THE ENDODONTIC MANAGEMENT OF AVULSED PERMANENT ANTERIOR TEETH

IAN GEOFFREY MARTIN B.D.S. (The University of Sydney)

Treatise submitted as partial fulfillment of the requirements for the degree of Master of Dental Surgery

Department of Operative Dentistry
Faculty of Dentistry
The University of Sydney
1990
ACKNOWLEDGEMENTS

I would like to thank the following persons for their assistance in the preparation of this treatise:

My supervisors, Dr. G.H. Hewitt and Professor W.R. Hume. Associate Professor R.W. Bryant and Dr. A.R. Carter for their advice and encouragement. I would also like to thank Dr. K.S. Lester and his staff for their help with the S.E.M. illustrations. I am grateful to the administration of the Westmead Dental Clinical School for the allocation of time for this work. Finally, I thank Mrs. J.A. Shelley for typing this treatise.
ABSTRACT

The anatomy, histology and pathology of the dento-alveolar complex in relation to avulsion injuries are discussed. Clinical treatment protocols in the management of this type of injury are considered.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Chapter/Section</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acknowledgements</td>
<td></td>
<td>i</td>
</tr>
<tr>
<td>Abstract</td>
<td></td>
<td>ii</td>
</tr>
<tr>
<td>List of Figures</td>
<td></td>
<td>iv, v</td>
</tr>
<tr>
<td>List of Tables</td>
<td></td>
<td>v</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>CHAPTER 1</td>
<td>Brief Historical Review</td>
<td>3</td>
</tr>
<tr>
<td>CHAPTER 2</td>
<td>Incidence and Aetiology of Avulsion</td>
<td>7</td>
</tr>
<tr>
<td>CHAPTER 3</td>
<td>Anatomy and Histology of the Tooth and Supporting Structures</td>
<td>12</td>
</tr>
<tr>
<td>CHAPTER 4</td>
<td>Histopathology of the Tissues Involved in Tooth Avulsion</td>
<td>30</td>
</tr>
<tr>
<td>CHAPTER 5</td>
<td>Treatment of Avulsed Teeth</td>
<td>59</td>
</tr>
<tr>
<td>CHAPTER 6</td>
<td>Effect of External Factors</td>
<td>85</td>
</tr>
<tr>
<td>CHAPTER 7</td>
<td>Endodontic Intervention</td>
<td>97</td>
</tr>
<tr>
<td>CHAPTER 8</td>
<td>Management of External Root Resorption</td>
<td>111</td>
</tr>
<tr>
<td>CHAPTER 9</td>
<td>Prevention of Traumatic Injuries</td>
<td>116</td>
</tr>
<tr>
<td>CHAPTER 10</td>
<td>Clinical Management of Avulsed Teeth</td>
<td>127</td>
</tr>
<tr>
<td>CHAPTER 11</td>
<td>Summary and Conclusions</td>
<td>131</td>
</tr>
<tr>
<td>BIBLIOGRAPHY</td>
<td></td>
<td>133</td>
</tr>
<tr>
<td>Figure</td>
<td>Description</td>
<td>Page</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>Figure 1</td>
<td>Scanning Electron Micrograph of Human Dentine</td>
<td>14</td>
</tr>
<tr>
<td>Figure 2</td>
<td>Light Microscope View of Human Tooth</td>
<td>18</td>
</tr>
<tr>
<td>Figure 3</td>
<td>Light Microscope View showing Human Tooth and Supporting Tissues</td>
<td>22</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Scanning Electron Micrograph showing Human Cementoblasts</td>
<td>24</td>
</tr>
<tr>
<td>Figure 5</td>
<td>Light Microscope View showing Human Cementum</td>
<td>25</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Light Microscope View showing Apex of Rat’s Tooth</td>
<td>26</td>
</tr>
<tr>
<td>Figure 7</td>
<td>Light Microscope View showing Apex of Rat’s Tooth</td>
<td>27</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Scanning Electron Micrograph of an Osteoclast</td>
<td>47</td>
</tr>
<tr>
<td>Figure 9</td>
<td>Radiograph showing Inflammatory Root Resorption</td>
<td>51</td>
</tr>
<tr>
<td>Figure 10</td>
<td>Radiograph showing Replacement Root resorption</td>
<td>54</td>
</tr>
<tr>
<td>Figure 11</td>
<td>Suggested Handling Method for an Avulsed Tooth</td>
<td>67</td>
</tr>
<tr>
<td>Figure 12</td>
<td>Placement of a Fishing Line Splint</td>
<td>76</td>
</tr>
<tr>
<td>Figure 13</td>
<td>Placement of a Rigid (wire) Splint</td>
<td>77</td>
</tr>
</tbody>
</table>
LIST OF FIGURES (CONTINUED)

Figure 14    Avulsion of a Central Incisor    78
Figure 15    Replantation of Tooth    79
Figure 16    Stabilization of Tooth    80
Figures 17 & 18    Radiographs Showing Apexogenesis    99
Figure 19,20 & 21    Sequence of Radiographs Showing Apexification Procedure    104-106
Figure 22 & 23    Illustration of a Mouthguard    118

LIST OF TABLES

Table 1    Osmolality of Different Storage Media    89
INTRODUCTION

The subject of this treatise is "The endodontic management of avulsed permanent anterior teeth". The treatise is primarily concerned with avulsion in humans, although reference is made to some studies in other animals. The work is restricted to the avulsion of anterior permanent teeth, i.e. canine to canine in both the maxilla and mandible.

A significant proportion of the population presents with traumatic facial injuries, often with concomitant tooth avulsion. In the past, lack of knowledge relating to healing of the dental tissues involved in traumatic incidents often led to inadequate treatment of the avulsed teeth. As more information comes to hand through new experimentation coupled with careful reporting of clinical treatments, an improved protocol for management can be formulated to give a better prognosis for the traumatised dental structures.

Avulsion, or exarticulation, involves the complete removal of the tooth from its bony socket. This is usually caused by a direct force to the mouth. Anterior teeth are much more susceptible to avulsion than posterior due both to their position in the dental arch and to their root shape. Posterior teeth
are seldom avulsed, but may be fractured by a direct or indirect force. Divergent root form in posterior teeth may reduce the incidence of avulsion.

The aim of this treatise is to review the literature relevant to this topic and to identify, in the light of current knowledge, a reasonable approach to this clinical problem. Areas of inadequate knowledge and matters which are controversial will be highlighted. In this review the following will be discussed.

1. The causes and incidence of avulsion.
2. The normal and abnormal anatomy and histology of the tooth and supporting tissues.
3. The histologic changes which occur in the involved tissues following avulsion.
4. Treatment of avulsed teeth. A distinction will be made as to the different methods of treatment associated with teeth that have closed apices or open apices (i.e. mature or immature teeth).
5. The types and treatment of root resorption.
7. Preventive measures that can be taken to reduce the chance of injury to the teeth and associated structures.
CHAPTER I

BRIEF HISTORICAL REVIEW

Tooth avulsion and trauma have been problems throughout man's history. Replantation of teeth has been performed for many centuries (Smith, 1975). Fauchard (1749) described the immediate replacement of teeth that have been removed by mistake, which were then "bound to the neighbouring tooth", as quickly as possible.

Hunter (1788) suggested that extensively damaged teeth should be "extracted and replanted, after subjecting the tooth to boiling water in order to cleanse it perfectly and to destroy its vitality entirely". He suggested that this treatment would prevent further destruction of the teeth, since the tooth was now dead and could no longer be a seat of disease (Cohen and Burns, 1984).

Wadsworth (1876) alerted the dental profession to replantation. In summary, his guidelines for the procedure were:

1. fill the root canal before replacing the tooth;
2. splint to prevent movement;
3. do not remove any healthy 'periosteum';
4. rinse in warm water;
5. work as rapidly as possible.

With the exception of the first step, his basic concept was the same as that advocated today.

In "3" above Wadsworth was probably referring to the periodontal ligament (Cohen and Burns, 1984). Wilkinson (1917) and Hess (1944) suggested that the state of the periodontal ligament can be a factor limiting successful replantation (Loë and Waerhaug, 1961). Healey (1953) believed that the success rate of reimplantation was proportional to the amount of periodontal ligament present on the tooth when reimplanted.

In the 1930's, Payne and Hankey had observed the morphology of roots undergoing resorption. Resorption was related to the damage to the periodontal ligament and loss of cementum from the root surface, and osteoclastic activity. Until recently, no distinction has been made between the different types of external resorption i.e. replacement and inflammatory. Down (1957) suggested that "the tooth must be sterilised before replantation". He also suggested that the avulsed tooth should be splinted for three months. Others considered it necessary to scrape the root surface and/or treat the root surface with an antiseptic agent, whether it be in clinical or
experimental situations (Loë and Waerhaug, 1961; Bell, 1970)

Another aspect of replantation that has changed is the treatment of the pulpal space. Healy (1953) presented a case report where a root canal therapy was undertaken prior to replantation. He advised the use of hydrogen peroxide and water to cleanse the root of the tooth. Down (1957) noted that it was "unlikely that the pulp will remain vital when a fully developed root is replanted". However, many different ways of treating the pulp space have been tried. Bell (1970) advocated root canal therapy prior to replantation. He also undertook an apicectomy of the tooth prior to replantation.

Lundquist in 1936 (in Healy, 1953) gave reasons for treatment, which were similar in some respects to present concepts.

He stated that "as far as the 'circulation of the tooth' and its adherent tissues are concerned, it is completely interrupted". With respect to splinting, "the immobilisation of the tooth during the early stages of rapid resorption and repair, prevents the possible stimulating effects on repair, which functional use may provide". He believed that "the tooth becomes in consequence a foreign body".
Hence, there have been methods of treatment considered for the replantation of avulsed teeth, for hundreds and perhaps thousands of years. Most methods have been of an empirical nature. However, today, a scientific basis of treatment is developing.
CHAPTER 2

INCIDENCE AND AETIOLOGY OF AVULSION

INCIDENCE:

Avulsion of teeth is usually associated with a traumatic accident. According to Andreasen (1981), the incidence of avulsion of the permanent dentition following trauma lies between 0.5% to 16%. Many studies show a varying incidence and this variation is due to a wide variety of factors (Jarvinen, 1979; Kemp et al, 1977; Meadow et al, 1984; Ravn, 1974).

York et al (1978) in a survey of 72 children, found 3% of traumatised teeth were avulsed. In contrast, Andreasen (1970) with a large survey involving 1,298 clinical cases of dental trauma, showed an incidence of 16% avulsion. He explained that these cases were attended in hospital, and that more severe injuries are likely to present in a hospital. Martin et al (1990) found an incidence of 13%, under similar circumstances.

In a study by Liew and Daly (1986), patients were treated who sustained traumatic injuries to the teeth outside normal business hours. In this study they found 11.2% of traumatised permanent teeth were avulsed. They concluded that "anterior dental trauma
after-hours may be more severe", compared to that seen during normal business hours.

In contrast, Davis and Knott (1984) undertook a study involving traumatic injuries during normal working hours. There were 313 cases involved, of which 5.2% involved avulsion. However, there is no indication of the relative proportion of avulsion between permanent and primary teeth. A study by Grossman and Ship (1970), looked at a small sample of 54 avulsed teeth. They found that there were more boys than girls presenting with avulsions in a ratio of 2.4:1. The majority of injuries occurred in the 10-14 age group.

Burton et al (1985) made a comparison of traumatic injuries between two different socio-economic areas, using consenting High School Students in a large sample of some 12,000 children aged 12-18 years of age. The overall avulsion rate in this sample was 2.5%, however, in the lower socio-economic area, the avulsion rate was almost twice that of the higher socio-economic group. Sane and Ylipaavalniemi (1988) undertook a study of dental trauma in contact team sports in Finland and they found that avulsion composed 1.3% of the total injuries sustained.

The great variation in incidence of avulsion reported depends on many factors, such as recording
criteria, whether a study is retrospective, and the availability of data. As mentioned, variability also exists when comparing injuries sustained and presenting during business hours with those in an after-hours situation. Considering the wide range of variations reported in the literature, one might conclude that in traumatic injuries involving the permanent dentition, an incidence of 8-10% of avulsion could occur.

There also is a trend in the distribution of teeth injured. A study by Lenstrup and Skellar (1959) using a small sample of 48 patients showed that the upper anterior teeth were more likely to be avulsed. Of these, the upper central incisors were the most affected. In a comparison of avulsions of maxillary and mandibular teeth, Andreasen (1970) found that maxillary teeth were more likely to be avulsed by a ratio of 10:1. A retrospective study by Jarvinen (1979), with a large sample of 1,614 children, showed that the most frequently injured teeth were the upper central incisors (81.7%).

Andreasen (1981a) suggests that avulsion involves a single tooth, but that multiple avulsions are occasionally encountered.
CAUSES:

The literature suggests that causes of tooth avulsion are varied. Any excessive force applied to a tooth will either lead to a fracture of the tooth, or the force will be directed to its supporting structures in a dislodging action; or there could be a combination of these effects. Andreasen (1970) considers that the type of impact may determine the type of injury. He states that a sharp blow may tend to fracture the tooth, whereas a blow that is blunt or padded may be transferred to the supporting structures and give a dislodging action. Davis and Knott (1984) support this finding. Other factors may be involved such as the energy of the force, the resiliency of the teeth and supporting structures and the angle of impact.

Grossman and Ship (1970) found that the greatest cause of avulsion in boys was fighting, while in girls the major causes were bike accidents and falls. Similarly, Liew and Daly (1986) reported that the main causes of avulsion were assaults and fights, bicycle accidents and motor vehicle accidents. According to Andreasen (1981a) the major aetiological factors in the permanent dentition were fights and sporting accidents.
Certain predisposing factors may feature in the incidence of traumatic injuries and hence that of avulsion. The "accident-prone" child is more likely to fall and be involved in trauma to the teeth. Andreasen (1981a) stated that the frequency with which these patients sustained repeated trauma to the teeth was 4 to 30%. A person with either an occlusal discrepancy and/or an incompetent lip may be more susceptible to trauma to the teeth.
CHAPTER 3

ANATOMY AND HISTOLOGY OF THE TOOTH AND SUPPORTING STRUCTURES

An appreciation of the anatomy and histology and the dento-alveolar apparatus is necessary to understand any pathologic process related to avulsion. A brief outline of the literature is presented.

The dento-alveolar apparatus consists of the tooth and the periodontium. The tooth is made up of a crown and root. The crown consists of dentine overlayed by enamel. The root consists of dentine covered by a layer of cementum. The pulp is present in both crown and root. The periodontium is the supporting mechanism of the tooth and is made up of the periodontal ligament, cementum, bone and gingiva. The tooth is held in position by the periodontal ligament, the fibres of which are inserted into bone and cementum.

When a tooth is avulsed, the root and socket are the most important consideration in relation to treatment.

A discussion of the components of the tooth and periodontium will follow.
TOOTH:

Enamel:

Enamel is a highly mineralised tissue, being 96% inorganic and 4% organic and water. The inorganic component is a crystalline structure called Hydroxyapatite. The basic units of structure are the enamel rods, which are arranged in an organised pattern. Enamel is formed by ameloblasts during formation of the tooth. Ameloblasts are rich in organelles associated with protein production, to yield an organic matrix (Ten Cate, 1985).

Dentine:

Dentine is a mineralised tissue composed of 65% inorganic substance, 20% collagen, 13% water and 2% other.

Several types of dentine exist. Mantle dentine is that dentine which is first formed, being 80-100 \( \mu m \) thick. It contains Korff’s fibres. Circumpulpal dentine is the remainder of the dentine laid down during tooth development. Predentine is the organic matrix that is adjacent to the odontoblast layer.

Within the dentine mass are dentinal tubules (Fig. 1). These form around the odontoblastic processes and run the full width of the dentine. However, there is some controversy with regard to this
Fig. 1  Scanning Electron Micrograph of coronal dentine of a human molar.

D = Dentine tubules;
P = Peritubular dentine;
I = Intertubular dentine
Bar = 1 μm.
(Ten Cate, 1985). As the tubules proceed away from the pulp, they tend to branch. Two types of dentine are associated with the dentinal tubules.

Peritubular dentine is that dentine which lines the tubules.

Intertubular dentine is located between the rings of peritubular dentine (Cohen and Burns, 1984).

The formation of dentine is called dentinogenesis and is undertaken by special cells called odontoblasts, which differentiate from mesenchymal cells in the dental papilla. These cells line the inner aspect of the dentine and have processes which extend into the dentine. Odontoblasts lay down the organic matrix of the dentine, and play a part in the mineralisation of this matrix.

Dentine tubules are wide in young teeth, and as teeth get older the tubules narrow due to the laying down of peritubular dentine (Ten Cate, 1985).

**Dental Pulp:**

The dental pulp is derived from the mesoderm, its developmental precursor being the dental papilla. It is characterised "as a loose, highly vascular and highly specialised connective tissue, which is required for the formation and support of dentine" (Durr and Sveen, 1987). "Being a connective tissue,
it is composed of cells, fibres, ground substances, blood vessels, lymphatic vessels and nerves.

Morphologically, the pulp consists of four zones (Fig. 2).

1. The odontoblastic layer is immediately adjacent to the predentine, and may also contain capillaries and nerve fibres.
2. The cell free zone is next to the odontoblastic layer and is about 40 µm wide. It contains a small number of cells, blood vessels and nerve fibres.
3. The cell rich zone contains fibroblasts, macrophages, plasma cells and lymphocytes.
4. The remainder of the pulp contains fibroblasts, collagen fibres, ground substance, blood vessels and nerve fibres (Cohen and Burns, 1984).

The cells contained in the pulp include:

1. fibroblasts which are spindle shaped cells that form and maintain the pulp matrix with collagen and ground substance;
2. odontoblasts which are highly differentiated and specialised cells, that produce dentine. They contain organelles associated with
protein production. They have their cell body on the periphery of the pulp cavity and processes extending into the dentine within tubules. Complex junctions exist between these cells e.g. gap, zonula occludens and zonula adherens; which may indicate the possibility of communications between odontoblasts.

3. Undifferentiated mesenchymal cells which are large polyhedral cells possessing cytoplasmic processes. These cells are pluripotential and act as precursors for cells of the pulp including odontoblasts, fibroblasts and macrophages (Ten Cate, 1985).

4. Defence cells e.g., macrophages, which are large oval or spindle shaped cells containing lysozomes; lymphocytes and plasma cells which are involved with the immune response (Ten Cate, 1985).
Fig. 2 Light microscope section of human tooth showing the pulp/dentine complex (x40)

O = Odontoblastic layer;
PR = Predentine;
D = Dentine;
CF = Cell Free Zone;
CR = Cell Rich Zone;
B = Blood Vessel.
Fibres:

Different types of fibres are contained in the pulp. Collagen makes up the greatest part of the fibre component of the pulp. Reticular fibres are located around blood vessels and odontoblasts. Von Korff’s organic fibres are associated with odontoblasts and predentine.

The cementing agent of this connective tissue is ground substance. It is principally composed of glycosaminoglycans, hyaluronic acid, chondroitin sulphate, glycoproteins and water.

Its functions include the support of cells and a medium of transport of nutrients from the vessels to the cells.

Vasculature and Lymphatics:

Blood vessels enter the pulp via the apical foramen, and usually consist of one or two arteriolar sized vessels (approx. 150 μm diameter).

Other smaller vessels may enter through accessory foramina or lateral canals. Durr (1987) "considers that for practical reasons there is no collateral circulation, and hence the pulp’s circulation is easily disrupted if the apical vessels are severed".

The main vessels run through the middle of the pulp and give out branches to pulpal extremities.
forming capillary networks of about 4-8 μm in diameter.

Arteriovenous shunts may exist, and these bypass the capillary network. Venules form a complex network which give an efferent flow from the pulp.

Regulation of blood flow in the pulp is under autonomic nervous control (Ten Cate, 1985).

The existence of a lymphatic system is controversial as it is difficult to distinguish between venules and lymphatic vessels morphologically (Seltzer and Bender, 1984). However, Hume (1988) considers the pulp to be well supplied with lymphatics.

**Innervation:**

The nervous supply of the dental pulp is composed of myelinated and unmyelinated large and small diameter sensory fibres. They enter via the apical foramen with afferent blood vessels. They eventually form an extensive nerve plexus in the cell free zone of the pulp - the Plexus of Raschkow. There are also sympathetic nerve fibres associated with blood vessel innervation (Ten Cate, 1985).
THE PERIODONTIUM:

The periodontium is the attachment apparatus of the tooth and is derived from the dental follicle. It consists of cementum, the periodontal ligament, alveolar bone and gingiva (Fig. 3).

Cementum:

Cementum is a hard connective tissue, which covers the root surface. It is partially mineralised with hydroxapatite, having an organic matrix of collagen and ground substance. It is not vascularised. The cementum becomes thicker as the apex of the tooth is approached. Coronally it is approximately 50 μm thick, and apically it can reach 200 μm in thickness.

There are two types of cementum. Acellular cementum is that layer immediately adjacent to the dentine. Cellular cementum covers the acellular variety and contains cementoblasts. There are two types of cells associated with cementum. Cementoblasts form the cementum and line the root surface (Fig. 4). They are round cells and have a cytoplasm containing organelles used for protein production. They retreat as the cementum is formed giving rise to the acellular cementum. If they become entrapped in the cementum, they form the second
Fig. 3  Light microscope section of human tooth showing root, periodontal ligament and alveolar bone (x40)

RD = Root dentine;
PL = Periodontal ligament;
B = Blood Vessel;
A = Alveolar Bone
cell, the cementocyte (Fig.5). These cells have a small cell body with processes occupying canaliculi in the cementum.

**Periodontal Ligament:**

This is a soft connective tissue that is situated between the bone of the socket wall and the cementum of the root. It ranges in width from 0.15-0.38 mm. Its function is to attach the tooth to the wall of the bony socket, and to act as a pain and proprioceptive organ. Many cells are associated with the periodontal ligament. Fibroblasts are involved in response to the continual remodelling of the ligament. They are able to degrade and synthesise collagen simultaneously. Hertwig’s epithelial root sheath, which determines the shape and number of roots of the tooth, is derived from the inner and outer enamel epithelium (Figs. 6, 7). Remnants of this root sheath are called the epithelial cell rests of Malassez.

Fibres present in the periodontal ligament are composed of collagen and are arranged in fibre bundles. Those fibres of the ligament which are embedded in the bone or cementum are referred to as Sharpey’s fibres, and can be partially or wholly mineralised. The blood supply for the ligament is
Fig. 4  Scanning electron micrograph showing cementoblasts on the root surface of human molar.

Bar = 10 μm
Fig. 5 Light microscope ground section of human tooth root showing cementum, with cementocytes and their canaliculi (x10).

C = Cementum;

CC = Cementocytes and their canaliculi;

D = Dentine.
Fig. 6  Light microscope section of apex of immature rat's tooth, showing Hertwig's epithelial root sheath (x4).

A = Alveolar Bone;
PL = Periodontal Ligament;
P = Dental Pulp
R = Root.
Fig. 7 Light microscope section of an immature rat's tooth showing the root apex and Hertwig's epithelial root sheath (x40).
derived mainly from dental arteries, which pursue an intraosseous course, and give off branches (intra-alveolar) that lead to the ligament.

The nerve supply of the periodontal ligament is derived from the inferior and superior alveolar nerves. Two types of nerve endings are present - pain and mechanoreceptor (the latter afford a proprioceptive function) (Ten Cate, 1985).

Alveolar Bone:

The alveolar process is that bone of the jaws in which the teeth are located. There is an outer and inner cortical plate, interposed with spongy or trabecular bone.

The outer cortical plate is composed of fine fibred lamellar bone supported by compact Haversian bone. The inner cortical plate is composed of bundle bone, and is often referred to as the lamina dura. The fibre bundles of the periodontal ligament are inserted into this bone (Ten Cate, 1985).

Dentogingival Junction:

The gingival region most closely associated with the tooth is the dentogingival junction, and is part of the periodontium. It is composed of the sulcular epithelium, which is stratified squamous epithelium,
and is an extension of the oral epithelium; and the junctional epithelium, which is derived from the dental epithelium, and contacts the tooth using a desmosomal system, forming a collar around the tooth.

The blood supply of these described tissues is derived from the continuation of the intra alveolar arteries as they pierce the adjacent alveolar bone.

The nerve supply is via the basal layer of epithelium in the dentogingival junction.
CHAPTER 4

HISTOPATHOLOGY OF THE TISSUES INVOLVED IN TOOTH AVULSION

When a tooth is displaced from its socket, it has undergone mechanical/physical injury and the tissues of the socket are damaged. Bacterial contamination will occur. Hence, there will be tissue reactions in response to the injury, the replantation of the tooth and bacterial contamination and reactions will occur in the pulp, the periodontal ligament and the immediate alveolar bone.

THE DENTAL PULP:

The dental pulp of an avulsed tooth has had its nutritional supply eliminated by the severing of blood vessels at the apical foramen. Infarction is virtually certain to follow due to the absence of any adequate collateral circulation (Andreasen, 1981; Stanley et al, 1978). Infarction is an effect of ischaemia and consequent anoxia.

The infarcted or necrotic pulp may become bacterially contaminated during its extra-alveolar period and even after reimplantation (Klinge et al, 1986).
Severe pulpal damage is more likely to occur in mature teeth with completed apices (Andreasen, 1981a) and infarction of the pulp is the most likely response following avulsion.

Cipriano and Walton (1986) using light and electron microscopy, reported changes in pulps that had undergone ischaemic infarction following trauma to the tooth. In the light microscope examinations, they noted that there were altered nuclei, no discernable neural elements, and less collagen. The electron microscope samples showed necrosis, nuclear degradation, relative avascularity, degenerating neural elements and disorganised collagen.

In pulpal tissue, the nuclei mainly undergo kariolysis, but pyknosis too, might occur. There is degeneration of the vasculature and its contents. Resolution of the infarcted tissue cannot occur, due to the lack of collateral circulation, and also because of the barrier-like mineral walls of the tooth. This restricts the inflammatory response and its consequent lysozomal and lysozymal actions. As there is no appreciable inflammatory reaction, there is no mobilisation of macrophages and hence no scavenging action. With no inflammatory response, there is no reparative process undertaken, and ischaemic necrosis of the pulp will occur (Stanley et al, 1978).
In the case of avulsed teeth, bacteria may invade the pulp via the apical or accessory foramina. This may lead to proteolytic digestion of the pulp. There is a possibility that bacteria may enter the pulp from the root surface via the dentinal tubules. Anaerobic bacteria tend to dominate the flora of the root canal (Bergenholtz, 1974).

Monsour and Adkins (1985) have noted that there is a wide variety of changes in pulpal tissues of transplanted teeth. These include degeneration and dedifferentiation of odontoblasts, metaplasia of pulpal cells and ossification in reparative tissues. They also suggested that pulpal tissues of mature traumatised or transplanted teeth, may retain or recover the capacity to maintain cells of a specialised nature. Transplanted teeth may respond similarly to avulsed teeth, although because of the short extra-oral period and operative care, prognosis is more favourable.

Maintenance of vitality is possible in teeth with open apices, because there is a greater chance of revascularisation of the infarcted tissue (Klinge et al, 1986; Heithersay, 1974; Johnson et al, 1985). Revascularisation occurs by ingrowth of granulation tissue into the root canal through the open apex. The granulation tissue replaces the infarcted tissue in
the pulp space (Andreasen, 1981a). Maintaining vitality of teeth with incomplete root development is important for two reasons:

1. the completion of apical root development; and,

2. the thickening of dentinal walls.

Normally, the completion of root development occurs only where there is a normal healthy pulp. It has been shown experimentally that if the apical part of the pulp remains vital after reimplantation, it may proliferate coronally, replacing the necrotic portion of the pulp (Klinge et al, 1986; Johnson et al, 1985).

Andreasen and Hjørtung-Hansen (1966) in a study of replanted human teeth, found that teeth with incomplete root formation had a very high rate of revascularisation. However, these teeth also had an extra-oral period of less than ninety (90) minutes.

Kristerson and Andreasen (1984) observed mature and immature teeth of monkeys, which were extracted and replanted with no extra-oral treatment. Histological examination at a later date showed that the amount of vital pulp was related to the stage of root development; vital pulp tissue was noted in teeth with immature roots, and almost totally non-vital pulp tissue in mature teeth. The extra-oral period for these teeth was eighteen (18) minutes, and
the root surfaces were kept moist with normal saline. This might not be representative of the clinical situation for avulsed teeth, where the extra-oral period and storage conditions may not be so favourable.

Numerous authors have undertaken research on revascularisation of pulpal tissue after replantation in immature teeth, usually involving dogs or monkeys (Monsour, 1971; Heyeraas and Myking, 1985; Skogland and Tronstad, 1981)

Monsour (1971) noted that after early degenerative changes, the pulp recovered to a normal histologic appearance for teeth that were replanted immediately. Teeth replanted after delay, showed a pattern of slow degeneration, followed by an ingrowth of tissue via the apical foramen. Necrotic tissue was replaced by this new tissue. There possibly needs to be a slow degeneration of the original pulpal tissue to allow replacement to occur. Rapid degeneration of pulp tissue may lead to abscess formation.

The ingrowth of new tissue may show areas of maturation and the differentiation of cells of a more specialised nature. Cells were noted similar to osteoblasts and located peripherally in the pulp, which were responsible for the deposition of an osteo-dentine-like material on the pulpal walls (clinically
this may be shown as partial or complete obliteration
of the pulp space).

Monsour (1971) also suggested that the
odontoblastic layer may regenerate or recover —
regeneration of nervous tissue appeared to be limited
to the apical third of the pulp. Hence, in these
teeth, there is a high probability of the tooth not
responding when pulp tested.

Heyeraas and Myking (1985) undertook a similar
study. They showed by angiography that
revascularisation does occur. Similar findings were
noted where necrotic pulpal tissue was replaced by
proliferation of mesenchymal cells and capillaries
through the apices or from apical pulpal tissue.

In a similar study by Skoglund and Tronstad
(1981), immature teeth were extracted and replanted.
Histologic and angiographic investigations were
carried out. After four days, the pulpal tissue
showed signs of degeneration and decreased enzyme
activity. Later ingrowth of tissue from the apical
area was evident and after 32 days repair of the
entire pulp had occurred. Regrowth and canalisation
of blood vessels was evident in the early phase of
tissue repair. They also noted that the original pulp
tissue may survive if revascularisation occurs within
four days of replantation. Kling et al (1986) noted
that teeth reimplanted within 45 minutes of avulsion
had a significantly increased frequency of revascularisation than those with a longer extra-oral period.

Skoglund et al (1981) noted that, if the original pulp tissue survived, a normal odontoblastic layer would remain intact. He found areas of calcification within the pulp tissue, and likened these to bone or a type of osteodentine.

PERIODONTAL TISSUES:

In avulsed teeth the periodontal ligament appears to be severed through the middle of the fibres equidistant between its insertions at the bone and cementum interfaces (Chamberlain and Goerig, 1980).

The sequence of healing, or necrosis, of the periodontal ligament will depend on many factors, the most important of which is the maintenance of cellular vitality (Blomlöf et al, 1983; Cvek et al, 1974; Chamberlain and Goerig, 1980; Lindskog et al, 1985; Proye and Polson, 1982).

After replantation, a coagulum forms between the parts of the severed periodontal ligament (Andreasen, 1981a). Over three to four days connective tissue proliferation and primary repair occurs.

Andreasen (1981a) suggests three different sequelae associated with healing and the periodontal ligament after avulsion and replantation:
1. healing with a normal periodontal ligament;
2. healing with replacement resorption; or,
3. healing with inflammatory resorption.

The normal healing of the periodontal ligament will be considered here. The other sequelae will be considered in a later chapter.

Heithersay (1974) suggests that normal periodontal healing occurs in a tooth that is replanted immediately under ideal conditions. Normal connective tissue repair should occur, with regeneration of collagen fibres, and laying down of cementum in areas of dentine exposure.

Proye and Polson (1982) examined the repair of the periodontal ligament in monkeys. They stated that the cell repopulation of the ligament was derived from the ligament, and adjacent bone marrow spaces. The process of reattachment of the periodontal ligament was mainly by the union of pre-existing collagen fibres with new fibres.

Nyman et al (1985) observed that in monkeys a normal periodontal morphology was found in replanted teeth if the periodontal ligament tissue was preserved. They confirmed that a vital periodontal ligament was essential for the establishment of a fibrous union of a replanted tooth. Groper and
Bernick (1970) also stated that if the periodontal ligament was to have a chance of regenerating, vital fragments should be located on the root surface.

Loë and Waerhaug (1961) performed studies on monkeys and dogs. They were interested in the effect of replanting teeth with and without vital periodontal ligaments. Those with vital ligaments essentially demonstrated a healed ligament of normal morphology. However, those with a necrotic or no ligament, showed areas of tooth resorption, with osteoclasts contained in Howship’s lacunae on the root surface, and subsequent ankylosis.

Klinge et al (1984) undertook research on dogs, where teeth were extracted and replanted (either immediately or at varying time intervals after extraction). The teeth which were replanted immediately mostly showed complete healing after three weeks. They noted minimal resorption and normal laying down of cementum. They suggest that this indicated the survival of the cementoblast layer. Hence there is the possibility of a vital periodontal ligament providing the cells for the regeneration of the elements of the ligament. Roots of teeth that were left to dry were either denuded of periodontal tissue or left with devitalised tissue. After
replantation, areas of replacement resorption and subsequent ankylosis were apparent. In such teeth, they suggested that progenitor cells came from the adjacent alveolar bone. This experiment comprised a small sample, and the findings should be viewed with caution. They mentioned that statistical analysis was undertaken, but no results were given.

Jonck (1966), in an interesting and controversial explanation of periodontal ligament repair after avulsion, claims that there are two phases of healing.

The first phase is a productive or catabolic phase and lasts for one to five days. The first two days show large numbers of leukocytes, macrophages and inert cells. The last half of the first phase shows fibroblasts proliferating into the socket along developing capillaries - he refers to this tissue as osteogenic granulation tissue and it exhibits an inflammatory reaction. The second phase, referred to as proliferative, anabolic or osteoblastic phase, is characterised by the formation of collagen.

As opposed to current thinking, Jonck considers that early reimplantation inhibits the successful healing of the tooth. However, his preparation of the teeth, prior to replantation included the "removal of the periodontal ligament with roughened gauze". Some teeth were replanted up to 3 weeks after extraction
during which time "the recipient's blood is the preferred storage medium for teeth".

This is somewhat different to the treatment regimen prescribed today. Note is made to the effect that after 2-4 years of observation results are very encouraging; but no details are given. The literature would indicate the observation period is insufficient.

It has been postulated by various authors, that the epithelial rests of Malassez may play a role in the healing of the periodontal ligament (Coccia, 1980; Løe and Waerhaug, 1961; Lindskog et al, 1983; Spouge, 1980). These are the remnants of Hertwig's epithelial root sheath, which is responsible for the formation of the root. The "rests" appear as discrete clusters, or islands of epithelial cells, within the periodontal ligament close to the cementum surface (Ten Cate, 1985). Other authors have described the rests as strands, clusters and columns.

Loë and Waerhaug (1961) proposed that these rests may act to limit root resorption in teeth replanted with a vital periodontal ligament, and that they play a role in the maintenance of the ligament.

Lindskog et al (1983) suggested that the elevated activity of certain enzymes (i.e., glucose-6-phosphate dehydrogenase) in the periodontal ligament, indicate a repair of the ligament. This enzyme is most likely
derived from epithelial cells. The epithelial cells of the rests produce a collagenase inhibitor, which might be similar to an "anti-invasion factor" shown to be present in roots.

_In vitro_, this anti-invasion factor inhibits osteoclastic bone resorption and helps maintain the periodontal ligament. Another factor in the normal repair of the periodontal ligament is the orientation of the fibres. Fibres can be orientated to give a functional arrangement, or may be arranged in a random fashion or parallel to the root surface. The outcome of periodontal repair, with regard to fibre orientation, depends on the type of mechanical stimulation to which the tooth is subjected during the healing period (Anderson et al, 1968; Hurst, 1972).

**CEMENTUM:**

Avulsion produces a wound to all the structures involved, i.e., periodontal ligament, bone and cementum. Healing of these structures involves a regenerative process (Lindskog et al, 1983a). Cementum is one of the components that will regenerate. Deeb et al (1965) suggested that cementum has no physiological relationship with the pulp, but is dependent upon the periodontal ligament.

Many authors have stated that the maintenance of
the integrity of the cementum is essential for the most favourable healing of the periodontal ligament and a reduction in root resorption (Kaqueiler and Massler, 1969; Lindskog et al, 1985; Nyman et al, 1982).

Nyman et al (1982) exposed an area of root surface and curetted all periodontal ligament and cementum. Their results showed that new cementum is laid down on these curetted surfaces. They also found that a connective tissue attachment can develop on the root surface. They conclude, as did Deeb (1960), that this reparative cementum is derived from cells of the periodontal ligament, which differentiate into cementoblasts. This experiment is not clinically similar to avulsion cases, where damaged periodontal ligament cells are required to differentiate. This may occur in an avulsed tooth with the right conditions.

Nalbandian and Frank (1980), working with cats, came to a similar conclusion.

Melcher (1985) stated that cells of the periodontal ligament are constantly differentiating. With respect to the periodontal ligament as a whole, and the cementum specifically, there is constant change during normal functional demands, and also during healing. Monsour and Adkins (1984) studying transplanted teeth in dogs, support this. They found
transplanted teeth in dogs, support this. They found that the extent and intensity of the production of cellular cementum was closely related to onset and intensity of functional stimulation. Such function usually resulted in the formation of irregular cellular cementum. Cellular cementum might fulfil a role in the repair of dental hard tissues, or as an adjustment to changing functional requirements or circumstances. Also observed were areas of root resorption repaired by cementum.

Another study by Monsour and Adkins (1985), using transplanted teeth in dogs, found that where apical closure was incomplete, cellular cementum was involved in the closure of the apex. They also considered that the breakdown of Hertwig's epithelial root sheath induced increased production of cellular cementum.

Lindskog et al (1985) thought that cementum was more resistant to resorption than bone or dentin. There may be a deficiency of factors (e.g., plasma proteins) in dentine that allows osteoclastic activity to occur. Cementum may contain inhibitors of osteoclastic activity. These factors may play a role in a reduced resorptive activity.

Andreasen (1981d) observed periodontal healing in monkeys. He noted that the major decisive factor with regard to periodontal repair, with no root resorption,
was the state of the cemental part of the periodontal ligament. He suggests that the less damage to the cementum, the less likely the chance of resorption.

Kristerson and Andreasen (1984) also using monkeys, discovered there was a greater chance of inflammatory root resorption in young mature teeth than in mature teeth. This could possibly be related to a protective action of a thicker cementum layer in mature teeth.

Lindskog et al. (1983) were interested in periodontal repair in replanted teeth. They concluded that the formation of reparative cementum is a common finding on denuded root surfaces. They also described another form of cementum - intermediate cementum. This is located between the outer layer of cementum and the layer of Tomes. They postulated that it promoted the attachment of reparative cementum to dentine. They believed that a better attachment of reparative cementum to dentine occurred only when the organic matrix of the dentine is exposed (i.e., collagen fibres). Following on from this, a bone specific protein called Osteonectin, has been identified (Termine, 1981). It selectively binds collagen to hydroxyapatite. This agent, or one related, is also present in dentine and may be involved in the attachment of new reparative cementum.
to dentine.

EXTERNAL ROOT RESORPTION:

External root resorption is loss of tooth structure from that root surface adjacent to the periodontal ligament. It occurs commonly after replantation (Andreasen gives an incidence of 80%-90%) and any prognosis must be carefully qualified (Lenstrup and Skeiller, 1959). Radiographically it can be differentiated from internal resorption using specific guidelines (Gartner et al, 1976).

Theories as to the actual mechanism agree that the process is similar to that in bone resorption (Andersson et al, 1984), but that there are noticeable differences between the active cells (Hammarström and Lindskog, 1985). Hammarström et al (1986) say that odontoclasts are smaller than osteoclasts (Fig. 8), have fewer nuclei and very small clear zones. Pierce et al (1988) refer to these cells as dentinoclasts. Hammarström and Lindskog (1985) suggest that the dental tissues have resistance to resorption and that the rests of Malassez promote resistance to resorption and reduce ankylosic fusion to bone. They say the periodontal ligament has an osteogenic potential with marked activity of alkaline phosphatase, and can generate a new lamina dura. Andreasen (1981a) agrees
with this view.

Conversely, Melcher (1970) is of the opinion that the periodontal ligament can suppress osteoblastic activity.

The periodontal ligament is initially linked to cementum and it has been suggested that cementum, particularly intermediate cementum, is important in the prevention of inflammatory root resorption (Hammarström et al, 1986c; Kaquelner and Massler, 1969).

It was noted above that Hammarström believes that dental tissues resist resorption and Lindskog and Hammarström (1980) also believe that they may include anti-invasion factors. Dental structures have been found, in vitro, to inhibit proteases and collagenase production. Resorption perhaps occurs where such protective mechanisms are reduced or lacking.

Pierce (1990) has undertaken studies on the physiological and pathological aspects of dentoalveolar resorption using electron microscopy and analytical procedures. She notes the activities of osteoclasts and dentinoclasts in relation to bone and root resorption, including regulatory factors.

Hammarström et al (1989) have made suggestions that root resorption that is associated with
Fig. 8 Scanning electron micrograph of an osteoclast in a Howship’s lacuna in the root of a human molar.

O = Osteoclast
Bar = 10 µm
Dentoalveolar ankylosis is initiated by endosteal osteoblasts, this process having a hormonal influence. Andreasen (1981a) relates the type of resorption, whether surface, inflammatory or replacement, to the degree of damage to the cementoblasts. The greater the damage, the greater the resorption. The damage to cementoblasts relates directly to the damage to the periodontal ligament and so these two factors must be closely linked.

Andreasen (1981a) and Andreasen and Hjørting-Hansen (1966) classify resorption into the three types: surface, inflammatory and replacement resorption; and these warrant further discussion.

**SURFACE RESORPTION:**

Andreasen (1985) described surface resorption as small resorptive cavities of the root surface, usually confined to the cementum and caused by uni- or multinucleated cells, or, where there were resorptive cavities in the cementum and outermost layers of the dentine, without an inflammatory reaction in the periodontal tissues (Andreasen, 1980; Andreasen and Hjørting-Hansen, 1966). It was usually repaired by newly formed cementum, some weeks after injury. Simultaneously, the insertion of new periodontal ligament fibres occurred (Hammarström et al, 1986c;
Johnson et al, 1985).

In relation to surface resorption, Andreasen (1981) drew attention to the apparently protective action of the intermediate layer of cementum, which separates the dentine and the regular cementum layer.

Scott and Zelikow (1980) suggest that surface resorption does not affect the prognosis. However, where areas of surface resorption are deep and contact dentine, and there is an associated periodontal inflammatory reaction, this could lead on to a more severe type of resorption, inflammatory resorption.

INFLAMMATORY RESORPTION:

Inflammatory root resorption is characterised by bowl-shaped areas of loss of root structure (Fig. 9). These areas are characterised by granulation tissue and inflammatory cells. Lymphocytes, plasma cells, polymorphonuclear leukocytes, uni- and multi-nucleated cells are present (Andreasen, 1981a; Andreasen, 1985). The inflammatory resorptive process can be very rapid and aggressive (Andreasen, 1981a), and may lead to loss of the tooth as early as two months after replantation (Andreasen, 1971). Clinically, the teeth are often loose, extruded and tender to percussion (Australian Dental Association, 1986).
The development of inflammatory resorption after replantation seems to follow four main stages (Andreasen, 1985).

1. Damage to the root surface initiates the resorptive process.

2. Eventually, this resorptive process will expose dentine tubules.

3. The tubules communicate with a necrotic pulp or a leukocyte zone containing bacteria, where toxins penetrate the tubules and initiate and/or sustain an inflammatory reaction in the periodontal ligament (Johnson et al, 1985).

4. The inflammatory reaction in the periodontal ligament stimulates the resorbing cells on the root surface.

Andreasen (1973), in a study in rats, felt that where the cementum remained intact, root resorption was not present. He suggested that resorption would occur where the toxic pulpal products could gain access to the periodontal ligament via a breach in the cementum.
Fig. 9  Radiograph showing inflammatory root resorption (arrowed) of an upper central incisor.
Hammarström and Lindskog (1985) also suggested that the activity of resorption in association with infection was enhanced by substances released from inflammatory cells (e.g., prostaglandins and lymphokines).

Coccia (1980) considered that a local immune response in an inflamed pulp may contribute to the initiation of inflammatory root resorption.

Andreasen (1981a) noted that the amount of inflammatory root resorption was proportional to the damage to the pulp, and was most significant in an immature tooth with an open apex. It seemed that an immature or young mature tooth is more likely to undergo inflammatory resorption (Chamberlain et al, 1980). Kristerson and Andreasen (1984) state this is because the younger tooth with its larger tubules, will allow greater diffusion rates of toxic products. However, they state that in immature teeth, with open apices, it is more likely the pulp will revascularise and remain vital, hence there is less chance of resorption. Kaqueler and Massler (1969) agree. On the other hand, young immature teeth are at risk of resorption due to the fact that they have a thinner protective cementum layer.
Hammarström and Lindskog (1985) also suggested that the activity of resorption in association with infection was enhanced by substances released from inflammatory cells (e.g., prostaglandins and lymphokines).

Coccia (1980) considered that a local immune response in an inflamed pulp may contribute to the initiation of inflammatory root resorption.

Andreasen (1981a) noted that the amount of inflammatory root resorption was proportional to the damage to the pulp, and was most significant in an immature tooth with an open apex. It seemed that an immature or young mature tooth is more likely to undergo inflammatory resorption (Chamberlain et al, 1980). Kristerson and Andreasen (1984) state this is because the younger tooth with its larger tubules, will allow greater diffusion rates of toxic products. However, they state that in immature teeth, with open apices, it is more likely the pulp will revascularise and remain vital, hence there is less chance of resorption. Kaqueler and Massler (1969) agree. On the other hand, young immature teeth are at risk of resorption due to the fact that they have a thinner protective cementum layer.
REPLACEMENT RESORPTION:

Replacement resorption is a process whereby there is resorption of the root structure and direct apposition of new bone against the resorbed root (Chamberlain et al, 1980; Cohen and Burns, 1984) (Fig. 10). The result of replacement resorption is ankylosis. Andreasen et al (1984) define ankylosis as the fusion of cementum or dentine with the alveolar bone, where its development in avulsed teeth is associated with injuries to the root and its periodontal ligament. Andreasen notes two types of initial ankylosis (1980c). One develops in response to a non-vital periodontal ligament on the root and socket wall. The other develops as a result of the periodontal ligament being necrotic on the root surface but vital on the socket wall. Hardy et al (1981) undertook a study on dogs. They postulated that ankylosis occurs where there is damage to the periodontal ligament with subsequent invasion and fusion of bone to cementum by osteogenic cells from the surrounding bone. This occurs before a functional periodontal ligament can regenerate. They are supported by Line et al (1974).

Histologically, replacement resorption can be observed two weeks following replantation.
Fig. 10  Radiograph showing replacement root resorption of an upper central incisor (root filled). Note lack of definition of the lamina dura.
Radiographically it is shown by the absence of a periodontal space, and can be recognised about two months after replantation.

However, replacement resorption can occur later than two months after replantation usually originating in the apical third of the root (Andreasen and Hjørting-Hansen, 1966; Coccia, 1980). Clinically the tooth is immobile and gives a high percussion sound, indicating ankylosis (Andreasen, 1981a; Cvek, 1974). In a young person, because of growth spurts, the tooth will be in infra-occlusion.

Andreasen (1985) states that replacement resorption can be of two types:

(i) progressive, where the root of the tooth is resorbed completely; or

(ii) transient, where a once established ankylosis disappears, and a normal periodontal ligament is re-established. The latter might be related to minor areas of damage to the root surface (Andreasen, 1981a; Johnson et al, 1985).

The aetiology of replacement resorption is unclear and Andreasen (1981a) suggests that it occurs due to the absence of a vital periodontal ligament. Lindskog et al (1985) concur with this view. They
have observed that teeth replanted with a necrotic periodontal ligament demonstrate a high incidence of replacement resorption and ankylosis. They postulate that the necrotic ligament might actively stimulate the resorption of the cementum layer due to the non-vitality of the cementoblasts. Cementum resorption can be activated by infection, bacterial products, inflammation, and/or necrotic debris. Perhaps these factors are responsible for the chemotactic attraction of macrophages and related cells, and subsequent activation of resorbing cells.

However, Hammarström and Lindskog (1985) state that the mechanism is "essentially unknown" and this appears to be the general feeling through the literature.

Necrosis of the periodontal ligament and its cells, is a result of damage to the ligament. Andreasen (1981b) decided after a study on monkeys, that replacement resorption was associated with the greatest loss of vital cementoblasts on the root surface. The damaged ligament was repopulated by progenitor cells from the adjacent bone marrow which have an osteogenic potential leading to subsequent ankylosis (Andreasen and Kristerson, 1981).

Damage to the root surface, periodontal ligament and cells, can occur by various means. These include
initial damage to the tooth when avulsed; the improper handling of the tooth prior to replantation, including inappropriate storage; and, the delayed replantation of the tooth (Andreasen, 1980 and 1981c).

Andreasen (1981e) questions the wisdom of performing root canal therapy prior to replantation, because both the time required, and mechanical handling, increase the chance of damage to the ligament. He also believes that antibacterial medicaments may damage the periodontal ligament (1981f). He considers several aetiologic factors leading to replacement resorption and ankylosis, and states that common causes are prolonged dry storage, storage in tap water and partial or complete removal of the ligament (Andreasen, 1980c).

Nyman et al (1985) feel that to reduce the chance of replacement resorption, there is need for a controlled cell repopulation by the periodontal ligament. This depends upon the maintenance of the vitality of cells in the ligament. In their study on monkeys, they found that connective tissue attachment failed to reform on root surfaces, which had been deprived of their periodontal ligament tissue. Melcher (1970) supports this view, and adds that perhaps the cells originating in the periodontal ligament exclude ingress of other cells.
Andreasen and Hjørting-Hansen (1966b) have noted that there is an increasing degree of resorption with increasing extra-alveolar time. Andreasen (1981c) also believes that damage to the periodontal ligament could result from trying to replant a tooth into a partially organised clot in the socket.

Coccia (1980) suggest that replacement resorption may be initiated by the absence of epithelial rests at the root apex. Löe et al. (1961) conclude that these cells maintain the integrity of the ligament, thereby avoiding ankylosis. Heimdahl (1983), in a clinical study, observed that it is reasonable to assume that after long extra-oral periods, the periodontal ligament will become non-vital, leading to replacement resorption and ankylosis. However, he states that an ankylosed tooth can be functional for a period of time. Hammarström et al. (1983) concur with this view. Klinge et al. (1984) believe that prevention of damage to the root surface is a crucial factor in the reduction of replacement resorption. This is supported by various authors with respect to the maintenance of vitality of the periodontal ligament (Andreasen, 1981c; Australian Dental Association, 1986; Johnson et al., 1985; Klinge et al., 1984; Van Hassal et al., 1980).
CHAPTER 5

TREATMENT OF AVULSED TEETH

Initial Treatment:

The avulsion of a tooth should be considered an emergency (Andreasen 1981a). It will involve initial treatment, thorough history taking and examination and consideration of neurological factors; as well as subsequent treatment. The treatment will begin when the patient contacts the dentist, either by telephone, in person, or via the Emergency Department of a hospital.

If by telephone, advice should be provided that will expedite the treatment of the problem. Kopel and Johnson (1985) suggest that the parent or patient should reposition the tooth in the socket. If unable to be repositioned, the tooth should be stored in cold milk, or in the parent’s or child’s mouth. Perhaps the latter suggestion is open to argument because of the risk of inhaling the tooth. Levenstein (1982) agrees, and adds that the tooth should not be washed under water if contaminated. Both authors then advise immediate attendance at the dental surgery.
Booth (1980) considers the above measures satisfactory. However, Korzen (1982) recommends the storage of the tooth in lukewarm water, if not able to be replanted. This is contradictory to the current regimen for emergency treatment.

While prompt action is required, Andreasen (1981a) emphasises the point that, if treatment is to be successful, a correct diagnosis must be made. This is achieved by a thorough examination, supported by an adequate history. Proper records will be of benefit in cases of litigation, or for insurance purposes.

Davis (1984) too, is insistant that proper records and careful documentation of a traumatic incident are essential for a correct diagnosis, and as a precaution against litigation.

Blackler and Widmer (1984) and Cohen and Burns (1984) support this view. This must be put in perspective with respect to the situation in hand and the expedient replantation of the tooth. Collection of relevant details will aid in the determination of a prognosis for the tooth.
History:

An adequate history of the immediate complaint and the general medical status of the patient is necessary. Andreasen (1981a) gives such an example.

1. Patient particulars i.e. age, sex, address.
2. When an injury occurred.
3. Where the injury occurred.
4. How the injury occurred.
5. Treatment administered elsewhere.
6. History of previous dental injuries.

The details elicited by these questions will aid in the development of a treatment plan. The time lapse between the accident and replantation is generally considered to be the most important factor in the overall prognosis. The place of accident may indicate need for a tetanus prophylaxis, or antibiotic cover. The cause of the injury and treatment already provided are important details. Knowledge of the medical status of the patient will shed light on any allergies, bleeding disorders, or other problems which may affect treatment. Korzen (1982), Davis (1984) and Blackler and Widmer (1984) all support this protocol.
Examination:

Examination of the extra-oral and intra-oral tissues is important for a proper clinical assessment. Andreasen (1981a) summarises these as follows:-

1. recording of extra-oral wounds and palpation of the facial skeleton;
2. injuries to the intra-oral tissues;
3. injuries to the teeth;
4. displacement injuries to surrounding teeth;
5. derangement of occlusion;
6. detection of alveolar fractures.

Korzen (1982) is concerned with examination of the avulsed tooth for fractures and to determine the state of root development.

Blackler and Widmer (1984) also suggests the need for a thorough clinical examination, and note a requirement for photographs and diagrams.

If the avulsed tooth/teeth cannot be located, then a chest x-ray should be taken to exclude inhalation.

Neurological Examination:

Trauma to the cranio-facial-oral complex may lead to damage of the central nervous system. Emergencies presenting for traumatic injuries to the teeth, especially avulsion, should be examined for
neurological disturbances (Kopel and Johnson, 1985). Blackler and Widmer (1984) say that signs to look for include loss of memory, bleeding from the head, disorientation, vomiting, prolonged headache, or loss of consciousness.

Davis (1984) mentions that oral trauma is "head trauma", and cautions that there is the possibility of a subdural haematoma. Observation of the patient for these conditions can be made during examination and history taking without causing alarm (Cohen and Burns, 1984).

**Radiographic Examination:**

Andreasen (1981a) and Davis (1984) state that all injured teeth should be radiographed. Andreasen adds that where there is a suspected jaw fracture involved, with an avulsed tooth, an extra-oral radiograph should be taken.

Radiographic examination can help determine the state of root formation and might disclose injuries affecting the tooth and its supporting structures e.g., fractured root remnants and alveolar fractures. Cohen and Burns (1984) suggest it can also be used for the detection of foreign bodies in the soft tissues, such as the lips and tongue, using a shorter exposure time or lower kilovoltage than normal. He also
advises the use of periapical, occlusal and panoramic radiographs.

**Indications and Contra-indications for replantation:**

There are definite contra-indications to replanting avulsed teeth. Andreasen (1981a) has indicated certain conditions that should be considered before replantation:

1. advanced periodontal disease;
2. the alveolar socket should be intact with no gross comminution;
3. there should be no orthodontic contra-indications.

Friend (1970) suggests the need for careful assessment in relation to caries and overcrowding. Heithersay (1974) says great clinical expertise is required to make a decision on replantation in cases of malocclusion.

Scott and Zelikow (1980) remark that the gross comminution of the alveolus is a contra-indication to replantation. Emergency medical precedences or patient motivation may preclude replantation.

**Psychological Aspects:**

Many authors have indicated the psychological advantages of replanting teeth (Ekstein, 1983; Feldman
et al., 1966; Harris, 1982; Korzen, 1982; Matusow, 1984a; Scott and Zelikow, 1980).

Replantation is important with regard to aesthetic considerations. It provides time for the patient to accept the inevitability of a prosthesis, as a replanted tooth should be considered only a temporary measure. In the case of a young patient, the retention of a replanted tooth is important, even if only for a few years. It negates the need for the wearing of a removable prosthesis, where a fixed appliance can be placed after exfoliation of the replanted tooth. Replantation is important from the aspect of development and function.

Finally, other very important aspects of treatment are reassurance and prognosis. The patient is likely to be nervous and agitated. The dentist must remain calm and be able to confer a comforting effect on the patient (Blacker and Widmer, 1984).

The patient or parent/s should be informed of the prognosis of the replanted tooth as well as the initial and follow-up treatment necessary.

Emphasis should be made that the replanted tooth is only a temporary measure, and that it is likely to be lost and need to be replaced by a prosthesis in the future (Andreasen, 1981a).
Emergency Treatment:

The overall aim of expedient replantation of an avulsed tooth is the preservation of the vitality of the periodontal ligament on the root surface (Feiglin, 1984). The avulsed tooth should be replaced as soon as possible (Camp, 1983; Cohen and Burns, 1983; Ehrmann, 1977; Larsen, 1987).

There should be minimal interference with the surface of the root and the periodontal tissues. If there is some delay in the replantation procedure, then the tooth should be placed in normal saline (Andreasen, 1981a). It has been found that minimal interference with the root surface possibly leads to a reduction in the amount of root resorption (Johnson et al, 1985; Andreasen, 1981a). Manipulation by the crown only is indicated (Fig. 11). Any contamination of the root surface should be removed by gently rinsing in saline (Cohen and Burns, 1984) or with gauze soaked in saline (Andreasen, 1981a). Rinsing the tooth in tapwater is not recommended as it can affect the viability of periodontal ligament cells on the root surface (Levenstein, 1982). Two recent
Fig. 11  Suggested method of the handling of an avulsed tooth prior to replantation.
articles establishing guidelines for emergency
treatment (Australian Dental Association, 1986; Camp,
1983) emphasise that the root should not be touched,
scraped or treated in any way. Andreasen et al (1985)
considered that apical resection does not enhance the
healing capabilities of either the periodontal
ligament or the pulp.

Current research and thinking suggests that for
the socket, minimal intervention is best. If there is
derangement of the socket wall due to fracture, this
will impede the correct repositioning of the tooth,
and reduction of the fractured alveolar bone and
correct repositioning of the fragments is indicated
(Andreasen, 1981a; Cohen and Burns, 1984). Where
there is no fracture, the tooth should be replanted
without removing the clot (Andreasen, 1981a; Larsen,
1987).

Andreasen (1980e) in research on monkeys, found
that the removal of the coagulum before replantation
had no influence upon periodontal healing. He is
concerned that interference of any kind could further
damage the already injured periodontal ligament on the
socket wall. However, if there is excessive coagulum,
this should be removed by gentle irrigation with
normal saline.

Camp (1983) agrees, recommending that the socket

68
should not be curetted, vented or surgically involved. Conversely, Matsson et al (1987) recommend saline irrigation of the socket before replantation. They state that, in dogs, this procedure promotes normal healing of the periodontal ligament. They argue that the coagulum acts as a chemotactic stimulus for osteoclasts and like cells thus increasing the chance of root resorption. They suggest that removal of the coagulum, by saline irrigation, will allow a greater chance of proliferation by connective tissue cells, leading to normal healing of the periodontal ligament. They also postulate that by removal of the coagulum, periodontal ligament cells on the root of the tooth will obtain a greater exposure to nutrients from the socket wall.

Once the tooth has been repositioned, a radiograph should be taken to ensure correct repositioning (Australian Dental Association, 1986; Cohen and Burns, 1984), and the occlusion should be checked (Andreasen, 1981a).

Current treatment with respect to the pulp of a replanted tooth indicates that it should be left until there has been some healing of the periodontal ligament (Andreasen, 1981a; Cohen and Burns, 1984). Experimental studies on monkeys (Andreasen, 1981c; Andreasen and Kristerson, 1981) where pulp extirpation
is performed prior to replantation, show a greater incidence of ankylosis and replacement resorption. This is due to the injury to the periodontal ligament on the root of the tooth while performing pulp extirpation. Hence, the tooth should be replanted without any endodontic intervention. However, in cases where the tooth has been out of the mouth for several hours, and the prognosis is poor, Heithersay (1974), suggests that there is little to be lost in carrying out endodontic treatment prior to replantation. Any latter endodontic treatment will depend on the maturity of the tooth and monitoring of pulpal status (Larsen, 1987).

In the past, many modalities of treatment have been undertaken in replantation procedures. These ranged from curettage of the socket (Chamberlain and Goering, 1980; Down, 1957; Grossman and Ship, 1970; Harris, 1982; Johnson et al, 1985; Matusow, 1987b) scraping of the root surface (Grossman and Ship, 1970; Lind et al, 1982; Massler, 1974) undertaking root therapy prior to replantation (Deeb et al, 1965; Eckstein, 1983; Heithersay, 1974) to performing an apicectomy prior to replantation (Massler, 1974; Marosky, 1978)
Splinting:

After an avulsed tooth has been replanted, it must be supported and maintained. This is referred to as splinting.

The American Association of Endodontists, in Korzen (1982), define a splint as a "rigid or flexible device used to support, protect or immobilise teeth that have been avulsed".

The purpose of splinting is to stabilise the avulsed tooth while repair of the periodontal ligament is occurring, provide protection from further injury (Kehoe, 1986), and to establish its correct position relative to the adjacent teeth (Heithersay, 1974).

Splinting is, in general, empirical with few scientific or experimental data to support the various techniques (Kehoe, 1986)

The type of splint and duration of splinting vary according to the individual situation (Hovland and Gutmann, 1976) and the clinician should understand the inherent advantages and disadvantages of each (Kehoe, 1986). Overall, the concept of long term rigid splinting has given way to short term functional or physiologic splinting (Antrim and Ostrowski, 1982; Andreasen, 1981a; Kehoe, 1986; Massler, 1974).

Marosky (1978) feels that stabilisation of an avulsed tooth is mandatory, in harmony with minimal
damage to the periodontal ligament.

Conversely, Matusow (1985) feels that "splinting per se, and its duration, do not appear to be significant factors in the prognosis of resorption with replanted teeth ...." This view is not held by authors of current concepts related to splinting.

Finally, it has been suggested that no splinting at all is advisable (Andreasen, 1980a; Feiglin, 1984) but this has little support in the literature. Andreasen (1981a) lists requirements of an acceptable splint.

1. It should allow direct, quick and easy application in the mouth.
2. It should not damage the tooth in its application.
3. It should immobilise the tooth in its normal position.
4. It should provide adequate fixation
5. Damage should not occur to surrounding supporting tissues.
6. There should be no interference with the occlusion.
7. It should not interfere with subsequent endodontic procedures.
8. It should be relatively aesthetic.
9. It should be easily removed with no destruction of tooth structure.
Davis (1984) feels that a good splint should be:
1. easy to apply;
2. easy to clean;
3. effective in stabilisation;
4. easy to remove.

Nooland and Gutmann (1976) also suggests that oral hygiene and expense are factors when deciding the type of splint to be used.

Type of Splint:

Many types of splints have been used over the years for stabilising avulsed teeth (Chamberlain, 1980; Camp, 1983; Eckstein, 1983; Harris, 1982; Kehoe, 1986; Lenstrup and Skeiller, 1959).

Simple acrylic resin splints can be used. The resin is flowed around the teeth to be splinted. Such splints may have adverse effects on periodontal and gingival health (Booth, 1980; Lenstrup and Skeiller, 1959). Cap splints have been advocated but have the disadvantages of requiring an impression, subsequent laboratory procedure and therefore delay in treatment (Andreasen, 1981a).

Composite resin splints have been used. They are easy to apply, but may affect gingival health if not finished correctly (Booth, 1980). However, the fracture resistance is low and the rigidity questionable.
Orthodontic brackets and arch wire have been used (Harris, 1982). Although the technique is time consuming it does offer a physiologic splinting technique and simplifies oral hygiene procedures.

Splinting with a mouthguard is an option (Eckstein, 1983) but introduces problems. The impression procedure may adversely affect the prognosis of the tooth, and the mouthguard needs to be worn constantly, until periodontal healing is sufficient to support the tooth. Hence, oral hygiene procedures are difficult to institute.

Camp (1983) suggests the use of sutures to hold the avulsed tooth in position "as a last resort", or where an unmanageable child is involved.

**Rigidity of Splinting:**

Antrim and Ostrowski (1982) consider that a splint, allowing physiologic movement of the replanted tooth allows functional orientation of the healing periodontal ligament fibres, and that this functional stimulation may depress osteogenesis (hence unwanted ankylosis). This he believes will enhance fibrous healing. Hamarström et al (1986c) support this opinion.

Johnson et al (1985) suggest that functional stimulation of the tooth may play a role in the repair
process of the periodontal ligament, and reduce replacement resorption.

Massler (1974) feels that rigid splinting, for a long period, results in fibres in the periodontal ligament being oriented parallel to the root surface and hence non-functional. Andreasen (1981a) has noted that rigid splinting of replanted teeth increases the amount of root resorption and ankylosis.

The literature indicates that the most frequently used splints used today are those constructed of acid etch composite resin (AECR) utilising nylon fishing line or orthodontic wire as the splinting medium (Australian Dental Association, 1986; Kehoe, 1986) (Figs. 12,13). The splint utilising the fishing line (of 20-30 lb. breaking strength) allows physiologic movement, but is not suitable where more stability of the tooth/teeth is required, as with an associated alveolar fracture, or where there is more than one tooth involved (Johnson et al, 1985). In these cases, the use of orthodontic wire, or wire of similar rigidity is indicated with a diameter of no less the 0.8 mm (Booth, 1980). The application technique of this type of splint is described widely (Andreasen,
Fig. 12 Illustration showing placement of a fishing line/acid etch composite splint.
Fig. 13  Illustration showing placement of a rigid stainless steel wire/acid etch composite resin splint.
Fig. 14  Clinical case showing avulsion of an upper central incisor.
Fig. 15  Replantation of the upper central incisor
Fig. 16 Application of a rigid splint.
1981a; Booth, 1980; Australian Dental Association, 1986) and seems to satisfy the ideal requirements of a splint. After application, radiographs should be taken to observe tooth position, and the occlusion should be checked (Figs. 14-16).

**Duration of Splinting:**

In the past, empirical views dictated that splinting periods be long, in the order of one to three months (Bellizi, 1974; Booth, 1980; Down, 1957; Ellis and Davey, 1970; Feldman et al, 1966; Jonck, 19661; Levenstein, 1982; Lenstrup and Skieller, 1959). Current concepts indicate these periods were too long and resulted in the greater incidence of replacement resorption and ankylosis (Nasjleti, 1982).

Andreasen (1981a) and Camp (1983) believe the splinting period should be about seven days for an avulsed tooth. Andreasen also feels this time is sufficient to provide adequate periodontal healing to support the tooth.

Levenstein (1982) and Booth (1980) suggest that an avulsed tooth should be splinted for one to two weeks where there is no alveolar fracture, but for up to six weeks where a fracture does exist. Duration of splinting of teeth with associated alveolar fractures will depend upon the severity and extent of the
fracture, which can be evaluated clinically at subsequent appointments.

Andreasen (1981a) notes the need for good oral hygiene procedures during the period of splinting, with careful tooth brushing and the use of antibacterial mouth washes.

Splinting in the mixed dentition is difficult due to the presence of partially erupted, missing and loose teeth. Therefore selection of an adequate splinting technique is important in these cases, particularly as many traumatic incidences occur in patients of this age (Andreasen, 1981a; Hovland and Gutmann, 1976).

Antibiotic and Tetanus Prophylaxis:

The literature is ambivalent as to whether systemic antibiotic treatment should be instituted at the time of emergency treatment. Recommendations for antibiotic coverage were not frequent ten or more years ago, when it was argued that there was no scientific evidence of their effectiveness (Frank, 1966; Massler, 1974; Scott and Zelikow, 1980) and their use was considered empirical (Marowsky, 1978). However, many authors advocated their use when there was contamination of the tooth and/or supporting tissues, or when medical conditions indicated their

More recently it has been shown that the institution of a systemic antibiotic is effective in reducing root resorption, both inflammatory and replacement (Andreasen, 1981; Feiglin, 1984).

Hammarström et al (1986a) and Hammarström et al (1986c) commented that evidence has shown systemic antibiotic therapy, instituted at the time of replantation, has been effective in the prevention of the infected necrotic pulp and inflammatory root resorption. They especially note that antibiotics must be given before bacterial invasion of the pulp occurs, as no systemic antibiotics can reach a necrotic pulp, and that intrapulpal application of antibiotics will be necessary if the pulpal space becomes contaminated.

The type and dosage of antibiotics is not stated in the literature. This could be left to the discretion of the clinician in view of the clinical situation.

Most authors feel that tetanus prophylaxis is indicated and generally recommended, especially where there has been contamination of the tooth or the supporting tissues (Australian Dental Association,
1986; Ehrmann, 1977; Johnson et al, 1985; Medford, 1982; Scott and Zelikow, 1980) while others feel that clinical judgment should be used (Massler, 1974).
CHAPTER 6

EFFECT OF EXTERNAL FACTORS

Preservation of a viable periodontal ligament is desirable, as this will determine the prognosis of the replanted tooth (Anderson, 1968; Blomlöf et al, 1981; Cvek, 1974; Chamberlain and Goerig, 1980; Cohen and Burns, 1984; Hammarström et al, 1986c; Johnson et al, 1985; Kauljar and Massler, 1969; Larsen, 1987). Andreasen (1981d) has concluded that the presence of a viable periodontal ligament is the most important factor in ensuring healing with minimal root resorption. The prevention of evaporation of tissue fluid from the periodontal ligament must be considered a primary goal if the tooth cannot be replanted (Hammarström et al, 1986c). It is obviously wise to preserve the tissue fluids in the periodontal ligament rather than keeping it in some artificial storage media. Blomlöf et al (1983) realise that replantation is not always possible at the time of the accident, and hence the tooth must be stored in a suitable medium until replantation can take place. Suitability of the medium is related to osmolality, ion strength and macromolecular content with minimal or no contamination by micro-organisms (Blomlöf et al, 1983).
Prognosis for an avulsed tooth is partially determined by external factors such as:

1. the extra-oral period;
2. storage media;
3. conditioning of the root surface;
4. role of the tooth socket.

**EXTRA ORAL PERIOD:**

This is regarded as being the most important determinant in the prognosis of the replanted tooth. The longer the extra-oral period, the more likely is the death of the cells in the periodontal ligament and subsequent incidences of replacement root resorption (Andreasen, 1980c; Andreasen, 1981c; Blomlöf et al, 1981b; Cvek, 1974).

Andreasen (1981e) considers that extra-oral endodontic treatment of an avulsed tooth is contra-indicated. He states it will increase the extra-oral period, and that careless handling of the root surface will lead to greater damage of the periodontal ligament cells. Possible toxic effects of medicaments used e.g., eugenol, may also affect the periodontal ligament viability.

Andreasen (1981c) believes that long extra alveolar periods could affect prognosis, because organisation of the blood clot in the socket causes
resistance to the replantation procedures, thereby adding injury to the periodontal ligament with subsequent root resorption. In a study on monkeys, Andreasen (1981d) concluded that ankylosis after a short extra-oral period is related to a damaged periodontal ligament. After a long extra-oral period, ankylosis is related to the socket wall and partial organisation of the clot.

A general concensus of opinion reveals that an extra-oral period of less than thirty (30) minutes will yield a high percentage of teeth with no replacement resorption (Andreasen and Hjørting-Hansen 1966a; Scott et al, 1980).

**STORAGE MEDIA:**

It is desirable to replant an avulsed tooth as soon as possible. However, some situations do not lend themselves to immediate replantation, and it is necessary to ensure maximum viability of the periodontal ligament in the interim (Hammarström et al, 1986c). The type of storage medium affects the periodontal ligament viability (Blomlöf et al, 1981b) and storage media should ensure that optimal conditions are provided (Blomlöf et al, 1983; Larsen, 1987).

Andreasen (1981a) has demonstrated the importance
of storage media because delay in replantation may occur where the situation precludes immediate replantation (e.g., an alveolar bony fracture; or where a dental professional or suitably trained person is not immediately available to replant the tooth (Andreasen and Schwartz, 1986)).

Many storage media have been proposed. The main factor that appears to be crucial to periodontal ligament viability is that of the osmolality of the medium (Andreasen, 1981c; Australian Dental Association, 1986). Andreasen reminds us that the osmolality of a solution is determined by the ion strength and concentration of dissolved non-ionised molecules. The osmolality of human blood and plasma ranges from 230-320 milliosmoles/kg. (Blomlöf et al, 1981a). The pH and temperature of the storage media are also important (Australian Dental Association, 1986).

Lindskog and Blomlöf (1982) noted that the composition of the storage medium is of minor importance compared to its osmolality (Table 1). They also stated that a hypertonic medium seemed to potentiate the damaging effect of bacterial contamination. Andreasen (1981e) too, addresses hypertonicity.

Milk has been favoured as the most suitable
TABLE 1. Osmolality of different storage media
(from Lindskog and Blomlöf, 1982)

<table>
<thead>
<tr>
<th>Medium</th>
<th>mOsm/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human blood and plasma</td>
<td>230-320</td>
</tr>
<tr>
<td>Eagle’s medium</td>
<td>291</td>
</tr>
<tr>
<td>Sucrose solution (physiologic)</td>
<td>287</td>
</tr>
<tr>
<td>0.9% NaCl</td>
<td>285</td>
</tr>
<tr>
<td>Milk</td>
<td>242</td>
</tr>
<tr>
<td>Saliva</td>
<td>71</td>
</tr>
<tr>
<td>Sucrose solution (hypotonic)</td>
<td>66</td>
</tr>
<tr>
<td>Tap water</td>
<td>16</td>
</tr>
</tbody>
</table>
storage medium for avulsed teeth (Australian Dental Association, 1986; Blomlöf et al, 1983; Courts et al, 1983; Lindskog et al, 1983b). Compared to other storage media, there appears to be a lower rate of external root resorption (Blomlöf et al, 1981b; Blomlöf et al, 1983). The osmolality of milk appears to be isotonic, having a range of 235-270 milliosmoles/kg. (Australian Dental Association, 1986; Blomlöf et al, 1981b; Larsen, 1987). Several authors note the fact that pasteurised milk has a low bacterial count (Australian Dental Association, 1986; Courts et al, 1983; Hammarström et al, 1986c; Lindskog et al, 1983b) and that it is ubiquitous (Courts et al, 1983; Blomlöf and Otteskog, 1980). Studies have been undertaken where teeth stored for up to six hours in milk have similar resorption rates compared to those that have been replanted immediately. Hammarström et al (1986c) and Lindskog et al (1983b) state that storage in milk yields a greater mitotic activity in cells compared to other storage media.

Saliva has been proposed as a storage medium. It appears that compared to teeth stored in water or left dry, rates of root resorption for teeth stored in saliva are less (Andreasen, 1981c) because the periodontal ligament cell viability is better maintained (Australian Dental Association, 1986).
However for extended periods of storage, its osmolality is too low and eventual rupture of cells will occur (Blomlöf et al, 1981b). Several authors (Australian Dental Association, 1986; Blomlöf et al, 1983) consider that the bacterial count in saliva is too great and this will encourage the occurrence of root resorption. Blomlöf and Otteskog (1980) consider saliva to be a poor storage medium.

Andreasen (1981c) considers that saline will reduce rates of external root resorption. However, the availability of isotonic saline solution is restricted. The use of tap water as a storage medium is contraindicated as it is detrimental to the healing of the periodontal ligament. Due to the hypotonicity of tap water, there is rupture of cells (cell lysis) with consequent marked external root resorption. Brief exposure for rinsing purposes appears to be satisfactory (Andreasen, 1980a).

Dry storage is definitely contraindicated, where cell death, replacement root resorption (Andreasen, 1980b) and subsequent ankylosis occur (Andreasen and Kristerson, 1981).

Many other storage media have been proposed. Plastic wrapping has been found to prevent dehydration (Blomlöf, 1983) and is considered an ideal storage medium (Larsen, 1987).
Jonck (1966) considers blood is a suitable storage medium, with ideal osmolality, metabolites and electrolytic balance. After an extra-oral period of three weeks he claimed to obtain complete healing after storing the avulsed tooth in the patient’s blood. Tissue culture media may be considered suitable in certain circumstances where availability is no problem e.g., Eagles Medium (Blomlöf, 1983; Kristerson et al, 1976). An interesting concept would place the avulsed tooth submucosally (Andreasen, 1980d), but this would not usually be practical.

Massler (1974) has suggested the placing of the tooth in a wet handkerchief and then in the refrigerator. Schwartz and Andreasen (1983) have suggested cryopreservation as a method of storage.

CONDITIONING AGENTS:

As mentioned previously, external replacement root resorption is mainly associated with a long extra-oral period and/or inappropriate storage media.

Many agents have been suggested to be applied to the root surface in order to reduce or eliminate external replacement resorption. Andreasen (1981a) states that agents such as formalin, alcohol and hypochloric acid have been applied to the roots of the teeth in order to reduce resorption. The usual result
is accentuation of the process. He also notes that methods have been attempted to interpose various materials, such as silicone grease, methyl methacrylate and fascia between root and socket, and these were equally unsatisfactory. Sterilisation of the root surface has been attempted, by caustic dressings (Chamberlain and Goerig, 1980) and acids (Hammarström et al 1986c) but this usually leads to necrosis of any remaining vital tissue, and a consequent increase in root resorption. It has been suggested that the periodontal ligament be removed where the extra-oral period is greater than ten minutes (Grossman and Ship, 1970). However, Andreasen and Kristerson (1981) have shown that this will increase the incidence of ankylosis.

Fluoride application to the root surface might decrease the rate of root resorption (Australian Dental Association, 1986; Barbakow et al, 1981; Bjorvajn and Massler, 1971; Hammarström et al, 1986c; Massler, 1974). However, other studies (Barbakow et al, 1980; Schulman et al, 1968) have not given any conclusive evidence of reduction in resorption. Several theories have been postulated as to the mechanism of how the application of fluoride might reduce root resorption. One is that fluoride can change hydroxyapatite to fluorapatite, which offers
resistance to resorption. Another is that fluoride has an inhibitory influence on the formation of the cells that are responsible for root resorption. Finally it is proposed that both factors act simultaneously (Bjorvajn and Massler, 1971; Chamberlain and Goerig, 1980).

Several authors have experimented using thyro-calcitonin with no success (Barbakow et al, 1978, 1980).

The use of citric acid to condition the root surface has been postulated. Klinge et al (1984) in a preliminary experiment in dogs, have indicated that conditioning of the root surface with citric acid, where there are areas of non-vital tissue remnants, will remove these remnants and demineralise the root surface, allowing exposure of collagen fibrils which will facilitate the attachment of new fibrils from the repairing periodontal ligament. Melcher (1985) suggests that collagen is chemotactic for fibroblasts, and that exposure of fibrils on the root surface will facilitate repair by periodontal tissue rather than bone. Nevins et al (1978, 1980) have suggested that decalcification of the root surface and subsequent enzymatic treatment deletes glycoproteins and leads to cross linking of cementum collagen, resulting in little or no resorption, in the short term.
Lindskog et al (1985) note that "the vitality of the periodontal ligament is the most important factor in preventing ankylosis fusion between bone and tooth". In a study in monkeys the root surfaces were treated with 10% sodium hypochlorite (NaOCl) and they concluded that the chemical removal of the necrotic periodontal ligament rendered the cementum less vulnerable to resorption.

**ROLE OF THE SOCKET AND BLOOD CLOT:**

Preparation of the tooth socket prior to replantation has been undertaken on an empirical basis in the past, where removal of the coagulum and curettage of the socket was performed (Down, 1957). Recent experimental data has given a scientific basis to this aspect of the replantation procedure.

**Coagulum:**

With respect to the coagulum remaining in the socket, current evidence has indicated that it should be left in situ (Larsen, 1987). Andreasen (1980e) suggests that the coagulum may aid in healing of the periodontal ligament; he also feels that its removal had no influence on healing. Where there has been a long extra-oral period, organisation of the coagulum may offer resistance to replantation. This may lead
to an increase in the damage to the root surface and
a possible increase in the chance of replacement
resorption (Andreasen, 1981c). In such cases gentle
irrigation with isotonic saline is indicated to remove
a partially organised coagulum (Chamberlain and
saline irrigation of the socket appropriate. They
feel removal of the fibrin coagulum will reduce bony
healing and enhance the proliferation of periodontal
connective tissue cells. This is expected to reduce
the chance of ankylosis.

**Socket:**

Chamberlain and Goerig (1980) indicated that no
curettage or venting of the socket should be
undertaken; Klinge et al (1984) agree, stating that
protection of the socket wall is necessary to preserve
any vital periodontal ligament remnants. Interference
may increase the chance of replacement resorption.
Conversely, Morris et al (1981) and Oswald et al
(1980) consider that the socket wall has a role in
healing where its partial removal delays root
resorption. They postulate that bone removal
increases the distance between the root and bone; that
the lamina dura is the site where new bone growth
occurs, and that its removal would decrease the chance
of ankylosis.
ENDODONTIC INTERVENTION

Immature Teeth – Open Apex:

Many teeth that are avulsed have an incomplete root development, and this can be seen at the time of avulsion. Levenstein (1982) has suggested that replanted teeth with open apices do not require endodontic intervention where there is lack of evidence of pathology either clinically or radiographically, as there is the possibility of pulpal revascularisation. Andreasen (1981a) states that the decision to endodontically treat avulsed teeth with open apices will also rely on the history and the information gained from reviewing each particular case.

The sequelae of replantation of an immature avulsed tooth will lie in two directions. Either the pulp will survive, and hence continued root development is likely to occur, or the pulp will become necrotic, leading to the arrest of root development.

Aberrant root development can occur such as the formation of cementum or osteodentine, development of a root fragment separate from the remaining root, or the possibility of pulp canal obliteration (Andreasen
Cvek et al (1974a), Johnson et al (1985) and Kaqelar and Massler (1969) agree that the favourable prognosis for pulp survival is proportional to the degree of immaturity of the tooth.

Webster (1984), notes that the endodontic management of a tooth with an open apex and divergent dentine walls is difficult with respect to obturation. He considers that the use of surgery in these situations is unsatisfactory with respect to the apical seal; and also the possible psychologically traumatic effect on a young patient. Ham et al (1972) agree.

**Apexogenesis and Apexification:**

Webster (1984) in his discussion on apexogenesis defines this as being "the physiological root end development and formation" (Figs. 17,18). He says that where an avulsed tooth has incomplete root formation, apexogenesis should be the objective. He emphasises continual review at regular intervals to observe clinical signs and symptoms for pulpal degeneration and necrosis. Methods of establishing pulp viability are numerous, and have been discussed by many authors and researchers (Andreasen, 1981a; Davis and Knott, 1984; Fulling et al, 1976; Jacobson,
Figs. 17, 18  X-rays showing apexogenesis (arrowed) of an upper central incisor over a 16 month period.
The criteria for determining pulp vitality include colour changes of the crown, reaction to percussion, radiographic signs of periapical pathology and/or arrested root development, sinus formation and pulp vitality testing. However, the latter is unreliable in the case of traumatised and immature teeth, and results should be viewed in the light of other clinical criteria (Jacobson, 1980). Gazelius et al (1988) have demonstrated that blood flow can be restored in luxated and avulsed teeth as measured by means of a laser doppler flowmeter. Gutmann and Heaton (1981a) would also aim at apexogenesis.

Klinge et al (1986) accept the possibility of pulpal revascularisation and regeneration in teeth with open apices, with the qualification that this could be adversely affected by any bacterial contamination. They emphasise that the shorter the extra-alveolar period, the greater the chance of revascularisation.

As with any tooth, when an immature tooth becomes necrotic, endodontic intervention must be undertaken. However, Barker and Mayne (1975), observed several cases where traumatised teeth, with apparent subsequent pulp necrosis, underwent an autoapexification. No endodontic intervention was
implemented.

Andreasen (1981a) has described the difficulty of removing necrotic tissue and obturating a root canal with an open apex and divergent walls.

Kerekes et al (1980) consider that apical closure or barrier formation is essential for an adequate apical seal. Zeldow (1967) considers that this closure should be attempted by biologic means as opposed to surgical intervention.

Apexification has been defined by Webber (1984) as a procedure which induces apical closure by the formation of hard tissue, or the continual apical development of the root in a tooth with a necrotic pulp and incomplete root formation.

Nygaard Østby (1961) noted the significance of the blood clot in healing, where the periapical tissues adjacent to the root apex had been lacerated. This lead to the formation of granulation tissue and its subsequent calcification and hard tissue formation.

However, Citrome et al (1979) found that a locally induced blood clot maintained the initial inflammatory response and did not result in hard tissue formation.

When the pulp becomes non vital, Hertwig’s epithelial root sheath will usually cease its function
of completing the formation of the root apex. However, its cells are very resistant to infection or inflammation, and its function may be restored if periapical conditions are favourable (Webber, 1984; Webber et al, 1981). Such favourable conditions may be promoted by the use of various materials e.g. calcium hydroxide (Frank, 1966; Kerekes et al, 1980). Andreasen (1981a) mentions numerous studies which have proved that intra canal treatment with calcium hydroxide will result in the formation of an apical barrier that will allow adequate obturation. He considers it to be the treatment of choice, as does Levenstein (1982) and Heithersay (1975).

Calcium hydroxide placed in the root canal has been proven clinically and experimentally in the treatment of non-vital teeth to induce healing of periapical inflammation and the formation of a hard tissue barrier (Figs. 19-21) (Andreasen, 1981a; Cvek et al, 1974a; Holland and de Souza, 1985). It has an antibacterial effect (Andreasen, 1981a; Cvek et al 1974a; Frank, 1979; Heithersay, 1974) and an osteogenic potential for the formation of cementum, osteodentine and interglobular dentine (Cvek and Sunderstrom, 1974b; Frank, 1979; Feiglin, 1985; Heithersay, 1970,1975). It has an alkaline pH, which may act as a buffer to the acidic nature of any
associated inflammatory process. It may have a physiologic effect on the surrounding tissues, such that the calcium ions will effect new capillary formation, and the hydroxyl ions will induce an osteogenic response. The necessary calcium salts could be drawn from the surrounding tissue fluids (Feiglin, 1985; Heithersay, 1975). The high pH of calcium hydroxide may act to denature any remaining pulpal tissue by leaching and arresting autolysis, thereby reducing the possibility of future periapical irritation (Kerekes et al, 1980). The calcified tissue produced can be laid down at the apex or inside the root canal (Heithersay, 1970).

Many other materials have been used within the canal to induce apical formation; antiseptic pastes (Cooke and Rowbotham, 1960), collagen calcium phosphate (Citrome et al, 1979; Nevins et al, 1976) and tricalcium phosphate (Koenigs et al, 1975; Roberts and Brilliant, 1975). Smith et al (1984) compared calcium hydroxide and barium hydroxide as agents for apical calcification. The calcium hydroxide exhibited repair and calcific bridge formation, whereas the barium hydroxide displayed an inflammatory response and a foreign body reaction. There seems to be no
Figs. 19-21  A sequence of X-rays illustrating an apexification procedure.
Fig. 20  Calcium Hydroxide Dressing
Fig. 21  Obturation with Gutta Percha
conclusive evidence that will enable any of these other materials to supersede calcium hydroxide as the material of choice.

With regard to the use of these materials, the literature is abundant with techniques (Andreasen, 1981a; Chawla, 1986; Coviello and Brilliant, 1979; Frank, 1966; Friend, 1970; Heithersay, 1970,1975; Koenigs et al, 1975). The time required for apexification varies and is largely dependant on the maturity of the root. Six to twenty four months is the suggested time frame (Andreasen, 1981a). Webber et al (1981) has detailed criteria to indicate completion of apexification using both clinical and radiographic indicators.

Final obturation after apexification procedures normally is undertaken with gutta percha, either using a chloroform dipped technique (Feldman et al, 1966) or a warm gutta percha technique (Andreasen, 1981a).

**Closed Apex:**

In an avulsed tooth which has a completed radicular configuration, the chance of pulpal revascularisation is negligible, and pulp necrosis should be anticipated (Andreasen, 1981a). There has been a multitude of endodontic treatment regimens with respect to pulpal extirpation. Harris (1982) feels
that endodontic treatment, either partial or complete, should be performed prior to replantation. Other authors consider that pre- or post-replantation endodontics will depend on various circumstances such as the extra-oral period and storage media (Massler, 1974; Marosky, 1978). Many others propose that endodontic intervention should be performed after replantation, at an interval of between five days and fourteen days (Barbakow et al, 1977; Chamberlain and Goerig, 1980; Cohen and Burns, 1984; Feiglin, 1984; Hammarström et al, 1986; Levenstein, 1982).

Scientific evidence suggests that prompt removal of the pulp is wise because of the incidence of inflammatory root resorption (Andreasen, 1980a; Barbakow et al, 1977; Cohen and Burns, 1984). Andreasen (1980a) has noted that subsequent endodontic procedures should not be continued until periodontal repair is nearly complete i.e. approximately two weeks. An intermediate dressing is necessary. In fact, it has been suggested a dressing be left in the canal for one (Cohen and Burns, 1984) or two (Feiglin, 1984) years.

Calcium hydroxide has been suggested as a routine endodontic dressing in avulsed teeth (Chamberlain and Goerig, 1980; Cohen and Burns, 1984; Feiglin, 1984; Levenstein, 1982). However, recent evidence has
indicated that it may lead to further damage to the periodontal ligament (Andreasen, 1980a; Hammarström et al, 1986c) and increase the incidence of replacement resorption (Hammarström et al, 1986b).

Recent research has shown that Ledermix(R), an anti-inflammatory and antibiotic preparation, has a therapeutic effect on the healing of the periodontal ligament when used as a root canal dressing (Abbott, 1985). The steroid component (triamcinolone) controls inflammation in the periodontal ligament. Pierce et al (1988) have shown that this component inhibits the spread of dentinoclasts by their detachment from the root surface.

Abbott (1985) advised that the antibiotic component of Ledermix (Demethylchlortetracycline) has an antibacterial effect.

Both Abbott et al (1988) and Pierce and Lindskog (1987) found that the active components of Ledermix penetrate the dentinal tubules and cementum, and become readily available at the root surface. This will inhibit the progression of inflammatory root resorption.

(R) Lederle Pharmaceuticals, West Germany
Abbott et al (1988), in their research on human teeth *in vitro*, have shown that the therapeutic effect of Ledermix is available for up to three months.

A permanent root filling with gutta percha is indicated, using conventional obturation techniques (Andreasen, 1981a).
CHAPTER 8

MANAGEMENT OF EXTERNAL ROOT RESORPTION

Root resorption is known to be a major cause of tooth loss following trauma, especially avulsion (Andreasen, 1981a; Schwartz et al, 1985).

As mentioned previously, external root resorption can be divided into three types: surface, replacement and inflammatory.

Surface resorption appears to be self limiting and will repair, with no intervention, by the laying down of cementum (Andreasen, 1981a).

Replacement resorption is related to the degree of non-vitality of the periodontal ligament. Hence, any procedure that maintains the viability of the periodontal ligament up to the stage of replantation will reduce the incidence of replacement resorption (Andreasen, 1981a; Schwartz et al, 1985).

Frank (1979) and Cheshire (1987) consider that there is no effective treatment for replacement resorption. However, Andreasen (in Chamberlain and Goerig, 1980) believes that calcium hydroxide may possibly retard the progression of replacement resorption. Heithersay (1975) disagrees.
Anderson et al (1985) feel that ankylosis may be reduced by masticatory stimulation during the period of healing of the periodontal ligament. They postulate this may occur by the in growth of blood vessels and rapid repopulation of necrotic areas of the periodontal ligament by fibroblasts, leading to the formation of a functional periodontal ligament.

Klinge et al (1984) suggest that replacement resorption can be reduced by removing any non-vital tissue on the root surface prior to replanting (resulting from adverse storage conditions or handling). He states that treatment of the root surface with citric acid will expose collagen fibres for reattachment of a periodontal ligament and that further experimentation needs to be undertaken in order to ascertain the source of connective tissue that repopulates the root surface.

In an experiment on dogs, Hardy et al (1981) felt that the interposition of polylactic acid between the root and socket wall inhibited ankylosis. They suggested that this occurred because the migration of osteogenic cells from the alveolus was prevented and the regenerating periodontal ligament was allowed to repopulate the root surface. However, the polylactic acid degraded before complete healing occurred, leading to some degree of ankylosis.
Inflammatory resorption in avulsed teeth is considered to be caused by a non-vital pulp, and can be controlled by endodontic intervention within fourteen days (Andreasen, 1981a; Chamberlain and Goerig, 1980; Cheshire, 1987; Frank, 1979; Hammarström et al, 1986c; Schwartz, 1985). Deeb (1960) considers surgical intervention is also required. Some authors consider that an interim dressing of calcium hydroxide prior to final obturation is indicated (Coccia, 1980; Chamberlain and Goerig, 1980; Heithersay, 1975). However, Pierce et al (1987) feel that the effect of calcium hydroxide may produce less than optimal results. They feel that the effect of calcium hydroxide on the periodontal ligament might be potentially harmful, leading to necrosis of the repairing ligament and subsequent ankylosis.

Consideration has been given to the effect of steroids on inflammatory resorption. In an in vitro study on cat’s bone marrow, Suda et al (1983) found that hydrocortisone affected osteoclasts by making them inactive. They suggested that in vivo, reduction of osteoclastic activity could involve cell rounding, thereby reducing cytoplasmic surface area. Pierce et al (1988b), using the steroid component of Ledermix in an in vitro study, found it had a direct inhibitory effect on dentinoclasts, by detaching them from the
root surface. A further study indicated that its use will lead to a change from inflammatory resorption to surface resorption and ankylosis. They consider that the combination of a steroid and antibiotic in Ledermix leads to a reduction in inflammation and bacterial contamination (Pierce et al., 1987). The antibiotic eliminated bacteria from the root canal and dentine tubules (Pierce et al., 1988b). Heithersay (1985) recommends the use of a 50/50 mix of calcium hydroxide and Ledermix as an interim endodontic dressing. He considers, with Abbott (1985), that the mixture leads to a slower release of the active components of Ledermix resulting in a longer lasting dressing.

Concern has been shown over the effect of this locally applied steroid component on the systemic steroid levels. Pierce et al. (1987) and Hume et al. (1981) consider that there is no significant effect.

Hammarström et al. (1986a) suggest that the use of systemic antibiotics subsequent to replantation will reduce the inflammatory response in the periodontal ligament caused by bacterial invasion.

Lindskog et al. (1986) consider that the use of ultrasonics could be important in the reduction of inflammatory resorption. They consider the use of ultrasonics will reduce the bacterial population in
the root canal and dentine tubules.

Pierce et al (1988a) have experimented with a hormone, Calcitonin, produced by the thyroid gland. They have found it is a potent inhibitor of osteoclastic bone resorption. It causes complete cessation of cytoplasmic mobility of osteoclasts by acting on a cell membrane receptor specific for these cells and possibly has the same effect on dentinoclasts. It may cause the detachment of dentinoclasts from their resorption lacunae and allow osteoblasts and/or cementoblasts to proliferate. In turn they might seal dentinal tubules, thereby eliminating bacteria and preventing an inflammatory reaction in the periodontal ligament.

Heithersay (1985, 1988) suggested the use of trichloracetic acid to destroy resorptive tissue. It is an escharotic agent, and should be used with caution. It should be used only where access is easy so that its application can be controlled and excessive harm to tissues will not occur, e.g., cervical resorptive defects. It yields a surface coagulation necrosis.
CHAPTER 9

PREVENTION OF TRAUMATIC INJURIES

"Hindsight explains the injury that foresight would have prevented". Author unknown, (in Cohen and Burns, 1984). In other words, prevention is better than cure. The statement is as relevant to the avulsed tooth as to any other accident.

Avulsion is a traumatic injury, as are other injuries such as crown fracture or luxation. Hence, any preventive measure described must involve all types of traumatic injuries to the teeth.

Many traumatic incidents can be prevented and the numbers greatly reduced (Tompson, 1982). However, as there is a variety of causes of such incidents, it is unlikely that all can be prevented (Cohen and Burns, 1984). Andreasen (1981a) notes that due to the many and varied aetiologic factors, preventive measures are hard to institute.

In considering the causes of injuries to the teeth, few can be anticipated. Perhaps injuries resulting from sporting activities, especially contact sports, can be prevented e.g. football, hockey, boxing. Awareness of and recognition of a trauma prone dentition and its possible correction is
essential e.g. protruding maxillary incisors and their orthodontic correction (Berkowitz et al, 1980; Chapman, 1985; Meadow et al, 1984). Sweet (1955) has observed that predisposition of the dentition to trauma may result from hereditary factors or habits, such as thumbsucking.

The mouth is a frequently injured area of the body amongst individuals who participate in contact sports. Other parts of the body that are injured are able to heal. This is not the case with the teeth, to which any injury may be permanent (Dennis and Parker, 1972; Tompson, 1982).

The methods of preventing traumatic injuries, most of which involve damage to the teeth, are many and varied. The main method of prevention is by the use of some device to protect the head, face or mouth. Davis and Knott (1984) have stated that the compulsory wearing of seat belts has resulted in a reduction in oral injuries.

The use of protective helmets in American Football was instrumental in the reduction of injuries to the mouth and head. It basically consists of a helmet with bars attached to protect the oral region (Andreasen, 1981a; Chapman, 1983; Cohen and Burns, 1984). Davis and Knott (1984) note the use of similar helmets by cricket players.
Figs. 22, 23  Illustrations showing a mouthguard and *in situ*.
The most expedient and tangible way of reducing injury to the dental structures is by the wearing of an adequately constructed mouthguard (Figs. 22,23) (Stenger et al, 1964). Many sporting bodies have found a marked reduction in dental injuries after the use of mouthguards (Andreasen, 1981a; Chapman, 1985; Davis, 1984). One report has claimed reductions in the range of 50-90% (Hughston, 1980). Mouthguards will either eliminate an injury or reduce its severity, depending on the magnitude of the blow.

Various authors consider mouthguards to be an essential piece of equipment for those involved in contact sports (Booth, 1980; Burton et al, 1985; Chapman, 1983,1985). Chapman (1983) suggested that mouthguards should be used both in competition and in training, as accidents are likely to occur in both situations. Chapman (1985) also conducted a survey with respect to the use of mouthguards in Rugby Union. He found that there was a significant relationship between reduced numbers of orofacial injuries and the use of mouthguards. He also discovered that a great majority of players were unwilling to compete without a mouthguard. He feels that mouthguards should be worn by children when they start playing organised sports, to accustom them to the use of the appliance.
Historically, the first mouthguards were used in boxing in 1913, in the crude form of trays made of rubber-like material (Chapman, 1983). Apparently, it was used to "soften the jarring pain of jaws being smashed together, shattering teeth, severely lacerating lips and transmitting spine tingling chin blows to the base of the brain" (Andreasen, 1981a).

There has been some resistance to the use of mouthguards as players and coaches felt that they detracted from the "rugged appearance" of the player (Stenger et al, 1964). Other problems which caused resistance to use were difficulty of communication and difficulty with breathing (Chapman, 1985; Hughston, 1980). Andreasen (1981a) outlined the various materials used in mouthguard construction. He mentioned vulcanised rubber (little used now), latex rubber, resilient methyl methacrylate, and finally the vinyl plastics. The latter are used almost exclusively today.


The functions of a mouth guard include:-

(i) reduction and distribution of force from direct frontal blows which may
cause tooth injury, and mainly
affording protection to the upper
anterior teeth;

(ii) protection of soft tissues, lips,
cheeks and tongue from lacerations and
bruising;

(iii) prevention of forceful closing of the
maxilla and mandible which may damage
teeth;

(iv) absorption of forces and bracing of the
mandible, thus reducing the chance of
a fracture;

(v) elimination of the transmission of
forces via the condyles to the glenoid
fossa, with subsequent possible
concussion and/or skull or condylar
fracture;

(vi) protection of the neck and reduction of
the incidence of injury by limiting the
transmission of forces to the cervical
spine;

(vii) provision of psychological benefits as
players feel confident that they are
less likely to receive injury to the
head and mouth;
(viii) replacing dentures, negating the possibility of their fracture and possible swallowing of the fragments.

The desirable properties of a mouthguard have been outlined by several authors (Chapman, 1983; Stenger et al, 1964). It should fit accurately providing good retention, thus preventing accidental dislodgement during violent actions. It should be comfortable, allowing proper breathing and speech. Ease of cleaning and durability are important. The material of choice should be odourless, tasteless and non-irritating to the oral tissues. Affordability is also important.

Andreasen (1981a) has suggested a design for a mouthguard with the following features. The occlusal surfaces of all teeth should be covered for protection, and the distal extension should include the tuberosities. The palatal tissues should be covered for only 4-6 mm. from the gingival margin, the edges of the guard tapering to a feather edge. A feather edge will help to avoid interference with breathing, speech or the triggering of the gag reflex.

Dennis and Parker (1972) advise that coverage should be limited distally to the distal of the upper first molars, and by covering only the ruggae.
Excessive extension may cause gagging or difficulty in breathing. Flanges should be underextended and thin, with no impingement of frena or muscle attachments.

Chapman (1975) mentioned that there is some disagreement as to whether the imprint of the opposing occlusion should be incorporated in the mouthguard. If too deep, it will weaken the material by decreasing its thickness. However, some degree of imprint is necessary in order to distribute forces more evenly, both in occlusal and lateral directions.

Dennis and Parker (1972) believe that the thickness of the occlusal coverage should not obliterate the interocclusal space, but should offer protection for the occlusion.

There are basically three types of mouthguard (Andreasen, 1981a; Dennis and Parker, 1972).

The first type is the stock type. It is made from plastic, and is of one size. It is held in place by closing the jaws together. No adaptation is attempted, hence it does not fit and has no passive retention. It’s major attraction is its low cost. Turner (in Cohen and Burns, 1984) and Chapman (1983) believe that they are useless.

The second type of mouthguard is classified as a mouth formed type. This consists of a tough
horseshoe-shaped outer shell with a softer resilient liner that adapts to the teeth and soft tissues. Other types have an outer shell into which a self curing resin is placed. The most widely used group of mouth formed guards are a thermoplastic shell of either polyvinyl chloride or polyvinyl acetate. They are softened by heating and adapted to the oral tissues. Should distortion occur, they can be resoftened and readapted. However, Dennis and Parker (1972) feel that to get good adaptation, the temperature of the materials needs to be beyond the tolerance of the tissues. They are also very inexpensive. Because of the better adaptation and hence improved retentive ability, they will offer better protection. One problem appears to be their bulky nature.

The final type of mouthguard is the custom made variety, fabricated from vinyl plastic on models of the wearer's arches. They conform best to the ideal qualities of a mouthguard (Chapman, 1983), are more expensive than the other types, but afford much greater protection. An advantage of this type of mouthguard over the others is that construction can be tailored to anatomical variations of the individual, and to the type of sport in which they are engaged.

A development of the custom made mouthguard is the bimaxillary mouthguard (Chapman, 1986). Both
arches are covered by one unit, the mandible opened to a level determined by strenuous breathing. Chapman maintains that wearers adapt to it easily and that the protection afforded is well worth the expense.

The second main method of reducing injuries is education, which involves three major areas.

1. Education of persons involved in activities where dental trauma is a risk e.g., contact sports.

2. The education of the parents of children engaging in such activities.

3. The education of the community at large with respect to these injuries.

Any education programme should be directed at making people, both individually and collectively, aware of the problems associated with avulsed teeth. One such programme was undertaken by the Australian Society of Endodontology in 1976, called "It's a Knockout" - an avulsed tooth campaign. The campaign involved production of a poster and publicity related to avulsion throughout Australia, including media coverage (Booth, 1980).

Benefits can be gained economically with respect to any subsequent treatment as a result of a traumatic injury which leads to the avulsion of a tooth. Knowledge gained from an education programme may lead
to either the expedient replantation, or storage in a suitable medium, of an avulsed tooth. This may lead to the reduction or elimination of root resorption, thereby simplifying treatment in the future. Aesthetically and functionally, the patient may also benefit, perhaps by the retention of the avulsed tooth, and eliminating the need for a prosthesis (Hughston, 1980; Tompson, 1982).

Kemp et al (1977) believe that a majority of cases of avulsion can be prevented. They indicate that education of participants, parents, coaches and other personnel involved in activities that could lead to avulsion, is desirable. Zadik et al (1980) agree.

Informed dental personnel including dentists, dental assistants and receptionists are an essential part of community education in trauma prevention. Education of the dental team can be achieved by means of seminars and maintained through familiarity with the dental literature.

Education should also be directed at steps taken once a traumatic injury has resulted, with respect to the expedient treatment of the injury (Booth, 1980).
CHAPTER 10

RECOMMENDATIONS FOR THE
CLINICAL MANAGEMENT OF AVULSED TEETH

Based on the current scientific literature, discussed in the previous chapters, the following recommendations can be suggested for the management of avulsed permanent teeth.

A. Emergency Treatment (by the educated lay person) could include.

1. If the tooth is contaminated, quickly wash with water to remove debris.

2. Replant the tooth as soon as possible, handling the tooth by the crown only.

3. Once replaced, the use of some method of fixation is advisable - e.g. foil splint, light finger pressure.

4. If unable to replant, then storage in a suitable agent to ensure viability of the periodontal ligament is mandatory. As a storage medium milk is optimal. Alternatively, plastic foil such as gladwrap; or isotonic saline (if available) are suitable.
5. Rapid conveyance to the dental surgery for continued treatment is advisable.

6. Once at the dental surgery, if not replanted, the dentist will replace the tooth in its socket. An adequate history and examination should be undertaken.

7. After replantation, the tooth should be splinted, using either fishing line (30lb.) and composite resin or 0.8mm stainless steel wire and composite resin, depending on the situation presenting. The splinting period is usually seven days, however, this period may be extended if there is a concomitant alveolar fracture. Clinical observation will indicate when the splint can be removed. Administration of antibiotics (normal course) and tetanus prophylaxis (if required) is wise.

B. Subsequent Routine Treatment.

1. After one week the patient is reviewed. Subsequent treatment will depend on the maturity of the tooth. A mature tooth with a closed apex will require pulp extirpation. After pulp extirpation a dressing of Ledermix is introduced into the canal. An immature tooth
will be left and observed for signs of pulp revascularisation (observation for a long period is recommended in order to ascertain the pulp status).

2. The patient is reviewed two weeks later, when either observation is continued (in the case of an immature tooth), or endodontic treatment is commenced (a mature tooth). A further Ledermix dressing is placed for a total of two months after replantation.

3. Where there are no complications, the root can be filled utilising normal obturation techniques and materials.

4. For an immature tooth where the pulp has become necrotic, an apexification procedure followed by routine obturation procedure is undertaken. Apexification requires the use of a calcium hydroxide based dressing (e.g. Pulpdent) until an apical barrier has been produced. This should be detectable both clinically and radiographically. This barrier must be able to withstand obturation pressures. Because the root canal may have a blunderbus shape, obturation using lateral condensation techniques may be inadequate. In these cases, a vertical or warm gutta percha technique may be indicated.
C. Treatment of External Root Resorption.

Inflammatory Resorption.

Where there is radiographic evidence of inflammatory resorption, the inflammatory response in the periodontal ligament must be controlled. Ledermix\(^1\) (an antibiotic/corticosteroid paste) as an intracanal medication, appears suitable for management. Once the resorptive process is stopped, resorption areas may be remineralised. This remineralisation may be promoted by the use of calcium hydroxide based dressing.

Replacement Resorption.

No treatment regime at present appears to be effective for combating the process of replacement resorption.

---

\(^1\) In the experience of the author, Ledermix alone appears to be clinically successful.
CHAPTER 11

SUMMARY AND CONCLUSIONS

This treatise has examined the literature in relation to the management of avulsed teeth. It has become apparent that there are areas of inadequate knowledge where there is need for further research. These include the mechanism of the repair of the periodontal ligament, the mechanism of replacement resorption and splinting techniques. There is also a need for a wider and more comprehensive education programme, both within the profession and to the public.

Amongst the literature, there is also a noticeable divergence of opinion in many aspects related to the avulsed tooth. These include the mechanism and treatment of replacement resorption; the effect of an anti-inflammatory root canal dressing; the effect of calcium hydroxide as a root canal dressing; the timing of root canal therapy; the selection of adequate storage media for the avulsed tooth; the type of splint and the duration of fixation procedure; the necessity for the use of antibiotics, their type and dosage; the treatment of the socket prior to replantation; conditioning agents for the
root surface in relation to root resorption; and aspects related to the design of mouth guards.

With further scientific research, these areas can be clarified, hopefully leading to a more predictable treatment regime for the avulsed tooth.

However, at the present time, it must be emphasised that a prediction of the prognosis of a replanted avulsed tooth can only be estimated, and that it should always be guarded.
BIBLIOGRAPHY

ABBOTT P. Personal communication, 1985.


ANDREASEN J. Analysis of topography of surface and inflammatory root resorption after replantation of mature permanent incisors in monkeys. Swed Dent J 1980(b); 4:135-44.


ANDREASEN J. Effect of extra-alveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys. Int J Oral Surg 1981(c); 10:45-53.


ANDREASEN J. The effect of pulp extirpation or root canal treatment on periodontal healing after replantation of permanent incisors in monkeys. J Endod 1981(e); 7:245-52.

ANDREASEN J. Relationship between surface and inflammatory resorption and changes in the pulp after replantation of permanent incisors in monkeys. J Endod 1981(f); 7:294-301.


BLACKLER S, WIDMER R. Trauma to anterior teeth. Written communication, Westmead Centre Dental Clinical School, Pedodontic Unit, 1984.


FAUCHARD P. The Surgeon Dentist or a Treatise on Teeth. 1746, Paris, Vol. 1, Chapter XXX.


GUTMANN J, HEATON J. Management of the open (immature) apex: 2 non-vital teeth. Int Endod J 1981(b); 14:173-78.


141


LARSEN R. Replantation of teeth - a review. Periodontology 1987; 8:3-10.


WADSWORTH H. Resetting and Transplanting Teeth. Dental Cosmos 1876; 18:577-582.


