter term is descriptive of the appearance of this material but is neglectful of the basic source of the deposition. The present study has not shown any suggestion of proliferation of cementum actively

formed by cementoblasts along the walls, but rather the process of repair has been due to metaplasia of the undifferential mesenchymal cell of the pulp. For this reason it would seem misleading to call this deposit cementum.

CLINICAL OBSERVATIONS AND ASSESSMENT

The comparative clinical and histological results further emphasise the inadequacy of judging material on a clinical basis - especially in relatively short term cases (up to 1 year). The difference in prognosis judged by the two methods of assessment was marked in both age groups.

In the younger group Clinico-radiographic success was 94°/₀ when all clinically assessable cases were considered. The percentage success of histologically assessable cases was 38°/₀ while 33°/₀ were considered doubtful. In the same histologically assessable cases 90°/₀ were clinico-radiographically successful and 5°/₀ doubtful.

In the older group 48°/₀ were histologically successful; 13°/₀ doubtful. Comparative clinico-radiographic figures for the same cases were 74°/₀ successful and 5°/₀ doubtful. As one case was not assessable histologically (A22) and Clinico-radiographically it was judged successful, the overall clinical success rate was 75°/₀ (doubtful 4°/₀).

The differences between the histological and clinico-radiographic results are significant.

Radiographic interpretation of the results of partial pulpectomy presents a problem. Complete bridging has been considered a sign of one type of healing of a completely healthy pulp but partial bridging may indicate chronic inflammation beneath the deficiency. Radiographically it seems very unlikely that small deficiencies in the bridge could be detected.

Internal resorption which occurred in the present study was observed radiographically in some but not all of the cases. This process, which

may be expected in many cases following partial pulpectomy merely indicates that at some stage the pulp has had or still has an increased blood supply. It is a radiographic sign to be expected in a large percentage of treatments but cannot be taken as positive evidence of a healthy reaction.

Deposition of cellular secondary dentine, thus repairing the resorbed dentine is a positive sign of repair, but chronic inflammatory lesions may still exist in the pulp and the prognosis may be unsatisfactory.

If the deposition of cellular secondary dentine goes to the stage of complete obliteration of the canal, it would indicate positive evidence of successful healing.

The converse is not however true. In the present study some of the pulps in the young group were healthy but showed no sign of bridging, internal resorption or subsequent repair by cellular secondary dentine. Thus radiographically no positive evidence from these factors would be available. Completion of root formation in a tooth which previously had an open apex may be the only positive evidence of a retained healthy pulp in young teeth.

Absence of any periapical change - either in the form of a rarefying osteitis or a condensing osteitis, is only of real value after a relatively long observation time. In the present study only 5 of the 25 teeth with pulp pathology showed positive periapical radiographic changes. Long term radiographic studies are therefore essential to assess the success of this treatment on a clinico-radiographic basis.

COMPARISON OF HEALING FOLLOWING THE VARIOUS FORMS OF VITAL PULP THERAPY

General differences in the healing observed following partial pulpectomy compared with studies dealing with other forms of vital pulp therapy are:

(1) Nature of bridge. The present study has not shown a canalised bridge and only one of consequence has been reported (Nyborg 1965). Bridge formation is generally in the form of a dystrophic calcification following partial pulpectomy with no differentiation of the underlying pulp into odontoblasts or "odontoblast-like" cells.

Pulp capping and pulpotomy procedures both have been shown to produce, in a percentage of cases, a bridge consisting superficially of a dystrophic calcific layer, but lined with tubular secondary dentine on the pulpal aspect. A layer of odontoblast-like cells, have been shown to line the barrier. The reason for this difference in response in the regions involved in pulp capping, pulpotomy and partial pulpectomy may be related to anatomical and physiological factors, which modify the reactions. In the studies of pulp capping and pulpotomy opinions differ whether odontoblasts from the undamaged bordering areas undergo mitosis and slowly line the bridge or whether odontoblasts differentiate from the undifferentiated mesenchymal cells of the pulp and form the new dentine under the bridge.

In the apical region the cuboidal shaped odontoblasts may not have the same power to undergo mitosis from the neighbouring areas or

alternatively the un-differentiated mesenchymal cell in this region may only be capable of producing a cell which can lay down a non-specific repair tissue rather than canalised dentine.

(2) Internal Resorption generally has not been reported following pulp capping but a study on pulpotomy by James et al (1957) reported the incidence as 20°/₀ of adult treated teeth. Internal resorption occurred in 10 (23°/₀) of the 44 histologically assessable teeth in the present study. Both pulpotomy and partial pulpectomy involve considerable trauma to the pulp stump, which produces the increase in vascularity resulting in internal resorption.

(3) Deposition of Cellular Secondary Dentine. The prolific deposits of this calcific scar tissue on the previously resorbed dentine surfaces would seem to be a feature of repair following partial pulpectomy which is not parallelled either in pulp capping or pulpotomy. It is, however, similar to apical repair by cementum following total pulpectomy. This deposition has been considered to be influenced largely by the blood supply - occurring when there is a reduction from the normal supply at any particular time. There are certainly anatomical differences in the blood supply to the various regions of the pulp as has been discussed earlier, and it is probable that differences in response are due to these factors. In the apical region, branching from the main vessels is minimal, and there are few capillary loops in the sub-odontoblastic layer. These features may be of some significance.

The nature of deposit indicated that this material had been laid down by an osteoblast type cell which could only have been derived from the undifferentiated mesemchymal cells of the pulp. The cells of the apical region, are in a transition zone between pulp and periodontal membrane, and produce this relatively non-specific calcific repair response. This repair reaction may proceed to complete obliteration of the canal.

GENERAL CLINICAL APPLICATION

The main aim of this study was to describe the pulp reactions following partial pulpectomy on a qualitative rather than quantitative basis. Some indication of the prognosis of this therapy has, however, evolved. Repair or resolution of the retained apical remnant has occurred in 43°/₀ of cases with experimental periods greater than seven days. Such a success rate is not satisfactory when compared with other forms of vital pulp therapy. Of these other forms of treatment, total pulpectomy appears to give the best prognosis.

Specific cases do arise, however, where the repair observed in this study may be used to advantage. Such cases may be:-

(1) Teeth of young patients with incompletely developed apices which may not be suitable for pulp capping or pulpotomy, but nevertheless would present great problems if treated by total pulpectomy. An excessively traumatised and contaminated coronal pulp following a fracture of the crown could be one type of case suitable for this treatment. In other instances, where vital pulp tissue has been retained apically, the remnant could be treated by this method to allow completion of root development.

(2) Excessively curved roots where instrumentation to the apex may be extremely difficult or impossible. This may apply particularly where such teeth require restoration by post-retained crowns.

(3) Retained remnants after attempted total pulpectomy. If there is no urgency to place permanent crowns or large inlays over such teeth with retained remnants, it has been shown in the present study that they have a reasonable chance of repair. Much time is saved and distress

to the patient is avoided, if such a retained remnant be treated as a partial pulpectomy. This should be followed by suitable re-examinations and re-assessments. Should deterioration occur the remnant can easily be removed and the root filling extended to the apex. Future improvements in the instrument used to amputate the pulp may so alter the prognosis for this form of treatment that the interesting repair reactions observed in this study may be used as a practical form of endodontic therapy.

SUMMARY AND CONCLUSIONS

1. Repair of the pulp remnant following partial pulpectomy and dressing with Calxyl occurred in both young and older patients.
2. In the young patients the reaction in the retained remnant was either repair or resolution.
3. Repair in both young and older groups, occurred by organisation of the tissues of the pulp under a superficial layer of necrosis, produced by the action of calcium hydroxide.
4. Definite zones were produced in the organising layers due to connective tissue repair which often led to calcification. The zones under the superficial debris or medicament zone and the layer of superficial necrosis were (a) Calcific bridge (b) "Dense zone" of hyalinised or fibrous tissue and (c) underlying pulp.
5. The pulps of the older age group showed a greater calcific repair response than the younger age group.
6. Complete bridge formation was a sign of one type of repair by a healthy pulp remnant.
7. Partial bridge formation was often associated with localised chronic inflammation within the pulp remnant.
8. The calcific bridge was slightly laminated and contained occasional cell inclusions.
9. No canalised dentine was deposited below the primary calcific bridge.
10. The pulp cells below the bridge were apparently fibroblasts and

were orientated parallel to the bridge. No differentiation of new odontoblasts occurred.

11. Internal resorption usually followed the inflammatory reaction due to the injury received by the pulp remnant. Increase in vascularity appeared to be the cause of this resorption.
12. Repair of the resorbed dentine occurred by the deposition of cellular secondary dentine (calcific scar tissue).
13. Cellular secondary dentine deposits could narrow the canal and line the primary calcific bridge. Complete obliteration of the canal could occur by this deposition.
14. Histological assessment gave considerably different results to clinico radiographic evaluation.
15. Success rate in the younger group was 38°/₀, doubtful 33°/₀. In the older group 48°/₀ were successful and 13°/₀ doubtful. The combined results gave successful prognosis in 43°/₀ doubtful in 23°/₀.
16. The pulp reactions following partial pulpectomy differ from those following pulp capping and pulpotomy. These differences are considered to be due to anatomical and physiological factors.
17. Of the various forms of vital pulp therapy, pulpotomy would appear to be the most applicable for teeth with incompletely developed roots. Total pulpectomy gives the best prognosis for teeth with completed apices.

There are, however, specific cases where partial pulpectomy may be the preferable form of treatment for both young and older age groups.

APPENDIX I

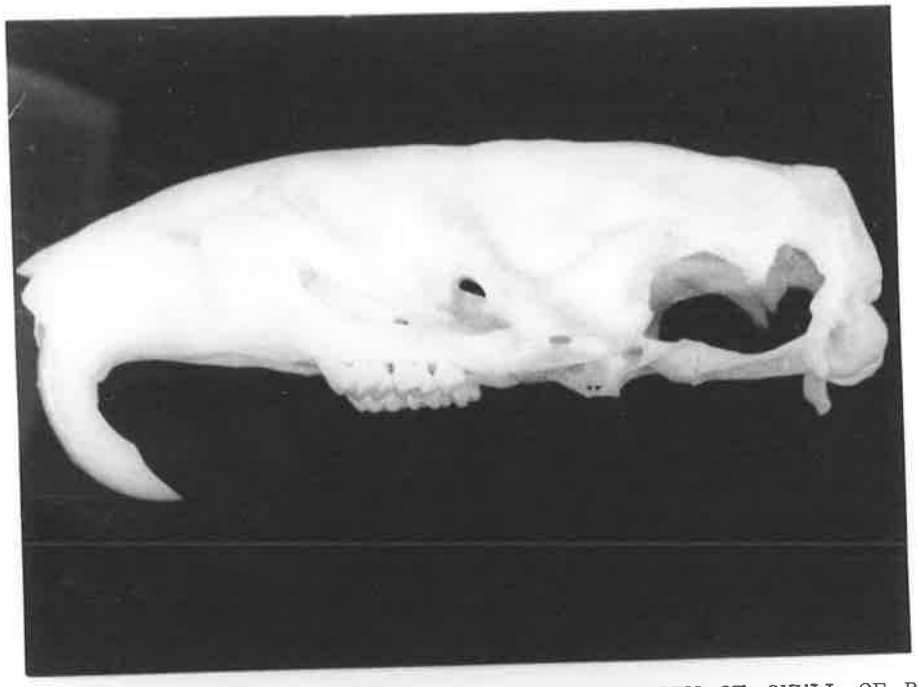
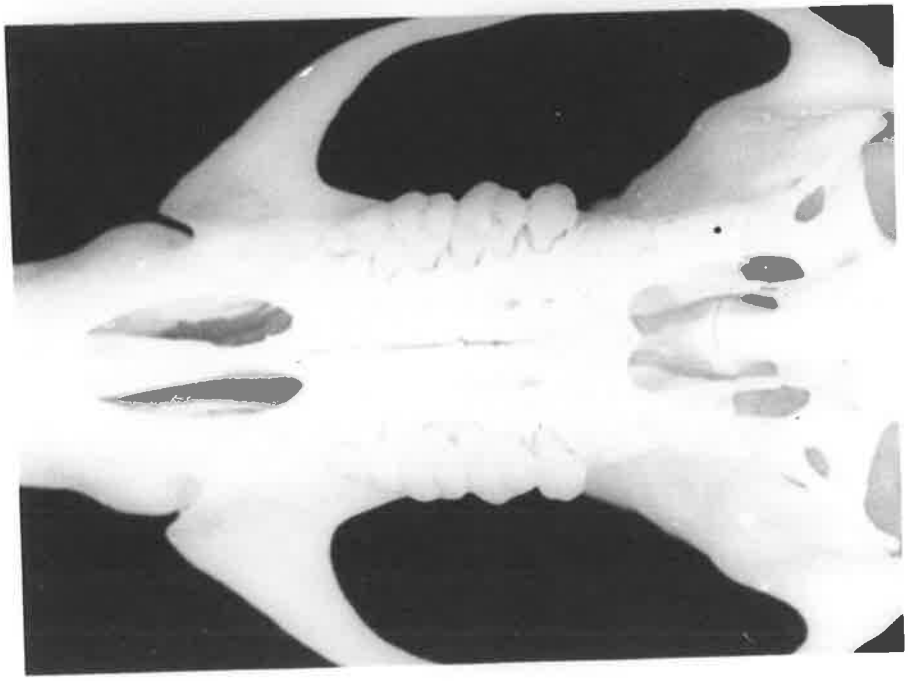


Fig 124, 125. LATERAL AND OCCLUSAL VIEW OF SKULL OF RAT



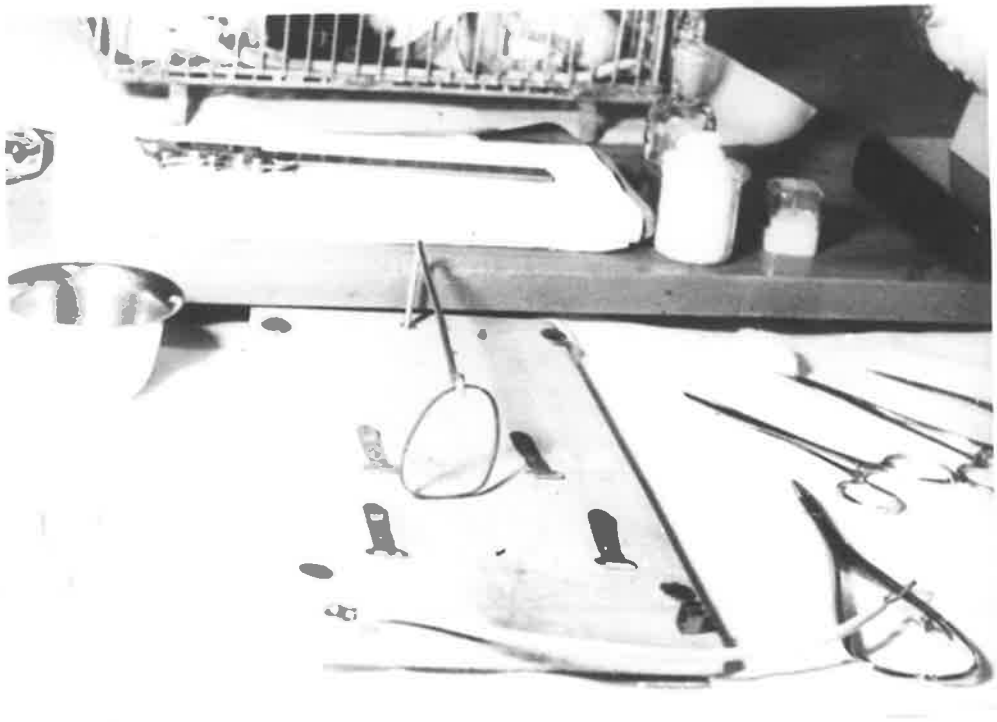


Fig 126. **OPERATING STAND**

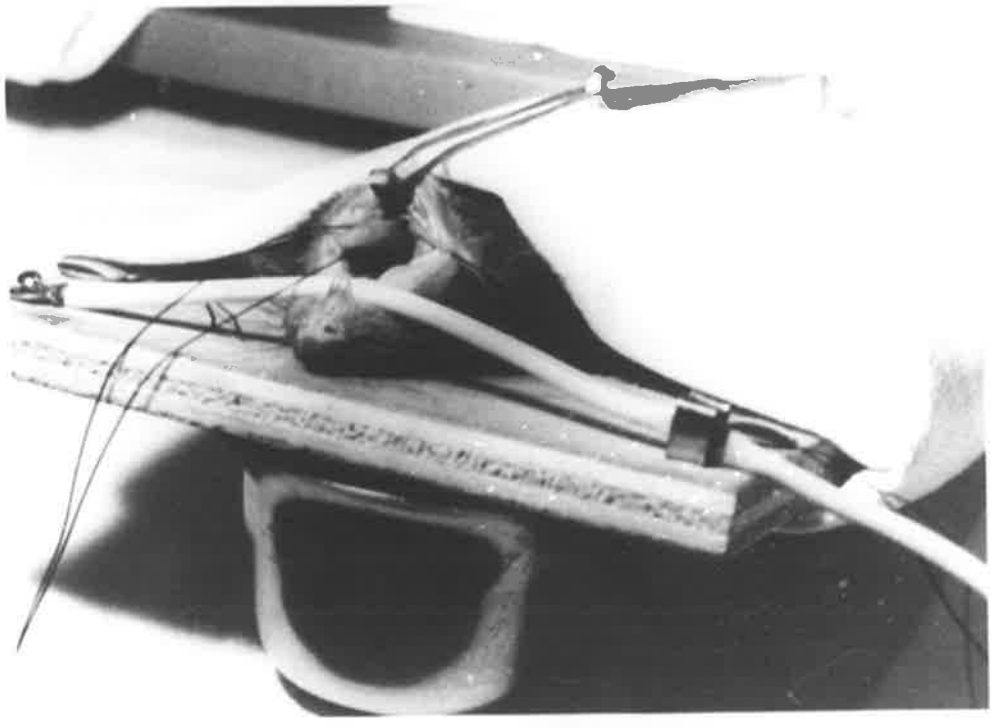


Fig 127. RAT SECURED IN OPERATING POSITION.

Fig 128. ACCESS TO UPPER FIRST MOLAR REGION

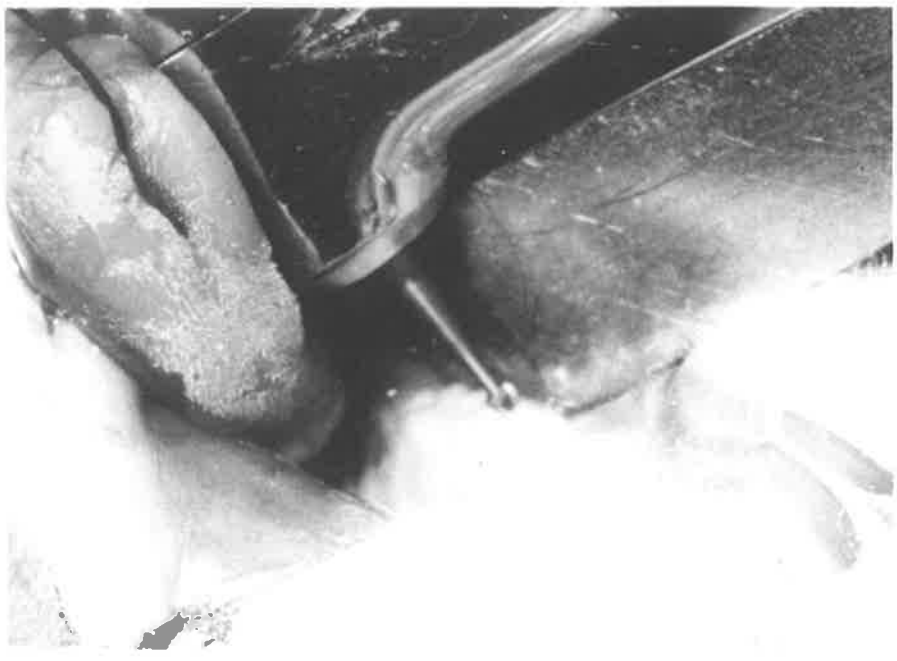


Fig 129. OPENING THROUGH THE MESIO-BUCCAL CUSP INTO THE PULP CHAMBER

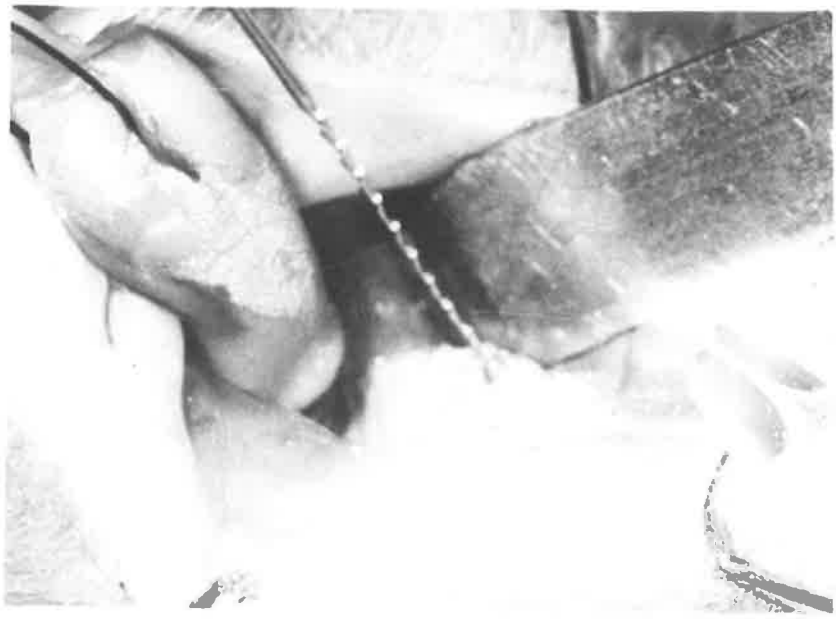


Fig 130. PULP AMPUTATION WITH FLATTENED NO. 2 HEDSTROM FILE

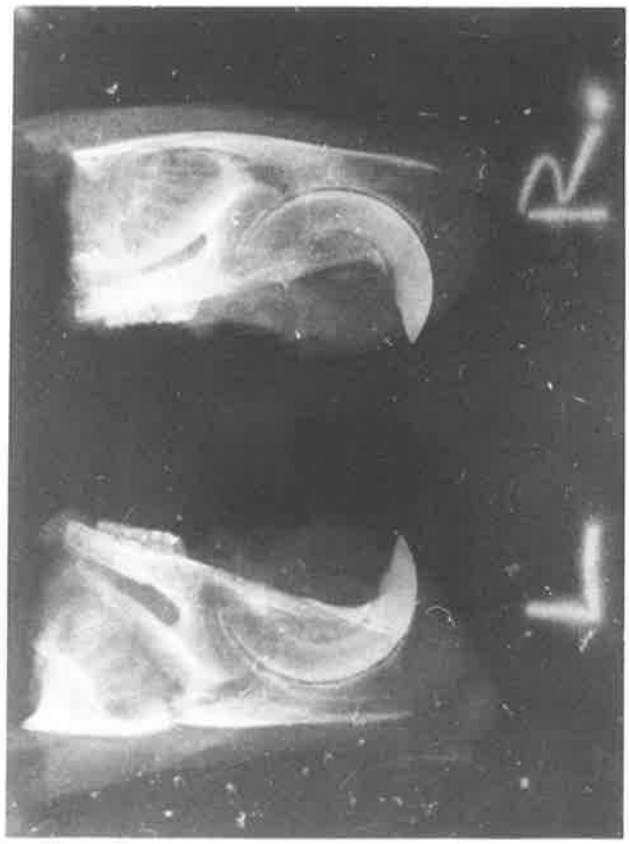


Fig 131. RADIOGRAPHS TAKEN AFTER SACRIFICE SHOWING THE RETAINED DRESSING WITHIN THE UPPER FIRST MOLAR.

APPENDIX II

RAT STUDY

No.

Date

Animal

Weight

Anaesthetic

Experimental Details:

Date Sacrificed:

Histological Report

APPENDIX III

HISTOLOGICAL TECHNIQUES EMPLOYED

FIXING SOLUTION - FORMOL-SALINE

Formaldehyde (40%)	100 ml
Sodium Chloride	9 gm
Tap Water	900 ml

The solution is buffered with magnesium carbonate to excess.

Minimum period of fixation is 24 hours.

Decalcification

All teeth were decalcified in buffered formic acid. The end point of de-calcification being checked radiographically. Specimens were neutralised in 5°/₀ Sodium Sulphate, then processed by a double embedding technique, clearing being effected by methyl salicylate (four days at 37°C). Wax infiltration was effected by 62°C using a wax with a melting point of 60°C. Three changes were effected the last under increasing vacuum max 21" Hg.

Haematoxylin and Eosin

Ehrlich's Haematoxylin	20 minutes
Tap water (Warm)	10 minutes
1°/₀ HCL in 70°/₀ alcohol - differentiate.	
Tap water (warm)	10 minutes
Eosin 2°/₀ W.S. Yellowish	30 seconds

Differentiate in warm tap water (Microscopic control)

Blot

Absolute

Xylol

Mount Xam

Result: Neclei blue, r.b.c's red connective tissue faint pink.

Periodic Acid Schiff

Schiff's reagent was prepared according to de Tomasi 1936 (Pearse Histochemistry Theoretical and Applied 1954, P.425)

Sections were first oxidised in Periodic Acid for five minutes.

Schiff reagent for one hour

Sulphurous water 3 rinses

Tap water for ten minutes

Counterstained in Ehrlich's Haematoxyling.

For the demonstration of Mucopolysaccharides.

Silver Technique

Reticulin impregnation (Stain Technology 21, 64-72, 1946, Lillie R.D.)
Demonstration of reticulin fibres depends on the impregnation of the tissue with a di-ammonical-silver complex (prepared by titration of 10°/₀ AG No. 3 against 880 NH_4OH). Followed by a reduction.
Tissues are first treated with an acidified Potassium Permanganate solution-bleached with oxalic acid and treated with Ferric Chloride. De-ionised water was used throughout. After reduction of the complex (by 10°/₀ Formalin) the section was 'toned' in gold chloride and 'fixed' in sodium thiosulphate. In some instances the sections were counter-stained with Van Gieson, in others simply dehydrated cleared and mounted.

Result: Reticulin fibres stain black.

Mallory's Trichome

5 minutes in 1°/₀ Acid Fuchsin in 1°/₀ acetic acid.

10 minutes in Mallory's aniline blue

Result: Collagen stain blue, nuclei stain red.

Van Gieson

Groot's Iron Haematoxylin (Stain Technology, 24. 1949, 157-163).

20 minutes

Warm tap water	10 minutes
De-ionised water rinse	
Van Gieson	10 minutes

Result: Collagen stains red nuclei stain yellowish

APPENDIX IV

No.

Date.

Name.

Age.

Tooth.

State of tooth.

State of pulp.

State of periodontal membrane.

Anaesthetic.

Details of treatment:

Post-operative history.

Date of extraction.

Radiographic report.

Histological report:

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