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IMPACTED TEETH ASSOCIATED WITH ROOT RESORPTION
OF ADJACENT TEETH IN THE PERMANENT
DENTITION

A Thesis embodying original work, submitted
in partial fulfillment of the requirements
for the Degree of Master of Dental Science
in the Faculty of Dentistry,
University of Sydney, December 1967

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Department of Preventive Dentistry.
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Impacted Tooth Associated with Resorption of Adjacent Tooth in the Permanent Dentition.

Introduction.

Occasionally, during routine radiographic surveys, or in full-mouth radiographic examinations preceding orthodontic treatment, an unerupted tooth is found in association with resorption of the root of an adjacent permanent tooth.

Although this is not a common occurrence, the implications of such a discovery have a direct bearing on the orthodontic management of the malocclusion present, and the most favourable procedure to adopt is sometimes a most difficult decision.

The aim of this study is to develop a rational approach to this problem, by an examination of a number of patients with this condition, and considering the various treatment plans which would apply, in the light of observations made on case reports found in the literature, and on a knowledge of the principles of root resorption and of dental impactions.

An impacted tooth and root resorption will be defined for the purposes of this work.

An impacted tooth is one whose full eruption into
the dental arch has been obstructed by any part of an adjoining tooth. (Adapted from Boucher 1963 p. 279). A tooth may erupt ectopically, and be obstructed by a normally positioned tooth, or normal eruption may be prevented by an aberrant tooth, or a combination of these factors may exist, resulting in insufficient room in the dental arch for all the erupting teeth. This is not the full definition of an impacted tooth, as teeth may become impacted for many other reasons, but this restricted definition will apply in this study.

Radicicular or root resorption may be defined as the loss of a varying amount of root substance, either cementum or dentine or both, by accelerated local cellular activities, stimulated by either local or systemic factors, or a combination of these. (Adapted from Boucher 1963 p. 316).

In this work, we are examining root resorption precipitated by a local factor, the presence of an impacted tooth, but as will be seen, there may be systemic factors present as well which influence local cellular responses.
REVIEW OF THE LITERATURE

The review of the literature will be divided into the following sections:

A. Impacted Teeth.
   Historical.
   The Aetiology of Impacted Teeth.
   Evolution of the Jaws.
   Civilization of Primitive Man.
   The Effects of Diet.
   Racial Hybridisation and Genetic Inheritance.
   The Endocrine Glands.
   Local Factors.
   Incidence and Prevalence of Impacted Teeth.

B. Dental Eruption.
   The Mechanism of Eruption.
   Historical.
   Theories on the Mechanism of Eruption.
   Eruptive Force.

C. The Mechanism of Resorption and Repair of the Teeth.
   Historical.
   The Osteoclast.
   The Aetiology of Resorption.
   An Hypothetical "Anti-Resorptive Factor".
   Replacement of Resorbed Dental Tissues.
   Frequency and Incidence of Root Resorption.
D. Impacted Teeth Associated with Root Resorption of Adjoining Teeth.

Teeth Most Commonly Affected.

Sex Distribution.

Age of Occurrence.

Fate of Affected Teeth.

Associated Symptoms.

Pulpal Condition of Resorbed Teeth.

Frequency.

E. Application in Clinical Orthodontics of the Conclusions Drawn from the Review of the Literature.
REVIEW OF THE LITERATURE

A. IMPACTED TEETH.

Historical.

Impacted and aberrant teeth are not the heritage entirely of modern civilized man. They have been found in the fossilized remains of both man and beast.

One of the earliest examples of an impacted tooth (Nodine 1943 p.440) was discovered in the Rancho La Brea pits in the jaw of a sabre-toothed tiger of the Pleistocene era. (Approximately 75,000 years ago). The last molar in this jaw was nearly horizontally impacted into the distal surface of the molar immediately anterior to it.

The earliest human specimen (Weinberger 1929) showing an impacted tooth is the Mousterian youth, found in Vezere, France in 1908 and estimated to be 40,000 years old. In this skull, which displays a nearly "ideal" occlusion, the lower left permanent canine is impacted, the deciduous canine still being present. The individual was a young male, about 16 years of age. Coincidentally, this type of impaction is uncommon in modern man.

References to impacted teeth may be found as far back as the fourth century before Christ. Aristotle (384-322 B.C.) observed that the "third molars erupt about the age
of 20 years. But in some cases, especially in women, they have been known to come forth, not without pain, very much later, even so late as 80 years." (Nodine 1943 p. 9).

Works, such as those of Pliny (23-79 A.D.) (Nodine 1943 p. 11) Hippocrates (460-377 B.C.) (Weinberger 1943) Vesalius (1514-1564) and Eustachius (1563) (Nodine 1943 p. 111) record the prevalence of impacted third molars, and the problems associated with their eruption.

Charles Allen, who published in Dublin in 1686 the first dental book in English, "The Operator for the Teeth", had some rather pertinent points to make on the impaction of third molars. He says "The four last of them seldom coming out before the one or two and twentieth years of our age; for which reason such teeth are called by some, the teeth of wisdom, because by that time we should have a full use of our Rational Faculty, though God knows how often it proves to be true". And later, "and without the help of Art, hardly ever appear before half our life is already passed away, and when the time of our growth is over". (Nodine 1943 p. 216).

Nearly ninety years ago, Fowler claimed that "defective condition of the third molars (impacted third molars) is no monopoly of the most highly civilized races, but may be found among the most abject and degraded of
the human species - Negroes, Melanesians, Australians, Tasmanians, etc." (Nodine 1943 p. 440).

Although there is evidence that primitive man suffered from the impaction of teeth, there is also evidence to show that more highly civilized man suffers from a far greater prevalence of impactions and in a wider variety of teeth. Some of this evidence will be considered in the section on the etiology of impacted teeth.
THE ETIOLOGY OF IMPACTED TEETH.

Evolution of the Jaws.

In primitive man, both ancient and modern, an efficient masticatory apparatus was a major factor in the survival of the individual.

To gain the full nutritional value from the available food supplies, which themselves, could only be obtained with the expense of great physical effort, a full breakdown of the sinewy meats and harsh roots would be necessary before this food was suitable for digestion.

The teeth and jaws of primitive man were virtually the only available mechanism for reducing this food. Any defect in the efficiency of this apparatus would most certainly have serious consequences for the individual.

It can be seen, relating the Darwinian theory of evolution, that for the survival of the human species, any unsuitable characteristics in the jaw mechanism would eventually be "bred-out". (Mead 1930).

Civilization of Primitive Man.

There is very little literature available on the effects of a civilized diet on the first generation after civilization of primitive man. In other words, the effects of partial loss of masticatory function on development of the full growth potential of the tooth bearing bones has
not been fully studied.

The question has been more thoroughly investigated in animals. Bjork (1950) states "From a morphological viewpoint, the effect of domestication is to widen the range of variation, i.e. to give rise to a greater variety of morphologic character", and later, when discussing Lundholm's work on domestication said, "In some animals there is a reduction in the size of the head, and thus also in the brain volume. In the latter case there may be no proportional diminution in the size of the teeth, corresponding to that of the jaws and brain case, so that there is less space available for them in the jaws, and crowding results, as in the domestic dog".

These studies would suggest that the effects of civilization with consequent partial loss of function may be one etiological factor in crowding and impaction of teeth.

Moss (1962) in his development of the functional matrix theory of growth, has also stressed the importance of fully functioning investing organs for the full development of the bony framework.

Mead (1930) quoting Dr. Wahl on the Eskimos, said "of the older generation, caries, malocclusion, and
impactions were rare and that the diet of these people consisted of a hard diet requiring mastication. It was found that in the younger generation where the diet consists of, or is interfered with, by the use of carbohydrates, candies, etc., where there has been considerable disuse of the teeth, caries have become apparent, as well as have malocclusions and impactions".

That the size of the jaw is diminishing in the evolutionary scale in man is clearly expressed by Hellman (1936) "... despite the progressive swelling of the human brain case, the face is continuing to withdraw beneath it. Accompanying this is a gradual diminution in size of the jaw bones with a consequent reduction in space for the accommodation for the full complement of the teeth!"

The Effects of Diet.

Diet can influence the development of the jaws and hence the prevalence of impactions in several ways. These influences can be listed as (a) Functional,

(b) Nutritional,

and (c) Attritional.

(a) We have examined briefly the role of function in development previously. Obviously the nature of the diet, whether highly refined, or very coarse, has an
influence on the amount of function required to prepare it for digestion.

(b). The nutritional requirements of the individual in reaching full physical development, depend almost entirely on the diet. Deficiencies in any of the essential elements can lead to faulty or distorted growth patterns, including a failure to achieve full jaw development. Salzmann (1957) says "The effect of diet on irregularities of the teeth is exerted through faulty structure and developmental growth of the bones and teeth".

(c). The part that attrition has to play in the development of occlusion, and the inclusion of the full complement of teeth in the dental arch, has been stressed by Barrett (1953), Begg (1954), and others. Speaking of tooth crowding, bi-maxillary protrusion, and a refined diet, Barrett writes "...these differences in occlusion and in tooth to basal bone relationship, may be due to the relatively small amount of interproximal attrition in the Yuendumu natives compared with the marked attrition seen in pre-white aborigines and may be the direct result of the present diet of the natives". Begg goes so far as to say that occlusion is not "normal" unless cuspal "meshing" has been virtually eliminated by wear to allow full mesial dental migration and compensation for interproximal attrition-
thus allowing the third molars to erupt and avoid this most common impaction found in civilized man.

Occlusal and interproximal wear is reduced in primitive man on a civilized diet (Cambell 1938). This in itself can predispose the individual to at least third molar impaction.

Racial Hybridisation and Genetic Inheritance.

Other general factors operating in the aetiology of impactions are racial hybridisation and genetic inheritance.

On the mixing of races, Bjork (1950) says "It is a known fact that racial mixture increases the varieties, as borne out by Fischer's investigation of Boer–Hottentot hybrids (1913). Naturally it is also feasible that hybrids will exhibit a wide variation in respect of size of teeth and dental arches, and hence also in crowding..." As the vast majority of white races are extremely mixed, the degrees of variation are becoming more extreme and the problems of impacted teeth more severe.

The battle over the implications of genetic heritage still rages, after seventy years of heated discussions.

Strict followers of Angle and his school claim the separate inheritance of the jaws of one and the teeth of another is impossible and therefore impactions of genetic origin of this type do not occur.

However, the points put forward by Calvin Case (1911),
and others in the famous "extraction debate" of 1911 still hold good and the weight of evidence strongly suggests that the greater proportion of cases which display crowding and impactions through basal bone deficiency are the result of inheritable variables in the jaws and teeth.

**The Endocrine Glands.**

The function of the Endocrine glands have both a direct and an indirect bearing on the etiology of impacted teeth. This description of the effects of the endocrine system on malocclusions has been adapted from Salzmann (1943). The particular type of deformity depends upon the stage of development at which the disturbance takes place and the duration of the disfunction.

**The Thyroid Gland.**

This gland controls general metabolism by the secretion of thyro-globulin and controls bodily growth and development by complementing the function of the pituitary gland. The symptoms relative to dental crowding and impactions manifest themselves in hypothyroidism as disturbances in growth and development of the skeleton, such as stunting of growth and infantile skeletal proportions and naso-orbital configuration, delayed and defective tooth development and epiphysial dysgenesis. Congenital hypothyroidism shows itself as cretinism and is acquired during intra-uterine life. Irregularly
shaped arches, crowding and impactions are frequently found in these cases.

**Pituitary Gland (Hypophysis).**

The anterior lobe of the pituitary controls practically all growth phenomena during infancy, childhood and adolescence. This control is exerted through the pituitary growth hormone and through the influence of the pituitary and other endocrine glands.

Hypopituitarism in early childhood results very often in the lack of, or a delay in growth and development of the cranium and face, crowding of teeth, tendency to develop a deep overbite, a high narrow palate, constricted arches, and a receding chin.

**Thyropituitarism** is a pluriglandular disturbance which retards calcification of the teeth and produces arrested and retarded physical development. In thyropituitarism, there is a combined deficiency of the thyroid and anterior lobe of the pituitary glands. This causes even greater malocclusion through narrowing of the arches and crowding of the teeth.

The Parathyroids, Adrenals, the Thymus, the Pineal body and Gonads have varying effects on the formation of the teeth themselves, but are not a major factor in the etiology of impacted teeth.
Subclinical disorders of the thyroid and pituitary glands, however, although not recognised, may be a major factor in the aetiology of underdeveloped basal arches and the impaction of teeth.

**Local Factors in the Aetiology of Impacted Teeth.**

**Introduction.**

The teeth are derived from the dental lamina. A knowledge of the dental lamina is therefore necessary to understand the aetiology of those teeth which become impacted in jaws where there appears to be ample space for their inclusion in the arch.

These impactions often seem to be the result of a disturbance in the normal development of the tooth buds from this lamina at a time when the dental organ is not supported by a bony frame.

A description of the dental lamina is included here.

**The Dental Lamina.**

Sicher (1962 p.35) in his text book of oral histology, states that the first sign of tooth development occurs during the sixth week of embryonic life (11 m.m. embryo). At this stage, the oral epithelium consists of a basal layer of tall cells and a surface layer of flattened cells. The epithelium is separated from the connective tissue by a basement membrane. Certain cells in the basal layer
of the oral epithelium begin to proliferate at a greater rate than do the adjacent cells. An epithelial thickening extends along the entire free margin of the jaws. This is the dental lamina.

From this shallow dental lamina arise in both jaws ten round to oval swellings, corresponding in position to the future deciduous teeth — these are the primordia of the dental organs.

In all teeth, except the permanent molars, the dental lamina proliferates at its deep end to give rise to the dental organ of the permanent tooth, while it disintegrates in the region between the dental organ and the oral epithelium. The dental organ is gradually separated from the dental lamina at about the time when the first dentine is formed.

The dental lamina, distal to the dental organ of the second deciduous molar, extends distally in the 140 mm. embryo. The permanent molars arise directly from this distal extension, the first permanent molar arising at about 4 months in utero (160 mm. stage), the second permanent molar at about the first year, and the third molar at about fourth or fifth year.

The distal extension of the dental lamina is responsible for the development of the permanent molars in the ascending ramus of the mandible and tuberosity
of the maxilla.

Centres of ossification of the maxilla and mandible appear at about the 39th or 42nd day of intrauterine life. In the mandible, this centre is situated at the junction of the mental and incisive branches of the mandibular nerve. Bone formation spreads rapidly and continues forwards towards the midline and backwards along the outside of Heckel's Cartilage and forms a bony trough around the inferior dental nerve and its branches.

Aberrations of the Dental Lamina.

This bony trough is filled with a gelatinous mass of mesoderm, into which the dental organs are growing. It is conceivable that any undue pressures at this stage of development may be responsible for a disruption in the position of the dental germ, resulting in ectopically placed or impacted teeth.

Aberrations of the dental lamina are responsible for supernumerary teeth, and complex and compound composite odontomas, all of which may be associated with the impaction of permanent teeth.

A tooth bud may fail to form, or fail to develop after formation, resulting in a congenitally missing tooth, the most commonly affected being the maxillary lateral incisor, the third molars and the mandibular
second premolars.

Lack of Co-ordination of Local Growth During Eruption.

"The movements of the teeth during eruption are intricate and are accomplished by minute co-ordination of growth of the tooth, growth of the alveolar bone, and growth of the jaws. Any break in this correlation may affect the direction of the movements; this in turn may lead to an impaction or embedding of a tooth". (Sicher 1962 p. 319). This quotation from Orban's Oral Histology and Embryology gives some idea of the factors influencing the eruption of a tooth into its correct position in the arch.

Teeth whose axial inclinations are beyond normal limits may continue to erupt in the direction of this axis and become impacted, especially if the guiding influence of the adjoining teeth has been removed. (Cryer 1965)(Rose 1958). This can be seen especially in the third molar which, because of its impaction, has the second molar removed from in front of it. In many cases the third molar continues in its former direction and becomes impacted against the first molar. The lower second bicuspid with a distally inclined axis may continue in this distal direction after the first permanent molar has been lost for some reason and become impacted into the mesial aspect of the second molar. (Salzmann 1966 p.
Teeth may be deflected from their proper path of eruption by the presence of retained deciduous root fragments, cysts, various types of tumors, scar tissue and retained deciduous teeth.

Premature Loss and Ankylosis of Deciduous Teeth.

The premature loss of deciduous teeth has been recognised for many years as a prime factor in the etiology of malocclusions and impactions. (Salzmann 1966 p. 287).

Where there is a skeletal discrepancy, such as is seen with Class II Divisions 1 or 2 malocclusion, the effects of an early loss of deciduous teeth is much more severe (Horowitz and Nixon 1966), and impactions are much more likely.

When a deciduous tooth is lost, particularly a molar or canine, there is a tendency for the permanent teeth, adjacent to the remaining space, to drift either mesially, as in the case of permanent molars, or collapse distally, as with the anterior teeth, into this space.

These teeth then tend to usurp the space necessary for the unimpeded eruption of the succedaneous teeth, resulting in either their full impaction or in a crowding out of the line of the arch.

An ankylosed deciduous molar may become "submerged", 

295) (Cryer 1965).
allowing the adjacent permanent teeth to tip and trap the permanent successor in the alveolar bone or deflect its eruptive path. (Salzmann 1966 p. 315). Granulation tissue from a lesion on a deciduous predecessor may also displace a tooth, and perhaps lead to impaction (Bloch-Jørgensen 1931).

Other Local Factors.

Other local factors in the aetiology of impacted teeth may be listed as follows:

(a) Pressure habits such as thumb sucking.
(b) The sequence of eruption of the permanent dentition.
(c) The eruptive path and distance travelled to attain correct position in the arch.

(a) Thumb sucking may collapse the lower anterior teeth distally (Salzmann 1966 p. 298 fig. 168b), with the possible impaction of the second bicuspid and the third molar.

(b) The sequence of eruption of the permanent dentition has an important bearing on those teeth most commonly found impacted.

The most frequently found sequence of eruption in the maxilla are: 6-1-2-l-5-3-7-8 (48.72%).

6-1-2-l-3-5-7-8 (16.01%) (Lo and Moyers 1953). Gates (1963) has also found these sequences of eruption
to be most common in New South Wales school children but did not express them in a percentage form.

It is most unusual for the central and lateral incisors to experience difficulties in eruption provided there are no obstructions such as supernumerary teeth or odontomes. If there is insufficient space left by the shedding of the deciduous predecessors, the deciduous canine may be shed also, the permanent centrals usurping the position of the permanent canine.

The first bicuspid is the next tooth to appear. It is uncommon for this tooth to become impacted into an adjoining permanent tooth. If there is a lack of space for its eruption, the deciduous canine may be shed to accommodate it.

It should be noted that in the maxilla only the canine and second bicuspid erupt between permanent teeth and if there is not sufficient space in the arch to accommodate them, they are likely to become impacted, the last of the two to erupt, the canine, being the most likely to find difficulty in this respect. The second permanent molar rarely becomes impacted, unless there is an acute shortage of arch length associated with some form of growth retardation.

The third molar is most commonly found impacted in the maxilla; being the last tooth to erupt, and erupting
at an age when growth of the tooth bearing areas is virtually complete, even minor arch length deficiency with normally sized teeth is transmitted to this area.

In the mandibular arch, the sequences of eruption most commonly found are: 6-1-2-3-4-5-7-8 (45.7%) or 6-1-2-3-4-7-5-8 (18.64%) (Lo and Moyers 1953).

This sequence applies also to New South Wales School children (Gates 1963).

It will be seen that after the eruption of the first permanent molar the sequence of eruption is from before back, any erupting teeth usurping the space of the more distally placed tooth where there is a space deficiency. This leaves the second bicuspid frequently vertically impacted between the first bicuspid and the first molar.

The lower canine is rarely found impacted as its eruptive force is usually great enough to displace the small-rooted lower incisor teeth. This may happen where eruptive sequence is altered and the first bicuspid erupts before the canine.

Again, the mandibular third molar is found impacted most frequently (see later) for the reasons given in the case of maxillary third molar impactions.

(c) Finally, even in those jaws whose development is sufficient to contain all the teeth, we find impactions,
Fig. 1. Skull of a four year old child dissected to show the relationship of the erupting permanent canine.
Fig. 2. Skull of a seven year old child dissected to show the position of the erupting permanent canine.
particularly of upper cuspids and (Dewel 1949 p. 83) mandibular third molars. These teeth originate some distance from their final position in the arch (see fig. 1 and 2) and any slight force which tends to deflect them initially can lead to severe displacements and impactions. (Marsh 1965).

The lower third molar develops in the ramus of the mandible, and only by sufficient resorption of the anterior border of the ramus can it erupt into normal occlusion. These molars rotate from a forward direction during their course of eruption as they come into occlusion. (Salzmann 1966 p. 115).

Dowel (1949 p. 84) on the subject of the impaction of the upper cuspids, will be quoted here at length. "Possibly the original cause of the cuspids impaction lies in the extent of eruption required of these teeth. Opportunity for deflection from a normal course increases in proportion to the distance a tooth must travel from its point of origin to full occlusion. To reach full occlusion the cuspid travels the longest and most roundabout course of any of the permanent teeth. As with all succedaneous teeth, the permanent cuspid germ branches off from the dental lamina that lies between and connects the deciduous cuspid bud to the oral epithelium. Thus the permanent
germ at first is of necessity occlusally and somewhat lingually to the deciduous crown. With the gradual eruption of the deciduous tooth and the natural increase in height of the alveolar process, the permanent cuspid germ shifts more and more to the lingual until the deciduous cuspid passes it into the dental arch.

"At the age of 12 months, calcification of the permanent cuspid is far enough along to show roentgenographically and in dissected specimens, and now the tooth is found not directly above the root of the deciduous cuspid as normally would be expected, but instead between the roots of the first deciduous molar. Since this is also the area where the first bicuspid is due to begin calcification at from 1½ to 2 years of age, the cuspid continues its apparent upward travels, or the other structures proceed occlusally with further vertical growth, until the area is cleared for the first bicuspid. At this time the permanent cuspid is located immediately above both the first bicuspid and the first deciduous molar — three teeth in a row, one above the other. From this point the cuspid must migrate forward at a greater rate than the normal forward growth of the maxilla and of the deciduous teeth to reach its final developmental position above the apex of the deciduous cuspid."
"All of this is occurring in the most crowded portion of the arch in an area where the cuspid crown in its earliest stages is not far below the orbit and at all times at a higher level than any of the other developing permanent teeth. In assuming any of these various developmental positions it may be deflected palatally in its direction of eruption. Normally, however, its ultimate developmental position is lingual to and above the apical plane of the deciduous teeth well up in the region between the nasal cavity and the maxillary sinus. Usually the base of its crypt is at a slightly higher level than the floor of the nasal cavity and so located that it is braced against the solid bone of the lower border of the malar process. From this area it slowly proceeds occlusally to its final location in the dental arch. In contrast, teeth which develop in a constant position comparatively close to the surface do not have, in the shorter and more direct distance they need to travel, as great an opportunity for directional deviation as that of the deeply placed cuspid."

"Teeth that are late in appearing seem also to have a greater responsibility in preparing their own way into the dental arch. Unlike bicuspids, which replace teeth equal to or greater than their mesiodistal diameters, the cuspid is preceded by a deciduous tooth which is by no means its equal in width. As it erupts, it wedges itself
between teeth already established in occlusion, and in
time of appearance is also competing for space with the
currently erupting second molar".

Benson (1967) has an interesting conjecture on the
aetiology of impacted third molars. He believes removal
of a bicuspid and rapid forward movement of first and
second molars by orthodontic means at an early age allows
the mesially directed lower third molar to tip downwards
even further and become almost horizontal. The guiding
influence of the distal root of the lower second molar
is thus moved away, resulting in a severely impacted
molar, rather than creating more space in the arch for
the molar to erupt. He points out that in some cases,
removal of a bicuspid can result in impaction of the third
molar, rather than prevent it.

To summarise this section on the aetiology of impacted
teeth, we have considered six general factors influencing
the shape and size of the jaws and the development of
occlusion and six local influences which have a bearing
on the individual tooth positions.

The general factors may be listed as:-

1. Evolution,
2. Domestication of primitive man,
3. The effects of diet (a) Functional,
   (b) Nutritional.
(c) Attritional.

4. Racial Hybridisation.
5. Genetic Inheritance.
6. The Endocrine System.

Local factors influencing tooth positions are:

1. Abnormalities of the dental lamina.
2. Co-ordination of local growth during eruption.
3. Premature loss and ankylosis of deciduous teeth.
4. Pressure Habits.
5. The sequence of eruption.
6. The Length and direction of the path of eruption.
Table 1: An analysis of 6,789 cases for the incidence of impacted teeth.

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<th>Type</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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Legend:
- Cases: Number of cases analyzed.
- Race: Classification of race categories.
### Table II

**Number of Impacted Teeth in 1462 Office Cases**

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<th>MAXILLARY AND MANDIBULAR</th>
<th>NO.</th>
<th>PER CENT</th>
<th>MANDIBULAR</th>
<th>NO.</th>
<th>PER CENT</th>
<th>MAXILLARY</th>
<th>NO.</th>
<th>PER CENT</th>
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<td>3</td>
<td>1.12</td>
<td>9</td>
<td>3.53</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>0.96</td>
<td>–</td>
<td>–</td>
<td>5</td>
<td>1.96</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lateral Incisors</td>
<td>1</td>
<td>0.02</td>
<td>–</td>
<td>–</td>
<td>1</td>
<td>0.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canines</td>
<td>25</td>
<td>4.8</td>
<td>2</td>
<td>0.74</td>
<td>23</td>
<td>9.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First Premolars</td>
<td>1</td>
<td>0.02</td>
<td>1</td>
<td>0.37</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second Premolars</td>
<td>11</td>
<td>2.1</td>
<td>9</td>
<td>3.39</td>
<td>2</td>
<td>0.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First Molars</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second Molars</td>
<td>2</td>
<td>0.04</td>
<td>1</td>
<td>0.37</td>
<td>1</td>
<td>0.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third Molars</td>
<td>461</td>
<td>89.76</td>
<td>248</td>
<td>94.01</td>
<td>213</td>
<td>83.81</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>518</td>
<td>100.0</td>
<td>264</td>
<td>100.0</td>
<td>254</td>
<td>100.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table III

<table>
<thead>
<tr>
<th>RACE</th>
<th>NUMBER OF MANDIBLES EXAMINED</th>
<th>NUMBER OF IMPACTIONS FOUND</th>
<th>PER CENT</th>
<th>NUMBER OF MAXILLAS EXAMINED</th>
<th>NUMBER OF IMPACTIONS FOUND</th>
<th>PER CENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Office Cases</td>
<td>1462</td>
<td>264</td>
<td>18.05</td>
<td>1462</td>
<td>254</td>
<td>17.3</td>
</tr>
<tr>
<td>White (Smithsonian)</td>
<td>888</td>
<td>45</td>
<td>4.68</td>
<td>350</td>
<td>10</td>
<td>2.85</td>
</tr>
<tr>
<td>Eskimo (Smithsonian)</td>
<td>1187</td>
<td>48</td>
<td>4.04</td>
<td>585</td>
<td>26</td>
<td>4.44</td>
</tr>
<tr>
<td>Peruvian (Smithsonian)</td>
<td>481</td>
<td>57</td>
<td>11.9</td>
<td>1345</td>
<td>158</td>
<td>11.75</td>
</tr>
<tr>
<td>Indian (Smithsonian)</td>
<td>230</td>
<td>14</td>
<td>6.09</td>
<td>301</td>
<td>23</td>
<td>7.64</td>
</tr>
<tr>
<td>Mongolian (Smithsonian)</td>
<td>37</td>
<td>2</td>
<td>5.4</td>
<td>117</td>
<td>8</td>
<td>6.84</td>
</tr>
<tr>
<td>Negro (Smithsonian)</td>
<td>117</td>
<td>4</td>
<td>3.42</td>
<td>83</td>
<td>2</td>
<td>2.41</td>
</tr>
<tr>
<td>Egyptian 12th Dynasty (Smithsonian)</td>
<td>6</td>
<td>–</td>
<td>–</td>
<td>115</td>
<td>9</td>
<td>7.82</td>
</tr>
</tbody>
</table>

Table 2. Number of Impacted Teeth in 1,462 Office cases.

Table 3. Frequency of Impacted Teeth in 6,389 cases of both Civilised and Primitive Man.

Taken from Mead (1930).
THE INCIDENCE AND PREVALENCE OF IMPACTED TEETH.

Incidence.

Mead (1930) examined some 6,389 cases for impacted teeth. Of these, 1,462 were studies of full-mouth x-rays of patients attending surgery for routine dental treatment.

The remaining 4,927 cases were skulls in the Smithsonian Institution, routinely x-rayed for impactions. No account was taken of sex or age or the complement of teeth in the arches examined. From Table 2, the order of frequency of impactions in 1,462 routine general dental patients in Washington was:

<table>
<thead>
<tr>
<th>Position</th>
<th>Tooth Type</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mandibular third molars</td>
<td>248</td>
</tr>
<tr>
<td>2</td>
<td>Maxillary third molars</td>
<td>213</td>
</tr>
<tr>
<td>3</td>
<td>Maxillary Canines</td>
<td>23</td>
</tr>
<tr>
<td>4</td>
<td>Mandibular second bicuspids</td>
<td>9</td>
</tr>
<tr>
<td>5</td>
<td>Maxillary Supernumeraries</td>
<td>9</td>
</tr>
<tr>
<td>6</td>
<td>Maxillary central Incisors</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>Mandibular Supernumeraries</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>Mandibular Canines</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>Maxillary second premolars</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>Mandibular first premolars</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>Mandibular second molars</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td>Maxillary Lateral Incisors</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>Maxillary second molars</td>
<td>1</td>
</tr>
</tbody>
</table>
(Mandibular Central and lateral incisors 0 impactions
Mandibular first molars 0 "
Maxillary first premolars 0 "
Maxillary first molars 0 "

Of the 1,462 cases studied, there were 276 patients displaying 518 impacted teeth between them.

Dachi and Howell (1961) did a further study on the incidence of impacted teeth. On the subject of Mead's work, they say: "Although these figures are indicative of the frequency with which impactions occur in patients, they are not truly representative of the incidence of impactions. The latter can be derived only from comparing the number of impacted teeth with the number of normally erupted teeth found in a population sample".

To summarise Dachi and Howell's work on the incidence of impactions, we find in a study of 3,874 routine full-mouth radiographs, the incidence of patients with at least one impacted tooth was 15.7 per cent.

The teeth most commonly found impacted, in order of frequency were the maxillary third molar, mandibular third molar, the maxillary cuspid and the mandibular premolars. This is at variance with Mead and Hellman, who put the incidence of mandibular third molar impactions greater than that of the maxillary third molar). The incidence of impaction was 21.9 per cent, for maxillary third molars and
17.5 per cent. for mandibular third molars.

The incidence of impaction among maxillary cuspids was 0.92 per cent.

No statistically significant predisposition towards bilateral impaction was noted among third molars, while a definite tendency toward unilateral impaction was found in maxillary cuspids.

Prevalence.

Sex differences in the impaction of teeth.

In his studies on the third molar tooth, Hellman (1936 p. 758) suggests that the incidence of impacted third molar teeth is greater in females than males. He gives figures of 9.51 per cent. in males and 23.8 per cent. in females showing at least one impacted third molar. Dachi and Howell (1961), on the other hand, referring to the incidence of third molar impactions, in their own work, state: "These figures indicate that, although a slight difference is apparent, it is not statistically significant. There are no sex differences in the incidence of impacted third molars in this study".

Dachi and Howell studied the sex differences in the incidence of canine impaction and concluded, "A difference noted between the females (1.17 per cent.) and the males (0.51 per cent.) was at the borderline of being statistically significant at the 5 per cent. confidence level".
Rohrer (1929) in his work on displaced and impacted canines found, "An enormous preponderance of displacements in favour of the female in the case of maxillary canines, amounting to 71.9 per cent, as against 28.1 per cent.

He explains this by suggesting that the female cranium, jaws and dental arches are smaller, therefore more prone to impactions. This is difficult to reconcile with the fact that together with the jaws the teeth of females are also generally smaller than those of males.

This has been shown by Moorrees (1959), who showed also that the largest sex difference was in the canines. These observations apply also to the teeth of Australian Whites, as shown by Sandilands (1966).

Prevalence in Primitive and Modern Man.

Mead (1930) has shown that impacted teeth are considerably more prevalent in modern man than in ancient or primitive man.

His findings are summarised in Table 3.

These findings bear out those points made previously in that section describing the general etiology of impacted teeth.
B. DENTAL ERUPTION

Definition.

The term "Eruption" describes the movement of a tooth from its intraosseous location in the jaw to its final position in the oral cavity. In man, this eruption process usually occurs throughout life, and does not cease with the attainment of full occlusion.

Eruption continues to compensate for occlusal wear.

Eruption may be passive or active. (a) Passive eruption is the apical movement of the epithelial attachment (gingival recession) in the erupted tooth, and is not pertinent in this work. (Sicher 1962 p. 243).

(b) Active eruption is the movement of a tooth from its bony crypt to full functional occlusion, and perhaps beyond, if there is no antagonist.

Three phases may be recognised:

1. Intraalveolar.
2. Intraoral.
3. Continuous eruption.

The Mechanism of Eruption.

(a) Historical.

One of the earliest references pertaining to the mechanism of eruption is found in Charles Allen's book, "The Operator for the teeth" published in 1686, as has been previously mentioned. On the process of eruption,
he says "Nature having perfected our growth, the BLOOD becomes hotter, stronger and its ENERGY more powerful by the firmness of the HEART... being able to surmount the resistance of the jaw and gums, forces the said teeth to come out of their cells and erupt".

Allen had thus foreshadowed the famous work of Constant (1896) which is the basis of the modern concept of the mechanism of eruption. John Hunter (1728-1793) described the gubernacular cords and canals. "When the incisors and cuspids of the new set are a little advanced, but long before they appear through their bony sockets, there are small holes leading to them on the inside, and behind the temporary sockets and teeth: and these holes grow larger and larger 'till at last the body of the tooth passes quite through them". (Scott 1953).

The gubernacular cords, described by the early anatomists, were lost sight of in the nineteenth century, and only recently have they been "re-discovered" in relation to the eruptive process (James 1923) (Scott 1953).

In 1835, Thomas Bell wrote on the subject of eruption and regarded the elongation of the roots as the precipitating cause. (Constant 1896), Kolliker re-iterated Bell's ideas in 1854 (Brodie 1959). Harris (1850 p. 114) in his textbook "The Principles and Practice of Dental Surgery" subscribed to a rather quaint and romantic theory on tooth
eruption, initially propounded by Delabarre. "...the passage of a tooth through the gum, or rather its escape from its matrix, is effected in precisely the same manner as is the birth of a child... the sac, which is attached to both the gums and the neck of the tooth, as the chief agent, and believes that the latter is raised from the bottom of the alveolus and ultimately forced through the dilated orifice of the former and the gums.

"This is the most rational theory that has been advanced; it explains, upon principles of sound physiology, this most wonderful and curious operation of the animal economy."

Pierce (1887) made some interesting suggestions on the eruption of a fully formed tooth in later life "The repeated closing of the jaws must exert to a large extent this mechanical force, just as the bung in a barrel is elevated by a blow being struck upon a stave on either side of it". He also suggested eruption was caused by an increase in the "pulpy mass or formative tissue and its calcification together with resorption of the overlying tissue of the advancing crown."

Gottlieb (1920) suggested that eruption was a similar process to the shedding of a foreign body (the enamel) from the tissue, shown in his work on rachitis and enamel hypoplasia.
Modern theories on the mechanism of eruption began with the work of Constant (1896) and have been extensively reviewed by Ness (1964) in the most recent work on this subject.

Modern Theories on the Mechanism of Eruption.

There are at least eight major theories on the eruption of the teeth. None of these theories is supported by all the evidence and the exact role of each of these processes is obscure. Some authors suggest a combination of several of these eight theories.

These theories will be enumerated below:

1. Growth and elongation of the root.
2. Growth of dentine and pulpal constriction.
3. Growth of periodontium, including alveolar bone.
4. Resorption of alveolar crest bone with subsequent tooth exposure.
5. Pressure from cellular proliferation of pulp.
6. Shrinkage of maturing collagen at the hammock ligament and periodontal membrane.
7. Elastic deformation of surrounding bone by masticatory forces.
8. Pressure due to periapical vascularity.
1. ROOT ELONGATION.

Growth and elongation of the root of a tooth pushing the crown into the oral cavity is the most simple and direct mechanism explaining dental eruption and was probably one of the first theories on this subject. (Hunter, Magitot, Nasmyth, Sarogen and Wedel cited from Massler and Schour 1941), (Bell cited from Constant 1896), (Kolliker cited from Brodie 1934). That this process plays only a minor role can be shown by the facts revealed by everyday dental observations, such as the eruption of rootless teeth, eruption after root elongation has ceased and the fact that a cuspid must travel a greater distance than its root length to reach occlusion.

Although root elongation is not the major factor in eruption, it may play a minor role and should not be entirely discounted.

2. PULPAL CONSTRICTION.

It has been suggested that the growth of dentine and subsequent constriction of the pulp may deliver enough force to propel the tooth coronally. This theory again does not explain the eruption of rootless or fully formed teeth, and the evidence given is purely circumstantial.

3. GROWTH OF PERIODONTIUM AND ALVEOLAR BONE.

"Underwood (cited by Tomes 1923) suggests that the connective tissue surrounding the tooth may function in
pulling the tooth into the oral cavity. He cites the teeth of continuous succession in fish and reptiles in support of this view. It is not certain whether Underwood intended to apply this theory to the teeth of higher forms. In the latter case, the theory would imply that the periodontal membrane would function to pull the tooth into occlusion. This theory is invalidated by histologic examination of the direction of the periodontal fibres during tooth eruption, which shows that the periodontal fibres are being pulled by the tooth, and do not themselves pull upon the tooth". (Massler and Schour 1941).

Brash (1928) on dental eruption says "Bone growth... raising erupting teeth from their crypts to the level of those already erupted, which are also moving upward at a slower rate. The proximate cause of the eruption of the teeth is a local intensification of the general upward growth of alveolar bone" and later "...the growth of cementum is an additional factor in the upward movement of teeth both erupting and erupted". He also discounts root elongation as a factor.

Alveolar bone growth is not the primary factor in eruption as is shown by Massler and Schour (1941), who explain that on extraction of a deciduous molar, the underlying premolar may erupt so rapidly as to leave the bone behind.
The alveolar bone depends for its existence on the presence of the teeth, and not the reverse. This theory can also be discounted by the fact that a tooth can erupt in a different direction to the general direction of growth trends.

Weinmann and Sicher (1944) have written "Developmental movements of tooth germs, eruptive movements of teeth and physiologic movements of teeth during their functional period are the result of differential growth of the tooth and surrounding bone... movement of the teeth is, therefore, but a component of the growth of the jaws in the elaboration of the genetically determined growth pattern".

4. RESORPTION OF ALVEOLAR BONE.

It has been postulated that resorption of the alveolar crest and opening up of the gubenacular canals would serve to expose the crown and allow it to erupt into the oral cavity (Aichel and Weidenreich, cited by Massler and Schour 1941). This theory can also be discounted by the fact that teeth can travel through bone and fail to reach the oral cavity.

5. PRESSURE FROM CELLULAR PROLIFERATION OF PULP.

Sicher (1942 p.402) suggested that pulpal growth against the "hammock-ligament" and in a confined space provides the impetus to eruption, whereas bone growth at the alveolar fundus provided the "force" after the root
had developed. Scott (1953) also suggested pulpal proliferation between the calcified tooth and the follicular base, along the path of the gubernacular canals as the mechanism of eruption.

Bryer (1957) does not agree with Sicher on the cellular proliferation theory. He observed an increased cellular proliferation of the pulp in rickets, hypervitaminosis D and in vitamin A deficiency and yet this was associated with a reduced eruption rate.

Harris and Griffin (1964) described the eruption of pulpless, rootless teeth and attributed their movement into the arch to "growth of the follicle".

6. SHRINKAGE OF MATURING COLLAGEN.

Shrimpton (1960) developed the theory that the shrinkage consequent upon the maturation of the principal fibres of the periodontal membrane and the "cushioned" "hammock-ligament" of Sicher in the prefunctional stage of eruption produces the "force" which causes teeth to erupt. Bone growth is purely adaptive.

It is difficult to reconcile this theory with the early eruptive phase when it is sometimes seen that the direction of the periodontal collagen fibres is unfavourable for lifting the tooth in the direction of eruption.

Shrimpton suggests also that eruption compensating for attrition is affected by the shrinkage of the collagen fibres
in the periodontal sling. Considering the direction of the gingival and interdental fibres, and the existence of an "intermediate - plexus" in the oblique fibres, this mechanism seems rather obscure, but could still be a factor in the difficult question of eruption of fully formed teeth.

7. ELASTIC DEFORMATION OF BONE

The elastic deformation of bone under masticatory stress has been shown by Picton (1962) and others. Picton described the distortion of the mandible and it is conceivable that this distortion could also occur in the maxilla.

Shadle (cited by Ness 1964) found that a porcupine quill, because of its shape and its barbs, could move through human tissues as much as 25,000 µ per day. This is an extreme instance of the way in which a body, embedded in tissue which is reversibly and periodically changing shape, can be translated because some feature of its shape gives a preferred direction to its movements.

Pierce (1887) as mentioned in the historical section, suggested that this mechanism may apply in the movement of fully formed teeth through the jaws and it does appear logical that minute movements can occur between an impacted tooth and the functional impacting tooth providing the necessary stimulus to root resorption.
8. PERIAPICAL VASCULARITY

Constant (1896) was the first to publish the theory of the vascular influence in eruption. He wrote "The blood pressure exerted in the vascular tissue which lies between a developing tooth and its bony surroundings is the active mechanical factor in the process known as the eruption of teeth".

Bryer (1957) in numerous experiments on the rat incisor came to the conclusion that the eruptive force is derived predominately from the tissue tension within the pulp and periodontal tissues and that this tension is dependent upon the dynamics of the blood circulation.

Haasler and Schour (1941) concluded "that the eruptive force or the force responsible for the tooth movement during eruption, may be related to the vascularity of the tissue which surrounds the tooth."

Ness (1964), relating the research of Bryer and many other workers and critically reviewing the results, concluded that there are possibly several factors operating to bring a tooth into occlusion, but the greatest single factor is probably periapical vascularity and its influences.

Sicher (1962 p. 314-315) has presented probably the most clear picture of the mechanism of eruption, and
possibly the most logical. He says "The eruptive movements of a tooth are the effect of differential growth....the growth of the pulpal tissue causes a slight increase of pressure in the confined space of the dental crypt. Resorption of the bone at the fundus of the crypt is prevented by the suspension of the tooth germ by the hammock ligament....the rise of pressure in the crypt is the stimulus for the initiation of tissue changes that will reduce the pressure. Thus, pressure does not build up but acts in a wave-like fashion, rhythmically rising and falling...its vertically erupting movement is aided by growth of bone at the bottom of the crypt, lifting the growing tooth, with the hammock ligament, towards the surface".

Sicher does not mention the theory of Bryer and others that blood circulation dynamics is the main erupting influence.

A human tooth erupts throughout life to compensate for occlusal wear, and generally moves mesially to adjust the occlusion in the case of interproximal wear. The mesial migration of teeth is explained usually by the anterior component of masticatory force, but vertical eruption is more difficult to account for.

Sicher suggests that cementum is being continually deposited throughout life, especially in the apical and furcation areas of the root not only to provide attachment
for the constantly renewing Sharpey's fibres but to provide
a mechanism to compensate for loss of occlusal tooth
substance through attrition.

Bone apposition at the fundus also provides a mechanism
to account for continuous eruption, but Slicher raises the
question whether the bone changes are primary and cause the
tooth movement or are only adaptive.

Brodie (1934), commenting on the status of knowledge
thirty years ago, wrote "the mechanism whereby the tooth
germs are able not only to travel with this bone, but
actually exceed it, remains unexplained".

Summary.

The exact mechanism of eruption is obscure and many
theories have been put forward to explain it. None of
these theories is the complete answer.

All that can be said is that the teeth erupt under
the influence and guidance of some tissue force or forces
which may change or be replaced by other forces at
successive stages of eruption.
Eruptive Force

When considering impacted teeth and eruptive forces, one must consider the active "force" exerted by an erupting tooth on the impacting tooth, and whether the magnitude, duration or constancy of such a force has any relationship to the initiation of a resorptive process in the root of an impacting tooth.

Sicher (1942 p. 201) does not believe any particular "force" such as a "measurable" orthodontic force, is involved. He believes that these are minute differences in tissue tensions, or disturbances in equilibrium. These forces are just strong enough to act as stimuli for re-establishment of the equilibrium and are enough for tooth movement in eruption.

In orthodontic therapy, extremely light forces, provided they are applied constantly, will move teeth. Schwarz (1932) suggests forces should be about blood capillary pressure (20-26 gm/sq.cm.) for the most favourable biologic tooth movement.

Forces which are considered as very light by orthodontic standards (Oppenheim 1944) (Smith and Storey 1952) are gross compared with the forces of eruption. This is not because large forces are necessary to promote the stimulus to osteoclastic activity. To control minute differences in tension and at the same time surmount the obstacles to
tooth movement such as interferences from occlusion and appliance inefficiencies, is beyond the ability of any orthodontic device so far invented.

Ness (1964 p. 57) asks the question "Do morphogenetic forces exist?" He concluded that eruptive movement is evidence of the existence of forces. Absence of movement is no indication that the forces had vanished, but merely reached a state of equilibrium.

Ness here quotes the work of Taylor and Butcher on the eruptive force of a rat incisor. They found a force of 2 grams retarded the unimpeded eruption rate, 5 grams prevented eruption and greater forces brought about a recession of the tooth into its socket. Ness chose a value of 4 grams as the unimpeded eruptive force of an incisor in a 150 gram rat.

An examination of the literature did not disclose any quantitative values for this force in the human dentition.

Maclillian (1927), writing on the pressure developed by an impacted third molar says "Pressure seems to be incompatible with physiologic laws...gentle pressure constantly applied causes absorption". He also says, speaking of the balance of forces in the process of eruption, "The two processes are so nicely balanced that no measuring device is sufficiently delicate to measure the difference in the factors involved".
He suggests that the secretory ability of a cyst brings about sufficient pressure to initiate resorption of the roots of the susceptible teeth.

**Factors Influencing Rate of Dental Eruption.**

The rate at which a tooth erupts is governed by many factors, both local and systemic. In assessing the "force" of eruption, we can only assume that those factors which influence the speed of eruption may also influence the force — although this may not necessarily be true.

With an erupting tooth or an impacted tooth, depending on the degree of root development and the age of the patient, it is conceivable that the force of eruption is not steady but waxes and wanes with various alterations in systemic and local conditions.

Bryer (1957) has shown the effects of altering these influences on the rat incisor. He observed that rickets and toxic excess of vitamin A and D reduced the unimpeded eruption rate of the incisors, and factors which increased the local vascularity of the periapical area increased the eruption rate.

The speed at which a tooth travels through bone may be influenced by the endocrine glands. They can influence this rate directly or indirectly, by interfering with the normal rate and direction of bone growth, the physical characteristics of bone, and the basal metabolic rate.
In hypothyroidism, eruption of the permanent dentition is delayed, the teeth develop slowly and show few structural defects, whereas hyperthyroidism produces an increase in metabolic rate with accelerated eruption of the permanent teeth. (Schour and Massler 1943).

Hypofunction of the pituitary gland greatly retards dental eruption, hyperfunction having the opposite effect of increasing developmental and eruptive rates.

In thyropituitarism there is a combined deficiency of the thyroid and anterior lobe of the pituitary glands which can lead to a considerable delay in eruption.

Disorders of the adrenals and the thymus glands at the time of eruption may influence the times and rate of eruption. (Salzmann 1966 p. 25).

Subclinical imbalance or disorders of these hormonal controlling mechanisms may have some effect on the eruptive mechanism and may be a factor influencing the force of eruption.

The nature of the tissue through which a tooth must pass may affect the eruption rate but not necessarily the "force" of eruption. Scar tissue and tough fibrous tissue, such as the anterior palatal mucosa can prevent further eruption completely.

Local inflammatory reactions such as are initiated
by surgical exposure of impacted teeth, by creating an increased tissue tension, may provide sufficient stimulus to allow the tooth to complete the eruptive process. (Mitchell 1960).
C. THE MECHANISM OF RESORPTION AND REPAIR OF THE TEETH.

1. Historical.

John Hunter (1728-1793) described the exfoliation of the deciduous dentition and recognised the fact that the roots were destroyed by some process before the teeth were shed. He did not actually speak of "resorption" but used the word "decay" to describe the resorption of the roots of the deciduous teeth and showed various examples of deciduous teeth in the process of shedding (Colyer 1913).

Harris (1850 p. 130) quotes Fauchard (1678-1761) and Bourdet as attributing the resorption of the deciduous teeth to a corrosive fluid, secreted for this purpose. He quotes Bunau (1743) as thining the roots were worn away by the erupting successors.

Delabarre (1819), also cited by Harris, raised the question as to the actual process of resorption of the deciduous tooth roots "Is there a dissolving fluid that acts chemically on the surrounding parts, or do the absorbents, without any intermedial, destroy everything that would obstruct the shooting up of the tooth?"

But it was not until 1856, that, according to Henry and Weinman (1951) external resorption of the permanent teeth was described by Bates. Since that date, resorption of the roots of the permanent dentition had received a considerable amount of attention by many researchers, but
it was not until the advent of the x-ray, discovered in 1895 by William Conrad Roentgen, that the prevalence of root resorption in the human permanent dentition was really appreciated. The first x-ray of the teeth was taken by Dr. W. Koenig of Frankfurt in 1896.

Dr. C. Edmund Kells, of New Orleans, was perhaps the first dentist to use the x-ray clinically, in 1896 (Bremner 1939) but it was at least fifteen years before the dental profession fully accepted the value of x-rays in clinical diagnosis.

It was at this time, in the years immediately before the First World War, that the problem of root resorption from orthodontic tooth movement was recognised fully. Ottolengui (1914) was probably the first to bring this effect of tooth movement by orthodontic appliances before the profession. He also described at this discussion root resorption in a second molar in association with an impacted wisdom tooth.

Root resorption in the permanent dentition associated with impacted teeth was recognised long before this. Hutchinson (1874) described a similar occurrence in a woman and references to this type of resorption can be found throughout the dental literature from this date (Canton 1883), (Verley 1898).

Research into the problem of root resorption gained
impetus in the 1920's with the works of Mumery (1920) Ketcham (1927) and Zemsky (1929). Mumery described at great length the pathology of internal resorption and named them "Pink Spots".

Ketcham was the first, and probably the only, worker to consider teeth which had lost large amounts of root structure from impactions from the point of view of the orthodontist and to assess their prognosis. He considered this event quite common.

Zemsky formulated a complicated classification of root resorption and included root resorption from impactions in a subsection of its own. (Group 6b).

The 1930's saw the greatest volume of work on root resorption in any decade, with the works of Hopewell-Smith (1930), Marshall (1930) (1935), Rudolph (1935), Becks (1936) Stuteville (1937)(1938) and others.
THE MECHANISM OF RESORPTION

Introduction.

All calcified tissues found in the human body may be subject to resorption. (Sognnaes 1963 p. 91).

"Resorption is, in essence, the putting into solution of a complicated structure, in such a fashion that it disappears, its end products entering the blood stream. Resorption always progresses inward from the surfaces of bone; it never arises within the deeper layers of the structure. Of the components of bone, a small fraction is already in fluid form, and hence easily disposed of; the remainder is in solid form, for the most part insoluble or soluble with great difficulty in aqueous fluids. In order to resorb bone it is necessary that it be reduced to substances soluble in water, and that these be transferred to the fluids of the body". This description of the resorption of bone (McLean 1956 p. 712) applies to cemental and dentinal resorption as well.

Resorption of bone is a normal physiologic process which is occurring constantly throughout the skeletal system and is essential to maintain the proper function of the bony framework. Old bone is being removed and replaced by new for the entire life of the individual and the skeletal system can be used as a "store-house" of salts which can be called upon to suit the needs of the body metabolism.
This property of bone is made possible by its system of anastomosing osteocytic canaliculi and by its lavish blood supply, which are necessary pathways for the flow of mineral salts. (Pritchard 1956 p. 18).

Dentine and cementum, on the other hand, resemble bone in their crystalline, mineralised structure, but do not have the properties of constant renewal possessed by bone, nor do they possess the vascular pathways and cellular qualities necessary for this process.

"It is noteworthy, in the case of dental resorption that the origin of the multinucleated osteoclasts (perhaps more appropriately called 'odontoclasts') cannot be attributed to the fusion of antecedent uninucleated cells within the tissues undergoing resorption. Neither enamel, dentine nor primary cementum contain interstitial cells". (Sognnaes 1963 p. 97).

From this it can be seen, that when examining the process of dental resorption, we can call a close parallel with bone resorption except for this one point. Cells derived from tissues adjacent to the resorptive process are responsible for the hard tissue destruction, unlike bony resorption, which can be brought about by differentiation of the bone cells themselves. (Pritchard 1956 p. 232).

This is mentioned here, as much of the work applied to dental resorption has been taken from investigations on bone.
It is generally accepted that local cellular (osteoclastic) activity is responsible in some way for the resorption of hard structures. (Boothroyd & Hancox 1963 p. 497).

This does not mean that osteolysis, or resorption without osteoclasts, cannot occur, although this mechanism, propounded by Frey in 1874, is usually regarded as academically interesting but difficult to demonstrate. (Bouyssou et al. 1965 p. 45) (Hancox 1956).

The Osteoclast.

When considering the resorption of cementum, dentine, or bone, we must first examine the cells involved.

The cells found in association with the destruction of the hard tissues of the human body are osteoclasts and giant cells.

These cells have been regarded in the past as separate cell types but they are now looked upon as different morphologic forms of the same cell (Irving and Handelman 1963), (Cameron 1967). Other cells, such as the histiocytes or macrophages arising from the reticulo-endothelial system, have been associated with resorption by some authors (Sullivan and Jolly 1957 p. 196), but these cells are generally not regarded as being primarily responsible for the direct destruction of bone, dentine and cementum.
Origin.

This description of the osteoclast is largely derived from Hancock's work in "The Biochemistry and Physiology of Bone" (1956 p. 213-240) and "Orban's Oral Histology and Embryology" (Sicher 1962 p. 243).

The origin of the osteoclast is still open to conjecture but it is generally thought to have a common precursor with not only the multinucleated giant cells but also the osteoblast and osteocyte.

This precursor cell, Sicher (1962 p. 213) calls the "undifferentiated mesenchymal reserve cell" or the "wandering" cell of Hancock (1956 p. 213). This is perhaps the "eiocyte" of Hopewell-Smith (1930) also.

The osteoclast may differentiate from these cells either by mitotic division of nuclei, without a following division of the cytoplasm or by the fusion of several uninucleated cells, (Hancock 1956 p. 230). It is not certain whether fusion or mitosis is responsible, but whatever the process, the osteoclast is almost always multinucleated, having up to 100 nuclei.

The actual stimulus which leads to differentiation of mesenchymal cells is not known. Resorption by osteoclasts may be partly genetic and partly functionally determined. Differentiation may be initiated by either chemical stimuli (from dead or degenerating hard tissues or hormonal influences) or from electro-mechanical stimuli
(Piezo-electric stimuli in stressed crystalline bone structures. (Shamos 1965)).

Microscopical Appearance in Fixed Tissue Sections.

Size.

Osteoclasts vary greatly in size, they range from 200,000 $\mu^3$, the largest cell in the mammalian body, containing a hundred or more nuclei, down to cells of two or three nuclei. The cells most commonly encountered are in the smaller range, from 2-5 nuclei.

Shape.

The variety of shapes is enormous, ranging infinitely between two extremes. Firstly, there are round or oval masses containing usually 10-20 nuclei. They appear to be separated from the bone by thin strands of connective tissue. The nuclei are round and vesicular. At the other extreme, the cells form thin sheets against the bone. The nuclei are usually flattened also. They may be in association with blood vessels and appear as endothelial cells.

When occupying Howship's Lacunae, the cells are roughly semicircular, with a flattened surface away from the bone. They may have one or two cytoplasmic lobes, with separate clusters of nuclei and each lobe may occupy its own lacuna. Some cells appear to have cytoplasmic branching processes, both short and long (or filiform). The mobile nature of the cell may account for these varying shapes.
Nuclei.

The nuclei are rounded or oval, with one or two nucleoli and are rather vesicular, but there is a considerable variation in appearance. They may be shrunken, crenated and box-like or frankly pyknotic.

Cytoplasm.

The cytoplasm of the osteoclast is usually coarsely granular but these granules are not always present. The staining reaction is variable, being basophil in young cells grading through oxyphil in the active phase to strongly oxyphil when the cells begin to degenerate. The most outstanding microscopical feature of the osteoclast is the "Brush", "striated" or "ruffled" border, found between the cell and the subjacent bone. (See Fig. 3).

This is the most important structural feature of the cell as far as bone absorption is concerned, and will be discussed in detail, the description being derived from the work of Boothroyd and Hancox (1963 p. 243-240).
Diagrammatic representation of the main features seen in osteoclasts with low-power electron microscopy. Bone matrix represented as black, above; osteoclast below. Seven nuclei (N) are shown. The rectangles lettered A, B, and C refer to zones discussed in detail in the text; zone A is the site of the traditional “brush” or “striated” border. P, pinoosome; M, mitochondrion; V, cytoplasmic vesicles.

Fig. 3. The Osteoclast.

Taken from Boothroyd and Hancox (1963 p. 498-501)
Explanation of Figure 3.

Zone C. "The cytoplasm overlying the matrix is usually poor in organelles such as ribosomes, rough or smooth walled sacs and vesicles, mitochondria, etc.; it has a finely granular amorphous appearance .... the cell membrane follows the contour of the bone edge closely except that here and there it seems to form short, blunt channels leading inward ... loose or detached bone crystals can sometimes be distinguished within these...

"Appearancees suggest that cytoplasm in this area was neither elaborating any secretion nor pouring it forth onto the bone surface".

Zone B. "This constitutes a transition zone whose length is very variable, it may be practically absent. The chief characteristics are the presence, first, of cytoplasmic vesicles gathered near the cell membrane and second, of more numerous and longer channels leading in from the surface. Bone salt crystals can be recognised on these..."

Zone A. "This is the "ruffled border mentioned previously. ". the border consists of a complex system of cytoplasmic folds and projections separated by cleftlike spaces of variable appearance, and an associated system of vacuoles and vesicles. At one end of the scale are relatively fine canals, often branching, which can sometimes be seen to terminate in saclike vesicles; bounded by a single membrane..."
Generally there are a multitude of other vesicles, structurally identical, in the near-by cytoplasm. At the other end of the scale...are broader channels leading into large vacuoles, probably pinosomes.

"Bone salt crystals can be recognised in both the finer and the broader channels and also in the more distal pinosomes..."

"Collagen occurs constantly...in our material... The fibrils seem to protrude from the surface of the calcified matrix and are closely invested by ruffled border folds. The interfold spaces continue outward into the cytoplasm in fine channels which may branch before ending in vesicular sacs. More rarely, collagen cross-bandung can be identified in the larger and more distal pinosomes... partly demineralized fibrils are also seen". (Boothroyd and Hancox 1963 p. 512)

Fate.

The ultimate fate of the osteoclast is obscure. Some cells undergo local degeneration and are no doubt phagocytised while others may find their way through the walls of the blood vessels, to eventually degenerate in the lungs or the spleen, (Hancox 1956 p. 230).

Heller, and others (as cited by Hancox 1956 p. 230) are of the opinion that osteoclasts may revert to osteoblasts or reticular cells. It seems that the osteoclast is an
"ephemeral" structure, which quickly appears in response to a stimulus, and disappears within 48 hours, either degenerating or reverting to another cell type. (Hancox 1956 p. 233).

Mode of action of the Osteoclast.

The hard tissues of the body which may be acted upon by the osteoclast are made up of an organic framework permeated to varying degrees by an inorganic substance in crystalline form, the hydroxyapatite crystal.

Dentine closely resembles bone. The main morphologic difference between bone and dentine is that some of the osteoblasts forming the bone are enclosed in the intercellular substance as osteocytes, whereas the dentine contains only the cytoplasmic processes of the odontoblasts.

The organic substance consists of collagenous fibrils and a ground substance of mucopolysaccharides. (Sicher 1962 p. 107).

This ground substance is regarded as a type of cement for the collagen fibres forming the bone or dentinal matrix. (Pritchard 1956 p. 7).

The mechanism of the processes involved at the junction between the ruffled border of the osteoclast and the surface of this complex combination of organic and inorganic substances - bone, dentine, and cementum, is largely conjectural. Despite this, certain theories have been advanced which are generally accepted as being the most likely sequence of
events in the resorption of hard tissues.

Hancox (1956 p. 238) and Boothroyd and Hancox (1963 p. 512) have studied this at both the light and electron microscope level. To remove bone, its three major constituents must be accounted for: collagen matrix, mucopolysaccharide ground substances (or cement), and mineral crystals.

These authors have found loose mineral crystals in the cytoplasm of the osteoclast and have concluded from this that either the collagen or the ground substance was digested first. Their later investigations showed denuded collagen present in the ruffled border of the active osteoclast.

They also pointed out that, cytochemically, osteoclasts contain a wide range of enzymes, sufficient to cope with both soft and hard tissue breakdown.

From this evidence, derived from embryonic avian bone, Hancox and Boothroyd came to the conclusion that, firstly, bone salt crystals were loosened from the bone matrix, probably by an enzyme capable of breaking down the ground substance.

This left denuded collagen fibrils protruding into the ruffled border and bone-salt crystals in sac-like vesicles within the cell itself. The collagen was disposed of by a continuous enzymatic digestive process at the bony face.
The fate of the bone crystals swept into the cell channels and vacuoles is left open but this comment is made "... acid phosphatase, leucine aminopeptidase, and B glucuronidase have been studied. The reaction product is located in the cytoplasm in the form of droplets and granules whose size and number seem to coincide quite well with those of the larger vesicles seen in electron micrographs". (Boothroyd and Hancox 1963 P.511).

The inference here is that the bone-salts are dissolved within the cell vacuoles themselves.

In conclusion, although in the main the mechanism of bone resorption has been described here, this mechanism may also be applied to the cellular destruction of dentine and cementum and is comparable with resorption of dental hard tissues.
THE AETIOLOGY OF RESORPTION

For the sake of convenience, we may divide those factors found associated with resorption of tooth substance into three groups.

1. Local Factors.
2. Systemic Factors.

1. Local Factors.

Local factors found associated with root resorption, in both the permanent and deciduous dentitions, may be listed as follows:

1. Infection.
   (a) Granulomas.
   (b) Root canal treatment.
   (c) Osteomyelitis.
   (d) Malignant tumours and cysts (see below).
   (e) Periodontal disease.

2. Pressure.
   (a) Erupting permanent teeth.
   (b) Benign tumours.
   (c) Orthodontic treatment (low forces).
   (d) Cysts and malignant tumours.
   (e) Malocclusion.
   (f) Impacted teeth.
3. Trauma.
   (a) A sharp blow.
   (b) Orthodontic treatment (high forces).
   (c) Excessive occlusal stress associated with poor occlusion.
   (d) Poor operative and prosthetic restorations.

4. Necrosis (local).
   (a) Replanted teeth.
   (b) Orthodontic forces (gross forces)
   (c) Osteomyelitis.

This list of local factors found associated with root resorption has been based largely on Zemsky's classification of root resorption (Zemsky 1929).

Some aetiological factors, such as "orthodontic treatment", fall into several groups, depending upon the conditions prevailing and the presence or absence of infection (cysts, malignancies).

It should be noted that any of these factors may be present singly or in combination, and yet resorption of the involved roots does not occur.

The converse is also true. None of these factors may be clinically demonstrable and yet resorption may occur, either affecting only one root or many roots.

This is "Idiopathic" resorption.

From this it is obvious that there must be other
necessary predisposing factors for the resorption process to begin.

These other factors may be general nutritional, hormonal, or metabolic disturbances, affecting the whole body or some condition influencing the local cellular microenvironment, the stimulus to resorption issuing either from the affected tooth or from the surrounding tissue.

Bouyssou et al (1965 p. 45) have developed an hypothesis linking ideopathic "amputating" root resorption with a disordered local vaso-motor (neuro-vascular) reflex.  

2. Systemic Factors.

Resorption of the roots of the permanent teeth is seen in various systemic diseases, but no one systemic disturbance has been reported which shows root resorption invariably so that both phenomena can be directly linked, either causally or as a diagnostic yardstick.

Sullivan and Jolly (1957 p. 193) have reported root resorption in connection with Paget's disease.

Hypothyroidism, hyperpituitarism and acute and chronic infectious diseases were linked with ideopathic resorption by Beck (1936).

The role of alkaline phosphatase has been considered by Black. "Up to date, there has not been any indication that liver dysfunction, or the alkaline phosphatase level, could have anything to do with root loss - in fact, in the patients so far referred, the only thing which most of
them have in common is some form of ectodermal dysplasia". (Black 1965 p. 34).

Dietary disturbance was listed by Ketcham (1927) as a possible predisposing factor in root resorption in orthodontic patients. Poor development of the teeth may be the result of a dietary disturbance or vitamin deficiency during the developmental period which may result in future resorption.

The role of the parathyroids has long been known in relation to calcium metabolism and the structure of bone (McLean 1956 p. 705) but has not been specifically associated with root resorption. (Bouyssou et al 1965, p. 117).

3. A Combination of Systemic and Local Factors.

It is possible that subclinical disturbances of the metabolic regulating mechanism of the body may be a predisposing factor in root resorption. This disturbance may not be great enough to initiate any observable pathology, but sufficient to create demonstrable circumscribed effects in the presence of some local influence.

We know this situation can arise in the general skeletal system. This quotation from Urist et al (1963 p. 437) is pertinent. "The occurrence of the disorder (osteoporosis) in patients with acromegaly, hyperthyroidism, diabetes, and hypogonadism was attributed to endocrine imbalance. It is also possible that endocrinopathy initiates
nonspecific degenerative changes and premature aging of the bone tissue. Hyperparathyroidism has specific effects on bone and causes osteoporosis by osteoclasia and osteocytolysis, and Cushing's syndrome inhibits the proliferation of osteoblasts and produces bone resorption without osteoclasts, but these conditions may also accelerate the aging process and deterioration of osteocytes. The retention of bone and control of the rate of bone mass, however, could be determined by an anti-osteoporosis factor. This seems to be associated with such phenomena as maintenance of blood flow, completion of osteons and development of structural bone, androgen and protein biochemistry, effects of mechanical stimuli of physical exercise upon bone tissue, liver metabolism, and the intracellular physiology of the bone cell.

"The point of view set forth above suggests the possibility that treatment of osteoporosis by exercise, with improved diet and injections of sex hormones, may stimulate the rate of capillary circulation of bone and thereby slow the process of aging of bone tissues."

These authors go on to suggest a possible genetic control over this "anti-osteoporosis factor" (Urist et al 1963 p. 439).

The hypothesis of the "anti-osteoporosis factor" resident in the body may also suggest the possibility of
an "anti-resorptive factor" present in the permanent dentition, deficient in varying degrees in those teeth which exhibit pathological root resorption and being almost entirely deficient in the deciduous dentition. **An Hypothetical "Anti-resorptive factor".**

It would be interesting to develop this hypothesis. We notice that in almost every case of transplanted or replanted teeth, eventual destruction of the root by resorption occurs (Thoma 1958). The explanation usually given for this is that the protective and resorption resistant cementoid always covering a vital root (Sicher 1962 p. 169 and 181) becomes non-vital, exposing the cementum to the action of the osteoclast.

This theory of the protective role of cementoid is open to doubt. We know osteoclasts have the power of destroying collagen, as can be seen in the resorption of the collagen matrix of bone, even though no specific collagenase enzyme has been located in this cell (Boothroyd and Hancock 1963 p. 512).

Young, quoting Carnes and Follis, reported that matrix need not be calcified at all to yield to resorption (Young 1963) although Reitan had previously indicated that osteoid tissue was not resorbed until it had become partly calcified (Reitan 1951).

This may be coincidental in the experiments of Reitan
as bundle bone formation occurred rapidly after the formation of osteoid tissue, perhaps too rapidly to allow the migration and differentiation of osteoclasts and the resorptive process to begin on the osteoid itself.

Further, in support of a root protective mechanism (anti-resorptive factor) other than cementoid, we have the seemingly contradictory circumstances of root amputation or apicectomy without resorption.

In root amputation, relatively large areas of dentine and damaged and denuded cementum are exposed to the action of the resorptive processes of the body, and yet the incidence of progressive root resorption is low in these cases, even though the tooth is non-vital, as are large areas of the root surface. A rounding-off of the root surfaces by cementum occurs, but rarely does resorption progress to the massive destruction of the root, as seen in replanted teeth.

A perusal of the literature and consultation with several leading Sydney oral surgeons (Wilson, Stacey, Helmore 1967) failed to produce any case histories of progressive resorption occurring after successful root amputation. Helmore (1967) suggested that resorption had occurred in some cases after perhaps ten years, but was uncommon when compared with replanted teeth. An examination of serial histological sections of two upper central incisors which had undergone successful root filling and
and apicectomy eighteen months previously proved interesting.

These teeth were extracted from a fifteen year old girl and showed an almost complete cemental covering of the dentine previously exposed by the surgery. A very few resorptive lacunae were visible, these mainly being filled with cementum.

Occasionally, an abrupt break would be found in the new cementum, and a minute, shallow lacuna would be visible.

There was insufficient investing tissue present to definitely distinguish active osteoclastic activity, and it would have been interesting to have observed whether this resorption was progressive or whether these lacunae would eventually be filled with cementum also.

From this it could be hypothesised that in the removal from the socket of the tooth to be replanted, some factor in the structure of the root, possibly in the organic substance (as this seems to be the first structure attacked), is lost or destroyed, leaving the root unprotected from the process of resorption.

A further interesting study would be to perform apicectomies on a series of experimental teeth, leaving the gingival cuff and alveolar mucosa intact but destroying the periodontal attachment and cementoid root covering from the apex to within two or three millimetres of the gingival margin.
The teeth would then be splinted out of function until re-attachment had taken place.

Would resorption occur on these teeth as if they had been replanted, or would they behave as root amputated teeth?

It has been shown that resorption can be produced experimentally by the application of inflammatory media to the teeth. (Obersztyn 1963 p. 673). In this study, the induced resorption was attributed to the inflammatory changes in the surrounding tissue, rather than a change in the tooth substance itself, although this possibility was considered. (Obersztyn 1963 p. 671).

Gottlieb and Orban in 1930 applied diathermy to the gingival papillae between the incisors of dogs. (Warner et al 1947 p. 478).

Histological sections were examined after one month and extensive internal and peripheral resorptive areas were in evidence. The authors concluded "...a toxic influence might be assumed. The high frequency current might have brought about degenerative changes in pulp and dentine, as it definitely did in the periodontal membrane, and thus produced the necessary toxic influence". (Warner et al 1947 p. 483).

In this case, damage to the periodontal membrane and perhaps the pulp could conceivably be reversible. It
would be unlikely that dentinal damage would be reversible, some factor in the dentine possibly being destroyed allowing osteoclastic resorption to begin, rather than a direct inflammatory reaction of the surrounding tissues initiating the process.

To develop this hypothesis further, we could conclude that root resorption was stimulated by an inherent deficiency of the dentine or cementum itself. This deficiency may occur congenitally as in deciduous teeth and possibly some of those teeth which display ideopathic resorption or may be created by local inflammation resulting in a breakdown of some factor of the organic framework of the root itself.

Root resorption occurring in association with an impacted tooth in the permanent dentition may be initiated by either a congenital lack of this "anti-resorptive factor", in the affected tooth or a localised breakdown of this factor through a reduced blood supply or some other traumatic influence.

Kronfeld (1932) considered the main factor in resorption of the deciduous teeth as a "kind of senescence" in the deciduous teeth themselves, resorption being an inevitable process hastened by eruption of the permanent successors. Young and Thomas (cited by Steadman 1942) regarded the deciduous dentition as having an inherent quality or predisposition to resorption. The inference here is that the permanent teeth do not normally have this quality.
The Replacement of Resorbed Dental Tissues.

Almost every tooth undergoes a certain amount of "physiologic" resorption of the root during its life which normally does not penetrate far beyond the cementum. These small areas of resorption are found most commonly on the mesial surfaces of the posterior teeth, corresponding to those surfaces of the teeth whose adjacent bone is undergoing resorption to allow physiologic mesial migration of the teeth.

Minor resorptions of the cementum may be repaired by either cellular or acellular cementum, and the anatomical shape of the root re-established. Sharpey's fibres of attachment are incorporated into this new cementum and the integrity of root and periodontal membrane restored. This is called "Anatomic Repair". (Sicher 1962 p. 181).

If the resorbed area is more extensive, the shape of the root may not be restored. In these cases, the lacunae may be covered with a "normal" thickness of new cementum, either cellular, acellular or both. The width of the periodontal membrane is re-established by an ingrowth of alveolar bone. Clinically, this may render the tooth difficult to remove if extraction is ever necessary. (Sicher 1962 p. 182).

With all forms of radicular resorption, there is almost always a concomitant process of repair. (Sullivan and Jolly 1957 p. 193) (Sicher 1962 p. 327).
Progressive resorption, as seen in deciduous teeth and some permanent teeth, occurs when the repair processes fail to match the rate of resorption.

There is no set pattern found in the repair of resorbed teeth. "The replacement process is intermittent and disordered and the resulting structure bears no relation to the normal form of the tooth, the new tissue frequently showing numerous resting and reversal lines. This replacement has been termed "metaplasia", as the new tissue is different from the original structure of the tooth. It is commonly seen when the resorption begins externally but can also occur in relation to internal ideopathic resorption". (Sullivan and Jolly 1957, p.193).

This quotation applies to the extensive "burrowing" types of resorption rather than the "accommodating" resproptions usually found with impactions. (See p. 110) The resting phase of that type of resorption which does not "burrow" or penetrate with "finger-like" projections is characterised by a process of repair with a more compact substance, resembling cementum, compact bone or a transitional and primitive osteocementum.

Dentine resorbed peripherally can never be replaced by new dentine, as odontoblasts cannot differentiate from any of the adjacent cells, but new orthodentine may be laid down occasionally after pulpal resorptions. (Bouyssou
et al 1965 p. 46). In large areas of peripheral destruction, an attempt at repair may be seen, utilizing compact bone, cancellous bone, osteocementum, and cellular or acellular cementum or any combination of these. The replacement tissue may be closely applied to the dentine or cementum, the junction being marked by a basophil reversal line, or it may occur freely in the resorbed area. Marrow spaces may even be found in the cancellous bone structure. (Thoma 1954).

As the resorption approaches the pulp, the odontoblasts may or may not become active and lay down a defensive layer of new dentine. The resorptive area may encircle the pulp without actually invading the pulp chamber or it may invade the pulp directly.

Histologically, the pulp may appear quite normal, even though only a thin dentinal wall separates it from the cavity of resorption. On the other hand, degenerative changes may occur in the pulp in the form of fibrosis and false denticles. Sullivan and Jolly (1957 p. 195) state that the pulp usually remains intact even though the amount of dentine between the lesion and the pulp be very small.

It seems that the larger the resorption cavity, particularly in ideopathic resorption, the more chaotic is the repair process, and the more likelihood there is of a less compact structure being laid down. This may result in
the lesion being entirely filled with a type of cancellous bone. (Sullivan and Jolly 1957 p. 195).

**Frequency and Incidence of Root Resorption.**

A study of the literature does not provide any statistics on the incidence and frequency of resorption from impactions or any other local factor.

Ideopathic resorption, on the other hand, has been studied by many authors. (Ketcham 1927, 1929), (Becks 1939), (Massler and Malone 1952, 1954), and several tables have been compiled to provide information as to the prevalence of resorption before and after orthodontic treatment.

This type of resorption lends itself to statistical study because of its relatively common occurrence. Black, in his thesis on root resorption in human permanent teeth, however, has rendered the majority of these figures suspect (Black 1965 p. 16). He says "It will be shown later that it is quite impossible to try and detect resorption of any multi-rooted tooth (except possibly the lower first molar) from the best possible intra-oral radiograph. It cannot be done. Any such investigation that makes such claims is immediately suspect of extremely subjective assessment of the evidence available".

On the frequency of resorption, he says "...the incisors show the highest frequency of resorption, both before and after (orthodontic) treatment. It can also be seen that
the frequency of any other group is far from clear..." (Black 1965 p. 16).

In examining the incidence of root resorption before orthodontic treatment Black provides a table (Black 1965 p. 11) summarising the works of various authors. These authors claim an incidence of from 1 per cent. in 385 cases (Ketcham) to 86.4 per cent. in 708 cases. (Massler and Malone). From this table, we can get no clear idea at all as to the true incidence of ideopathic resorption.

In Black's review of the literature on the difference in susceptibility to ideopathic resorption between males and females, he concluded that females generally exhibited slightly more resorption than males (Black 1965 p. 11).

To summarise, this section on the incidence and frequency of ideopathic resorption, all we can say is that the incisors show the highest frequency of resorption, and that females exhibit slightly more resorption than males.
D. A Review of Those Cases of Impactions Associated with Resorption Found in the Literature.

The available dental literature from 1874 to 1966 was examined for any records of impacted teeth associated with root resorption of the impacting tooth in the permanent dentition. Forty-one cases were found with sufficient data to include in the Table (4).

The bibliography for Table 4 will be found in Appendix A, on page 87.

Analysis of Table 4.

There were 51 resorbed teeth in 41 patients.

Of the 51 resorbed teeth, 36 were extracted, 10 were retained, the fate of 5 not being recorded.

There were 39 females, 11 males and 3 of unrecorded sex.

Incidence of Resorbed Teeth.

There were 24 anterior teeth resorbed, comprising 9 upper central and 15 upper lateral incisors.

There were 27 posterior teeth resorbed, comprising 20 second molars, 5 first molars, 1 upper first premolar, and 1 upper second premolar.

Of 20 second molars resorbed, 6 were upper and 14 were lower molars.

Of 5 first molars resorbed, 2 were upper and 3 were lower molars.
Incidence of Impacted Teeth.

In the 44 impacted teeth involved, 19 were upper cuspids, 19 were third molars (6 uppers and 13 lowers), 4 were second bicuspids (2 upper and 2 lower), 1 was a lower second molar and 1 a lower supernumerary between the roots of a lower second molar.

Occurrence of Painful Symptoms.

In the 41 patients, 17 experienced pain to a varying degree, 22 experienced no pain and 2 had no record as to painful symptoms.

Of the 17 patients experiencing pain, 16 had the posterior teeth involved, 1 the anterior teeth.

Of the 22 patients experiencing no pain, 8 had the posterior teeth involved, 16 the anterior teeth.

Age of Occurrence.

Of the 41 patients involved, 19 were between 10 and 20 years old, 8 were between 20 and 30 years old and 11 were over 30 years old. There was no age given for 3 patients.

Of the 17 patients with anterior teeth resorbed, 16 were between 10 and 20 years of age and 1 was 27 years of age.

Of the 25 patients with posterior teeth resorbed, 6 were between 10 and 20 years of age, 16 were over 20 years. In 3 cases no age was recorded.
<table>
<thead>
<tr>
<th>No.</th>
<th>Resorbed</th>
<th>Impacted</th>
<th>Sex</th>
<th>Age in Pain</th>
<th>Symptoms</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>8</td>
<td>M</td>
<td>24</td>
<td>vague</td>
<td>Second molar removed 26 months after removal of impaction. Resorption progressive.</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>3</td>
<td>M</td>
<td>12</td>
<td>none</td>
<td>2 extracted. Bisected by canine.</td>
</tr>
<tr>
<td>3</td>
<td>21/12</td>
<td>3/3</td>
<td></td>
<td>27</td>
<td>none</td>
<td>Canines and incisors retained. Resorption became static.</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>5</td>
<td>F</td>
<td>20</td>
<td>none</td>
<td>6 removed.</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>8</td>
<td>M</td>
<td>33</td>
<td>none</td>
<td>Lymphadenitis from pericoronitis of 8.</td>
</tr>
<tr>
<td>7</td>
<td>same patient</td>
<td>as above</td>
<td>none</td>
<td>Both molars removed.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6a</td>
<td>12</td>
<td>3</td>
<td>F</td>
<td>21</td>
<td>none</td>
<td>Canine removed. 2/3rd of roots of 12 destroyed. Resorption ceased. 12 vital after 2 years.</td>
</tr>
<tr>
<td>b. 1</td>
<td>3</td>
<td>F 12</td>
<td></td>
<td>12</td>
<td>none</td>
<td>Canine removed. 1 vital and firm after 6 months.</td>
</tr>
<tr>
<td>No.</td>
<td>Resorbed</td>
<td>Impacted</td>
<td>Sex</td>
<td>Age in years</td>
<td>Pain Symptoms</td>
<td>Comments</td>
</tr>
<tr>
<td>-----</td>
<td>----------</td>
<td>----------</td>
<td>-----</td>
<td>--------------</td>
<td>---------------</td>
<td>----------</td>
</tr>
<tr>
<td>c. 2</td>
<td>2</td>
<td>same patient</td>
<td>F</td>
<td>12</td>
<td>none</td>
<td>2 removed. Canine erupted</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>as above</td>
<td></td>
<td></td>
<td>none</td>
<td>6 removed</td>
</tr>
<tr>
<td>d. 7</td>
<td>8</td>
<td>M</td>
<td>68</td>
<td>pain</td>
<td></td>
<td>Both molars removed</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>F</td>
<td>30</td>
<td>pain</td>
<td></td>
<td>7 removed</td>
</tr>
<tr>
<td>8</td>
<td>supernum.</td>
<td>M</td>
<td>30</td>
<td>pain</td>
<td></td>
<td>Both teeth removed</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td>2 removed</td>
</tr>
<tr>
<td>10</td>
<td>7</td>
<td>8</td>
<td>M</td>
<td>20's</td>
<td>pain</td>
<td>Both teeth removed</td>
</tr>
<tr>
<td>11</td>
<td>21</td>
<td>3</td>
<td>M</td>
<td>19</td>
<td>pain</td>
<td>2 pushed 2 into 1. Half root of 1 destroyed. 2 notched. 123 removed</td>
</tr>
<tr>
<td>12</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>50</td>
<td></td>
<td>7 extracted</td>
</tr>
<tr>
<td>13</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>47</td>
<td>pain</td>
<td>Both molars extracted</td>
</tr>
<tr>
<td>14</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>38</td>
<td>pain</td>
<td>Both molars extracted</td>
</tr>
<tr>
<td>15</td>
<td>7</td>
<td>8</td>
<td>M</td>
<td>45</td>
<td>pain</td>
<td>Both molars extracted</td>
</tr>
<tr>
<td>16</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>36</td>
<td>pain</td>
<td>Both molars extracted</td>
</tr>
<tr>
<td>17</td>
<td>7</td>
<td>8</td>
<td>M</td>
<td>30's</td>
<td>pain</td>
<td>Both molars extracted</td>
</tr>
<tr>
<td>No.</td>
<td>Resorbed</td>
<td>Impacted</td>
<td>Sex</td>
<td>Age</td>
<td>Years</td>
<td>Symptoms</td>
</tr>
<tr>
<td>-----</td>
<td>----------</td>
<td>----------</td>
<td>-----</td>
<td>-----</td>
<td>-------</td>
<td>----------</td>
</tr>
<tr>
<td>18.</td>
<td>7</td>
<td>3</td>
<td>M</td>
<td>3</td>
<td>none</td>
<td>pain</td>
</tr>
<tr>
<td>19.</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>30</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>20.</td>
<td>21</td>
<td>3</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>21.</td>
<td>7</td>
<td>3</td>
<td>M</td>
<td>26</td>
<td>pain</td>
<td>pain</td>
</tr>
<tr>
<td>22.</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>15</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>23.</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>14</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>24.</td>
<td>7</td>
<td>3</td>
<td>M</td>
<td>51</td>
<td>pain</td>
<td>none</td>
</tr>
<tr>
<td>25.</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>16</td>
<td>none</td>
<td>None</td>
</tr>
<tr>
<td>26.</td>
<td>45</td>
<td>3</td>
<td></td>
<td></td>
<td>None</td>
<td>No data</td>
</tr>
<tr>
<td>27.</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>50</td>
<td>pain</td>
<td>none</td>
</tr>
<tr>
<td>28.</td>
<td>1/1</td>
<td>3</td>
<td>F</td>
<td>19</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

TABLE 4. Continued
<table>
<thead>
<tr>
<th>No.</th>
<th>Resorbed Tooth</th>
<th>Impacted Tooth</th>
<th>Sex</th>
<th>Age in Years</th>
<th>Pain</th>
<th>Symptom</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>29</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>47</td>
<td>pain</td>
<td></td>
<td>Both molars removed</td>
</tr>
<tr>
<td>30</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>18</td>
<td>pain</td>
<td></td>
<td>No data</td>
</tr>
<tr>
<td>31</td>
<td>7</td>
<td>7</td>
<td>F</td>
<td>12</td>
<td>none</td>
<td></td>
<td>No data</td>
</tr>
<tr>
<td>32</td>
<td>6</td>
<td>2</td>
<td>F</td>
<td>10</td>
<td>none</td>
<td></td>
<td>Molar removed</td>
</tr>
<tr>
<td>33a</td>
<td>1</td>
<td>3</td>
<td>F</td>
<td>19</td>
<td>none</td>
<td></td>
<td>1 removed</td>
</tr>
<tr>
<td>b</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td></td>
<td>2 removed</td>
</tr>
<tr>
<td>34</td>
<td>7</td>
<td>8</td>
<td>F</td>
<td>over 40</td>
<td>none</td>
<td></td>
<td>Both molars removed.</td>
</tr>
<tr>
<td>35</td>
<td>2</td>
<td>3</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td></td>
<td>2 removed</td>
</tr>
<tr>
<td>36</td>
<td>2</td>
<td>2</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td></td>
<td>2 removed</td>
</tr>
<tr>
<td>37</td>
<td>6</td>
<td>5</td>
<td>F</td>
<td>13</td>
<td>none</td>
<td></td>
<td>Molar removed.</td>
</tr>
</tbody>
</table>

Reference to case numbers in Appendix A. P. 185
Sex Differences.

Of the 41 patients displaying impacted teeth associated with resorption, 27 were females, 11 were males, and no data was given as to the sex of 3.

Of the 24 anterior teeth resorbed, 17 were in females, 3 in males and there was no record as to the sex of the patient for 4 teeth.

Of the 27 posterior teeth resorbed, 14 were in females, 10 in males and for 3 teeth there was no record as to the sex of the patient.

Conclusions Drawn from the Analysis of Table 4.

1. Teeth Most Frequently Affected.

Resorption from impactions was found most commonly in those areas which show the highest incidence of impactions (see p. 34). From Table (4) we find 20 second molars and fifteen lateral incisors affected. Looking at Table 2 on page 33 we see 461 third molar impactions but only 23 canine impactions in a given sample. If we consider that the incisor area shows the greatest incidence of ideopathic resorption (see page 34), this may explain the relatively high proportion of lateral incisors resorbed from canine impaction in relation to second molars resorbed from third molar impactions.

The high proportion of central incisors involved was the result of -
(a) Lateral incisors being completely resorbed by the canine, which eventually impacted into the central incisor,
(b) Congenital absence of lateral incisors allowing canine impaction into the central incisor, and
(c) The horizontal impaction of canines affecting the apices of both lateral and central incisors.

The first molars were involved by the impaction of the second bicuspid in all cases except one (case no. 31) which showed the relatively unusual impaction of a second molar.

When one considers that the incidence of impactions is highest in the third molar and upper canine areas and the incidence of ideopathic resorption is highest in the anterior teeth, it is not surprising that the upper lateral incisor and the upper and lower second molar teeth are most commonly found associated with resorption from impacted teeth.

2. Sex Distribution.

From Table (4) we find 27 females and 11 males involved.

Of the anterior teeth resorbed, 17 were in females and 3 were in males. This correlates well with previous data given on the incidence of impacted canines (p. 34), and the incidence of resorption related to sex (p. 84).
<table>
<thead>
<tr>
<th>Tooth</th>
<th>Females</th>
<th></th>
<th></th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Eruption ages</td>
<td></td>
<td>Eruption ages</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median</td>
<td>Earliest</td>
<td>Latest</td>
<td>Median</td>
</tr>
<tr>
<td>Upper</td>
<td>Yr. mth</td>
<td>Yr. mth.</td>
<td>Yr. mth.</td>
<td>Yr. mth.</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>10</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>9</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>8</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>10</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>8</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>1</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>11</td>
<td>1</td>
<td>9</td>
<td>11</td>
</tr>
</tbody>
</table>

| Lower | | | | |
|-------|---------|---------|---------|
| 1     | 5      | 11      | 4       | 11      | 7       | 2       |
| 2     | 6      | 11      | 5       | 8       | 8       | 4       |
| 3     | 9      | 5       | 7       | 10      | 11      | 4       |
| 4     | 10     | 3       | 8       | 0       | 13      | 0       |
| 5     | 11     | 1       | 8       | 6       | 14      | 3       |
| 6     | 5      | 11      | 4       | 10      | 7       | 2       |
| 7     | 11     | 3       | 9       | 4       | 13      | 7       |

Table 5. Median age of eruption of the permanent teeth of New South Wales school children. Earliest and latest eruption ages include 90 per cent of children. Five per cent may erupt earlier and five percent later. Taken from Gates (1964).
The incidence of impacted canines is higher in females than in males, the incidence of resorption is higher in females than in males, and is also higher in anterior teeth than in posterior teeth.

Of the posterior teeth involved, we find more equal figures for male and female, being 14 females and 10 males affected. From page 34 we find the incidence of impacted third molars is very slightly higher in females than in males and also the incidence of ideopathic resorption is a little higher in females than in males.

3. Age of Occurrence.

Resorption of the anterior teeth was found mainly in the age group of 10-20 years, mostly in early teen-age. In one patient, (ref. no. 3) resorption began in early teenage but by the age of 27 years had become static, even though the impaction remained. Because the cuspid is normally actively erupting at this time (see Eruption Chart of Gates p. 93) we can expect to find most anterior resorptions occurring in this age group.

In the bicuspid areas, we again find a correlation between eruption times and the discovery of resorption.

The impacted second bicuspids associated with resorption were all in early teenagers except one (ref. no. 4) in a 20 year old female.

Impacted third molars associated with resorption of the second molar showed no strict correlation with the age of
eruption of the third molar.

As can be expected, there was no case before the age of 18 years, one at 18 years of age and the remainder distributed evenly between the ages of 24-68 years of age.

There was no way of knowing in any of these cases when the resorptive process started. It may have commenced during the active eruptive phase of the third molar and become static, only to resume its course later in life, or it may have been a slow, steady process extending over a number of years. These cases of resorption were mainly discovered after painful symptoms had become evident, which usually meant pulpal involvement and hence extensive tooth destruction.

4. Fate of the Affected Teeth.

Of the 51 resorbed teeth, 36 were extracted, 10 were retained and the fate of 5 was not recorded. Most of those posterior teeth removed were extracted immediately on discovery because of painful symptoms. Only one posterior tooth (ref. No. 1) was allowed to remain under observation after the impacted tooth had been removed. This was an upper second molar. 26 months after the

+ The approximate average age of eruption of the third molars for American University students is 20.5 years for both male and female (Hellman 1936 p. 754).
removal of the impacted tooth the resorption had progressively destroyed the root to such an extent as to cause pain, necessitating extraction. There is no record as to whether the resorption was of the "burrowing" type or the "accommodating" type (see later).

Of the nine remaining teeth retained, all were anterior teeth, the tooth responsible for the resorption being the canine in every case.

In each of these cases, the impacted tooth was removed, except in one (ref. No. 3) the resorption remained static after the removal of the impaction and all the teeth retained their colour and vitality for the periods of observation ranging from 6 months to 2 years.

In case No. 3, the bilaterally impacted canines were observed for 18 years. They had brought about the resorption of the four anterior teeth and at the age of 27 years, about one third of the roots of the centrals and laterals had been lost. At this stage, the resorptive process appeared static.

In case No. 35, the canine was impacted into the disto-cervical area of the tooth, initiating a type of "burrowing" resorption. The process progressed until sufficient tooth structure was lost to allow complete eruption of the cuspid. The resorption continued even after the eruption of the cuspid. The lateral was extracted when almost the entire crown had become pink
from the granulation tissue replacing the dentine.

A similar situation occurred in case No. 33 (b). A labiocervical cavity appeared in the lateral against the disto-labially placed canine. When the cavity was opened, a resorptive area was found for a considerable distance down the root surface. The pulp was exposed from the cervical area almost to the apex. The lateral incisor was extracted and the canine drifted into the line of the arch. There had been no painful symptoms.

5. Associated Symptoms.

In the 41 patients involved, 17 experienced pain to a variable degree and 22 had no pain associated with the condition.

All those patients who experienced pain had the posterior teeth involved, except one (case No. 11). All these cases had reached an advanced stage in the process of resorption and an exposure of the pulp was almost always present.

As pain in the region was the reason why most of these areas were investigated, it follows that painful symptoms must always be associated with those cases shown in Table (4).

This is not so in the anterior region. These areas are usually investigated if a tooth is late in appearing in the mouth or if there is some irregularity of the
anterior teeth; thus the condition is usually diagnosed at a fairly early stage.

This does not fully explain the lack of painful symptoms in those cases of resorption of the anterior teeth. Resorption can continue to the full destruction of the root and still no painful symptoms occur (case No. 9).

Twice as many posterior teeth exhibited pain as those posterior teeth which showed no symptoms but the pain was always associated with a late stage of the process. The pain felt in the posterior region was usually quite severe except in a small number of cases. The degree of pain is difficult to assess in a study of the literature but from an examination of these cases presented in Table (4) it seems that there must be some actual pulpal degeneration of the resorbed tooth before marked pain occurs.

Minor pain could occur for many reasons, just as pain can occasionally be felt in reasonably sound teeth. Masticatory stress on a tooth with a markedly shortened root could bring about discomfort which might well be recorded as pain by the patient and attributed by the examining dentist to the actual resorptive process.

Why this pulpal degeneration occurs in some teeth and not in others is not known. Case No. 21, showed a
series of pulpal abscesses on histological examination. These could be the result of bacterial invasion although the pathway of entry of the bacteria into the pulp is obscure.


Comments on the condition of the pulp were made in all cases where the teeth were retained. The pulp appeared to remain vital and react normally to vitality tests even after gross root loss, and remained so until the end of the observation periods.

In three cases, the extracted, resorbed teeth were subjected to an histological investigation (case Nos. 5, 21, 29).

In case No. 29, the only remarks made on the pathology of the pulp were the presence of a large pulp stone almost obliterating the pulp chamber and the posterior root canal practically occluded by secondary dentine.

The author comments "The cause of the patient's pain can easily be explained by the large area of exposed dentine and the presence of such a large pulp stone in the pulp chamber".

This explanation is certainly easy but not necessarily accurate, as will be seen later.

In case No. 5, a pathology report was supplied "the distal root shows evidence of an old resorptive process..."
and at the moment is inactive. The coronal portion of
the pulp shows a heavy deposition of secondary dentine
with a well delineated border of odontoblasts. There
is some congestion of the blood vessels and evidence of
reticular atrophy..." Pain was not present in this case.

Case No. 21, in a search of the literature, was the
only example of a relatively detailed description of the
histological appearance of the pulp of a tooth which had
been resorbed by an impacted tooth. The description is
quoted here in full.

"... at the site of the (resorptive) cavity the
dentine is very markedly absorbed. It became very thin,
the pulp chamber in this region being covered only with
a very thin layer of dentine. The pulp is very markedly
atrophied, the odontoblastic layer is almost totally
absent. In the pulp chamber, several pulp-stones are
present showing globular arrangements; some of them,
continuous with the dentine, are of the so-called
"Dentikel" type. In the pulp chamber, there is also an
abscess filled with detritus near the absorbed wall. In
the root canal, there are calcifications, some old
abscesses filled with a necrotic mass and also new
abscesses filled with pus cells, each surrounded by a
capsule. The apices show absorption; in the lacunae,
osteoclasts are embedded. The periodontal membrane is thick, and contains a large number of inflammatory cells.

The impacted third molar was also sectioned and studied histologically, but appeared "normal".

It seems from this scant evidence that pain symptoms are associated, not with the resorptive process itself, but with a subsequent degeneration of the pulp of the impacting tooth. This pulpal degeneration does not always occur, even in very advanced resorption, as will be seen later in this work.

7. Frequency of Resorption from Impactions.

Comments on the frequency of resorption occurring from the presence of an impacted tooth in the permanent dentition range from "not uncommon" (Sealey 1955) to "comparatively rare" (Springer 1956). Sicher (1966) speaking of resorption from impacted third molars, states "This has been observed frequently on the lower second molars, due to the oblique position of the erupting third molars... It can be so extensive that the pulp may be exposed. When the pressure is relieved during the normal movement of the third molar, repair by apposition of cementum may follow. Such resorption was observed in about two-thirds of investigated jaws".
Dachi and Howell (1961) attempted to record the incidence of impacted teeth which were causing resorption of adjacent erupted teeth. They wrote "It was found, however, that the difficulty in interpreting mild resorption from periapical radiographs made this task impossible".

From these observations we can say that mild resorption in the roots of the permanent second molars from adjacent erupting teeth is the rule rather than the exception, but in almost every case this resorption is impossible to detect radiographically and eventually heals by a deposition of cementum.

For this resorption to progress to the stage of becoming radiographically evident is quite uncommon.

To obtain material for the histological study of resorbing teeth in this thesis, a thorough search was made of the dental x-rays of patients attending the United Dental Hospital, Sydney. One resorbed upper first molar was discovered and prepared for microscopic examination.

Consultation with Orthodontists, Oral Surgeons, and general practitioners in Metropolitan Sydney resulted in two upper lateral incisors being prepared for sectioning.

One other case, a lower first molar, was discovered
but this tooth was not available for either sectioning or observation.

In a personal correspondence with Silas J. Kloehn of Wisconsin U.S.A. (1967), he stated that in twelve years of general dental practice he removed "a great number of impacted third molars". He found six or eight lower second molars displaying resorption of the distal root initiated by the impacted third molar and only one maxillary second molar resorbed by an impacted third molar.

This gives some indication of the frequency of radiographically demonstrable resorption from impactions. A percentage figure cannot be placed on this condition but it can be seen that it is a reasonably rare occurrence associated with impacted permanent teeth.
E. THE APPLICATION IN CLINICAL ORTHODONTICS OF THE
CONCLUSIONS DRAWN FROM THE REVIEW OF THE LITERATURE

INTRODUCTION

Impacted teeth may be discovered in general radiographic surveys prior to any form of general dental treatment.

More specifically, a radiographic search may be made of any area in the mouth to locate teeth which are late in erupting, or of any area where a tooth should normally appear, but there is insufficient room in the dental arch for its inclusion.

The radiographs will either reveal a congenital absence of the tooth, an abnormal position or a definite obstruction in the path of the erupting tooth.

This obstruction may be in the form of a pathological condition such as a cyst or tumor, a supernumerary tooth, or the root of a tooth normally found within the arch.

DIAGNOSIS.

On locating an impacted tooth, a series of x-rays must be taken to determine

(a) the position and degree of angulation of the impacted tooth, and

(b) the condition of the roots of the impacting tooth.
(a) **Radiographic Location of the Impacted Tooth.**

The exact position of an unerupted tooth cannot be assessed by one x-ray film alone. A single x-ray film is a two-dimensional shadow picture of a three dimensional object. In favourable circumstances two films will give an accurate three dimensional localisation of an impacted tooth, but at times several films are necessary.

The teeth most commonly found impacted are the third molars and the upper canines.

The exact position of the buccal teeth is usually a relatively simple procedure, but the localisation of an impacted maxillary canine or incisor tooth can be difficult.

There are four major methods for determining the location of impacted teeth.

1. **The Parallax or Shift Method**

This method was recorded by Clark in 1909 (Seward 1963). If two periapical films are placed successively in the same position and exposures are made using two different horizontal or vertical angulations of the x-ray tube, then the relative positions of the images of the teeth will differ. From this, the bucco-lingual relations of the impacted tooth can be worked out. If the image of the impacted tooth moves (in relation to the impacting tooth) in the same direction as the tube is moved, it
must be further from the tube than the impacting tooth and hence, palatally or lingually placed. An idea of the distance between the unerupted tooth and adjacent teeth can be gained by the extent of the apparent relative movement of the teeth.

The disadvantages of this method are that it requires considerable "mental gymnastics" to discover the bucco-lingual position of the tooth and that bucco-lingual distances cannot be accurately assessed (Wäggener 1960).

2. Stereoscopy.

If two x-rays are taken with the film in the same position and a shift of the tube equivalent to the distance between the examiner's eyes, then the resulting films can be viewed in a stereoscope. The main disadvantage of this technique is that the closer an unerupted tooth is to the adjacent teeth, the less the sense of depth. The result also depends upon an optical illusion which is not a good foundation for accurate diagnosis.

3. Multiple Exposure Method

This method is similar to the Parallax method. Two or three images are recorded on the same film with a shift of the x-ray tube between exposures. The more widely separated the images of the unerupted tooth, the further it is from the film. If the two or three images are nearly superimposed, then the unerupted tooth is palatally
or lingually placed.

This technique has similar disadvantages to the parallax method and is difficult to apply when the unerupted tooth is close to the erupted teeth.

4. Radiographic Views at Right Angles.

This method of viewing impacted teeth provides the maximum information as far as spacial relations are concerned. A three-dimensional mental image can be created without the "working-out" necessary in other methods. (Waggener 1960).

A conventional periapical view can be taken of the impacted tooth. This x-ray gives data on the vertical position and mesio-distal angulation of the tooth.

To derive the necessary information for a three dimensional mental picture, an occlusal film taken at right angles to the periapical film is essential. This film establishes the labio-lingual and mesio-distal position of the tooth, but gives little information on angulation, especially in single-rooted teeth.

Any other projections, such as true-lateral and postero-anterior cephalometric radiographs may also be useful in building up a true picture of the position of the tooth.

In patients to be treated orthodontically, an examination of the study models prior to x-ray localisation
of an unerupted tooth may provide information as to the most desirable angulations of the film and x-ray tube for the initial periapical film.

Rounded protruberances on the buccal or palatal mucosa, or the angulation of the impacting tooth may give some clue as to the position of the impacted tooth.

(b) **Assessment of the Radicular Condition of the Impacting Tooth.**

Having determined the exact position of the impacted tooth, further radiographs may be necessary to establish the condition or degree of resorption of the root of the impacting tooth.

Those x-rays taken to determine the position of the tooth are usually sufficient to recognise any dilacerations or unusual anatomic deviations in the root but may give little information on possible resorptive cavities or ankylosis.

Black, in his thesis on root resorption in the permanent dentition (Black 1965 p. 150) has shown that an exacting radiographic technique is required to detect small areas of resorption in the root surface of a tooth, necessitating the use of a long cone technique.

Precise angulations of the x-ray beam and accurate exposure values are essential, but even with a "perfect" x-ray small resorptive cavities may escape detection. (Black 1965 p. 11).
With resorption associated with impacted teeth, the diagnosis is made more difficult by the dense crown of the impacted tooth and the fact that the impaction may be buccally or palatally placed in relation to the x-ray beam.

The follicle of the unerupted tooth may be enlarged, even to the extent of a follicular cyst. The shadow of this follicle may overlap the image of the root of the impacting tooth, giving the appearance of a resorptive cavity.

To detect root resorption as early as possible, the x-ray must be taken at a tangent to both the curvature of the root surface and the curvature of the crown at the point or area of impaction.

If the root is dumbbell-shaped, as is sometimes seen in the distal root of a lower molar, the resorptive cavity may be masked radiographically by the bulk of the root on either side.

Any deviation from this tangential plane will result in the images of the impacted and impacting teeth overlapping. Unless the resorption is considerable, this overlap may completely obliterate the radiolucent resorbed area.

If the impacted tooth is placed in such a way that the point of impaction cannot be viewed tangentially, then the
area of resorption will not be detected until it has become quite extensive.

The Nature of the Resorptive Process.

The type of resorption initiated by an impacted tooth has an important bearing on the prognosis.

The resorption may be one of two types, "burrowing" resorption or "accommodating" resorption.

"Burrowing" Resorption.

Seward (1963b) has referred to this type of resorption and has described its radiographic appearance; "an irregular, mottled radio-lucency affecting the dentine". This mottled appearance is due to the varying amounts of replacement tissue distributed irregularly around the resorptive cavity and the "channel-like" progress of the resorptive cavity. The replacement tissue may be cancellous or cortical bone, osteocementum, or cementum; and these varying repair tissues produce a hazy margin to the walls of the lesion when studied radiographically.

The profile of the intact pulp canal may at times be seen superimposed over the hazy shadow of the resorptive cavity. This feature may be used to distinguish the "peripheral" type from the "pulpal" type of lesion.

This "burrowing" resorption appears to be the same as that described by Goldman (1954 p. 527) as "spontaneous intermittent" resorption which he distinguishes from
ideopathic resorption, "which is accompanied by no repair mechanism". This is also the "peripheral intra-dental resorption" of Bouyssou et al (1965 p. 251).

This type of resorption may be initiated in an aberrant pulp canal (Colyer and Sprawson 1931) and, as will be seen, by the impaction of an adjacent tooth.

"Burrowing" resorption may progress at an undiminished rate after removal of the impacted tooth. For this reason, it is necessary to distinguish the type of resorption before any orthodontic treatment plan is made.

This resorption is also similar to that type of resorption seen in completely embedded teeth (Stephne and Austin 1945), (Goldman 1954 p. 530) and may form tunnels, canals and finger-like projections into the dentine completely encircling the pulp but not often invading it. (Sullivan and Jolly 1956 p. 1957).

The pathogenesis of "burrowing" resorption is distinct from that of "accommodating" resorption.

"Burrowing" resorption is so often associated with marginal periodontitis that a casual link is highly probable, if not always obvious. (Bouyssou 1965 p. 438). A cervical impaction may initiate this process, not by direct pressure resorption, but by creating an environment whereby marginal inflammation and granulation tissue may occur. This granulation tissue may then invade the adjacent
root substance in the typical "burrowing" manner.

This resorptive process then becomes independent of the presence of the impacted tooth.

Treatment.

Teeth which display "burrowing" resorption may be regarded as being in the arch only temporarily unless surgical procedures are instituted. (Bouyssou 1965 p. 421).

Root filling and apicectomy is necessary to arrest the process of resorption occurring in the apical portion of the tooth.

Shallow cervical resorptions require gingivectomy, thorough curettage of the resorptive cavity with a round bur and the placement of an amalgam or gold inlay restoration.

Deeper cervical cavities may necessitate root filling, besides the placement of a metal restoration in the resorbed cavity.

The weakened root may be reinforced by placing a rigid metal post in the root canal to prevent fracture by occlusal stresses. (See fig. 4).

"Accommodating" Resorption.

This is the "amputating" resorption of Bouyssou et al (1965 p. 46). It can occur idopathically (as can the "burrowing" type) when it may be either progressive, to complete destruction of the root, or purely local.
Fig. 4. Diagramatic representation of methods of treatment of "burrowing" resorption. Taken from Bouyssou (1965 p. 424).
The term "accommodating" resorption is used here to describe that type of resorption which proceeds at a similar rate to the advancing impacted tooth and which extends just sufficiently to accommodate the advancing portion of the crown of the impacted tooth.

In "burrowing" resorption the lesion can advance independently of the impacted tooth and may continue even after the impacted tooth has been removed.

In "Accommodating" resorption, the resorptive process is dependent upon the presence of the crown of an impacted tooth and progresses in an orderly fashion, the concavity of the lesion matching in shape the convexity of the advancing crown. The resorbed area of the tooth appears quite smooth, as if made by a dental bur.

Radiographically it appears as either a clean cut amputation of a root apex or a semilunar bay in the side of a tooth.

The pathogenesis is distinct from that of the "burrowing" type, and the resorptive process is dependent upon the presence of the impacted tooth. Removal of the impacted tooth in most cases will result in the cessation of the process of resorption.
Fig. 5. Diagramatic representation of various types of dental resorption. 1. External Amputating Resorption. A. Localised, B. Progressive, C. In Included Teeth. 2. Intradental Resorptions. A. Pulpal or Central, B. Peripheral or Periodontal, C. In Included Teeth. Taken from Bouyssou (1965 p. 244).
The Effects of Orthodontic Movement of Erupted Teeth on Unerupted Adjacent Teeth.

Ottolengui (1914) was concerned with the distal movement of an erupted first molar onto an erupting or already impacted second molar. From his observations on impacted teeth bringing about resorption of the root of the impacting tooth he said "any forcing of this first molar backward against the second would, in my view, possibly inaugurate resorption of the distal surface of the first molar".

He applied this theory clinically by waiting for sufficient eruption of the second molar to allow its enamel crown to come in contact with the enamel surface of the tooth which he intended moving distally.

A search of the literature failed to reveal a further discussion on this subject or any examples of cases where the distal movement of a first permanent molar has resulted in resorption of its distal root by impaction on the unerupted second molar.

Silas J. Kloehn, acknowledging the work of Oppenheim (1944) re-introduced, after the second World War, the use of cervical traction for the distal movement of first permanent molars.

In a personal correspondence with Dr. Kloehn (1967) he stated that obvious resorption of the distal root of
of the first permanent molar had never been a complication of this distal movement in his many cases, even when forced onto an unerupted second molar.

He said that he had moved first molars posteriorly as much as 8 mms. measured cephalometrically, without any detectable resorption of the distal root. Kloehn explains that the cancellous nature of the tuberosity region "permits good adjustment and distal movement" of the unerupted second molars.

There has been considerable doubt as to whether cervical traction actually moves molars distally. It has been stated that cervical traction simply holds the first molars in their existing position while forward differential growth of the maxilla and mandible gives the impression of distal movement of the first and second molars.

Ricketts (1960) and Klein (1957) stated that first molars can actually be moved distally, whereas King (1957) felt that the molar was merely held in position while the face grew forward.

Graber (1955) summarises general thought on the scope of extraoral force. He says there is evidence that bodily distal movement of the first molars may occur, but not routinely, unless the second molars are extracted.

He also suggests that possible impaction of the
Fig. 6. Approximation and resorption of the roots of adjacent teeth as a result of loss of coronal contour through extensive caries (Orban 1928).
second and third molars may result from excessive distal tipping of the first molar.

It is conceivable that resorption of the roots of the second molar in these cases could occur at a time when the third molars are attempting to erupt, that is, when orthodontic observation of the patient has normally ceased.

Miles (circa 1963) in his review of the literature on extraoral force, has left the question of the possibility of distal bodily movement open. He concludes that the maxillary teeth normally do show forward movement with growth, but not nearly as much as when a restraining appliance (extraoral force) is used.

From the above observations, it can be concluded that extraoral traction applied to the upper first permanent molars cannot be considered as directly responsible for clinically demonstrable damage to the roots of this tooth by impacting it against the unerupted second molar.

However, it is possible that orthodontic treatment utilising extraoral force without extraction of the second molars may result in the impaction at a later date of the erupting third molars.

This could conceivably lead to a resorption of the roots of the second molar, but is a most unlikely direct
sequence of extraoral force.

Orban (1928) has shown how a large carious lesion in the distal of a lower second molar has allowed the third molar to drift in such a way that the interseptal bone has become resorbed (Fig. 6.).

This has allowed approximation of the lower third of the distal root of the second molar and the mesial root of the third molar, resulting in a surface "accommodating" resorption of both roots.

A similar situation could conceivably arise in the overparalleling of roots using uprighting springs in the Begg Technique of orthodontic tooth movement.

The third stage of this technique requires the uprighting of tipped bicuspids, cuspids and laterals in bicuspid extraction cases (Begg 1955 p. 144). This is achieved by paired, opposing uprighting springs applied to the crown of the tooth, allowing the teeth to rotate axially around a point corresponding to the bracket on the crown of the tooth.

If these springs are left in position for too long a period it would be possible to impact the apices of the adjacent teeth against each other, resulting in a similar resorption to that seen in fig. 6.

Overtreatment is a requirement for success in the application of the Begg technique. Rotated teeth should
be over-rotated, the first molars are ideally placed in a mild Class III relationship and teeth which have been tipped into extraction spaces should be uprighted and "torqued" slightly beyond the upright position (Begg 1965 p. 350).

The danger here may lie in "overtorquing" these tipped teeth until their apices become impacted against each other, resulting in excessive root damage by resorption.

The Erupting Maxillary Canine.

A common problem facing both the general dental practitioner and specialist orthodontist is the displacement of the upper lateral incisor by an ectopically erupting permanent canine.

This displacement may take the form of the "ugly-duckling" stage of dental development, where the large canine crown in a restricted apical base displaces the apex of the lateral incisor mesially. This gives the effect of tipping the crown of the lateral distally. The four upper incisors then give the appearance of "fanning-out".

A labially placed canine may tip the lateral incisor apex palatally or a lingually placed canine may tip the lateral apex labially, resulting in the lateral incisor crown becoming interlocked behind the lower anterior teeth in occlusion.
From our previous observations, it would seem most
inadvisable to try and position this lateral incisor
orthodontically without first making sure the crown of
the canine has moved away from the root area of the
lateral.

To press the root of the lateral incisor against the
crown of an erupting cuspid may well initiate a damaging
resorption of the root of the lateral incisor.

This applies to the moving of any root against an
obstructive erupting crown, but perhaps more so in the
anterior region because of the higher resorptive potential
of the anterior teeth.

That this is not a sequel of the distal movement of
first molars onto unerupted second molars may well be
because of the fact that the first molar is not actually
moved distally. If slight distal movement is achieved,
the cancellous nature of the tuberosity region may
allow for an easy distal movement of the second molar
before resorption commences.

A minor amount of resorption of the distal root
surface of the lateral incisor from the erupting canine
may be the rule, rather than the exception, as Orban has
shown for the second molar. (p. 101).

If the canine crown is in such a position that it
cannot erupt because of impaction it would be necessary
to surgically expose the crown, and ligate it to an
arch wire with a view to orthodontically moving the
canine crown away from the roots of adjoining teeth
before any attempt was made to move these teeth.

An erupting upper canine crown seen in the proximity
of the root of the lateral incisor is not a danger to
that root per se, as the normal position of an erupting
cuspid is closely associated with the lateral incisor
root apex (see fig. 2), but to torque the apex of the
lateral incisor against this crown may initiate serious
"accommodating" resorption of the lateral incisor root.
ORIGINAL WORK

Histological Studies of Resorbed Teeth Found In Association with Impacted Teeth.

AIM.

To examine the effects of resorption initiated by an impacted tooth on the root and pulp of an adjoining human permanent tooth, using histological methods.

MATERIALS & METHODS.

(a) Materials.

The material consisted of three teeth displaying varying degrees and types of resorption initiated by impacted adjoining teeth. For convenience in reference, the teeth will be lettered A, B, and C.

TOOTH A.

This tooth was an upper left permanent lateral incisor surgically removed from a female aged 12 years 0 months.

History.

A routine general dental examination was carried out in November 1965. No caries was evident. Although the first molars were in good Class I relationship, there was evidence at this stage of a lack of room in the dental arches for accommodation of the full complement of permanent teeth.

Oral hygiene was good and there was no oral pathology obvious. The patient was in good health and appeared of
average height and weight for her age group.

There was a small amalgam restoration present in a lower deciduous molar.

The patient returned in September 1966, concerned about a pink discolouration of the upper left lateral incisor. The area of gum corresponding to the interdental gingival papilla between the distal of the lateral incisor and the mesio-incisal angle of the semi-impacted canine appeared swollen and chronically inflamed.

Full mouth x-ray examination revealed a large cavity in the distal aspect of the left lateral incisor, approximately 7 mms. in length, extending from within 3 mms. of the incisal edge to within approximately 11 mms. of the apex and to a depth apparently involving the pulp chamber and root canal.

The upper left canine appeared impacted into the resorptive cavity on the distal aspect of the lateral incisor.

The upper right canine appeared impacted into the distal aspect of the upper right lateral incisor towards the gingival third of the root.

The lower right second premolar was impacted into the mesio-gingival aspect of the lower right first molar but was in an upright position.

Clinical examination revealed an inelastic and restrictive oral musculature. Orthodontic treatment using
Fig. 7. Tooth A.
B. Periapical X-ray taken April 1967.

Fig. 8. Tooth A. Left lateral view of partially impacted Canine Sept. 1966.
Fig. 9. Palatal view of Tooth A after extraction.
Fig. 10. View of serial section of Tooth A. Note enamel loss through decalcification. E. epithelialised granulation tissue, P. pulp, D. dentine, P.M. periodontal membrane, A.B. alveolar bone (PAM stain).
full bands, edgewise brackets and .016" round wire was commenced in January 1967.

Resorption of the distal surface of the lateral incisor had progressed sufficiently at this stage to allow sufficient room for the full eruption of the left canine.

In April 1967, the upper left lateral incisor was x-rayed and surgically removed. The x-rays revealed that the resorptive process had progressed in the six months since the previous x-ray, despite the fact that the impacted canine had been orthodontically moved away.

The entire crown appeared involved except for a small portion of the mesio-incisal dentine.

The resorption had progressed apically and distally also but the outline of the pulp and root canal could still be discerned superimposed on the area of resorption. The incisal two-thirds of the tooth appeared to be affected by a hazy, mottled radiolucency.

There was no history of pain, except for mild discomfort when hard food was bitten. The resorption had undermined the palatal enamel to such a degree as to allow it to break up on mastication. The exposed resorptive granulation tissue had then become epithelialised, giving the typical appearance of a pulpal polyp.

**TOOTH B.**

This tooth was an upper right first permanent molar,
Fig. 11. Lateral view of dental cast showing Tooth B.

Fig. 12. Elevated bite-wing X-ray showing second premolar impacted into Tooth B.
surgically removed from a female aged thirteen years and three months.

**History.**

The patient was of average height and weight for her age group, with a low caries experience. Oral hygiene was good. There was a severe Class II division 1. malocclusion present; with a slight tendency to crowding in both upper and lower jaws.

The mandibular right buccal segment occluded palatally with the maxillary teeth, the upper first molar being overerupted, giving the appearance of an exaggerated curve of Spee.

The upper left second premolar was slightly hypoplastic.

The upper right second premolar was not visible in the arch, although there was almost sufficient room to accommodate it. The first molar and the first premolar had drifted mesially and distally, respectively, to partly usurp the space required for the second premolar.

A full mouth x-ray survey revealed the missing upper second premolar to be impacted into the buccal roots of the first molar and inclined distally at 60° to the occlusal plane.

The tooth appeared well formed but was rotated through 90° in the long axis. The presence of a large coronal sac obscured the apical area of the first molar but it was
evident that there was resorption of the mesio-buccal root of the first molar.

X-rays taken four months later showed little or no change in the amount of resorption present.

There was no history of pain and the patient was unaware of the condition. The case was treated by the extraction of the four first molars. Full orthodontic banding and the application of the Begg Technique was the treatment plan considered. The upper right first molar was surgically removed and prepared for sectioning.

**TOOTH G.**

This tooth was an upper left permanent lateral incisor removed from a female aged thirteen years 0 months.

**History.**

The patient was of average height and weight for her age group and had a moderate caries experience, there being Class 2 amalgam fillings in the four first molars.

Oral hygiene was fair, with calculus present on the lingual aspect of the lower incisor teeth and moderate marginal gingivitis in the lower anterior region.

There was mild Class I malocclusion present which could be described as a tooth-to-base bone discrepancy case resulting in slight crowding of the anterior teeth.

Clinical examination revealed the upper left deciduous
canine in position, and the left canine unerupted. The upper right canine, as well as all the other permanent teeth due at that age except the upper second molars, were fully erupted.

Full mouth x-ray revealed the upper left canine ectopically placed and impacted into the disto-apical third of the lateral incisor. The apical third of this lateral incisor appeared to have been resorbed, as well as most of the root of the deciduous canine even though the permanent canine appeared radiographically some 4 mms. distant from the remnants of the deciduous tooth.

These x-rays also revealed a "snubbing" resorption of the roots of the lower left second premolar, the upper left first premolar and the upper right first premolar. The lower central and right lateral incisor apices appeared slightly affected also, but this could not be claimed with certainty.

This "snubbing" of the roots of the premolars appeared rather as idiopathic resorption than a lack of development of the apices. The walls of the root canal appear parallel or slightly convergent to the apical foramen, which is visible on the x-ray. This is an indication that resorption has occurred. (Black 1965 p.151).

The patient was referred to Wade House, Royal Alexander Hospital for Children for a physical examination. No
Fig. 13. Tooth C. after extraction.

Fig. 14. Frontal view of dental cast showing Tooth C.
abnormalities were found. Coagulation studies, serum calcium, phosphorus and alkaline phosphatase levels were all within normal limits in the blood tests performed, and nothing was found which could be linked with a predisposition to root resorption.

The left lateral incisor and granulation tissue from the resorbed area were removed for sectioning. Orthodontic treatment was commenced using a full band technique with edgewise brackets and .014 and .018 in. round arch wires to align the teeth and make room for the impacted canine. The canine was surgically exposed and ligated to the arch wire.

METHOD.

All teeth examined were fixed in a 10 per cent neutral buffered formol-saline solution immediately after removal from the mouth.

All sectioning was performed on a Reichert Rotary Microtome after decalcification and embedding and all sections were of a thickness of 6 μ.

TOOTH A.

Two hundred and eighty serial sections were made, all cut in a labio-lingual plane parallel to the long axis of the tooth.

There were seven sections on each slide, and every second slide was stained with Harris' Haematoxylin and
Fig. 15. Periapical X-ray of Tooth C. showing impacted canine. Note appearance of idiopathic resorption of first premolar.

Fig. 16. Appearance of idiopathic resorption of lower left second premolar in patient from whom Tooth C. was removed.
and Eosin (H & E) (Gurr 1963 p. 47).

The remaining twenty slides (140 sections) were stained with Mallory's Phosphotungstic acid and Haematoxylin (P.A.H.) (Gurr 1963 p. 49).

**TOOTH B.**

Two hundred and forty-five sections cut serially in a bucco-palatal plane, parallel to the long axis of the palatal root, were made. These sections included the apices of the three roots and the pulp chamber.

Three stains were used on this tooth, using alternate slides. One third of the slides were stained with Harris' Haematoxylin and Eosin, one third using Mallory's Phosphotungstic acid and Haematoxylin and the remaining third were stained with Periodic acid-Schiff (P.A.S) (Gurr 1963 p. 63), with added Harris' Haematoxylin for the nuclei.

**TOOTH C.**

Thirty two serial sections were cut in a labio-lingual plane parallel to the long axis of the tooth, including the pulp chamber.

Forty sections were also made of the granulation tissue found in the resorbed cavity.

All these sections were stained with Harris' Haematoxylin and Eosin.

The resulting five hundred and ninety seven sections
Fig. 17. Tooth A, immediately prior to extraction.
Note pink discolouration and inflamed distal gingival papilla.
were examined histologically using a Leitz Ortholux binocular microscope.

Magnifications of 25 to 1000 times were used, oil immersion being used at a magnification of X1000.

The colour photographs used to illustrate this work were taken through a Leitz microscope using a Leitz microphotographic camera.

Kodacolour-X 35 mm. film was used, with electronic flash.

RESULTS.

HISTOPATHOLOGY

TOOTH A. The Pulp:

In this tooth, the pulp had not been invaded at any point by the resorptive process.

A continual thin wall of dentine surrounded the pulp, which in the coronal portion of the tooth, was almost completely surrounded by the resorptive cavity. (see fig.10).

A column of dentine in the mesial portion of the crown was the only remaining support to the incisal dentine and enamel, except for the delicate "tube" of dentine around the pulp.

The pulpal tissue appeared relatively normal (Cameron 1967).

The degree of fibrosis may have been at the upper limits of normality for a person of that age but could
not be construed as pathological. (Cameron 1967).

There were no aberrant calcifications of any kind in the pulpal connective tissue or adherent to the pulpal walls.

The size of the pulp chamber appeared normal, and a continuous layer of odontoblasts lined the pulpal walls. (See fig. 18 and 21).

Even in those areas where the resorptive cavity appeared within 0.5 mm. of the pulp, the underlying odontoblasts did not appear to be affected and were laying down new predentine at a rate equal to those odontoblasts further apically, as evidenced by the uniform layer of acidophilic predentine.

Altered odontoblastic activity was not a feature in the vicinity of the resorption. There was no increased deposition of secondary protective dentine, nor was there any decrease in the odontoblastic activity through necrosing influences of the proximity of the resorptive process.

The apical blood vessels appeared slightly enlarged, and were engorged with red cells.

This effect was probably the result of the "pumping" action which occurs during the process of removal of the tooth from the alveolus, (Cameron 1967).

There appeared to be no pulpal inflammation whatsoever,
Fig. 18. Pulp of Tooth A. in the vicinity of the resorptive process. N. nerve bundle, P. pulp, O. odontoblastic layer, D. dentine, G. granulation tissue. (P.A.H. stain original x 80).
Fig. 19. Osteoclastic activity in dentine surrounding coronal tip of pulp in Tooth A. O. masses of osteoclasts, D. dentine, P. pulp. (H. and E. stain. original X 24).
Fig. 20. High power view of osteoclast in Tooth A. Nine nuclei are visible. D. dentine, O. osteoclast. (H. and E. stain. Original X 1000).
either bacterial or traumatic.

There were no aggregations of inflammatory cells, and no areas of necrosis within the pulp to indicate inflammatory breakdown of the pulp tissue.

The Area of Resorption.

The process of resorption had commenced at the distal dentino-enamel margin at the point of impaction of the erupting canine.

The resorption was of the "burrowing" kind (see p. 110) and finger-like processes of granulation tissue had penetrated the dentine and progressed both labially and lingually around the pulp chamber.

These processes had become enlarged and confluent until the entire coronal dentine had become resorbed, except for a small portion of the dentine in the incisal area and a column of supporting dentine in the mesial margin of the crown.

As a result of this undermining, the enamel in the gingival third of the palatal aspect of the crown had become thinned by resorption until it had finally broken away, exposing the resorptive granulation tissue to the oral cavity.

This tissue had then taken on a thick epithelialised layer. A similar process had commenced at the disto-labial gingival margin.
Channel-like resorptive processes could be seen extending apically from the body of the resorptive tissue, but no osteoclasts could be found in these channels.

Masses of multinucleated osteoclasts could be seen surrounding the dentine at the coronal tip of the pulp (see fig. 19 and 20) and at the small portion of dentine remaining in the incisal tip of the crown, denoting active resorption.

No osteoclastic activity could be found elsewhere associated with the resorptive cavity.

A process of irregular repair was going on in conjunction with the resorptive process.

A large triangular section of irregular bone-like substance was laid down on the palatal aspect of the thin wall of dentine over the pulp.

This was laid down directly on the dentine.

Irregular areas of thin cellular and acellular cementum were being laid down on nearly every resorbed surface, except towards the incisal portion of the crown.

The Soft Tissue in the Resorbed Cavity.

The soft tissue in the area of resorption was an irregular granulation tissue, interlaced with bundles of fibrous tissue such as is seen in the periodontal membrane, (fig. 21).

The tissue was quite vascular, with isolated areas of intense activity of inflammatory cells, particularly
Fig. 21. Epithelialised granulation tissue in close proximity to pulp in Tooth A. P. pulp, D. dentine, N. nerve bundle, E. epithelial layer over G. granulation tissue. (P.A.H. stain. Original X 24).
near the oral surfaces.

Giant multinucleated osteoclasts were found in profusion applied to the dentine of the incisal portion of the crown, as well as the line of enamel on the labial aspect of the crown, but were absent elsewhere.

That area of the granulation tissue which had become exposed to the oral cavity by loss of the palatal enamel had developed a heavy layer of prickle cells which stood out in strong contrast with Mallory's P.A.H. stain.

The surface cells were extremely flattened but it is doubtful whether true keratinisation had taken place.

Sullivan and Jolly (1957 p. 198) described the presence of large numbers of histiocytes (also known as "resting wandering cells" of Maximow or "macrophages"), in their sections and intimated that these were the cells concerned with the resorptive process.

These cells could not be found in any large numbers in this series of sections.

The apex of the tooth had also undergone slight resorption. An irregular area of lacunar resorption which had undergone repair with cementum could be noticed, but no osteoclasts were present to denote active resorption at the time of extraction.
TOOTH B. The Pulp.

In this tooth, resorption had commenced at the apical region of the mesio-buccal root, and the apical foramen had been directly involved by the process of resorption.

Three quarters of this root had been destroyed by this process of resorption but despite this, the pulp had remained relatively normal. (Cameron 1967).

That description of the pulpal tissue given previously for Tooth A would apply also to Tooth B with some minor exceptions, noted as follows:-

The degree of fibrosis of the pulp of Tooth B was greater than that found in Tooth A but could not be considered as pathological in a first permanent molar in a thirteen year old girl.

There was evidence of several minor irregular free calcifications in the pulp and root canals (see fig. 22) as well as in the palatal root canal which had not been attacked by the resorptive process.

These took the form of free false denticles, and were very small. Sicher (1962 p. 157) has classified these under regressive pulpal changes, and states that they are on the borderline of pathologic changes. Sullivan and Jolly (1957 p. 196) describe these false denticles as being relatively common under these circumstances.
Fig. 22. Tooth B. Palatal root apex. Note calcifications in pulp near apex. P, pulp, P.C, pulpal calcifications, D, dentine, (P.A.S, stain, Original x 24).
Provenza (1964 p. 284) states that they are relatively common under any circumstances. He says two thirds of the pulps in the 10 to 20 year age-group and nine-tenths in the 50-to 70 year age-group demonstrate calcific regression of one kind or another.

The nidus of a false denticle may be organic detritus, such as pulp cells or thrombi (Provenza 1964 p. 284). Thrombi in the pulp could occur in cases of resorbed teeth by occlusal trauma of the loosened tooth against the erupting impacted crown.

The blood vessels of the apical areas were slightly enlarged and engorged with blood, but no signs of pulpal inflammation were present.

**The Area of Resorption.**

The resorption displayed in Tooth B was of the "accommodating" type (see p. 112) presenting a rather more linear progress, in contrast to the irregular finger-like extensions found in Tooth A.

The remarkable feature in this case was the complete lack of osteoclasts or any cells which could be linked with resorption. On some aspects of the resorbed root, cellular and acellular cementum and cementoid could be seen covering the resorbed dentine in varying thicknesses.

In other parts, dense fibrous tissue lay closely approximated to the dentine, which seemed to be undergoing neither resorption nor repair.
Thick layers of bone-like tissue could be found on that part of the affected root which had not undergone resorption, taking the form of a cellular hypercementosis.

Thick layers of cementum could also be found on the palatal and disto-buccal roots.

Inflammatory cells were absent in the tissue surrounding the resorbed root, being also absent in the pulp. (Cameron 1967).

**TOOTH C. The Pulp.**

The resorptive process had commenced on the disto-palatal aspect of the root apex and progressed to the destruction of one third of the root.

The pulp was essentially normal histologically, (Cameron 1967) except for the presence of a large false denticle towards the apex of the root canal, attached to the distal pulpal wall for about half the length of the denticle (see fig. 24).

A description of the pulp would be similar to the description of the pulp in Tooth B in most aspects, except that in Tooth C there was only one large denticle, as opposed to several small denticles in Tooth B.

There was no evidence of any inflammatory degeneration; and a continuous layer of normal odontoblasts lined the pulp chamber. (fig. 23).
The Area of Resorption.

The resorptive process present in Tooth C was of the "accommodating" type, as seen in Tooth B.

As in Tooth B, the progress of resorption was in a more linear fashion, and the shape of the resorptive cavity closely matched the shape of the advancing impacted crown.

A careful examination of all the sections revealed no osteoclasts nor cells which could be linked with the resorptive process.

The tissue from the resorptive cavity was of a very dense, fibrous nature, showing little vascularity, and no aggregation of osteoclasts or macrophages could be identified in it.

There was no sign of any inflammatory cells present.

A large irregular thickening of the cementum occurred around the root end near the area of resorption.

This thickening decreased as the dentino-enamel junction was approached and the cementum in the vicinity of the dentino enamel junction appeared normal.

The greatly thickened cementum near the root end showed many reversal lines and was of a cellular nature.

Although the process of resorption had progressed in a linear fashion, deep finger-like processes could be seen invading the dentine in several places, particularly
Fig. 23. Tooth C. Section of normal pulp. D, dentine; P.D, predentinal layer; P, pulp, (H. and E. stain. Original X 250).
Fig. 24. False denticle in pulp of Tooth C.

(H. and E. stain. Original X 24).
near the pulp chamber.

These processes gave the appearance of very localised areas of "burrowing" resorption. All these deep channels were filled with a cementum-like substance, with only a small number of cells.

Most of the resorbed surface to the labial of the root canal was covered with a thin layer of cementum, thickening as the labial root surface was reached.

On the palatal side, of the root canal, little repair could be seen of the resorbed area, but neither was there evidence of an active resorptive process.

Deeper resorptive lacunae were filled with a loose fibrous connective tissue, but an extremely dense and closely applied fibrous tissue characterised those areas where there were no deep lacunae.
DISCUSSION.

The Condition of the Pulp.

In considering the effect of the resorptive process on the pulp of the affected tooth, the outstanding feature in most cases is the lack of any marked degenerative processes occurring in the pulp.

In Case 21 in the review of the literature, advanced degenerative processes had occurred in the pulp, but there is no evidence to show when this degeneration had commenced.

It may have begun prior to the commencement of resorption through bacterial invasion or traumatic influences but cannot be certainly linked with the presence of a resorptive process.

In Case 5 the pulp appeared relatively normal, but with "evidence of reticular atrophy", and "a heavy deposition of secondary dentine with a well delineated border of odontoblasts". This patient was 33 years of age and a considerable deposition of secondary dentine and evidence of reticular atrophy is not unusual for a "normal" second molar in this age group. (Provenza 1964 p. 281).

In Case 29, the presence of a large pulp stone almost obliterating the pulp chamber and the posterior root canal nearly occluded by secondary dentine were noted. This condition again may not be unusual in a
47 year old female and cannot necessarily be linked with the resorptive process.

The presence of pulpal calcifications in the form of false denticles seems to be a feature in many of those cases examined. The significance of these denticles has been considered on page 148.

The effect on the pulpal tissue of both the "burrowing" and "accommodating" types of resorption, even in relatively advanced cases, in the young patients examined in this work, seems to be almost negligible.

The condition of the tooth prior to the commencement of resorption, the age and health of the patient and the nature of the occlusion of the tooth must all have a direct bearing on the degree of atrophy of the pulp, just as these factors influence the pulpal condition of an unresorbed tooth.

It is conceivable that extensive root loss associated with a heavy occlusal load will lead to an early degeneration of both pulp and periodontium, but this degeneration cannot be associated directly with the resorptive process.

The Resorptive Process.

The resorptive process may be one of two types, either "accommodating" or "burrowing". In Tooth C, there is evidence of both types occurring in the one tooth.

It seems that the "burrowing" type of resorption is
not the direct result of the impacted tooth, although its presence in limited areas was noted in Tooth C. Rather, the impacted tooth resulted in an inflammation of the gingival tissue in the vicinity of an adjoining tooth, the resultant granulation tissue seemingly initiating the resorption, the impacted tooth then erupting into the cavity formed.

This resorptive process is capable of progressing even after the impacted tooth has been moved away.

In the "accommodating" type, the process is intermittent. In both Tooth B and Tooth C, the process was dormant, and no evidence could be found that active resorption was progressing.

Active repair had commenced in many places, and even layers of cementum were being laid down on the newly exposed dentine.

It is important to distinguish between the two types in making a prognosis.

It is reasonable to assume that surgical removal of an impacted tooth initiating "accommodating" resorption will result in the cessation of resorption.

The usefulness of the resorbed tooth must then be assessed in terms of the length of the remaining root structure and the force of occlusion on that tooth.

These two factors must be considered together and
a conclusion made as to their effects on the pulp and periodontium.

Where extraction is necessary prior to orthodontic treatment a decision must be made as to which teeth are best extracted.

There is a distinct possibility, that a tooth which has been resorbed by an impacted tooth, even if the impaction is removed, will further resorb under the influence of orthodontic movement.

These teeth should be regarded as a poor "risk" if it be necessary to move them over large distances.

It may be necessary to remove these teeth, rather than more "strategic" ones, when long term orthodontic treatment is considered.

The Repair Process.

In Tooth B and Tooth C, where considerable root loss was a factor, the cementum beneath the remaining intact periodontal membrane became greatly thickened, decreasing in thickness as the cemento-enamel junction was approached.

The reason for this is difficult to find. It could be either a response on the part of the periodontal tissues to maintain the root area of attachment or a local disturbance of the primary undifferentiated mesenchymal cells to form cementoblasts rather than
cementoclasts (or osteoclasts).

In Tooth A, although no active osteoclasts was evident at the apex at the time of extraction, there was an indication of resorption having occurred.

This had been repaired by an irregular layer of cementum, again greatly thickened on the labial aspect of the tooth.

In the area of "burrowing" resorption, little attempt had been made at cemental repair, except in small areas towards the labio-gingival aspects of most sections.

Most resorption lacunae were filled with loose connective tissue, under strong bands of dense fibrous tissue.

This lack of organised repair tissue may be related to the rapidly progressive nature of this type of resorption.

The large wedge of bone-like tissue in the palatal resorptive area described previously may be an attempt at organised repair, indicating a cessation of the resorptive process in that area or may be a haphazard process as a result of "accidental" differentiation of osteoclasts.

Clinical Implications.

Where "accommodating" resorption is radiographically
visible, the "clinical usefulness" of the resorbed tooth must be assessed, considering the following conditions:
1. The position of the affected tooth in the arch.
2. The amount of root loss.
3. The position of the resorptive cavity on the root.
4. Whether there is a malocclusion present.
5. Whether orthodontic treatment will be undertaken.

To illustrate, several clinical examples may be considered.
(a) An upper lateral incisor has one third of its root destroyed in the apical area by an ectopically erupting canine, and there is no other malocclusion or dental pathology present.

Provided surgical exposure and alignment of the canine by orthodontic therapy is feasible, the lateral incisor may be regarded as a sound tooth.

If three-quarters or more of the lateral incisor root is missing, this may still be the treatment of choice.

The lateral incisor may be retained until occlusal stresses result in a breakdown of the periodontal tissues. A bridge may then be used to replace the tooth.

If occlusal forces are not heavy, this breakdown may never occur.
(b) An impacted third molar has resulted in resorption of the middle third of the distal root of a lower second molar, the resorptive cavity extending half way to the pulp.

If the coronal condition of the second molar and the occlusion are good, surgical removal of the third molar would be considered.

If the resorptive cavity extends into the pulp, or pain from the pulp of the impacting tooth is present, extraction of the second molar would be the treatment of choice.

Provided the patient is of a suitable age and the third molar appears sound radiographically, it may erupt to replace the lost second molar.

(c) In a severe Class II division I malocclusion, an impacted second premolar has brought about root resorption of the first permanent molar. No matter how much root resorption of the molar was evident, in most cases it would be the treatment of choice to extract the first molars, particularly in the arch displaying resorption.

The extraction of these molars would provide sufficient anchorage for the reduction of the malocclusion, as well as sufficient room for the eruption of the impacted premolar.

In any of the above cases, the closer the resorptive
cavity is to the neck of the tooth, the more difficult it is to distinguish between the "burrowing" type and the "accommodating" type of resorption. This makes diagnosis less certain.

If the tooth displaying resorption must be moved orthodontically through large distances, periodic x-rays of the tooth must be taken during treatment.

These teeth should be regarded as having a high resorptive potential and are hence poor orthodontic "risks".
"Resorption Associated with Impacted Teeth in the Human Permanent Dentition."

Summary and Conclusions

Frequency.
Radiographically detectable resorption associated with impacted teeth can be regarded as a relatively rare occurrence.

Histologically detectable resorption from impacted teeth, on the other hand, may be regarded as the rule, rather than the exception, but seldom progresses to a radiographically demonstrable extent.

Incidence.

The incidence of root resorption initiated by an impacted tooth is highest in those areas which show the highest incidence of impactions and ideopathic resorption.

The upper and lower permanent second molars and the upper lateral incisors are most commonly affected.

The second molars and upper lateral incisors are almost equally affected, although the proportion of third molar impactions is far in excess of upper cuspid impactions.

This perhaps can be explained by the higher susceptibility of anterior teeth to ideopathic resorption.
Age of Occurrence.

In the anterior and bicuspoid areas a high rate of resorption is associated with the active eruptive phase of the impacted tooth.

The resorptive process commences when the erupting tooth becomes impacted and probably continues in an intermittent fashion until active eruptive movements cease.

The eruptive process may then cease altogether or become active intermittently when minute movements of either the impacting or the impacted tooth occur.

In the molar region there was no correlation between the eruptive phase of the impacted molar and the discovery of the resorptive process.

This was probably due to the fact that the presence of resorption was discovered accidentally in many of the cases.

This resorption could have commenced with the eruptive phase of the impacted tooth and ceased when the eruptive force had diminished.

Eruptive Force.

The eruption of a tooth can continue after full root development has occurred, but with a diminished rate. This mechanism of eruption is obscure.

From this it can be concluded that the resorptive process initiated by an impacted tooth will not necessarily
cease on the completion of root development in the impacted tooth.

Sex Differences.

Resorption initiated by an impacted tooth is more common in females than in males. This applies only to the upper anterior region, where the incidence of both impactions and idiopathic resorption is higher.

There seems to be little significant difference in the molar region.

Nature of the Resorptive Process.

Two types of resorption have been associated with impacted teeth. These are "burrowing" and "accommodating" resorption.

"Burrowing" resorption cannot be regarded as a direct result of pressure from the erupting tooth, but rather as a consequence of local inflammation of the periodontal tissues associated with the impaction. This type of resorption cannot be relied upon to cease on removal of the impacted tooth.

"Accommodating" resorption is a direct result of the presence of an erupting impacted tooth.

This type of resorption will cease on removal of the impacted tooth.

Effect on the Pulp.

The resorptive process has little direct effect on
the pulpal tissue of the resorbed tooth. The presence of false denticles and minor pulpal fibrosis has been associated with the resorptive process but with no greater frequency or severity than found in "normal" dental pulps of a similar age group.

Degenerative pulpal conditions may occur in teeth which have suffered severe root loss.

This cannot be directly attributed to the resorptive process. It is more likely the result of the extensive root loss and relative increase in occlusal stress on the reduced periodontal tissues.

Pulpal pain is not associated with the resorptive process.

Pain will only occur when pulpal degeneration or bacterial invasion occurs, or possibly from the irritating presence of pulpal calcifications.

Repair of the Resorbed Dentine.

Cementum or bone-like repair tissue may be laid down while resorption is occurring in other areas.

It is most likely that a covering of cementum over the resorbed dentine will be laid down when the process of resorption ceases.

In radiographically demonstrable resorption, it is unlikely that the root contour and dimensions will be re-established on cessation of the resorptive process.
Orthodontic Implications

Where "accommodating" resorption is radiographically visible, the "clinical usefulness" of the resorbed tooth must be assessed, considering the following conditions:

1. The position of the affected tooth in the arch.
2. The amount of root loss.
3. The position of the resorptive cavity on the root.
4. Whether there is a malocclusion present.
5. Whether orthodontic treatment will be undertaken.

The use of extra-oral force on the first molars before the eruption of the second molar may, through distal tipping of the first molar, bring about mild impaction of the second molar.

Severe resorption of the roots of the first molar as a result of this type of impaction could not be considered as a likely consequence. Histologically demonstrable resorption may occur but could be regarded as readily repaired by cemental deposition.

Forcible "torquing" of roots onto erupting or impacted teeth, on the other hand, may result in severe root damage. This situation would not arise in a properly diagnosed and carefully treated malocclusion.
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APPENDIX A.

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