A CRITICAL REVIEW OF LITERATURE PERTAINING TO

IMPACTED AND UNERUPTED TEETH

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This critical review of literature concerning Impacted and Unerupted Teeth is submitted in support of candidature for the Degree of Master of Dental Surgery.

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- PART I -

THE AETIOLOGY OF IMPACTED AND UNERUPTED TEETH.

Teeth which fail to erupt concern not only the oral surgeon, but also the orthodontist. A natural denture, marred by non-eruption of one or more teeth, displays malocclusion. In many of these cases, a satisfactory solution may only be attained by the closest co-operation between these specialists.

Unerupted teeth and malocclusion, in most cases, may be regarded as mere symptoms of failure of development of the dento-facial structures. Broadbent[1] noted : "Any buckling of the lower dental arch is commonly attributed to the so-called pressure of the erupting third molars. Factual evidence....would acquit the wisdom teeth of the afore-mentioned charges, and include them, along with the incisors, as co-sufferers from failure of the facial skeleton to attain its complete adult size and proportions." It follows that these factors which predispose to malocclusion, may also predispose to, or cause, non-eruption of teeth.

Textbooks of oral surgery and oral pathology tend to dismiss briefly, the aetiology of unerupted teeth. Some present a list of local and systemic factors, usually without amplification. Others lay emphasis on the oft-quoted theories of miscegenation, and "small jaws of modern man" without submitting these theories to critical analysis.

The cause of failure to erupt may be obvious in some cases - such as impaction of a tooth against a supernumary. A plausible, though unproven theory, may explain other cases - such as congenital displacement of a tooth germ to produce the aberrant tooth. The cause in the remaining cases may be far from obvious, and this is especially applicable to most third molar impactions. It is not sufficient to claim that lack of space is the cause of failure to erupt- lack of space is, after all, a symptom of some underlying anomaly. Likewise, failure of eruptive force is no satisfactory explanation -

many factors influence the physiology of eruption ; which of these factors is at fault ? The oral surgeon, and the orthodontist, if they are to be in a position to prevent future occurrences, and to treat effectively, cases of non-eruption of teeth, must be intimately acquainted with those factors which interfere with the attainment of growth potential of the dento-facial structures ; furthermore, they must understand why teeth may fail to erupt, even when the genetically predetermined growth potential has been attained.

It is important to understand that no one factor is responsible for lack of space in the jaws, but rather a combination of factors - genetic, evolutionary, metabolic, endocrine, nutritional and any other factor which will affect the growth and development of the cranial and dento-facial structures. Thus it is evident that only through a sound knowledge of the basic sciences, can one become cognizant of the aetiology of impacted, aberrant and unerupted teeth. Therefore, it is proposed to tackle the problem of aetiology, by consideration of the literature pertaining to :-

A. Growth and development of the cranial and dento-facial structures.

B. Embryology of the teeth.

C. The process and physiology of eruption.

D. Data concerning the dentition and facial development of ancient man and his forebears.

E. Data concerning dentition and facial development of man, living under primitive conditions.

F. The effects of specific developmental, nutritional and endocrine disturbances on growth.

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A. GROWTH AND DEVELOPMENT OF THE CRANIAL AND DENTO-FACIAL STRUCTURES.

1. Bone.

Bone is a living tissue, described as the most plastic, most adaptable tissue of the body, never static, but ever undergoing remodelling in adaptation to forces, functional or external. Besides its mechanical functions, it has the biological function of storing calcium and phosphorus. Any disease, endocrine or nutritional disturbance affecting the balance of calcium and phosphorus between bone and other body tissues and fluids, will produce a distortion of the growth pattern of bones which may, or may not, revert to normal if and when, the distorting factors have been removed. Weinmann and Sicher[3] repeatedly emphasised that "Bone is a tissue, a bone is an organ."

2. Growth of Bone.

"One of the most important advances in bone biology is the conclusive demonstration that there is no interstitial bone growth. Growth of bone always occurs by addition of new bone to free bone surfaces ; in other words, by appositional growth." [3]

3. Growth of Bones.

"It cannot be overemphasised that bones do not grow only by growth of bone." Weinmann and Sicher,[3] in support of this seemingly incongruous claim, illustrated it by the growth of the cranium which relies for its enlargement upon :-

(i) Cartilagenous growth (e.g., at the spheno-occipital synchondrosis).

(ii) Proliferation of connective tissue (seen at the sutures).

(iii) Surface apposition of bone.

It is necessary to understand clearly, the precise contribution to the growth of the cranium and dento-facial structures made by each of these processes. A condition such as achondroplasia, will interfere with the growth contributions made by the interstitial

growth of cartilage, sutural and appositional growth remaining unaffected. Osteogenesis imperfecta, a developmental disturbance of the skeleton, affects growth by retarding sutural and appositional bone deposition, growth by cartilage proliferation being unaffected. In each case, a distortion of growth pattern must result.

4. Growth of the Skull and Mandible.

Two dominant schools of thought on this subject have evolved, and voluminous contributions in support of the views of each are to be found in the literature. The American school of thought, formulated largely by Weinmann and Sicher[3] and followed by Strang, Salzman, and recently by Moore[4], is based on the belief that the sutures of the cranium and facial bones are active, primary growth sites, proliferation of sutural connective tissue with subsequent conversion to bone, constituting the growth force. Growth at the synchondroses by cartilage proliferation they claim to be of equal importance. The diametrically opposite view is held by Scott[5], who claims that expansion of the cranium is primarily due to enlargement of the brain, which separates the bones of the cranium, causing tension at the sutures ; in response to tension, the sutural width is restored by bone apposition to the bones concerned. Scott agrees on the role played by the synchondroses. Weinmann and Sicher attribute the growth of the maxillary complex, in its downward and forward thrust from the cranial base, to primary active growth of the facial sutures ; Scott considers that the enlargement of the eyeballs and growth of the cartilage of the nasal septum thrust the maxillary complex away from the cranial base, causing tension of the sutures, which react by bone deposition, sutural growth being passive. In my opinion, the views of Scott are more credible, for the following reasons :-

(i) The histology of the sutural connective tissue shows that there is a density of Sharpey's fibres inserting into the bone ends : this clearly reflects an adaptation to tension.

(ii) The high degree of co-ordination between cerebral and cranial growth, suggests that the latter is dependent on the former. In hydrocephalus, the cranial growth responds to the increased demands dictated

by cerebro-spinal fluid accumulation in the ventricles although in severe cases, there may be separation of the sutures. In microcephalus, the interdependence of cranial expansion and cerebral enlargement is clearly demonstrated.

5. Growth of the Cranial Base.

The facial skeleton is suspended from the anterior and inferior aspects of the cranial base. Growth of the facial skeleton is inseparably linked with, and dependent upon, growth of the cranial base.

Ford[6] stated : "It is generally accepted that cranial growth is largely dependent upon the growth of the brain, and therefore conforms to the neural pattern of growth (very rapid in the first 2-3 years, falling off rapidly and almost finished by 7-8 years). Facial growth, however, is independent of neural growth, and conforms to the general skeletal pattern shown by most bones and muscles, which grow fairly evenly from birth to adult life, with a characteristic spurt at adolescence. The cranial base, being the junctional region between the cranium and face, might be expected to show a growth rate intermediate between the neural and general skeletal rates." Ford found that the base as a whole did follow an intermediate pattern rate, but the individual growth sites showed either the neural or the skeletal growth rate. He also found that the cribriform plane, intimately connected with the frontal lobe, follows the neural rate and after the age of 7 years, is immutable. The nasion, so long regarded as a fixed point, moved upwards during growth, in relation to the cribriform plane.

Scott[7] indicates that the cranial base may be divided into three growth sites :-

(i) From basion to the anterior border of the pituitary fossa ; growth occurs by proliferation of the cartilage at the spheno-occipital synchondrosis, at the skeletal growth rate; the synchondrosis ossifies at 18 - 20 years ; growth at the synchondrosis, active for two decades, thrusts the facial skeleton forward in relation to the vertebral

column.

(ii) From the anterior border of the pituitary fossa to the foramen caecum : this site includes the cribriform plate, and growth occurs at the spheno-ethmoidal and spheno-frontal sutures ; growth at this site, neural in pattern, is completed at 7 years.

(iii) From the foramen caecum to the nasion : growth is of skeletal pattern, and relies on apposition to the anterior surface of the frontal bone, and is related to the pneumatisation of the frontal sinus.

The nasal septum at birth, is a cartilagenous plate, part of the nasal capsule, and is continuous above and behind with the cartilage of the cranial base ; its lower border is embraced by the bilaminar superior border of the vomer, which is braced against the hard palate, and its anterior portion articulates directly with the maxillae, from the vomer to the anterior nasal spine. From the cartilage of the nasal capsule, the ethmoid bones form. By the age of seven years, the cribriform plate, perpendicular plate of the ethmoid, and the vomer are fused, thereby fixing the facial skeleton in relation to the cranial base.

6. Growth of the Maxillary Complex.

Weinmann and Sicher[3] believe that the maxillary complex is thrust downward and forward from the cranial base by active growth at the "parallel" sutures (fronto-maxillary, zygomatico-maxillary, zygomatico-temporal, and the pterygo-palatine). The sutural growth school of thought now emphasises that the main centre of growth is at the fronto-maxillary sutures, and at the tuberosity. The tuberosity growth is believed to drive the maxillary complex forwards, by appositional growth to the tuberosity, which is braced against the pterygoid process (with intervention of the palatine bone).

Scott criticises this view on the following

grounds :-

(i) The apparent growth at the fronto-maxillary suture is assessed against the position of the nasion; the nasion, Ford[6] found, creeps up the frontal bone during growth.

(ii) The maxilla is grasped between two almost parallel sutures - the palatomaxillary and the zygomaticomaxillary - which lie in an almost antero-posterior plane. Growth at these sutures could not possibly drive the maxilla down and forwards ; Scott believes that accommodational growth at these sutures, permits the maxilla to slide between them, thrust by the nasal septal growth.

(iii) It is erroneously believed that the tuberosity is buttressed against the pterygoid process ; Diamond[8] pointed out that the posterior surface of the maxilla grows into the pterygopalatine fossa during childhood and adolescence, and does not become buttressed against the pterygoid process until adulthood. Furthermore the alveolar bulb[9] of the maxilla projects backwards on the outer side of the lateral plate and undergoes a process of alternating expansion and contraction, as the molar teeth develop within it, and move forwards into occlusion. Thereafter growth at the posterior border of the maxilla does not necessarily involve growth at the deeper palato-maxillary or palato-pterygoid sutures.

7. Growth of the Mandible.

The main growth centre is the condyle. It is covered, during its active growth period (up to 25 years) by cartilage, on the surface of which is a layer of fibrous connective tissue ; this acts as a perichondrium. Therefore the condyle exhibits both appositional and interstitial methods of growth.

Growth at the condyle is responsible for the overall increase in length of the mandible (glenion to gnathion) and increase in the height of the mandibular ramus. There is considerable appositional growth at the posterior border of the ramus : this, together with resorption of the anterior border of the ramus, also

controls the width of the ramus (which doubles from birth to adulthood) and determines the length of the body of the mandible. Apposition to the posterior border of the ramus also maintains the gonial angle constant, and effects a decrease in the condylar angle during growth.

The height of the ramus determines the height of the face. It provides the space into which not only the mandibular, but also the maxillary teeth erupt, and their alveolar processes grow.

The length of the alveolar process depends upon the vertical growth of its bone, the obliquity of the gonial angle, and the degree of prognathism or retrognathia of the alveolar process (Bjork[10]), and the extent of the resorption at the anterior border of the ramus. Resorption at the anterior border is not accepted by all authorities : some believe that the presence of osteoclastic activity at this site is part of the process of attachment of the temporal muscle to the coronoid process (Symons, 1953[7]).

Growth in width of the mandible is determined by apposition to its external surface, with resorption at its internal surface (that is, the thickness of the bone, not the intercondylar width). The backward divergence of the arch of the mandible ensures increase in intercondylar width, with increase in overall length. Growth in width is not possible after 9 years of age.

Scott[7] presented a novel view of the mechanism of condylar growth : "It is usually stated that growth of the condylar cartilage thrusts the mandible downward and forward from the glenoid fossae. It may be more accurate to say that growth of the cartilage permits of growth of the condyle upwards and backwards so as to maintain contact at the temporo-mandibular joint, as the mandible is carried downwards and forwards by the growth of the upper facial skeleton. The head of the condyle is very similar to the ends of the clavicles and it is more accurate to state that growth of the clavicles takes place to maintain contact with the sternum at one end and the scapula at the other, rather than say that growth of the clavicle thrusts the scapula away from the sternum." This implies that the mandible is not independ-

ent of the upper facial skeleton, and therefore the growth of the cranial base, upper facial skeleton, and the mandible is co-ordinated.

8. Facial Development as a Whole.

Krogman, referring to facial growth in general, estimated that "in the first 5 years, 78% of height, 85% of width, and 82% of depth have been achieved." It is difficult to understand how such specific assessments can be gained, but accepting their reasonable accuracy, Krogman's deduction is of interest ; "This means that after 5 years, only 15-20% of growth increments remain as avenues of possible adjustments."

Recent work by Symons (1951) [7] and Dixon (1954) [7] on the mandible and maxilla, indicates that these bones are built up of quite distinct developmental parts. These are the neural, alveolar, ramal and muscular processes of the mandible, and the neural, alveolar, zygomatic and palatal processes of the maxilla. The independent origin of these parts increases the probability of a gene-regulated developmental existance.

Wylie states that"there is a growing conviction that there is no such single entity as a normal facial pattern, and that dento-facial anomalies are in a large measure occasioned by a random combination of facial parts, no one of which is abnormal when taken by itself, but each one of which may fit badly with the other parts to produce a condition which may be called dysplasia."

Stockard,[12] crossed the long-jawed Saluki with the broad-jawed Basset hound, and found that the offspring showed the inheritance of the maxilla from one parent, and the mandible from the other. "Genetically, there is evidence that the growth of the upper face is under the control of factors other than those which control the growth of the lower jaw."[12]

9. Alveolar Bone.

It was formerly thought that the growth of the jaws is dependent upon the development of the teeth. Certainly alveolar bone growth is dependent upon the presence of teeth, and particularly upon their eruption; that the alveolar bone growth is dependent on the presence of teeth, is seen in congenital anodontia : the skeletal development of the jaws is usually within normal limits, while the alveolar process remains rudimentary. Alveolar bone disappears on loss of the teeth, by disuse atrophy.[13]

Alveolar bone develops in the first place as a protective capsule around the crown portions of the developing teeth. It gains attachment to the teeth only they erupt, and form a root portion. Alveolar bone plays little part in the eruptive force of teeth.

As the growth in height of the ramus thrusts the jaws apart, alveolar growth is necessary to maintain the teeth in occlusion. However, teeth do not require the stimulus of occlusion to erupt, as eruption continues after loss of the antagonists. Growth of the alveolar bone is a process quite distinct from that of tooth eruption : growth of the alveolar process carries with it the teeth, whether they are developing in their crypts, erupting, or are in post-eruptive occlusion ; it is well seen in ankylosis of a tooth, the developing alveolar process growing upward to engulf the stationary tooth.

There appears, however, to be some genetic factor associated with alveolar growth. Alveolar growth may fail, in length and width, to produce constricted arches, closed bite, and to produce crowding and impaction of teeth, with retarded eruption, for no apparent reason.

Growth in length, width and height of the alveolar process occurs as a result of surface deposition of bone upon its buccal surface, with resorption of bone on the lingual surface. As Scott[12] points out, this growth of alveolar bone is to a large extent, independent of the basal bone of the jaws, as is readily seen in the

chinless anthropoids and in the early human species. Bjork[10] drew attention to the importance of alveolar prognathism in the lengthening of the alveolar processes.

A great deal of controversy exists concerning the mechanism whereby the alveolar arches increase in length and width. It was formerly believed that the teeth remained fixed in the arches after eruption, and that space for the permanent molars had to be provided entirely by addition of alveolar bone at the back of the jaws. It was believed that the jaw elongated between 3 and 18 years. Scott[12] postulated that the forward and outward migration of the teeth with their alveolar bone takes place, and that there is no significant growth at the back of the jaws after the third year. Symons[7], as previously mentioned, asserted that resorption at the anterior border of the ramus is associated with the attachment of the temporal muscle, and not with alveolar arch lengthening. The ramus doubles its breadth from 3 to 18 years, and concurrently augments the body length to the same extent. It will be seen that function stimulates the forward and outward migration of teeth and their alveolar bone, and in the human, the full expression of this process is probably to be found only in man living on a primitive diet - such as the Australian aborigine and the Eskimo.

The function of the "alveolar bulb" relies upon the forward migration of the dentition. An understanding of the bulb is necessary to the explanation of third molar impaction.

10. <u>The Alveolar Bulb</u>.

The alveolar bulb (first described by Brash, in 1924), and later by Scott[9], is a bone-encapsulated cavity which is part of the alveolar process. In the upper jaw it projects backwards beneath the orbital cavity, and in the lower jaw, it occupies the front of the mandibular ramus. In man, the bulb contains only one calcified molar crown at a time, whereas in the pig, manatee, and the kangaroo, it attains a massive size, and may contain three calcifying

molars at varying stages of development. In the lower jaw of man, the bulb is the crypt of a molar. At first the crypt contains the calcifying crown of the first molar, and the germ of the second molar. Later, as the first molar moves forward, the back part of its crypt becomes divided off as a separate compartment which becomes the crypt of the second molar and contains the germ of the third molar. The cavity of the bulb in man opensin front of the ramus at the base of the coronoid process. At this opening, the gubernacular cord becomes continuous with the submucosa overlying the retromolar triangle. The bulb in the maxilla functions similarly ; it projects into the pterygomaxillary fossa, expanding and contracting periodically as one molar is drawn from it and the succeeding molar commences its development. Scott[9] stated that :

> "There can be little doubt that the teeth which develop in the alveolar bulb migrate forward and that this migration forward.... is associated with a forward migration of the whole dentition. This migration is not brought about by the pressure of eruption of the most distal of the molar series ; this would not explain the forward migration of the last of the series, the third molar. It is not brought about by the growth movement of the interalveolar septa as a result of mesial deposition and distal resorption of bone. Such a reconstruction of the socket does, however, take place as the teeth migrate. The movement of migration is not just a simple forward movement. In the upper jaw, it is forwards, downwards, and especially in man, outwards. In the lower jaw, it is forwards, upwards and outwards."

Scott[9] pointed out that the developing molar teeth in the alveolar bulb are attached to the submucosa of the retromolar region, by the molar gubernaculum ; as the alveolar bone grows by deposition of bone by the periosteum of the gum " the teeth are drawn forwards out of the bulbs, as a bucket is drawn out of a well. "

11. <u>Tooth Development and Facial Growth</u>.

It has been suggested that there is ready evidence of the independence of the ontogenetic development of the teeth from

that of the jaws. This is reflected in such phrases as "apical base deficiency", Ludstrom's term for lack of correlation between the jaw size and tooth size, and "large teeth of one parent, with small jaws of the other". The work of Stockard on hybridisation of dogs clearly proved the independence of development of teeth and facial growth. Fleming,[12] in a study of Anglo-Negro hybdrid offspring, found cases in which one jaw showed Negro characteristics of wide dental arch, large teeth and no crowding, while the other jaw showed smaller teeth which were crowded in a deformed arch. Nodine[14] considered that the high incidence of impacted third molars found in modern European races may be partly attributed to hybridisation, and expressed the view that :

> "A great deal of human malocclusion is probably the consequence of the continual intermingling and intermarriage within historical times of what were isolated inbreeding communities in prehistoric times."

Cauhepe[15] claimed that most characteristics of the teeth are dependent upon heredity. Their number is specific in each species, and if occasional variation occurs, such as missing third molars or maxillary lateral incisors, these variations, it may be shown in many cases, are transmitted as dominant characters. Form, and the dimensions of the teeth are determined by heredity, and they cannot be modified by environmental factors because they are fixed too early by calcification. With Cauhepe, I cannot agree in full, on the absence of environmental influences : Hutchinson's incisors are ready examples of environmental effects upon the morphology of the teeth ; Miles[16] reported that maternal rubella at an early stage of pregnancy, can cause dental malformations, retarded eruption, and the absence of teeth.

12. <u>Function in Relation to the Growth and Development of the Jaws</u>.

The following points taken from the textbooks of Strang[1] and Salzman,[2] with regard to function, will be mentioned :-

(1). The growth pattern and the growth potential of the skeletal bases of the jaws are determined genetically ; the view that function-

al stresses are of great influence in producing structural enlargement and perfection is no longer held.

(ii) It follows that the genetically determined pattern allows for adequate strength of design to meet anticipated functional demands; the internal strength of the jaws, by experiment, is able to withstand forces eighty to ninety times as great as the maximum functional stress.

(iii) Function is a necessary adjunct to growth : it aids in the attainment of growth potential through a stimulation of blood supply ; myofunctional exercises cannot stimulate growth in excess of the genetically determined potential ; hypofunction, will, however, predispose to underdevelopment of the jaws.

(iv) Function modifies the internal structure of the jaws (number, strength and trajectoral arrangement of the trabeculae) and muscle attachment through heavy function, increase the ruggedness of outline.

The distinction between alveolar bone and skeletal or basal bone is important : the latter is of genetic determination, whereas the alveolar bone is solely dependent for its existence upon the development and eruption of teeth. Alveolar bone is limited by basal bone development, and functional stresses play an essential role in the development of the alveolar process. Brodie[1] states that "the teeth and alveolar process should be looked upon as passive though responsive victims of a continuous interplay of muscular forces, their positions dictated by the resultant of these forces....." The forces mentioned by Brodie may be grouped as follows :-

(i) The <u>posterior component of force</u> .- The dental arches are acted upon by the tension produced by a ring of muscle, viz, the muscle band formed by the orbicularis oris, buccinator, and the superior constrictor of the pharynx ; this band on each side, is attached to the cranial base through the constrictor muscle. The teeth and their plastic supporting alveolar process are prevented from drifting forward in relation to the cranial base by this muscular band. To the orbicularis oris are attached the other facial muscles, all of which

pull against the anterior section and "cheek" teeth of the dental arches. The supra-hyoid muscles pull backwards on the mandible, when the hyoid bone is fixed.

2. The Anterior Compenent of Force.- To counterbalance the posterior component, is the relatively feeble pull of the anterior fibres of the temporal muscle, the superficial fibres of the masseter muscle, the buccinator as it wraps around the tuberosity and the distal molars, and the pterygoid muscles. Yet the most powerful forward propelling force is that exerted upon the teeth in mastication. Strang[1] defined the anterior component of force as " a powerful forward propelling force associated with the inclined plane action and arising from the normal mesial axial inclination of the buccal teeth....." This force is directly proportional to the masticatory forces exerted, and is dependent, for its fullest effects, upon the integrity of the dental arch (i.e., the absence of extracted teeth). It is this powerful component of force which drives the teeth of the dentition in their forward migration, which is partly responsible for the alveolar prognathism that lengthens the arches, in preparation for the eruption of the molar teeth of the permanent dentition.

However, just as sound ample development of the alveolar process is aided by the normal functioning of these muscular forces, so may deformity of the alveolar process, with attendant crowding, malalignment and impaction of the teeth occur, in perverted functioning of the same muscles. Mouthbreathing, hypotonicity, thumbsucking result in protrusion of the maxillary teeth and maxillary alveolar process ; muscle tics, abnormal swallowing habits and hypertonicity limit the lateral dimension of the arches.

B. EMBRYOLOGY OF THE TEETH AND THE DEVELOPMENT OF OCCLUSION.

1. The Dental Lamina.

The dental lamina develops during the sixth week of embryonic life, as an epithelial thickening near the free margins of the jaws. It is continuous, and runs archwise along the crest of the ridge. From the dental lamina arise the ten tooth buds of the deciduous teeth of each jaw. From the lingual aspect, the secondary lamina arises, which gives rise to the tooth buds of the permanent successors of the deciduous teeth. The origin of the permanent molars differs from that of the other permanent teeth. They are the only permanent teeth which arise directly from the dental lamina. At the seventeenth week in utero, the dental lamina extends distally, and gives rise to the tooth germ of the first permanent molar. With further growth of the jaws, the dental lamina extends distally back from the enamel organ of the first molar to give off the bud of the second, and at the age of 4 years, the bud of the third molar.

The dental lamina finally becomes fenestrated, and resorbed. Remnants of the dental lamina persist as epithelial isles, and may differentiate into enamel masses and supernumary teeth.[18]

2. Abnormalities of the Dental Lamina.

Redundancy of the dental lamina may produce supernumary and supplemental teeth, complex and compound composite odontomas. Dentinomas, the exact mechanism of origin of which is not understood,[17] also probably arise from an aberration of the lamina. The composite odontomas form in place of a normal or supernumary tooth, grow to a considerable size in many cases, causing the impaction of one or several teeth. The dentinoma is always associated with a normal tooth, and, lying to the occlusal of that tooth, causes its impaction.

Abnormalities of tooth size may occur. When a tooth is larger than normal, its eruption into occlusion is prejudiced and the eruption of adjacent teeth may be secondarily impeded.

Megadontia, of rare occurrence, is nevertheless an important predisposing factor to impaction, especially as the jaw size and tooth size appear to develop independently.

Abnormality of form is seen in geminated teeth, the dilated odontoma, and dilaceration of the root.

Cystic degeneration of the enamel epithelium of the developing tooth germ may result in a primordial cyst (occurring before, and precluding enamel formation). Cystic degeneration of the reduced enamel epithelium following enamel completion, gives rise to the dentigerous cyst. The adamantinoma, of controversial origin, is generally considered to be a neoplasm arising from epithelium capable of forming enamel : by its expansion within the jaws, it may displace and impact, erupting teeth.

It should be remembered that the dental lamina may fail to form a tooth bud, or the tooth bud, if formed, may fail to develop. The affected tooth is termed congenitally missing. Teeth most frequently suppressed are the maxillary lateral incisor, the third molars, and the mandibular second premolar.

3. <u>The Aberrant Tooth Germ</u>.

When a developing tooth follows a path of eruption well outside that of normal deviation for the tooth concerned, it is presumed to have developed from an abnormally located tooth germ, or from a tooth germ with an abnormally orientated axis.[14, 20] Nodine[14] attributed abnormality of location of the tooth germ to redundancy of the dental lamina. He attributed abnormality of axis to pressure of unknown origin, brought to bear upon the tooth germ, at a very early stage of its development ; he drew attention to the embryology of the dental lamina : "prior to the complete encapsulation of the tooth germ by the growing follicular wall, and for some time after the completion of this wall, the deciduous tooth follicles are found swinging in a membranous trough....filled with loose semi-gelatinous mesoderm

which completely surrounds the follicles." That abnormal pressures to the tooth germ in this early developmental stage do occur, and do produce abnormality, is supported by the suspected aetiology of geminated teeth, which many feel, arise from coallescence of two adjacent tooth germs, the extent of gemination depending upon the time of occurrence of pressure.[13] Apart from Nodine's hypothesis, I have found no other reference to its aetiology. The teeth most frequently affected, are the maxillary canine, the mandibular second premolar, and the third molars.

4. <u>The Development of Occlusion</u>.

Schour, by staining methods, was responsible for the first accurate data on the development of the teeth. Broadbent, by means of the standardised cephalometric radiographic technique, produced the first accurate and scientific data on the process of tooth eruption and the development of occlusion.

"At birth, the jaws contain the partially calcified crowns of twenty deciduous teeth and the clearly defined crypts of the first permanent molars.. They occupy the lower half of the upper jaw and the upper half of the lower jaw." It should be noted that the body of the maxillae is occupied almost entirely by the developing teeth, the antrum at this stage being rudimentary only. The mandibular first molar crypt lies not within the body of the mandible, but in the confines of the ramus, high above the level of the occlusal surfaces of the deciduous teeth. The maxillary first permanent molar crypt lies high up in the tuberosity, below the floor of the orbit. With subsequent jaw growth (and mesial migration of the dentition), the location of the permanent first molar crypt changes. Each successive molar occupies a similar position.

"Monthly registrations of the same child indicate that the eruption in its broadest sense, begins with the appearance of the crypt in the bone, and includes the migration of this crypt through the bone even before calcification is detected by the

roentgenogram....."[1]

Logan and Kronfeld[20] mentioned that : "the germs of the permanent incisors, permanent canines, and bicuspids are originally placed on the occlusal side of the deciduous teeth. In the course of the growth of the maxillary ridge, and during the occlusal movement of the deciduous teeth, the permanent tooth germs are left behind. Thus, they become located on the lingual side of the deciduous teeth, and at the same time, are surrounded and encapsulated by the lingual portion of the bone. As the deciduous teeth move occlusally, the permanent tooth germs finally assume a position above or between the roots of the deciduous teeth."....." At birth, the permanent central incisors, lateral incisors, and cuspids have already reached a position lingual to the deciduous teeth, and the bicuspids are still on the occlusal side of the corresponding deciduous molar teeth."

At about one year, the permanent canines, when they begin to calcify, lie between the roots of the deciduous first molar, a position later to be occupied by the first premolar. As the deciduous teeth move toward the occlusal plane, the permanent incisor and canine crypts, including their partially developed crowns, migrate forward at a greater rate than the forward movement of the deciduous teeth themselves. Thus, the permanent canine eventually lies apical to the deciduous canine root by 2½ years of age. With the growth of the deciduous root, the canine lies to the lingual of its apical portion. With later resorption of the deciduous root, the canine follows in its wake, wedging its way between the erupted lateral incisor and first bicuspid.

Thus it is seen that the permanent canine must pursue a devious route before its eventual eruption. Its early lingual alignment explains why palatal impactions are more frequently met than buccal impactions. Transposition of the canine with the first premolar is not uncommonly met, and, less commonly, with the lateral incisor. However, regardless of its devious course, the permanent maxillary canine is impacted mainly on account of the sequence of

eruption of the permanent teeth. Nodine[14] believed that the canine, occupying the corner of the arch, is more exposed to developmental pressure, favouring aberration of its axis ; aberrant canines are of frequent occurrence.

The first permanent molar has, by the third year, migrated downward and forwards from its high position in the ramus, into the body of the mandible ; its occlusal surface, prior to eruption, is directed mesially and lingually ; the lengthening of the alveolar arch permits its rotation and vertical eruption. The maxillary first molar, likewise migrates, until it lies in the tuberosity, its occlusal surface directed buccally and distally. Growth of the jaws permits it to erupt vertically.

5. <u>The First Molar Relationship</u>.

In civilised man, the first permanent molars meet end to end on eruption. The maxillary second deciduous molar is almost equal in width to the second pre-molar, and the eruption of the latter does not incur a mesial drift of the first molar. However, the second mandibular deciduous molar is wider mesiodistally, than its successor, and the eruption of the second premolar permits a mesial drift of the first molar, thereby establishing the normal mesial interdigitation of the mandibular first molar with its antagonist. In the Australian aborigine, abrasion of the deciduous teeth permits the mesial shift of the mandibular teeth into an edge-to-edge relationship, and permits the first permanent molars to erupt initially into normal cuspal interdigitation, according to Begg.[21]

Thus, it is most important that the deciduous molars are maintained in functional occlusion until they are shed normally, for they guide the first permanent molars into correct occlusion. Premature loss of the deciduous molars, in permitting unrestrained mesial drift of the first permanent molars, results in a loss of space for the eruption of the premolars, and consequently predisposes to impaction.

C. THE PROCESS AND PHYSIOLOGY OF ERUPTION.

1. Current Theories of Eruption Force.

Several theories have been expounded to explain this perplexing mechanism, among which are the following :-

(i) Alveolar bone growth.- Brash and others have ascribed the most important role in eruption to the growth of the jaws, and regard the teeth as passive agents in the process of eruption. It would not serve to explain the eruption, against the direction of jaw growth, of inverted teeth. It must be conceded that jaw growth is essential to normal eruption of teeth into functional occlusion, but evidence suggests an inherent active movement of the teeth.

(ii) Elongation of the root.- The simplest explanation would be active pressure by the elongation root against the resistant floor of the bony crypt, to produce a peripheral movement. Teeth may erupt in the absence of root formation, or despite normal root formation, may remain unerupted. Broadbent[1] noted that : "Unlike the usual description of tooth eruption by apposition at the root end that is believed to force the crowns toward the plane of occlusion, our studies on the living children clearly disclose that a tooth at various times, may progress in three distinctly different ways : first, the growing tooth may remain stationary while its forming end grows away from the incisal or occlusal surface of the bone ; second, at another time, it may migrate relatively rapidly through the bone with little increase in its length ; third, the increase in tooth length and the migration through the bone may occur simultaneously."

(iii) Pulpal pressure theory.- Constant[18] suggested that blood pressure is the impelling force in eruption, pointing out that the vascular supply of the pulp and tissues beneath the teeth is greater than above it.

(iv) Cellular proliferation theory.- Noyes[18] accounted for eruption as follows : "The force exerted by the growing tooth is the

result of the multiplication of cells in the tooth germ, and is exactly comparable to the forces exerted by the multiplication of cells in any position." He compared the forces of eruption to the force of a rootlet which can cleave concrete by osmotic pressure.

(v) Pulpal constriction theory.- The constriction of the pulp, by the ingrowing of the apical portion of the root, exerts the eruptive force.

2. The Research of Bryer (1957)[22]

Bryer (1957), in his essay entitled "The Physiology of Tooth Eruption", very considerably elucidated the mechanism of tooth eruption. A brief summary of his findings is considered pertinent to the study of impacted and unerupted teeth.

The basis of past experimental work on the eruption of teeth, using the continuously erupting lower incisors of rats in functional occlusion, is fallacious. The rate of eruption of incisors in occlusion, is only as rapid as attrition permits. Bryer substituted the non-functioning incisor, by grinding it out of occlusion, and was therefore able to calculate the "unimpeded eruption rate" of the tooth, when the rat was subjected to various dietary, circulatory, surgical and endocrine disturbances. He correlated the unimpeded eruption rate with histological evidence, and clinical and radiographical findings. He made the following observations :-

(i) Acute protein deficiency.- This exerts very little effect. As oedema sets in, there is a change in tissue tendion, and eruption is accelerated. As the rat becomes moribund, the eruption rate diminishes.

(ii) Rickets.- A profound fall in eruption rate is noted. Osteoid tissue accumulates, which should, on the alveolar bone growth theory, cause an increase in eruption rate. A fall in vascularity of the pulpal and periodontal tissues is noted.

(iii) Calcium and phosphate decrease.- Amelogenesis and dentinogenesis is inhibited, and the eruption rate remains unchanged.

(iv) Hypervitaminosis D.- In toxic doses, vitamin D causes a fall in the eruption rate, and hypotension.

(v) Vitamin A deficiency.- A severe fall in eruption rate is produced, which is restored to normal on administration of the vitamin. Bryer noticed a fall in vascularity and pavementing of leucocytes.

(vi) Cobalt subcutaneously injected.- Polycythaemia is produced. Eruption rate was found to fall by 5-7%. Increased viscocity, a sluggish flow of blood, and a reduction in tissue tension is evident.

(vii) Fluoride.- Increase in fluoride consumption markedly disturbs the process of amelogenesis and dentinogenesis ; the eruption rate and vascularity remain unchanged.

(viii) Circulatory disturbances.- Ressection of the inferior dental nerve in one group, and removal of the superior cervical ganglion in the other group, was carried out. Removal of the vascular tone produces an eruption rate so rapid, that the root cannot "keep pace", and trails its odontogenic epithelium.

(ix) Pulpectomy.- The disturbance to the vascularity so produced, causes a marked fall in eruption rate with buckling of the root ; Rushton noted the same buckling of the roots in cases of cleido-cranial dysostosis.

(x) Excision of the formative end of the root.- Root elongation was prevented. After a slight retardation of eruption rate, the rate is augmented, by the inflammatory response to surgery.

Bryer came to the following conclusions :-

(a) If the alveolar bone growth theory is correct, vitamin A and D deficiency, by increasing the alveolar bone or osteoid tissue formation, should increase the eruption rate. Instead a fall was noted, and that buckling of the root occurred, indicates that root elongation per se, does not cause eruption.

(b) The pulpal constriction theory, which concerns dentinogenesis, is untenable, as a major contribution at least, since fluorides, acute

protein deficiency, did not disturb the eruption rate, even though dentinogenesis was inhibited.

(c) The cellular proliferation theory may play a subsidiary role.

(d) All evidence supports the vascular theory, with blood pressure the eruptive force.

Bryer extended his studies of eruption physiology to include the influence of the endocrine glands. At the time of publication of his essay, the clinical observations had not been correlated with histological and radiographical findings ; he felt justified, however, in making the following predictions :-

(a) Chemical and surgical thyroidectomy.- A fall of 37% in eruption rate is induced; normal eruption rate may readily be restored by the injection of thyroxine.

(b) Parathyroidectomy.- No alteration in eruption rate is induced, despite inhibited dentinogenesis. The injection of parathormone does not affect the eruption rate.

(c) Hypophysectomy.- A severe fall in eruption rate is produced over a period of 9 months. The fall in rate is more severe if ACTH or thyroxine is also lacking. An increase occurs if ACTH or thyroxine is restored, but only by the restoration of all three hormones, is eruption rate normal.

(d) A "tumour" of the hypophysis.- Growth hormone injections cause a marked increase in eruption rate.

3. The Process of Eruption.

It is well known that eruption may be active, or passive. Passive eruption - the apical migration of the epithelial attachment in the erupted tooth - is not considered germane to the study of impacted and unerupted teeth.

Active eruption occurs when the tooth passes from

its pre-eruptive location, to its final position in functional occlusion. It does not cease with the attainment of occlusion : attrition reduces the substance of the occlusal , incisal and interproximal surfaces of the tooth, and eruption continues to maintain the teeth in occlusion at the established "vertical dimension". Active eruption may therefore be divided into three phases : intra-alveolar, intra-oral, and continuous eruption.

Active eruption of a tooth from its site of development to functional occlusion, depends upon the normal location of the tooth germ, its normally orientated axis, normal morphology of its crown and root, normal eruptive force, a path of eruption free of impediments, and adequate space for its accommodation in the dental arch.

4. Factors Interfering with Normal Eruption.

(i) Abnormal location or abnormal axis of the tooth germ.- Should the tooth germ be located in an abnormal site, or should its axis be abnormally aligned, the developing tooth must necessarily pursue an aberrant path of eruption. The extreme degree of abnormality of axis is seen in the inverted tooth ; a lesser degree is evident in the transverse buccal tooth. A tooth may be found at a considerable distance from its normal developmental location : the mandibular third molar has been found in the neck of the condyle.

(ii) Abnormal morphology. Mechanical interference with eruption may occur through abnormality of shape. In congenital conditions such as abnormal crown size, true gemination, dilated odontoma, and in acquired conditions such as dilaceration of the root, impaction or failure to erupt is the usual sequella.

(iii) Failure of the eruptive force.- Failure of the eruptive force generally results in multiple unerupted teeth, and the causes have already been discussed (vide supra).

(iv) Obstruction of the path of eruption.- This may be caused by

(a) 'foreign bodies' - supernumary teeth, retained roots, tumours, cysts ;

(b) over-retention of the deciduous tooth ;

(c) ankylosis of a deciduous molar ;

(d) premature loss of deciduous predecessor to produce a bone and fibrous scar ;

(e) the dentigerous and eruption cyst.

(v) Lack of space in the dental arch for eruption.- This condition may result in tooth impaction, bone impaction, or gum impaction; it arises from

(a) basal bone deficiency;

(b) alveolar arch deficiency ;

(c) an inherited predominance of tooth substance over jaw size ;

and (d) a lack of timing between eruption time and jaw growth.

5. The Mechanism of Eruption of Surgically Exposed Teeth.

Teeth long past their normal eruption time, may erupt on removal of the obstruction or cause preventing their eruption. The technique of inducing eruption of embedded teeth is termed surgical exposure; it involves the freeing of the crown of overlying bone and the elimination of any impediment (such as a supernumary tooth, or fibrous scar tissue), in addition to which, the unerupted tooth is "wiggled". Orthodontic traction may, or may not be required.

The reason why "wiggling" stimulates eruption is not understood and a search of dental literature failed to reveal the publication of a discussion of the possible mechanism. Bryer's evidence would suggest that the inflammatory response of the pulpal and periodontal tissues to trauma, may accelerate eruption by an increased local blood pressure and tissue tension.

It is a commonly held opinion that an embedded tooth

is stimulated to eruption by an overlying artificial denture. It seems reasonable to believe that, in elderly persons at least, the tooth is exposed by alveolar resorption and gingival ulceration. Active eruption is precluded, when the embedded tooth is ankylosed.

6. The Sequence of Eruption.

The eruption times of the permanent teeth are given by Schour in the following form :-

First molars.....5 - 7 years
Central incisors.....6 - 8 years
Lateral incisors.....7 - 9 years
First premolars.....8 -10 years
Mandibular canines.....9 -11 years
Second premolars.....10 -12 years
Maxillary canines.....11 -13 years
Second molars.....12 -14 years
Third molars.....17 years and any time later

From this chart, the sequence of eruption for the maxillary teeth, may be tabulated as follows :-

6 1 2 4 5 3 7 8

It is noted that only the maxillary canine and the maxillary second premolar erupt between two permanent teeth. The central incisors rarely experience difficulties in erupting, unless their path of eruption is obstructed by either a supernumary tooth or an odontoma.

The first molars are guided into normal eruption by the deciduous dentition. Should there be a lack of co-ordination between their eruption times and jaw development, the first permanent molar may become impacted against the deciduous second molar.

Should lack of space interfere with the eruption of the lateral incisor (e.g., a midline diastema), it will impinge upon the root of the deciduous canine. The permanent canine, which erupts four years later, cannot prevent the distal eruption of the

lateral incisor, and the root of the deciduous canine is resorbed, and the tooth exfoliated prematurely. The lateral incisor may thereby erupt in good alignment by usurping the space intended for the canine.

The first premolar usually erupts without difficulty, assuming normal resorption of its predecessor. If, however, the first deciduous molar has been prematurely lost by caries, the forward drift of the teeth distal (the first permanent molar and the second deciduous molar) will cause mesial eruption of the first premolar, with resorption of the deciduous canine, or, if it has already been shed, into part of the space intended for the permanent canine. Impaction of the first premolar is uncommon.

The second premolar must erupt between the first permanent molar, and the first premolar. If the second deciduous molar has been prematurely lost by caries, the first permanent molar drifts mesially, and the second premolar suffers space deficiency - it either erupts palatally, or remains vertically impacted between the adjacent permanent teeth.

The maxillary canine erupts between the already erupted lateral incisor and the first premolar. It has already been shown that its space in the arch may be usurped by the adjacent teeth. Even if the deciduous canine has not been prematurely lost, any failure of jaw development will adversely affect the eruption of the canine.

The second molar, erupting distal to the first molar, at a stage when a few years of jaw growth still remain, rarely becomes impacted or malposed.

The eruption sequence of the mandibular teeth may be tabulated as follows :-

6 1 2 3 4 5 7 8

In the normal sequence, only the mandibular second premolar must find accommodation between two already erupted permanent teeth. It is not surprising that this tooth suffers space deficiency

and therefore impaction and maleruption more frequently than any other mandibular tooth with the exception of the third molar. Second deciduous molar loss by caries is usually disastrous for the second premolar - rapid mesial migration and tilt of the first permanent molar follows, and results in irrevocable space deficiency for the second premolar.

The mandibular canine is impacted less frequently than the second premolar, and the maxillary canine. As the mandibular canine erupts in advance of the first premolar, space deficiency is transferred distally, by usurpation of the first premolar space. The first premolar, however, manages to erupt normally, despite slight distal eruption of the canine, by usurping part of the second premolar space. In addition, the mandibular incisors are weak teeth, and are unable to deflect the mandibular canine, when space is lacking : the mandibular incisors frequently are overlapped.

It does happen occasionally that the normal sequence of eruption is not pursued. With eruption of the mandibular first premolar in advance of the canine, the latter may become buccally deflected, and erupts into a buccal malposition. Impaction of the mandibular canine by space deficiency rarely occurs.

Nodine[14] listed the order of frequency of impaction as follows :-

i. mandibular third molar
ii. maxillary third molar
iii. maxillary canine
iv. mandibular second premolar
v. maxillary central incisor
vi. mandibular canine
vii. maxillary second premolar
viii. mandibular first premolar

It is not surprising to find that the mandibular and maxillary third molars show a higher frequency of impaction than the other teeth. They are the last teeth to erupt, and usually eruption takes place after jaw development is complete. Therefore they are acutely affected by all factors leading to lack of jaw development.

Prevention of impaction of the maxillary canine,

and the second premolars may be effected by the serial extraction of teeth. The principles guiding this practice have been described by Dewel,[23] Hotz,[24] Kjellgren[25] and others. The technique is based upon the predictions which may be made through knowledge of the eruption sequence in relation to the incidence of impaction.

Lo and Moyers[26] found that

(i) the most frequently occurring sequence of eruption seen in the maxillae, is

6 1 2 4 5 3 7 8 ;

(ii) the most frequently occurring sequence of eruption seen in the mandible is

6 1 2 3 4 5 7 8 ;

(iii) a combination of these eruption sequences provides the greatest incidence of normal occlusion ;

(iv) in the maxilla, the most unfavourable eruption sequence is seen when the second molar erupts earlier than either the premolars or the cuspid ;

(v) in the mandible, the most unfavourable sequence of eruption is seen when the canine erupts later than the premolars, and when the second molar erupts before either the premolars or the canine; and

(vi) there is a prognostic value in studying the sequence of eruption in radiographs prior to the loss of the deciduous teeth.

Begg[21], from his studies in malocclusion found in the Australian aborigine and the Caucasian, observed that :

"There is not complete harmony and synchronisation of growth, development and times of eruption of teeth with the development and increase in size of the jaws. For example, crowding of the anteriors may be found in jaws with more than sufficient space posteriorly for eruption of the third molars. Such cases may be due to hereditary early eruption or

late jaw development, rather than to size disparity of tooth and bone. Again, all teeth may be in textbook normal occlusion, except that the third molars are impacted."

D. THE DENTO-FACIAL DEVELOPMENT AND DENTITIONS OF PRIMITIVE AND ANCIENT MAN AND HIS FOREBEARS.

1. The Evolution of Man.

It is generally believed that present day man, Homo sapiens, the only surviving representative of the genera Hominidae, has emerged as follows : lower ape, man-like ape, fossil ape-man, and Homo sapiens (Huxley[27]).

The terms "higher" and "lower" races have very little meaning.[27] Present day human beings, regardless of their different physical characteristics, have a common ancestry, belong to the one species, and are mutually fertile. The emergence of numerous major ethnic groups, and innumerable sub-groups, showing distinctive physical variations, such as colour of skin, type , colour and texture of hair, nostril characteristics etc., is explained by Huxley[27] as follows :-

"The differentiation of the main groups from the stem of the species could only have occurred in areas in which, over a more or less prolonged period of time, they were to a considerable degree isolated from the rest of the human stock. Only so could inbreeding and selection have brought about markedly divergent and relatively pure racial types."

Thus we hear of the term "inbreeding isolates",[28] referring to groups which have acquired distinctive characteristics, which have become features of that group. The greater number of sweat glands in the Negro, and the reduction of their numbers in the yellow skinned peoples, are probably adaptations to hot and dry conditions respectively. A white skin is a disadvantage in the Tropics, a wide nostril in the Arctic.

Bjork (1950)[29] studied the dento-facial development of the baboon, the gorilla (man-like ape), Pleisianthropus (fossil ape-man), and Homo sapiens (as an example of which he chose the Bantu), by submitting a skull of each to cephalometric radiographic analysis. By this means, he was able to study the developmental changes from an evolutionary point of view. He summarised his findings as follows :-

(i) "Increasing brain volume is accompanied by a corresponding increase in size of the brain case. The forehead is prominent."

(ii) "The shape of the skull suffers a gradual change, brought about by a deflection of the cranial base, which in the lower vertebrates, consists of more or less a flat plate, such that the rear part of the cranial base is swung progressively forward in relation to the forward horizontal portion. This occurs with the increasingly upright stance of the body, with a progressively forward displacement of the spinal column on the underside of the skull. In the human skull, the two parts of the cranial base form a fairly acute angle."

(iii) "The degree of prognathism diminishes, so that the snout is progressively retracted. This diminution is partly connected with the increasing breadth of the skull and consequently of the jaws, and partly with a shortening jaw length."

As the skull becomes broader, the condyles move apart, to produce jawswhich are broader and therefore less prominent. The shortening of the jaws affects the alveolar portion more than the basal portion of the arches. This makes the chin more prominent than ~~than~~ the mid-facial profile, the profile becomes more orthognathic, and the incisors more vertical, with the teeth more closely aligned. Crowding and impaction of teeth have been reported in all ethnic groups to varying extents. Different ethnic groups show different degrees of prognathism, alveolar and basal. Within any one ethnic group, there are considerable individual variations within "normal" limits.

Evolutionary changes may explain the development of

many physical characteristics, but it must be remembered that evolutionary changes in the vertebrates occur over a period of time, measured not in decades, but in tens of thousands of years. Evolution by no means explains the rapid changes found in people who depart from their primitive mode of life, to what is known as the civilised mode of life. Numerous articles in the literature describe the remarkable changes in the occlusion and alignment of teeth found when members of an ethnic group depart from the primitive way of life of their ancestors, and assume the customs of the European.[14, 30, 31] Barrett[32] described the regular alignment of teeth in "continuously developing functional occlusion" of the "bush" Australian aborigines ; those aborigines of the cities and towns show crowded arches, within a generation or two. Of the Eskimos, Waugh[30] found that "the denture growth....was quite universally perfect, provided the individual had not been in contact with any of the food materials used by his more cultured, but physically defective white brothers."

What constitutes a "primitive" and a "civilised" mode of existence ? Waugh[30] described the diet of the Eskimo, which consists of the red meat of seal, walrus, sea-lion, whale, caribou, reindeer, bear, trapped animals and birds, also fish of various kinds; the fish and animal meat are left in the sun to "cure", and become very hard, requiring much rugged chewing. This rugged mastication is a great stimulus to alveolar bone development, and the grit incorporated in the food promotes attrition. Even young children have little other food after weaning, and deciduous teeth show advanced attrition. Barrett[32] aptly described the diet and chewing habits acquired with civilisation thus : "Mastication in the manner of our primitive ancestors has become unnecessary by the use of the mincing machines and of knives and forks and by intensive selection and preparation of food. Chewing a mouthful of food usually consists of a few weak masticatory strokes prior to swallowing ; many foods need no more than a squash with the tongue against the palate. Abrasive particles are carefully excluded from foods during preparation and cooking." The results of civilised diet upon masticatory power is illustrated clearly by the findings of Waugh[30] using the gnathodynamometer : Eskimo children with deciduous dentitions can exert greater power with their masticatory muscles than

a picked group of American athletes, and Eskimo adults can register 4-5 times the power of the same athletes.

2. Domestication.

Bjork[29] attributed the rapid changes occurring in dento-facial development during the transition from the primitive to the civilised state, to "domestication". He stated that :

"Regarding the cultural changes, we do not yet know what effects a change from a primitive to a civilised mode of life has on the human being. The question has been thoroughly investigated in relation to animals." ....."By comparing the effect of captivity and domestication on different animals, it may be possible to throw some light on the problem."....."By the effects of domestication is meant the changes which take place in successive generations of a species of animal when its mode of life becomes dependent upon the human being.... even wild animals held in captivity in parks, zoological gardens etc., are subject to marked changes of a morphological and psychological nature."

Lundholm[29] dealt with the problem of domestication very thoroughly in relation to the horse ; one feature is the reduction in size of the body. Some animals show a reduction in the size of the head and thus of brain volume. In the latter case, there may be no proportional diminution in the size of the teeth, resulting in crowding, as seen in the domestic dog. Yet another feature is an increase in the breadth of the head in relation to length, as seem in the pig and lion when domesticated.

Bjork[29] illustrated the rapid effects produced by domestication upon the ourang outan Jacob, reared in captivity in Copenhagen zoo. The superstructure of the cranium showed marked differences in outline due to reduced muscle function. There was a marked reduction in prognathism, the basal skeletal reduction being more marked than the alveolar prognathism, and resulted in a horizontal position of the incisors, and an open bite anteriorly.

Lundholm[29] felt that the reduction of freedom and the change in diet are not sufficient to explain these marked changes. One very evident difference between wild and tame animals is that the latter reach puberty at an earlier age, and consequently they have a longer life of sexual maturity, which implies a different hormone balance than in wild animals. The activity of the growth hormone is inhibited by the onset of puberty, and this is well illustrated in Eunuchoid Gigantism. Hereditary factors may also be influenced, suggested Lundholm. The well known distinction between modification and hereditary change is that the former is due to environmental influences, whereas the latter are caused by mutation. Changes in hormone balance would therefore be assumed to have the effect of increasing the mutation frequency. It has been recognised for years, that urban children reach puberty at an earlier age than rural children, and differences are evident therefore in their skeletal development.

To what extent the civilised mode of life will influence the variety in occlusion and spacing of the teeth, and the mechanics of such changes, is not clear.

E. A COMPARISON OF THE DENTITIONS OF CIVILISED, PRIMITIVE AND ANCIENT MAN.

In 1880, Professor Fowler[14] claimed that "defective condition of the third molars (impacted third molars) is no monopoly of the most highly civilised races, but may be found in the most abject and degraded of the human species - Negroes, Melanesians, Australians, Tasmanians etc..." He called attention to several specimend in the Museum of the Royal College of Surgeohs, London, in which teeth other than third molars are impacted in the jaws of 'primitive' people.

The oldest human specimen to show an impacted tooth is the Mousterian youth, estimated at 40,000 years old: Nodine[14] quoted his age as about 16 years, and drew attention to the retention of a mandibular deciduous canine, with impaction of its successor; Hellman [33] described the specimen as showing the "incomplete eruption

of the third molars" which he took to be "early indications of the fate that seems to be overtaking our dentitions". I am uncertain of the validity of Hellman's claim, in view of the estimated age of the Mousterian youth - an age at which partial eruption may well be normal for the third molars.

A comparison of the incidence of impaction of the mandibular third molars in various races, primitive, ancient, and civilised, should be of aid in the determination of the aetiological factors of impaction and non-eruption.

1. <u>The Australian Aborigine</u>.

I have been unable to find mention of impacted mandibular third molars in the Australian aborigine living under primitive conditions, in the dental literature. There is a specimen at the Royal College of Surgeons, London (No. 1086), of an Australian aboriginal woman with second deciduous molar retention, and the second premolars obliquely angulated and buried/

This race is reported to show perfect dental arches, with teeth in good alignment, and characterised by progressive, severe attrition - interproximal and occlusal or incisal - which reflects the vigorous masticatory function and the coarseness of their diet. Barrett[32] emphasised the importance of attrition and continuous eruption in the maintenance of a functional occlusion. The adoption of the civilised diet results in early deterioration of the dental mechanism within the first generation.

Begg[21] in his excellent essays on "Stone Age Man's Dentition" did not agree that the Australian aborigine is immune from malocclusion, impaction, and lack of space within the jaws. A summary of his findings is justified :-

(i) "Correct occlusion (as seen in the aborigine) is not a static condition." Rapid occlusal or incisal attrition eliminates cuspal restraint, and permits the dentition to fulfil the necessary mesial migration (under the influence of the anterior component of

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force) to produce an alveolar prognathism sufficient to increase the arch length for the accommodation of the teeth. Freed from cuspal interference, the mandibular teeth migrate forward more rapidly than their antagonists, until overbite is eliminated, and the mechanically efficient "edge-to-edge" relationship of the incisors is acquired. Continuous eruption is necessary to compensate for occlusal or incisal attrition, mesial migration for interproximal attrition, and these processes produce a 'continuously developing functional occlusion', as Barrett termed it.

(ii) The deciduous dentition undergoes early attrition, and cuspal interference is eliminated; the mandibular deciduous teeth migrate mesially to overcome overbite, and assume the edge-to-edge relationship. When the first permanent molars erupt, they immediately gain the normal molar interdigitation that is assumed in civilised man only after the replacement of the unworn deciduous molars by their narrower premolar successors.

(iii) The interproximal attrition reduces the mandibular dental arch width by a total of 14.7 mms (average), prior to the eruption of the third molars. Thus an average of 7 mms. space for the erupting third molar is donated by the teeth mesial to it, at the expense of their contact points and occlusal or incisal portions.

(iv) Impaction of the third molars and crowding of the teeth in civilised man is attributed by Brash to the evolutionary reduction in jaw size occurring in advance of the evolutionary reduction in tooth size. Thus "large teeth in small jaws", "apical base deficiency" are terms handed down from textbook to textbook, in sections on the aetiology of malocclusion and impaction. Begg's insight is displayed in :

"Anthropologists observing the low incidence of tooth irregularity and crowding in primitive man and its high incidence in civilised man, consider that there has been much more evolutionary reduction in the size of civilised man's jaws, than of his teeth since Palaeolithic times. These observers do not realise that the great attritional tooth reduction appreciably reduced the incidence of tooth crowding of primitive man and that the absence of tooth attrition accounts for much

of the tooth crowding in civilised man."

Indeed, Stone Age man inherited (and we still inherit) excessive tooth substance so that that which remains after attrition, would be sufficient to fill without crowding, the tooth bearing parts of the jaws. That is, "apical base deficiency" is a normal hereditary pattern, and lack of function with its attendant attritional reduction of coronal arch width, through refined diet, is the main cause of crowding of the teeth found in civilised man.

(v) The Australian aborigine suffered his share of malocclusion. Begg examined 800 pre-European skulls, and found that 12% showed Class II Division I malocclusion, 3% showed Class III malocclusion, and 1% Class II Division II malocclusion. Class I malocclusion no doubt occurred, but attrition rapidly eliminated cusps, and the irregularity was then self-curing. Class II Division I malocclusions caused no inconvenience, free lateral jaw movements being possible, and so this form of malocclusion showed a high survival rate in this inbreeding , isolated community. However, Class III and Class II Division II malocclusions by abnormal attritional pattern, often caused pulpal exposure in the incisors by puberty ; such noxious malocclusions naturally showed a low survival rate.

(vi) Begg found that the total incisor diameter ($\frac{21}{12}$) in cases of crowding in the aborigine was greater than in a similar number of cases showing perfect alignment. He found similar results in civilised man. And thus he demonstrated that tooth size was a factor as variable as inherited jaw size. This led him to state "the main mass of malocclusions is produced by the working together of two separate but almost equally variable entities - tooth size and jaw size. Many of thecases of malocclusion in the Aborigine could be attributed to unfavourable combinations of these two factors."

(vii) No indications of the incidence of impacted and unerupted teeth were, unfortunately, supplied.

2. <u>The Greenland Eskimo</u>.

Pedersen,[19] in his extensive studies of the Eskimo, noted the excellent alignment of the teeth in well developed jaws ; severe attrition was a consistent finding. Radiographs of 45 skulls of ancient Greenlanders revealed only two cases of impacted third molar. Radiographs of 87 modern East Greenlanders revealed 3 cases of lower third molar impaction. It should be noted that there is a particularly high incidence of congenital absence of the third molar in the Eskimo and other Mongoloid peoples (Malayans show the same incidence of third molar agenesis according to Tratman[34]). Waugh and Goldstein[18], in their studies of American and North-East Greenland Eskimos, confirmed the findings of Pedersen. All noted the rapid deterioration of the dentitions when Eskimos adopt "the foods of commerce".

3. <u>The Andes Indian</u>.

Price[31] noted that whereas the Andes Indian, who lived under primitive conditions, displayed broad, perfect arches; his offspring reared on civilised diet showed crowded, narrow arches with all the signs of malocclusion.

4. <u>Ancient and Modern Egyptians</u>.

Curtis[14] examined hundreds of the pre-dynastic Egyptian skulls, and skulls of the Nubians of the Merotic period. No impacted teeth were found, the jaws were uniform and true to type, the bony supporting tissues of all teeth were markedly developed and there was ample room for the third molar, even for fourth molars; attrition was marked, and reflected a coarse simple diet. He compared these skulls with those of a later period. From 2,000 B.C., impactionss were discovered in all periods, and the teeth did not show the same attrition, nor were the jaws as highly developed. From 500 A.D., the diet became soft and pasty, and required no masticatory effort.

5. <u>Polynesians of Pukapuka (Danger Isles)</u>

Davies[35] found that of the 221 Polynesians he exam-

ined, who exhibited clinical evidence of the presence of all third molars, 44.8% showed lower third molars in poor occlusion or actually impacted. There was an incidence of 14.4% of impacted lower third molars. Davies had no radiographic facilities, and his studies would not have included those buried impacted third molars, as their presence was not clinically evident. One would expect, therefore, a somewhat higher incidence of impacted lower third molar than his estimate of 14.4% This group has negligible contact with European civilisation, and showed a moderate degree of attrition, the severity of which showed no correlation with the incidence of third molar impaction.

6. <u>The South African Bantu</u>.

Middleton Shaw[19] reported an absence of impaction in the South African Bantu.

7. <u>The West African Negro Races</u>.

Mitchell[36] in a dental survey of Nigerian students of various races and of different geographic regions, found an incid - ence of 12.6% for impacted and malerupted mandibular third molars. Radiographic facilities were not available for the survey, and the above assessment included only those students with clinical evidence of the presence of all third molars. Of the 436 cases selected, 55 showed impaction and gross maleruption of the lower third molar; 24 mesioangular, 15 distoangular, and 19 vertical impactions were recorded, together with 16 severe linguoverted and one marked bucco-verted mandibular third molars. There is no doubt that a radiographic assessment would have shown the incidence of impacted and malerupted lower third molars to be higher still. The incidence of mesioangular impactions was found to be 3.25 times higher in the group of students with crowded lower anterior teeth, than in those whose anterior teeth showed diastemata or normal alignment. The native diet is largely cereal, with a little meat and fish ; beans, yams, cassava, guinea corn, pounded to a pulp, and made into cakes (which are fried), or

into a glue-like gel requiring little mastication.

8. The Japanese.

Ito[37] estimated that the incidence of impacted mandibular third molars in the Japanese is at least 35%. He attributed the high incidence to the unusually dense cortical bone of the mandible which he found, required a greater exposure of radiation for a dental film. He found the bone to be dense from a surgical viewpoint. It would seem a rather too subjective type of evidence to be reliable, v and yet,the remarkable density of Japanese bone has been handed down in the literature, as an aetiological factor in third molar impaction. Cryer[14] attributed the reason for impaction of the lower third molars in the Japanese to the almost vertical condylar angle of 108' ; he assumed that therefore the length of the alveolar process is short. Bjork[10] showed that a near vertical ramus does not dictate alveolar space deficiency, as compensation occurs by alveolar prognathism. Although I have no evidence to support my opinion, I feel that the Japanese people usually display considerable alveolar prognathism.

9. The Chinese.

Montelius[14] in Peiping,China, found that Chinese males have a 31.4% incidence of lower third molar impaction. He also found that Caucasians in Peiping showed a 9.64% incidence. His survey included radiographic investigation, and the numbers in each group was 1,000 subjects.

10. Australian Students.

Lilienthal[38] found that 35.5% of Australian students examined, possessed one or more impacted third molars.

11. New Guinea Natives.

Cameron, Sinclair, and Goldsworthy[39] found that only 2% of 200 individuals examined, possessed impacted teeth.

Summary and Conclusions.

The following points arise from consideration of the dentitions and dento-facial development of ancient, primitive and civilised man :-

(1) Impacted teeth have been found in one of the few discovered specimens of Neanderthal man ; impacted teeth have been found in all ethnic groups ; they have been reported in the higher apes.

(ii) Those races showing relative freedom from impactions and malocclusion, have in common : a high degree of attrition ; consistently uniform, true to type arches ; and a diet which is coarse and simple, requiring the maximum of masticatory function. The Australian aborigine, the Greenland Eskimo, and the ancient pre-dynastic Egyptians and Merotic period Nubians all displayed the above features. It has been shown that once the diet becomes refined, malocclusion and impaction become rife in the first generation to be reared on the "foods of commerce."

(iii) The rapid onset of deterioration of the mechanism cannot be explained by evolution, which knows the time scale measured in thousands of years. To explain the phenomena, two interesting theories have been published :-

(a) Bjork and Lundholm considered that domestication of the human may produce changes which are similar to those found in domestic animals, and wild animals, reared in captivity. Lundholm showed that hormonal changes occur during domestication, viz, the earlier onset of puberty, which may explain the reduction in dento-facial development, and the development of malocclusion.

(b) Begg considered that lack of attrition - interproximal, occlusal, and incisal - militates against the attainment of adequate arch length by alveolar prognathism and lateral denture migration, and by the reduction of the coronal arch length to compensate for an inherited predominance of tooth substance over jaw accommodation.

(iv) Malocclusion is produced by the working together of two

equally variable factors - tooth size and jaw size. Hybridisation tends to increase the degree of disharmony between these two entities and should a small skeletal jaw development be inherited with large teeth, malocclusion and third molar impaction are very likely sequellae.

F. THE EFFECTS OF SOME RELEVANT DEVELOPMENTAL ABNORMALITIES ON THE ERUPTION OF TEETH.

1. Ankylosis of the Deciduous Molar.

Resorption of the roots of deciduous teeth is by intermittent phases of osteoclastic resorption, alternating with rest phases, in which reparative activity occurs. Osteoclasts of the periodontal membrane resorb cementum and dentine ; then follows the reattachment of the periodontal fibres to the dentine by cementoblasts.[18] Ankylosis of dentine and bone may occur, which, Vorhies et al.[40] considered, is due to the loss of viability of the cementoblasts. Once fusion of the tooth to alveolar bone has occurred, continuous eruption is not possible, and the tooth remains static within the alveolar process, whilst the adjacent teeth continue to erupt, with concomitant alveolar bone growth. The ankylosed tooth appears to sink within the alveolar process (which is the origin of the unsatisfactory term "submerged" tooth) whereas in fact, the tooth remains stationary and is engulfed by the alveolar growth to its mesial and distal. Eventually the crown is covered by gingival tissue, and the teeth to its mesial and distal, tilt over its crown - indeed, they often contact.

Erroneous theories as to its aetiology have arisen from clinical and radiographical appearances, due to misapprehension of the pathology : some authorities believed that the tooth had never erupted, simply having failed to erupt - an ankylosed deciduous molar, its crown at a level below the alveolar crest, may show radiographical evidence of an occlusal restoration, which invalidates this theory ; others considered that the deciduous molar is actively depressed by the adjacent teeth tilting and possibly contacting over its crown ; others again, explained the condition by the assumption of a

localised failure of growth of the alveolar process which thwarts continuous eruption. There is little doubt that fusion of the dentine to the alveolar bone is the primary cause in the majority of cases.

Dechaume and Cauhepe[41] found 63 retained and ankylosed deciduous molars in 40 of 60,000 children examined. The first deciduous molar was ankylosed as frequently as the second, and the mandibular deciduous molars were affected four times as frequently as the maxillary deciduous molars. In only 4 out of 36 cases examined radiographically, was the permanent successor congenitally absent.

The criteria for the diagnosis of ankylosed teeth is listed by Dunn et al.[42] as follows :-

(i) Loss of occlusion with the opposing molar.

(ii) Complete lack of mobility.

(iii) Incomplete, or lack of, periodontal membrane (radiographically interpreted).

(iv) Percussion gives a solid sound, not a dull, padded sound as in normal teeth.

(v) Invasion of the pulp by bone may, or may not be visible radiographically.

The sequellae of ankylosis of the deciduous molar are as follows :-

(i) Impaction of the permanent successor.

(ii) Mesial tilt of the tooth distal, over the crown of the ankylosed molar, and distal tilt of the tooth mesial; these adjacent teeth may even contact.

(iii) Permanent and absolute loss of alveolar height, unless surgical excision of the tooth is affected early ; delayed excision further increases the bone deficiency.

(iv) Antro-oral fistula may follow the late excision of an ankylosed maxillary deciduous second molar.[40]

(v) Pericoronitis, and Vincent's infection, are possible complications when the crown is covered in part by the gingivae.

2. Supernumary Teeth.

Supernumary teeth may be defined as teeth in excess of the normal number of the deciduous or permanent teeth. Such teeth may closely resemble in anatomy, teeth of the normal series - they are then called supplemental teeth ; or they may be conical or tuberculate in form - they are called supernumary teeth.[13] It is doubtful if the distinction is of importance.

Saarenmaa[43] found their incidence in Finnish recruits to be 0.64% (no mention is made of the method of investigation). McPhee[13] found an incidence of 0.3% in British school children. Aitcheson[13] considered them to be of higher incidence in the black races and the West African Negro is particularly prone to supernumary teeth.[36]

Their aetiology is not understood, therefore several theories are quoted in current literature. Atavism is the most popular theory, and it is claimed that supernumary teeth represent a throw-back to a primitive mammalian dentition. The mammalian dentition is three incisors, one canine, four premolars, and three molars in each quadrant. Saarenmaa[43] found 87% of supernumaries in his series were in the incisor region, whereas only 4% were found in the premolar region ; since twice as many premolars as incisors have been eliminated in the human dentition, a higher frequency should be observed in the premolar region. Again, fourth molars and paramolars of Bolk are found, especially in Negro people,[13 ,36] whereas the mammalian dentition index provides for only three; Wood[44] and Oehlers[45] reported three supernumary premolars in addition to the normal two, in the right mandible of a Jamaican and a Malay respectively, which is in excess of the four premolars of the typical mammalian dentition ; the rare supernumary canine has been reported.[13 , 43, 215, 217] Therefore the theory of atavistic tendency may be discarded. Dichotomy of the tooth germ to produce two teeth may explain the isolated case, such as the two smaller lateral incisors of the maxilla, but it cannot explain the aetiology of supernumary teeth in general. The most likely theory, in my opinion, is proliferation of the dental lamina to produce additional tooth germs : such a theory would be difficult to prove, except by a fortuitous histological discovery. Hitchin[46] drew attention to the genetic factor in the

aetiology of supernumary teeth : he found identical mesiodens in monozygotic twins, one in one set, two in another set. Saarenmaa[43] endeavoured to investigate the genetic factor, but found no familial tendency in the twenty Finish families in which one member possessed a supernumary. He concluded that a dominant inheritance is unlikely, and that a recessive inheritance would be difficult to trace.

It is often claimed that supernumary teeth erupt in advance of the permanent teeth, and usurp their position in the arch. A search through dental literature has failed to reveal an investigation of the eruption times of supernumary teeth. Published radiographs suggest, however, that apical closure of the mesiodens is in advance of the permanent maxillary central incisors in some cases, but occurs later in others (radiographs published by Ennis[47] confirm these observations). In West African Negroes, the eruption of the normal series premolars is rarely affected by the presence of supernumary premolars, as apical closure of the latter teeth occurs after 20 years of age in most instances, and the supernumary premolars either remain impacted, or generally, erupt lingually to the arch in the mandible, palatally in the maxilla.[36] Hill[48] noted negligible root formation in two mandibular supernumary premolars discovered in a 16 year old male, although, interestingly enough, the supernumary premolars lay to the occlusal of the second premolar, which was therefore prevented from erupting. Cowan[49] noted in a female, aged 15 years, 4 months, that apical closure in a mesiodens was complete, whereas minimum root formation had occurred in three supernumary premolars.

Supernumary teeth are common findings in cases of clefts involving the alveolar process, and have been a consistant finding in cases of cleidocranial dysostosis.

Supernumary teeth may block the eruption of permanent teeth, may cause a diastema between normal teeth, creating thereby, secondary space deficiency which may cause the impaction of later erupting teeth. An example is seen in the diastema between the maxillary central incisors, caused by a mesiodens ; the lateral incisor, erupting in a distal position, resorbs prematurely the deciduous canine,

with the result that the permanent canine is impacted. Supernumary teeth may themselves be impacted and embedded, and may consequently suffer and cause the same ill-effects as impacted teeth of the normal series.

3. Cleidocranial Dysostosis.

This is a rare condition of unknown aetiology. It is not necessarily hereditary in origin, but may be transmitted by a Mendellian characteristic (Miles[16]). Either parent can transmit it to children of either sex, i.e., the children have a fifty-fifty chance of being normak.

The four cardinal signs of the disease were published by Marie and Sainton[50] in I897 :-

(i) Aplasia of one or both clavicles.

(ii) Exaggerated development of the transverse diameter of the cranium.

(iii) Delay in closure of the fontanelles.

(iv) Hereditary transmission.

It was, until recently, considered to be a developmental disturbance affecting intra-membranous bones only. Meinmann and Sicher[3] claimed that "other than intra-membranous bones are involved"; "the disease is much more generalised than was formerly supposed"; and "whether we can speak of a disturbance of bone and dentine formation or of a disturbance of skeletal development is not yet clear."
Hutchinson[51] described an atypical case in which multiple unerupted teeth occurred with minor developmental defects of the skull (wormian bones, metopic suture) and spinal column, and with normal clavicles.

The maxillae are retruded, and may be underdeveloped with negligible pneumatisation of the maxillary simuses ; the palate is high, arched and narrow ;[13,50] the mandible, reported to be of normal size, shows relative prognathism. Zygomatic, nasal, lachrimal bones

may also be underdeveloped.

Dental abnormalities seen in cleidocranial dysostosis are :-

(i) Deciduous teeth may commence eruption at normal times, but the process may not be completed until 6 years of age (Kjellgren[52]).

(ii) The deciduous teeth are generally retained to maturity (Seldin[50]).

(iii) Finer roots in erupted deciduous teeth have been noted by Rushton[53].

(iv) Permanent molars may erupt as they have no predecessors, yet their eruption is delayed and uncertain ; the other teeth however, generally remain embedded (Seldin[50]).

(v) The roots of embedded teeth form, but are buckled and folded (Seldin[50]).

(vi) The roots of embedded teeth show an absence of cellular cementum, which in normal teeth, and in the erupted deciduous teeth of cleidocranial dysostosis, covers the acellular cementum of the apical half of the root - Rushton found that 8 out of 9 embedded teeth removed from 5 persons with the disease, lacked cellular cementum, which is necessary for continuous eruption.[53]

(vii) Rushton[53] noted that the embedded teeth are often conical, and show defects of the surface of the enamel, which hypoplastic defects he attributed to pressure of the developing tooth against the bony crypt.

(viii) Supernumary teeth are common findings - Kjellgren[52] reported fourteen in one case.

(ix) Geminated teeth have been reported by Stones.[13]

The aetiology of the disease is unknown, and its pathology not understood due to the lack of histological material available.[3] I feel that the buckling anf folding of the roots of the unerupted teeth in cleidocranial dysostosis may aid in the determination

of the pathology.

Buckling and folding of the roots of teeth have been noted by :-

(i) Bryer,[22] in roots of the incisors of rats subjected to a vitamin A deficient diet ; the eruption of the incisors was greatly retarded, there was vigorous osteogenesis at the fundus, and the roots were buckled and folded ;

(ii) Massler and Schour,[50] in roots of teeth in animals subjected to hypophysectomy : eruption was much retarded, and the roots were buckled and folded.

Buckling and folding of the roots could be regarded as the result of active root formation by dentinogenesis, concomitant with retarded eruption : that is, elongation of the root, at a greater rate than provision of space by active eruption. If that is the explanation, one should find evidence of buckling and folding in the roots of the impacted tooth, the eruption of which is actually prevented before root formation is complete. Instead, the roots are found to have achieved normal formation, and to have elongated to their normal length, without any evidence of deformity, buckling or folding (unless the impacted tooth is forced against cortical bone, such as the orbital plate, antral wall, or mandibular cortex, when splaying or folding is noticed). That the roots of impacted teeth do elongate, is proved by the impacted mandibular third molar, the roots of which inactively elongating, may abut against the cortical tunnel of the inferior dental canal, bifurcate, grow beyond the canal, and may invaginate the canal and contents within a root foramen. It seems reasonable to postulate, on the above evidence, that the roots of impacted teeth do not show buckling and folding, because space for their elongation has been provided by osteoclastic resorption of bone. In Bryer's case (vide supra) not only was eruption retarded, but, at the fundus of the developing tooth, active osteogenesis (which is stimulated by vitamin A deficiency - Mellanby[13]) occurred, not the osteoclastic action necessary when root elongation proceeds at a greater rate than eruption : the result was the concertina-like

compression of the plastic predentine.

It is agreed by all authorities that the aetiology of cleidocranial dysostosis is unknown, and that the mechanism underlying its pathological manifestations is not understood. I feel that, in the light of present knowledge, there is some justification in assuming that the mechanism of bone and dentine resorption is at fault, and submit the following reasons in support of this view :-

(i) Eruption of the deciduous teeth is considerably delayed ; eruption of the permanent molars may occur, but is usually much retarded. Normal eruption depends not only upon the inherent eruptive force, but also upon bone resorption.

(ii) The deciduous teeth are retained on account of failure of osteoclastic resorption of their roots, and active eruption of their permanent successors.

(iii) The successors of the deciduous teeth almost invariably fail to erupt : they are placed more deeply in the jaws and are covered by more bone than the more superficially placed permanent molars and deciduous teeth in pre-eruptive position. Their non-eruption cannot be attributed to failure of eruptive force - which would affect all teeth more or less equally. Failure of resorption of the overlying bone, and the normal shedding of their predecessors, is the more feasible explanation.

(iv) Buckling of the roots of embedded teeth, I consider, is due to concurrent failure of eruption, and failure of provision of space for the elongating root, by apical bone resorption.

(v) Hypoplasia of the enamel is seen sporadically, in the teeth. This cannot be due to a systemic factor, as amelogenesis and dentinogenesis are normal, and the distribution of the hypoplastic deformities would be generalised throughout the dentition. Therefore a local factor must be sought : local pressure to the enamel organ is a reasonable hypothesis, and it seems possible that by failure of bone resorption, pressure to the enamel organ would occur, due to the

failure of crypt expansion.

(vi) Should resorptive propensities of bone be deficient, it is reasonable to assume that formative processes dominate. Bernier[54] noted that : "The changes in bone are chiefly sclerosis. Osteocytes are sparce, as is the formative tissue." The sclerotic nature of bone in cleidocranial dysostosis has not been stressed in the literature. However, Blumenthal and Catonia[55] reported a case of cleidocranial dysostosis in which they noted "thicker and denser bone" requiring "longer exposures" for the "desired result" (referring to radiographic technique). The radiographs they published showed a radiopacity of bone which was marked, but less marked than in Marble Bone disease.

If cleidocranial dysostosis is indeed a condition in which, among other factors not understood, osteoclastic activity is diminished, one would expect to find that this condition possessed certain features in common with osteopetrosis - a condition of bone in which osteoclastic inactivity is a widely accepted characteristic. Osteopetrosis will therefore be considered.

4. <u>Osteopetrosis (Marble Bone Disease)</u>.

This is a rare osteodystrophy. It is of unknown aetiology, although Albers-Schonberg (1907) mentioned inbreeding as an important factor.[13]

It is characterised by the following features :-

(i) General involvement of the skeleton, with thickening of the bones, which radiographically show increased opacity, and histologically increased apposition of lamellae and reduced resorption, to produce a thickened cortex and reduced marrow spaces. The bones are brittle, due to excessive mineralisation, and are prone to fracture. Due to a relative diminished blood supply, the bones are susceptible to necrosis.

(ii) Stenosis of foramina (causing defective hearing and vision) and secondary anaemia are sequellae.

(iii) Dental anomalies, such as delayed eruption and susceptibility to caries, have been reported.[13]

(iv) The bones most seriously affected by sclerosis, are those formed in cartilage[54]

(v) Anatomical landmarks (such as the outline of the paranasal sinuses) are obliterated in radiographs.

The recent investigation of a case of osteopetrosis by Bergman and Engfeldt[55] reveals that :-

(i) The eruption of the deciduous teeth is greatly delayed : in the child studied, at 18 months of age, the deciduous incisors erupted, with no further eruption of teeth by the age of 5 years, the remaining deciduous teeth being embedded.

(ii) There was aplasia of the maxillary second deciduous molars and of the mandibular second premolars.

(iii) There was malformation of the crowns and roots of many deciduous teeth and permanent teeth. Deformity and buckling of the roots of some teeth, and enamel hypoplasia of others, was evident.

(iv) No mention was made of the presence of supernumary teeth.

Bergman and Engfeldt reasoned that the enamel defects could be due to the systemic factor which caused the skeletal effects. They considered this theory to be unlikely, as not all five embedded teeth examined histologically, showed hypoplasia. However, it should be remembered that not all teeth suffer hypoplasia in congenital syphilis. Another argument against the systemic type of enamel hypoplasia is that amelogenesis and dentinogenesis are usually equally affected (as in rickets, fluorosis etc). They concluded that the enamel defects arose from local causes, such as pressure to the enamel organ. They stated :"...a condition of normal tooth growth and eruption is active ~~bone~~ remodelling of the surrounding bone. In osteopetrosis, the resorption of bone is reduced." The stunting and buckling of

the roots of the embedded teeth, occurring with active, normal dentinogenesis, reflects lack of space for their elongation, which space may be provided by (a) active eruption, or (b) bone resorption at the fundus.

5. <u>A Comparison of Cleidocranial Dysostosis and Osteopetrosis.</u>

These conditions have the following features in common : delayed eruption and shedding of the deciduous teeth ; multiple unerupted teeth ; enamel hypoplasia in many, not all, unerupted teeth, associated with normal dentinogenesis ; deformation of the roots of embedded teeth, characterised by stunting and buckling. The two conditions are particularly similar in respect of dental anomalies.

The most outstanding differences noted are : the type of bone affected ; the degree of sclerosis ; and the effects upon the clavicle. Osteopetrosis, according to Bernier,[54] affects in particular, those bones formed in cartilage ; cleidocranial dysostosis affects the membrane bones, but it must be recognised that the distribution of the condition is more widespread than was previously thought,[3] and there is a considerable degree of variation in the extent to which the clavicle is involved - some believe that aplasia of the clavicle need not be associated, for a diagnosis of cleidocranial dysostosis to be correct. Sclerosis is particularly accentuated in osteopetrososis, as the name implies ; sclerosis, although a reported finding by Bernier[54] and by Blumenthal and Catonia,[55] is not, it seems, a consistent and marked feature of cleidocranial dysostosis.

To suggest that the two conditions are but variants of the one disease, would be unjustified in view of the scant knowledge of the histological, and biochemical aspects of these dystrophies. However, it is my opinion that a common, underlying mechanism - viz, defective bone and dentine resorptive propensities - is shared.

Both conditions are rare. The literature pertaining to cleidocranial dysostosis is not abundant - cases have been reported

by Arnott, Corbett and McEncroe,[58] Seldin,[50] Kjellgren,[52] Blumenthal and Catonia[55], Hodgson,[56] Chipps[57] and only a few other authors. The literature pertaining to osteopetrosis is sparce. Consequently no opportunity should be lost in the investigation of these conditions. Detailed histopathologic, biochemical and radiographic investigations should be carried out in all suspected cases. In view of Albers-Schonberg's observations, an exhaustive family history should be gained, and the possibility of consanguinity explored - each condition is considered an hereditary disease. Radiographic investigation should include not only the skull and mandible, but the entire skeleton, in order that the distribution of the skeletal involvement can be revealed.

6. <u>Hereditary Enamel Hypoplasia - : A cause of Multiple Unerupted Teeth.</u>

Hereditary "brown teeth", called also amelogenesis imperfecta hereditaria, is an anomaly of enamel formation. It can be sharply divided into two types :-

(I) Hereditary enamel hypocalcification. This condition is characterised by a qualitative defect of enamel which remains in the matrix stage. A normal thickness of matrix is laid down, but calcification fails to occur. The enamel remains soft and chalky, absorbs exogenous stains rapidly, and soon is lost by abrasion. Radiographs show unerupted teeth to be of normal form, though the enamel is indistinguishable from dentine. <u>Eruption is normal</u>.

(ii) Hereditary enamel hypoplasia. This condition is character-

ised by a quantitative defect of enamel of all teeth, permanent and deciduous. The enamel is thin, but well formed, glossy and hard. Due to its thinness, the underlying dentine produces a yellow discoloration which is not due to exogenous stains. All cases of this anomaly show multiple unerupted teeth.

Why do teeth affected by hereditary enamel hypoplasia fail to erupt ? Weinmann et al. studied 9 members of an Italian family having the disease. All nine members showed arrested eruption of a varying number of teeth. Those teeth most commonly affected were the second and third molars of both jaws, followed in frequency by the maxillary cuspids. All embedded teeth showed irregular defects in parts of the crown filled with bone. Most of the unerupted teeth were in normal pre-eruptive position and their long axes were at right angles to the occlusal plane. This held true of the molars, which had migrated from their primary positions in a horizontal plane, to the vertical position. The following conclusions can be drawn :-

(i) aberration of the tooth germ, and impaction may be ruled out as causes of non-eruption;

(ii) a systemic factor inhibiting eruptive force would affect all teeth;

(iii) defective bone resorption (as in cleidocranial dysostosis) is not the cause, as intra-osseous migration of the developing teeth appears to occur normally;

(iv) radiographic findings indicate a similarity to coronal resorption and ankylosis.

Normally, once the enamel matrix has been laid down, the ameloblasts unite with the outer enamel epithelium to form the reduced enamel epithelium. This secondary cuticle protects the enamel from the vascular connective tissue, and, on account of its presence, the erupting tooth effects resorption of bone in its path of eruption. In hereditary enamel hypoplasia, it may be assumed that the reduced enamel epithelium does form, but soon degenerates, so bringing the vascular connective tissue into contact with the enamel. From the

cemento-enamel junction, cementum is deposited on the lateral surfaces of the crown, progressively towards the incisal or occlusal surfaces ; cementoid tissue is resistant to osteoclastic action like osteoid tissue, and therefore protects the enamel from resorption. However, before it may reach the incisal or occlusal surface, resorption of enamel occurs ; the crown is hollowed out and the defects filled with vascular connective tissue, which later is converted into bone. The pulp is rarely exposed, on account of predentine, which resists resorption by osteoclasts. Eruption comes to a standstill, unless the area of resorption is small and the tooth nearly erupted. The pathology is similar to coronal resorption with ankylosis, which occurs usually, after the tooth has been embedded for years - in hereditary enamel hypoplasia, coronal resorption is not secondary to non-eruption, but its cause.

Weinmann and his co-workers published in detail, their clinical findings of each of eleven cases studied. Radiographic investigation of these cases showed radiographic evidence of coronal resorption in many, but not all, unerupted teeth. An analysis of their findings is presented :-

Age (years)	Teeth unerupted (number)	Teeth resorbed (number)
44	10	6
42	7	5
41	6	5
22	11	4
20	12	8
17	6	3
16	18	8
16	10	nil

G. SUMMARY AND CONCLUSIONS.

Teeth may fail to erupt into normal functional occlusion, for the following fundamental reasons :-

1. Abnormal location, or axis, of the tooth germ.
2. Failure of the eruptive force and associated mechanisms.
3. Lack of space in the dental arch. This may be due to
   (a) basal bone deficiency,
   (b) alveolar bone deficiency,
   (c) primary predominance of tooth over jaw size.
4. Obstruction of the erupting tooth by impediments in its path of eruption.

A classification based on the above, will be presented.

Group 1 : Abnormal location, or axis, of the tooth germ.

Such teeth necessarily pursue an abnormal path of eruption, and therefore are termed "aberrant".

Causes.-

(i) Genetic.- Tooth germ location and axis is genetically determined. There is no published evidence to prove that abnormal location or axis is inherited. In view of the proven hereditary tendency to overlapping of the maxillary central incisors, by the lateral incisors, I feel that an investigation of the family history in cases of aberrant teeth, would be fruitful.

(ii) Congenital.- Nodine[14] attributed abnormal axis to intra-osseous pressure during the early developmental period; he felt, although he had no evidence to prove, that such systemic maternal conditions as syphilis and tuberculosis, may set up pressures which could displace the tooth germ. Intra-osseous pressure is a popular

assumption - true gemination of teeth is attributed to fusion of tooth germs due to pressure effects, and, in view of the incidence of gemination in cleidocranial dysostosis, it appears to be a logical hypothesis.

(iii) Traumatic.- Fracture of the jaw, severe infections, surgical correction of cleft palate, injudicious use of an elevator in removal of deciduous teeth have been known to rotate or displace, a partly formed tooth in its crypt. That a tooth may move freely without strangulation of its blood supply, has been proved, by the deliberate correction of axial abnormality, by the practice of "surgical orthodontics" described by Holland.[60]

(iv) Premature loss of teeth.- Loss of the permanent first mandibular molar at a time when the mandibular second premolar is still unerupted, in many cases causes the latter tooth to rotate about its apex, and change its axis from the vertical to the distoangular position. Should the second mandibular molar be present, the second premolar will drive against its mesial root ; when it is extracted, the second premolar assumes a horizontal axis, and migrates towards the ramus.

Group 2 : Failure of the eruptive force and associated mechanisms.

Bryer[22] showed that the eruptive force is largely supplied by the blood pressure of the rich vascular beds of the dental papilla and periodontal membrane. Cellular proliferation of the papilla, and pulpal constriction play subsidiary roles in eruption.

Eruptive force may be frustrated by (i) failure of resorption of the deciduous predecessor, (ii) failure of resorption of overlying bone, (iii) ankylosis due to coronal resorption, and (iv) dilaceration of the root.

Causes of eruptive force failure.-

(i) Nutritional.- Deficient intake of vitamin A and D

(or adequate intake, with failure to utilise them), seriously retards eruption. Vitamin C has been shown by Dalldorf and Zall(1930)[13] to retard eruption in guinea pigs.

(ii) Endocrine.- Hypothyroidism, hypopituitarism, lack of ACTH, Bryer[22] has shown , retards eruption considerably. Mongolism, probably due to endocrine disturbances, significantly retards eruption.

(iii) Genetic.- It is unlikely that eruptive force may be defective through hereditary factors per se. Stones[13] reported a case of hereditary retarded eruption, and published an interesting, though incomplete genealogical chart.

b. Failure of the deciduous teeth to resorb.- Retention of the deciduous teeth usually accompanies delayed eruption of the teeth. Ankylosis of the deciduous molars occurs once the cementum loses its vitality (cementum may be resorbed, but not cementoid tissue). Delayed resorption may at times be an inherited defect (Stones[13]). Delayed resorption is seen in cleidocranial dysostosis and plays an important role in failure of eruption of their successors, as it is not unusual to find that the permanent molars do erupt normally.

c. Failure of bone resorption.- Failure of teeth to erupt may often be correctly attributed to premature loss of their predecessors. The sockets fill with a bone scar, which overlies the permanent successors' and not infrequently, the eruptive force may be insufficient to stimulate its resorption. Surgical exposure with the removal of all sclerotic bone, overlying the crowns of the unerupted teeth, usually permits eruption without the assistance of orthodontic traction.

d. Failure of the reduced enamel epithelium.- Should the integrity of this tissue be breached, as in hereditary enamel hypoplasia, the enamel is invaded by the vascular connective tissue, and, ankylosis ensues.[59]

e. Dilaceration of the root.- The root of the erupting tooth may be injured by trauma (jaw fracture, extraction of the deciduous predeces-

sor). This deforms the epithelial sheath of Hertwig, the form of which is the blueprint of the developing root. An angular bend is seen also when a tooth is deflected from its path of eruption. Dilaceration prevents the eruption of the tooth affected. This factor should not be overlooked when the prognosis of surgical exposure is considered.

Group 3 : Lack of space in the jaws.

Sub-group A : Basal bone deficiency.- The basal bone of the jaws, in growth pattern and potential, in genetically determined. The presence or absence of teeth has no significant effect upon the development of the jaw skeleton, and only the minimum amount of function is necessary for full attainment of basal bone growth potential. There is no correlation between tooth and skeletal jaw size, which are determined independently. Muscular function moulds the coronoid process and sites of muscle insertions, is responsible for the internal reconstruction of bone, but does not affect the form and size of the jaw skeleton. The degree of basal bone development limits the potential growth of the alveolar processes.

Causes.-

(a) Genetic (i) Evolutionary.- There has been an evolutionary reduction of facial prognathism and a dimimution in facial height (Brash and Bjork). Brash considered that jaw reduction is in advance of that of tooth size and number. Begg warned that preponderance of tooth substance over jaw size may be intentional, to compensate for attrition.

(ii) Cross-breeding.- Nodine considered that the bulk of human malocclusion may be attributed to "the continual intermingling of and intermarriage within historic times of what were isolated inbreeding communities in prehistoric times."

(iii) Domestication.- Wild animals held in captivity, produce offspring which show basal bone changes : Bjork reported diminution of the basal bone jaw growth in an ourang-outan reared in captivity. Lundholm noted earlier sexual maturity in domestic animals

compared with wild members of their species, and suggested that the hormonal balance upset would contribute, if not cause, the basal bone deficiency observed, and might also affect the mutation frequency. Domestication is seen in man when he departs from his primitive to a civilised mode of life, and a similar change in skeletal development as observed in other animals, should occur.

b. Congenital.

(i) Conditions in the mother.- Faulty diet, diseases of serious character (syphilis, tuberculosis, rubella during the early months of pregnancy) are thought capable of altering the foetal metabolic functions by lack of nutriment, and possibly by toxins. Congenital syphilis may produce gummatous destruction of the nasal septum or the spheno-occipital synchondrosis, thus causing underdevelopment of the maxillae.[14]

(ii) Conditions in the embryo.- Injury to the foetus by a blow to the mother's abdomen, and faulty intra-uterine posture of the foetus, could cause a deformity of the growth pattern : most authorities consider these causes unlikely.

(iii) Developmental abnormalities.- Cleft palate occurs during the 8th-11th week in utero ; the severe jaw deformity and malocclusion seen in childhood, is considered to arise from the surgical closure; unoperated cases are often found to possess jaws normal in development and free of malocclusion. Hare-lip occurs at the 5th w.i.u. Surgical closure predisposes to alveolar rather than basal bone deficiency, on account of lip tension by scar contraction. Oxycephaly, a congenital developmental condition, causes premature ossification of certain cranial sutures ; its origin is unknown, its pathology not understood; Nodine[14] and Stones[13] reported that there is underdevelopment of the maxillae, with crowding of the teeth, and the second and third maxillary molars may fail to erupt. Achondroplasia -a congenital or hereditary condition - may be responsible for underdevelopment of the maxillae with crowding and impaction of teeth; Nodine[14] reported a case with retention of the deciduous teeth, absence of, or greatly retarded eruption and malocclusion of the permanent teeth. Cleidocranial dysostosis

is responsible for underdevelopment and retrusion of the maxillae, and is associated with dental anomalies .

c. Parturient or natal.- Forceps delivery may cause injury to the child's jaws. Moulding is generally self-correcting. Should damage occur to the temporo-mandibular joint, ankylosis of the joint may ensue, with underdevelopment of the mandible.

d. Post-natal.

(i) Nutritional.- It is generally agreed that growth potential cannot be attained without adequate nutrition. Strang[1] stated that growth in the child is retarded by vitamin B complex, C and D deficiencies.

(ii) Endocrine.- Hypopituitarism causes underdevelopment of the jaws, although endochondral growth is more seriously affected than that of membranous bones.Hypothyroidism also causes underdevelopment of the jaw skeletons.

(iii) Constitutional diseases in childhood.- A short childhood illness probably has no appreciable effect upon skeletal growth. Todd[1] felt that : "The cause of deficiencies in facial growth is not as a general rule to be found in definite clinical disease, but rather in long continued poor health or constitutional disturbances for the most part subclinical in expression...."

(iv) Local.- Any inflammatory lesion destroying the condyle (osteomyelitis of the mandible, middle ear infection, scarlatina , radionecrosis) will result in underdevelopment of the mandible; an inadequately treated fracture dislocation of the condyle, by depriving the mandible of its growth centre, results in basal bone deficiency. The earlier the condyle damage, the greater will be the skeletal deficiency.

Group 3, Sub-group B : Inadequate Alveolar Bone Growth.

The hereditary factor is thought to play only a minor role in alveolar bone growth : occasionally, for no apparent reason, alveolar bone growth is found to be deficient, and this is seen in cases of constricted arches, infra-occlusion of teeth, and open bite anteriorly, Alveolar bone depends for its presence upon the teeth, and their eruption ; its growth potential is limited by basal bone development. Function is the most important element in the attainment of alveolar growth potential : the anterior component of force drives the dental arches forward in relation to the basal bone, and this force is proportional to the masticatory stress. The degree of alveolar prognathism, however, is limited by the facial muscles and tongue : perverted muscle function is an important factor inhibiting the full expression of alveolar bone growth.

Causes.

(i) Lack of masticatory function.- By reducing the power of the anterior component of force, the mesial migration of the teeth in relation to their basal bone support, is inhibited, and the degree of alveolar prognathism is limited : the greater the mesial and lateral migration of the alveolar processes, the greater is the alveolar arch length. The soft, refined foods of the civilised diet is largely responsible for the under-development of the alveolar processes.

(ii) Premature loss of the deciduous teeth.- The anterior component of force is able to drive the dental arches forward only when the teeth are in normal contact. Should the deciduous molars be extracted prematurely, the erupted first permanent molars, driven forward by this force, cannot themselves transmit this force to the teeth mesial to them; therefore, the permanent molars tilt forward, close the space intended for the accommodation of the premolars, and a net loss of space and alveolar prognathism results.

(iii) Habits.-

(a) Sucking habits tend to increase the alveolar prognathism of one

jaw, whilst inhibiting the prognathism of the other, with a lateral growth reduction of both jaws.

(b) Abnormal swallowing, with tongue thrust against the maxillary incisors, and simultaneous contraction of the lip and cheek muscles, inhibits mandibular prognathism, favours maxillary prognathism, and constricts both arches.

(c) Mouthbreathing and upper lip incompetence produce a similar clinical picture, although they have a different prognosis. The maxillary teeth are proclinated (due to a lack of lip tension), the arches are constricted (due to mandible "dropping" which reduces tongue pressure to the alveolar arches), and the mandibular alveolar prognathism is seriously inhibited, especially in the case of the incompetent upper lip, due to the powerful contraction of the mentalis muscle, in an effort to appose the lips during swallowing.

(d) Muscular tics and habit spasms are highly individualistic, and therefore produce diverse growth pattern deformities.

(e) Hypertonicity of the facial muscles produce marked deformity. "The tooth arches are held in a vice, as it were, and their forward and lateral growth markedly checked."[1]

Nutritional deficiencies, endocrine upsets, and constitutional diseases affect the growth of the alveolar bone primarily, as well as secondarily by underdevelopment of the skeletal jaw bases. It is difficult to assess the qualitative and quantitative effects upon the alveolar arches by these systemic factors.

Begg noted that only after cuspal attrition occurs, can the teeth and their alveolar support migrate forward and laterally, freely.

Group 3, Sub-group C : Preponderance of Tooth Substance over Jaw Size.

It is now well established that tooth and jaw size are genetically independent. Hybridisation tends to predispose to a

greater disparity. Only in inbreeding communities is harmony between these two variables likely to be attained.

Jaw growth is not completed until the end of the second decade, whereas the form of the last tooth to develop, is already fixed by the 9th year. Jaw growth is therefore considerably more susceptible to systemic influences than tooth development. Whereas the development of the jaws is inhibited in the hypopituitary dwarf, the cretin, and the progeria dwarf, the teeth are of normal size. Any systemic factor which causes underdevelopment of the jaws, results in a secondary preponderance of tooth substance.

Local factors may considerably affect the alveolar bone development, without influencing tooth size or number : muscular habits, perverted swallowing, and other factors upsetting the muscle environment of the teeth and alveolar processes, inhibit alveolar arch development, thereby causing a preponderance of tooth substance.

There is a very slow evolutionary reduction in the number of teeth evident. Although congenital absence of teeth occurs, it is thought that tens of thousands of years must elapse before the third molars and maxillary lateral incisors are eliminated from the human dentition.

Macrodontia of individual teeth predispose towards impaction; dilated odontomes, geminated teeth upset the tooth size/arch length ratio.

Group 3, Sub-group D : Lack of co-ordination between tooth eruption and jaw growth.

Begg suggested that many cases of malocclusion may be hypothetically explained by lack of correlation between eruption of teeth, and jaw growth. Dixon[264] believed that the impaction of the first permanent molars against the second deciduous molars is due to "the lack of timing of the respective processes of tooth eruption and increase in arch length."

Group 4 : Obstruction of the erupting tooth by impediments in its path of eruption.

A tooth may be prevented from erupting into occlusion by :-

i. An adjacent tooth or teeth. This is rarely the primary cause of impaction and usually occurs as a result of lack of basal and alveolar bone growth.

ii. A retained, ankylosed deciduous tooth. The primary cause of impaction is retention of the deciduous tooth, due to ankylosis.

iii. A supernumary tooth. The presence of the supernumary tooth reflects abnormal function of the dental lamina.

iv. A retained root, of a deciduous or permanent tooth.

v. A dentigerous cyst. It is thought that dentigerous cyst formation is a reaction to failure of the tooth to erupt.

vi. A tumour. The adamantinoma and the ossifying fibroma may cause one or several teeth to remain embedded, should the tumour occur during childhood. The unerupted tooth not only becomes impacted against the tumour, but is displaced by the expansive growth of the latter. Any central tumour occurring during childhood is liable to cause the impaction of erupting teeth.

The term "impacted tooth" is used rather loosely : a lower third molar may be described as "tooth impacted" when it collides with the root or crown of the second molar ; "gum impacted" when its occlusal surface is covered by an operculum ; "bone impacted" when its eruption into occlusion is prevented by the ascending ramus.

- PART II -

THE PATHOLOGY OF THE IMPACTED AND UNERUPTED TOOTH.

Introduction.- An unerupted tooth may produce symptoms, or it may be asymptomatic ; it may be innocuous to the patient, or it may be the seat of serious pathology. The asymptomatic unerupted tooth is not necessarily innocuous, indeed, it may be associated with advanced harmful developments, rendered potentially more harmful as the patient does not seek treatment, being unaware of any abnormality.

A sound knowledge of the possible effects of the unerupted tooth on its environmental tissues, local and remote, and the effects of the environmental tissues on the unerupted tooth, is necessary for the reasonable assessment of such questions as the following:-

1. Can an unerupted tooth, or a partly erupted tooth, be left in situ with safety ?

2. Is the practice of prophylactic odontectomy[61,62] at an early age of development, a justifiable procedure, when non-eruption is deemed inevitable ?

The unerupted tooth has always been a challenge to the profession, and always will be, as the prevention of non-eruption in civilised races lies not within the field of dentistry, but within the Utopian field of Eugenics. C.Bowdler Henry[61] in 1937, warned the dental profession of the gravity of the problem : "The complications attending the maleruption of the third molar vary from minor discomforts to very grave illnesses, even to death." And, the dangers of these complications is hardly less today, despite the development of antibiotic and chemotherapeutic drugs.

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The pathology of the malposed tooth varies according to its degree of eruption. There are three distinct clinical degrees : the completely bone-embedded tooth ; the partly erupted tooth ; and the apparently embedded tooth, which, in fact, lies partly within bone, partly within soft tissue, but which clinically, has no connection with the oral cavity.

IS THE COMPLETELY BONE-EMBEDDED TOOTH STERILE ?

This question is of obvious significance : the controversy of focal infection still rages, although not so fiercely as in former decades. Thoma[62] feels that "focal infection of dental origin is definitely a factor in some somatic diseases." In 1948, an Australian research group[63] listed three unproven assumptions as a basis of the concept of focal infection, viz, (i) the area at the apex of a dead tooth is infected, (ii) these toxins are absorbed from this area, and (iii) these toxins exert an influence on susceptible tissues elsewhere in the body. It would, in my opinion, be foolhardy to ignore possible areas of dormant or chronic infection, on the grounds of one's disagreement with the concept of focal infection: too little is known about the nature of the ailments supposedly caused by a focus of infection. If the completely embedded tooth is a focus of infection, then it should not be left in situ, unless'the cure is worse than the disease!

Ito(1929)[64] carriedvout bacteriologic investigations of the pulps of partly erupted mandibular third molars, extracted on account of pericoronitis. In each case, the pulp, which he freed from the tooth by splitting, on culture, produced a growth of streptococci. The technique of surgically removing an impacted tooth is unsatisfactory for such bacteriological investigations. No precautions will evade the inevitability of oral contamination.

Lucas (1929)[65] carried out similar studies, and in each of 5,000[600] mandibular third molars removed, a positive culture was produced, from the capsule. Durbeck,[65] accepting the validity of Lucas' findings, stated that : "In the completely covered impacted teeth this case of/ functionless structure is an excellent medium for a haematogenous bacterial invasion, and in cases in which there is communication with the oral fluids, bears the added brunt of invasions by the micro-organisms from that source. Its status may be considered similar to that of the vermiform appendix, the prophylactic removal of which frequently bears the blessing of good surgical judgment during laparotomies for other purposes." It may be gleaned from his statement

that Durbeck believes that completely embedded teeth should be removed as the follicle must inevitably become infected by the haematogenous route.

Logan[66] criticised the methods of Ito and Lucas, on two grounds : (i) that oral contamination, even in the simple extraction, is almost impossible to avoid ; (ii) that "the presence of micro-organisms" and " infection" are not synonymous. The finding of bacteria on the capsule and in the pulp does not necessarily indicate that these tissues are infected. "Infection is characterised by the actual presence of vascular and cellular reaction of the tissue to the presence of bacteria." The tissue reaction may be demonstrated histologically.

Logan's Experiments.- Autopsy specimens from two individuals were studied histologically. One was a block containing the erupted maxillary second molar, the completely embedded third molar, the supporting tissues and the oral and antral mucosa. The other was a block comprising an embedded mandibular third molar, with supporting tissues, oral mucosa.

Logan's Findings.-

1. The follicle was in all respects like the healthy follicle of a tooth undergoing physiological intra-osseous eruption. No inflammatory changes were noted.

2. The periodontal membrane was typical of the unerupted tooth: thinner than that of a functional tooth, with fibrous tissue not orientated in bundles, as in the erupted tooth. There was no inflammatory change. The lamina dura was intact and thin.

3. The pulp showed (i) atrophy, (ii) fibrosis, (iii) vacuolisation of the odontoblastic layer, (iv) a large number of pulpal calcifications, and (v) no vascular or cellular reaction.

Pulpal fibrosis and pulp calcification have been described by Kretsehmer and Seybold[66] as evidence of infection. Stafne and Szabo[67], Hill[70] and Willmen[66,70] indicate clearly that pulpal calcifications are not due to the effects of pathogens, but are physio-

logical changes ; 90% of adults teeth show pulp calcification, and it has been found in the normal teeth of children. Logan demonstrated the identical pulp changes in the normal erupted teeth of the same autopsy specimens. Hill found that the pulp, with age, changes : the amount of embryonal tissue and stellate cells diminish, vacuolisation of the odontoblastic layer occurs, and fibrosis develops.

Logan's Conclusions.- "From the normal appearance of the tooth follicle and from the total absence of any inflammatory reaction in or around the completely embedded teeth, it appears that such teeth merely represent a continuation of the pre-eruptive state." Kronfeld[71] agrees. The work of Kotanyi (1924)[72], Bauer (1926)[66], Kellner (1929)[66] on block specimens from human jaws, and Worman (1929)[68] on sections of a dog's jaw bearing several partly erupted and embedded teeth, confirm the findings of Logan. Worman found no inflammatory changes in embedded and extensively ankylosed teeth; however, if the embedded tooth became exposed to the oral cavity, local inflammatory changes ensued.

THE PARTLY ERUPTED TOOTH, AND THE TOOTH APPARENTLY EMBEDDED.

Gunter (1942)[69] states that "the nearer an impacted tooth approaches the gum, the more evident the pathology. An embedded tooth may partially or completely emerge from the investing jawbone but from general appearances, remain completely covered by the gum, which may be tense, inflamed, occasionally necrotic." He continues : " A communication becomes finally established during eruption with the mouth cavity whenever an impacted tooth breaks through the bone. Such an opening may be so minute as not to be visible, thus becoming an obscure portal of entry for saliva, food particles and micro-organisms to the capsule of the erupting tooth, ending with bone infection."

Once a communication with the oral cavity is effected bacteria may enter. A gum flap covers the crown, and offers excellent conditions for the culture of bacteria, and infection is further enhanced by trauma to the gum flap from extrusion of the opposing tooth. Anaerobic and micro-aerophilic bacteria are especially favoured by such a nidus, and therefore the partly erupted tooth may be a most signif-

-icant factor in the aetiology of cervico-facial actinomycosis, Vincent's infection, and the more common pyogenic infections which not infrequently spread to involve the jaw and the adjacent soft tissue spaces. Of infections arising from partly erupted teeth, a detailed account will be presented in a later section.

RESORPTION OF THE CROWN OF THE EMBEDDED TOOTH.

Stafne and Austin (1945)[70] made a study of resorption of embedded teeth, which lead them to the following conclusions :-

I. Any tooth which remains completely embedded, particularly over a long period, is liable to undergo resorption - enamel, dentine and cementum all being involved.

2. The incidence of resorption is higher in the later decades of life, although resorption may occur fairly early.

3. There appears to be a wide variation in the rate and extent of resorption, which may occur over a long period or, after a relatively short period of activity, may be arrested completely.

4. The resorption in itself is probably of no clinical significance. It is symptomless. Surgical removal of the resorbing embedded tooth is difficult, due to the ankylosis between tooth and bone.

The Mechanism of Resorption.- "Most often the resorption originates on the surface of the enamel, less frequently, at the cemento-enamel junction, and rarely, on the surface of the cementum."[70] Degeneration and disappearance of the reduced enamel epithelium must occur before resorption takes place. The enamel, once exposed to the vascular connective tissue, is a "foreign body" and resorption follows, by osteoclasts in Howship's lacunae. Enamel and dentine, when destroyed, are replaced by vascular connective tissue, in which spongy bone and marrow spaces are formed. In advanced cases of resorption, most of the tooth structure has been replaced, scattered islands of dentine alone remaining within the bone. Resorption is wrongly regarded as caries ; dental caries cannot occur in the completely embedded tooth. Histological study distinguishes between caries and resorption : in the former, the

dentinal tubules are plugged with bacteria, in the latter, dental tissue is removed by osteoclasts.

Radiographic Appearances.- The clear dense outline of the enamel surface is lost, and the radiolucent shadow of the follicular space is absent. The enamel surface is irregular ; sometimes large portions of the crown are missing, and replaced by connective tissue to simulate caries. The defects in the crown eventually show replacement with spongy bone. Many such teeth give a mottled appearance suggestive of multi-focal involvement.

Incidence.- Resorption of embedded teeth is not uncommon, in the latter decades of life especially. Stafne and Austin studied 300 embedded teeth which showed resorption, and 240 of these were completely embedded, the remainder partly erupted. Although the authors did not mention it, clinical experience suggests to me that many of those partly erupted teeth probably suffered resorption while embedded, and subsequently became exposed through alveolar resorption.

Of the 300 resorbing teeth, 8 were deciduous molars and 66 were supernumary teeth (61 of which were mesiodentes) ; the remainder were permanent teeth. Analysis of the permanent teeth is shown :-

	Maxilla	Mandible
Central incisor	4	1
Lateral incisor	1	0
Canine	106	17
2nd Premolar	2	7
3rd Molar	64	24

It is noted that resorption of embedded teeth is more common in the maxilla than the mandible. The mandibular third molar is the most frequently embedded tooth, yet is less frequently resorbed than either the maxillary third molar, or the canine.

The maxillary canine, the mesiodens, and the maxillary third molar are the teeth most frequently affected by resorption. The mesiodens, Stafne and Austin noted, is often almost completely destroyed by resorption, by 35 years of age, and it is the

tooth most deeply embedded.

Hitchin[71] found that 8 out of the 109 impacted canines studied, showed resorption, 5 of which occurred in edentulous patients, with a long denture history. No guide to the frequency of resorption may be gleaned from these figures, as the incidence must be correlated to the period the tooth remains embedded.

Stafne and Austin found that 6 of the 300 embedded resorbed teeth studied, showed cyst formation. They observed that "in such cases, resorption no doubt takes place before the development of the cyst." Three canines, one mandibular third molar, one mandibular second molar, and a mesiodens, underwent cyst formation and resorption.

Helzberg[72] reported a case of dentigerous cyst involving the mandibular first molar, in a boy aged 9 years : the entire crown of the tooth had undergone resorption, with replacement firstly by vascular connective tissue, then by bone. Only the roots of the tooth were found in relation to this dentigerous cyst, and that active resorption was occurring, was indicated by the osteoclasts lined on the occlusal surface of the roots, within their Howship's lacunae. The case remains an enigma, and no parallel case is to be found in the literature.

DEPOSITION OF CEMENTUM ON THE CROWNS OF EMBEDDED TEETH.

Kronfeld[73] noted that if a tooth remains embedded over a long period of time, the enamel epithelium may become atrophic and disappear. Then the connective tissue of the follicle may deposit cementum directly on the enamel surface. Frequently, this deposition of cementum occurs near the cemento-enamel junction, and then extends by spurs or tongues, toward the crown. Kronfeld noted that in some embedded teeth, the fissures of the occlusal surface were plugged with secondary cementum.

Stones[13] observed that coronal cementum was a physiological feature of certain herbivore teeth. In man, it is rare,

he noted, and should be regarded as pathological. I am not happy about the choice of the term 'pathological' and prefer to regard coronal cementum deposition, and coronal resorption as physiological processes, innocuous, and associated with ageing processes in the embedded tooth.

HYPERCEMENTOSIS OF THE ROOTS OF EMBEDDED TEETH.

Stones has found that hypercementosis of the roots of embedded teeth is more commonly found than coronal resorption ; Stafne and Austin[70] do not agree. The aetiology of hypercementosis of embedded teeth and of erupted teeth is obscure. There is considerable diversity of opinion as to whether the condition is a physiological or pathological process. It is an important aspect of the question of embedded teeth, and in the past, any manifestation of cementum deposition to the roots of mature teeth, erupted or otherwise, was sufficient reason for their extraction.

Hypercementosis may affect the individual tooth, or it may be generalised. Generalised hypercementosis is frequently associated with Paget's disease of bone[74] ; Kupfer[62] noted that it was present in 80% of persons afflicted with toxic goitre ; Stones[13] claims that cases in which there is a gouty diathesis, or a hereditary tendency, have been reported. Obviously, generalised hypercementosis rules out chronic infection. In individual teeth, chronic infection of the non-vital tooth, appears to predispose to diffuse hypercementosis. Kellner (1931) made a study of the degree of hypercementosis in patients, with some teeth in function, others afunctional : he found that diffuse cementosis occurred in the afunctional teeth to a greater degree than in functional teeth. Functional teeth have a wide periodontal space, and little cementum deposition ; afunctional teeth a narrow periodontal space, and progressive cementum deposition. Logan[68] noted that embedded teeth have a narrow periodontal space, and in comparing the histological appearance of an embedded mandibular third molar, with the erupted third molar of the opposite side, in the one autopsy specimen, found "the root of the embedded tooth is covered by very heavy layers of cementum, this being considerably

thicker than that of the corresponding tooth on the opposite side..."

Burchard and Inglis[75] (1915), referring to hypercementosed teeth, made the following claims : "Neuralgia, functional blindness, functional deafness, chorea, epileptiform fits, paralysis, cardiac neuralgia, insanity and other related conditions, have been cured by the extraction of hypercementosed teeth." Bunting [75] (1929) echoes these claims in his textbook of oral pathology. How similar to the great variety of conditions'caused' by impacted teeth, and 'cured' by their surgical removal.

Zemsky [75](1931) appears to have made the first sound pronouncement on hypercementosed teeth : " Hypercementosis, having been found on unerupted, impacted and malposed teeth which presented no pathological signs, is a condition which cannot be due ~~regarded~~ in all cases, to infection as some writers believe, and therefore cannot always be regarded as pathological." I am of the opinion that hypercementosis of the embedded tooth and the erupted tooth which is not in occlusion, is a reflection of lack of function, and, rather than a pathological disease, is a modification to environmental conditions ; generalised hypercementosis, however, is not a sign of disuse, but a dental manifestation of a systemic disease or disturbance such as Paget's disease, gouty diathesis, toxic goitre, and should be regarded as a signal for a thorough systemic investigation of the patient so affected.

PATHOLOGICAL GEMINATION OR CONCRESCENCE OF TEETH.

Pathological gemination denotes mineralised union between two teeth after the roots have been formed. Thoma[62] attributes the cause to traumatic injury, or crowding of the teeth associated with pressure resorption of the interdental tissues : this may be followed by a deposit of cementum on the resorbed surfaces, joining the teeth together. Stones[13], however, takes the view that infection, apical, or periodontal, is the underlying cause, destroying the interdental bone, "until the roots are in contact, except for an intervening portion of chronic inflammatory tissue that has taken the place of

the remains of the periodontal membrane." Of these two views, I feel that pressure resorption of interdental bone, with perhaps the pressure resorption of contacting roots, followed by cementum repair and fusion is the probable aetiology. Destruction of the periodontal membrane by pocket formation is likely to cause cementum deposition to cease, rather than to stimulate it.

The teeth most commonly affected are the impacted maxillary third molar, and the second molar ; Stones has reported the concrescence of the impacted maxillary second premolar, and the first molar ; Thoma has reported the concrescence of the impacted canine with the two maxillary premolars. I have not seen nor read of, the concrescence of the mandibular third molar with the second molar ; usually the crown of the third contacts the second molar. However, in a distoangular impaction of the third molar, the force of the roots against the apical portion of the roots of the mandibular second molar, is sufficient at times, to displace the latter ; quite conceivably there could be concrescence of the third and second molars of the mandible.

Pre-operative radiographs do not always lead to the diagnosis of fusion. Superimposition of the roots of one molar over the roots of the other could simulate concrescence. An X-ray at right angles may help to demonstrate fusion. It happens, however, that concrescence of teeth usually is not realised, until the attempted removal of one or the other tooth. It is best to be ~~beware~~ aware of the teeth which may fuse, and the circumstances under which fusion usually takes place. In such cases, a broad flap should be raised, in order that both teeth may be removed, should fusion subsequently be demonstrated. Concrescence should be suspected in third maxillary molar impactions, of the horizontal distal, distoangular, and occasionally the vertical types - the roots of such teeth may actually be "intertwined" with those of the second molar.

MIGRATION OF THE UNERUPTED TOOTH.

Erupted teeth are not static, but undergo migration in an occlusal and mesial direction, to compensate for attrition. Although scant information has been published on the topic, unerupted teeth are capable of a surprising degree of mobility.

Cooper (1955)[76] reported a most interesting case which demonstrates the mobility of the embedded tooth throughout life. A medical practitioner aged 38 years, complained of excruciating pain spreading to the head and neck of two days duration, and acute twinges preceded by exertion over two years. A radiograph revealed an inverted lower third molar encroaching on the inferior dental canal. Due to a lesion associated with the first molar, radiographs of the region had been taken over a period of sixteen years, and included the third molar. The position of the third molar varied as follows :-

(1) At 23, the third molar was unerupted and mesioangular, but not in contact with the second molar.

(2) At 28, it was horizontal, and transversely placed, whether to the buccal or lingual could not be determined.

(3) At 31, it was horizontally and mesially inclined, still not in contact with the second molar.

(4) At 38, it was inverted, and either buccally or lingually deflected, and radiographically encroaching on the mandibular canal.

Wahl (1947)[77] reported a similar case, involving an unerupted lower incisor and supplemental tooth, the positions of which varied over a period of 6 years as follows :-

(1) At 11, the teeth were horizontal and lingual to the lower right central and canine.

(2) At 12, lingual to the premolars.

(3) At 14, apical to the premolars and first molar.

(4) At 16, buccal to the premolars and first molar, but lying near the lower border of the mandible.

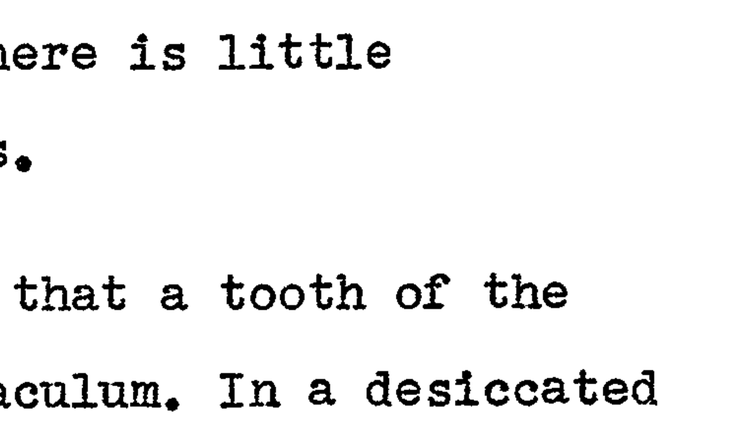
The unerupted third molar which lies in the neck of the condyle poses a problem. By what method could it have assumed such a position ? One possible explanation is that it was formed in this location : this explanation is not feasible, when the dental lamina and mandibular growth physiology is considered. It could have erupted into the condyle neck, as a result of aberrant tooth germ axis : such an explanation would be credible, if the occlusal surface lay toward the condyle. However, Nodine reported two cases in which aberrant eruption is an unsatisfactory explanation :-

1. An inverted mandibular third molar, its apices directed towards the articular surface, was discovered embedded in the condylar process ; cystic displacement was not evident.

2. An inverted third molar, embedded in the coronoid process, its apices directed towards the tip, was discovered.

To have assumed such positions, migration in an anti-eruptive direction must have occurred. It is, of course, possible that the tooth germs were aberrant in axis, and eruption to these remote regions took place, whereupon the teeth 'turned turtle' in a manner similar to the case reported by Cooper: there is little space for mobility in these constricted locations.

Atkinson (1950)[79] pointed out that a tooth of the molar series erupts along the line of the gubernaculum. In a desiccated skull in which there was discovered an aberrant third molar, the gubernacular channel disclosed the direction of migration, which was obviously bodily and unrelated to eruptive force. The gubernacular channel is a definite canal similar to the inferior dental canal. Atkinson was mainly interested in the fate of the gubernaculum, if such an aberrant tooth is removed, without curettage of the gubernaculum.



Eruptive force seems to be relatively ineffective when the crown is covered by a layer of solid bone ; the number of surgical exposures of lower premolars necessitated through premature extraction of their predecessors attests. Migration, however, appears to proceed in solid bone without difficulty,

and the process is independent of eruptive force.

Conclusion.- Intra-osseous migration of an embedded tooth may be explained by the constant remodelling of bone. Weinmann and Sicher claim that the mandible is rebuilt every three months, simply be the process of resorption and apposition, necessitated by the short life-span of the osteocyte. It appears to me a feasible explanation that the embedded tooth is caught up in the ebb and flow of constant bone remodelling, and this theory, is lent support by the migration of metal implants in the mandible, such as alveolar or lower border wiring, and schrapnel.

Migration of tooth-impacted teeth is illustrated by those cases of mandibular third molar impaction in which the contents of the inferior dental canal pass through a foramen in the root : a lateral jaw radiograph not infrequently shows the canal to make an abrupt curve at the point of intersection with the third molar root, and this reflects migration of the third molar after the invagination of the canal contents.[74]

Significance of Intra-osseous Migration.- An embedded tooth may migrate to remote regions of the jaw over a period of years. A lower third molar, embedded in a location easy of surgical access, may, if left in situ, migrate to a surgically inaccessible site such as the condylar process. If the contents of the mandibular canal pass through a perforation in the root of an embedded third molar, migration of such a tooth could be the cause of neurological symptoms.

DENTIGEROUS CYST FORMATION.

Definition.- A dentigerous cyst belongs to the group of odontogenic cysts, which are derived from epithelium connected with the formation of the tooth. Into the cyst cavity of the dentigerous cyst, projects the crown, in whole or in part, of an unerupted tooth, according to whether the cyst is of the central or lateral type.

Classification.- Is the odontogenic cyst a tumour, or is it a separate entity ? Bland Sutton (1922)[13] considered it to be a neoplasm, produced by irritation. There is evidence to suggest that radicular cysts resolve on successful root canal therapy of the causative tooth

in many cases. Denston (1948)[80] believed that residual periapical 'areas', radicular cysts included, undergo in most cases, complete resolution "unless there is some activating factor present, that has not been removed when the causal factor was eliminated" Once cells have been stimulated to neoplastic activity, their growth is henceforth autonomous, and removal of the cause does not interfere with the inherent potentiality of unrestrained proliferation. It would appear that the radicular cyst at least, is not a neoplasm, but an inflammatory reaction - hence the term 'cystic granuloma' which is sometimes used to describe it. The radicular cyst is considered by most authorities to be a reaction to an irritant, which is frequently not of purulent bacterial origin, as the cyst in these cases is regarded as sterile. Denston considered that the irritant, if of bacterial origin, must be of low virulence, and that if the irritant is virulent, the presence of a sinus, either external or through the pulp canal, is essential. There is no agreement to date, on the nature of the irritant causing the dentigerous cyst, and the theories which have been proposed, will be discussed in a later section.

While it may be claimed on sound evidence that the odontogenic cyst is not a neoplasm, there is every reason to suspect that the epithelial lining of such cysts possesses a definite neoplastic potentiality. Cahn (1933)[81] published cases illustrating the transition of odontogenic cyst to adamantinoma. Carpenter and Thoma (1933)[82] and Thoma and Proctor (1939)[83] reported similar cases of adamantinomatous transition to confirm Cahn's observation. Bernier et al. (1942)[84] claimed that 33% of all adamantinomas originated from odontogenic cysts. Even more alarming changes have been levelled against the odontogenic cyst epithelium, for Gerke, in 1948, reported two cases of odontogenic cyst , observed over a long period, which suddenly gave rise to squamous cell carcinomata. Trauner (1948)[85] found four cases of squamous carcinoma which developed from dentigerous cysts of the unerupted maxillary canine (two cases) and of the mandibular third molars. Thoma[62] stated : " Primary carcinoma does not occur in other bones but in the jaws it develops from cell rests remaining from the enamel organ and sheath of Hertwig, from enclaved epithelium of the location of facial fissures, and from the lining of cysts." Lucas[86]

(1954), though emphasising that he was not sceptical of the neoplastic potentiality of cysts, nevertheless published that he felt that it is often difficult to distinguish between mere hyperplasia of cyst epithelium, and frank neoplasia ; neoplastic changes, he considered, may arise from such epithelium, but not as frequently as claimed; he was of the opinion that many adamantinomas claimed to have arisen from odontogenic cysts, were in the first instance, unilocular adamantinomas, of cystic type. There is sufficient evidence to indicate the neoplastic potentiality of cyst epithelium, and such conservative techniques as marsupialisation of odontogenic cysts should be condemned - thorough enucleation of the cyst lining being the safest measure against neoplastic complication.

The Mechanism of Cyst Growth.- James (1926)[13] believed that the odontogenic cyst arose through proliferation of epithelial cells, with the central cells undergoing necrosis, on outgrowth of their blood supply. Necrosis is followed by liquefaction, and there is increase in size of the mass through uninterupted internal tension.

Toller(1948)[13] showed that in the degenerating cell mass, complex protein molecules of very high molecular weight are broken down to more numerous molecules of lower molecular weight, and a rise in osmotic pressure results. Tissue fluid therefore diffuses into the central zone according to the laws of osmosis, to dilute the degeneration products. Toller believed that osmotic pressure produces a heightened hydrostatic pressure within the cyst, which causes its progressive expansion. Should rapid epithelial cell proliferation initiate cyst formation by outgrowth of blood supply, one would expect to find cyst development in the basal cell carcinoma : according to Kramer, of the Eastman Dental Hospital, London, a fortunate section of a basal cell carcinoma will show cyst formation occurring within a cell mass - the superficial nature of the lesion, however, prevents extensive cystic expansion, and the cysts rupture to the surface, thereby causing the ulceration which is typical of the lesion. It would be logival to assume that the epithelial cell masses of the squamous carcinoma should likewise manifest cystic degeneration : the epithelial cells of the squamous carcinoma, due to their keratin-producing potential, on

outgrowth of their blood supply, do not liquefy, but degenerate to a central keratin plug, to give the typical "epithelial pearl."[87]

Aetiology of the Dentigerous Cyst.- There are two schools of thought concerning the aetiology of the dentigerous cyst : it is thought to have an intra-follicular origin by some, an extra-oral origin by others.

1. The intra-follicular theory.- Thoma[62] stated " that the central type of dentigerous cyst forms in that part of the enamel organ overlying the occlusal surface where amelogenesis is just being completed, and the epithelial cells are undergoing retrograde changes." He continued that "the lateral type of dentigerous cyst is formed like the central one except that it develops at a time when the central part has become atrophied. It occurs,therefore, in the lateral part of the enamel organ, where the stellate reticulum persists." Two conclusions may be drawn from Thoma's statement : (i) that the dentigerous cyst arises from the enamel organ ; (ii) that cystic degeneration affects the stellate reticulum, at a time before it atrophies to unite with the other cell layers of the enamel organ, to form the reduced enamel epithelium. Weinmann and Gilette[88] criticised these views on the ground that maturation of the enamel would be affected, because diffusion from the follicular vessels would be interrupted by the cyst ; hypoplasia is not a characteristic of the enamel of the tooth involved in a dentigerous cyst. The argument is not valid, however, as Lefkowitz, Shapiro and Bodecker (1947)[89,89a] showed that the source of the minerals which mature enamel, diffuse from the vessels of the dental papilla.

Thoma limited his conception of dentigerous cyst formation to a short interval after the enamel matrix formation. The stellate reticulum disappears as a separate entity, as soon as the reduced enamel epithelium forms. The secondary cuticle forms before the normal eruption time of the tooth . It is difficult to apply his theory to those cysts which arise from embedded teeth, long after the normal eruption time. That a dentigerous cyst may arise in the embedded tooth long after its eruption time, is believed by Rushton, who advocated prophylactic removal of embedded teeth from the jaws of persons with cleido-cranial dysostosis to prevent cyst development , and by

by Stafne and Austin[70] who found that cysts developed in some cases, after coronal resorption of the embedded tooth had been established.

2. The Extra-follicular Theory.- Bloch-Jorgensen (1930)[13] believed that the dentigerous cyst forms by the eruption of a permanent tooth into a cyst cavity, formed either by a radicular cyst of a deciduous tooth, or from epithelial debris, outside the follicle of an unerupted tooth, such as the so-called epithelial coils or the globes epidermiques. Malassez, a supporter of the extra-follicular school, believed that the tooth erupts into the cyst which obstructs its path of eruption, the epithelium of the follicle uniting with the cystic epithelium, in the same manner as union with the epithelium of the oral cavity. Weinmann and Gilette (1958)[88] published microphotographs of actual eruption of a tooth into a cyst cavity, and thereby proved that some dentigerous cysts at least, have an extra-follicular origin. Pekarsky(1950)[90] described a cyst of the mandible which contained the crowns of seven teeth, which lends support to the view that teeth may erupt into a cyst cavity.

Stones[13] takes the view that several different origins are possible in dentigerous cyst formation. Tratman[91] also believes that several different origins are possible, and in his opinion, these are by the eruption of a permanent tooth into a dental cyst of a deciduous or a permanent tooth, and by a "cyst of eruption".

What Factors Initiate the Formation of a Dentigerous Cyst?

1. The Inflammatory Theory.- Those who believe that the dentigerous cyst is caused by a radicular cyst of a deciduous or permanent tooth, which embraces the crown of an unerupted permanent tooth, believe that pulpal infection is necessary. Bloch-Jorgensen attributed the cysts forming from extra-follicular epithelial debris, to inflammation, such as is caused by peritonsillar infection, stomatitis, or gingival ulceration. Fischer (1911)[13] verified the theory by producing cysts in a cat, by artificially inducing abscess formation.

It is unlikely that many dentigerous cysts have their origin in radicular cysts of the deciduous predecessors. Periapical infection of the deciduous teeth involves the deciduous molars more frequently than any other teeth. A radicular cyst of the maxillary canine

would be a rare occurrence indeed, as the deciduous canine is the least susceptible in the maxilla, to caries and traumatic injury. And yet, the maxillary canine is the tooth most frequently involved in dentigerous cyst formation, of those permanent teeth with deciduous predecessors.

How common is radicular cyst formation in the deciduous dentition ? This has been a subject neglected by the majority of writers on oral pathology. Denston [80] claimed to have seen only two true radicular cysts of deciduous teeth, and attributed this rarity of occurrence "to the comparatively short duration of the chronic infective process and to the prevalence of acute infective processes." Sprawson [80] was the first to describe a dental cyst of a deciduous tooth (1922). Although the aetiology of chronic periapical lesions in the deciduous teeth is the same as in permanent teeth, the lesions of the deciduous teeth are "subject to greater variations in size and structure, and are characterised by a predominance of cellular elements over the fibrous components." Of the 50 periapical lesions Denston studied in deciduous teeth, 4% were cystic granulomata, the tooth incidence was molars 88%, and cuspids 12%, the position incidence was uppers 21%, lower 79%. It is evident, therefore, that radicular cysts of the deciduous dentition are rare, and the radicular cyst incidence of maxillary deciduous canines extremely low. On these grounds, I consider that radicular cysts of the deciduous dentition, play an extremely minor role, if any, in the formation of dentigerous cysts of the permanent dentition.

2. The Mechanical Theory.- Centano (1944) [13] considered that a dentigerous cyst arises from mechanical difficulties encountered during eruption and which act on the coronal aspect of the tooth follicle and enamel organ. As a result of the irritation, it does not follow the usual process of involution, but cystic formation occurs in the epithelium concerned in the process of tooth formation. In substantiation of this theory, the teeth most frequently involved in cyst formation, are the teeth which show the highest incidence of impaction. In descending order of frequency, the teeth most commonly involved are the third molars, the maxillary canine, and the mandibular second premolar.

3. The Familial Factor.- A familial factor has been recognised in the aetiology of the dentigerous cyst. Beyrent (1938) [92] found dentigerous cysts in four members of one family. Thoma and Blumenthal (1946) [93]

reported dentigerous cysts in four generations of a family of 30 members, of which twelve had one to three cysts each.

4. Individual Susceptibility to Dentigerous Cyst Formation.- Multiple dentigerous cysts are an occasional finding : although there are several cases reported, it must be remembered that the finding of a single cyst is probably regarded as rather commonplace and would not be reported in the literature, unless associated with an unusual feature, whereas the finding of multiple cysts would more likely be published. Oliver (1934)[94] reported dentigerous cysts involving all four third molars, both maxillary canines, and a mandibular second premolar. Seeman (1937)[95] reported dentigerous cysts involving right maxillary, and right and left mandibular second and third molars, and the left maxillary third molar. Individual susceptibility to dentigerous cyst development has been reported also by Bennett (1937)[96], Myers (1943)[97], McGregor (1949)[98], Catania (1952)[99], Tam (1955)[100], Caldwell and Thompson (1955)[101], It may be concluded that a small percentage of the population have a particular susceptibility to the formation of dentigerous cysts. This would suggest that the local accidental factor of peritonsillar infection, stomatitis, periapical infection of the deciduous teeth, if it does cause dentigerous cysts, would certainly not explain multiple cystic involvement, nor the noted familial tendency.

5. Congenital Malformation.- Kronfeld[102] suggested that a congenital malformation of the epithelium of the enamel organ could be responsible for the development of the dentigerous cyst. He did not offer his reasons, nor illustrate with examples, but the theory is an attractive one. Hereditary enamel hypoplasia is characterised by congenital 'weakness' or 'malformation' of the reduced enamel epithelium, and dentigerous cyst formation is also abnormal behaviour of the reduced enamel epithelium.

Discussion.- The evidence available indicates to me that there are two important factors in the aetiology of the dentigerous cyst : (i) that a predisposition to cyst formation is found in some individuals, and this predisposition may be inherited; (ii) the predisposition, congenital or inherited, does not affect all teeth of the dentition equally, the cysts

occurring not at random but almost always in association with those teeth most commonly impacted.

Does dentigerous cyst formation cause 'impaction' of the affected tooth, or does impaction or non-eruption cause cyst formation ? It is probable that in most cases, a dentigerous cyst is secondary to impaction or non-eruption, but the occasional case does suggest that the dentigerous cyst development prevents the eruption of the involved tooth : Tam's case of bilateral mandibular first molar cystic involvement is probably a case of primary cyst formation. Fish (1948)[103] stated that neither infection of a deciduous tooth, nor impaction, could explain the established cyst of a third molar of a 15 year old boy (which case he described).

The exact mechanism of cyst formation is vague. This is probably due to the very scant knowledge of the physiology of the enamel organ and its final developmental stage, the reduced enamel epithelium. So very little is known of the function of the stratum intermedium, and the stellate reticulum, during enamel formation ; and of the nature of the secondary cuticle, the reduced enamel epithelium fabricates, during formation of the epithelial attachment. The reduced enamel epithelium, when its intended function is upset, permits (i) resorption of the enamel, (ii) deposition of cementum on the enamel, (iii) dentigerous cyst formation.

<u>The Resorption of the Enamel and Dentine of the Crown of a Tooth Involved in a Dentigerous Cyst</u>.- Protzel (1955)[104] reported a dentigerous cyst with early ameloblastic changes, involving an 'anomalous' molar in the mandibular first molar region, of a Negro female aged 37 years ; the crown showed radiographic evidence of 'internal resorption'. Helzberg (1948) reported complete coronal resorption of a tooth in a dentigerous cyst, in a boy aged 9 years.During a routine examination, the left mandibular first molar was noted missing. Radiographs revealed " a radiolucent area in the body of the mandible in this region. This large area (2 cms. diameter) was outlined clearly by normal bone. In the centre of the area were two molar roots, and at their occlusal surface, a radiopaque image having a 'cotton wool' appearance, was evident." Microscopic examination of decalcified sections of the roots showed " the dentine at the occlusal surfaces of the roots was undergoing resorption. The presence of Howship's lacunae and a few scattered giant cells having the appearance of osteoclasts

attested to this. It cannot be concluded that the entire crown has been resorbed, because the possibility of incomplete formation of the crown must be considered. But it is certain that resorption has accounted for the loss of some tooth substance, and that the destruction is not caused by caries. If caries has caused this process, bacteria would have been seen in the dentinal tubules.The dentine which has been resorbed is being replaced by fibrous tissue and at many places, by bone. The pulp was normal. The irregular radiopaque shadows seen in the Xrays of the occlusal surfaces of the roots, is spongy bone."

The above case reported by Helzberg is difficult to understand, and there is no parallel case reported in literature to my knowledge. Resorption of the crown of a tooth in a dentigerous cyst has been noted when the cyst becomes infected.

PRESSURE EFFECTS OF AN IMPACTED TOOTH ON THE ADJACENT TOOTH.

In its attempt to erupt, an impacted tooth exerts considerable force on the root or crown of the tooth which impedes its eruption. The following sequellae have been reported :-

(i) Root resorption of the adjacent tooth.
(ii) Displacement of the adjacent tooth.
(iii) Splitting of the root of the adjacent tooth.

ROOT RESORPTION OF THE ADJACENT TOOTH.

If an erupting tooth drives against the root of an adjacent tooth, thereby becoming impacted, pressure resorption of the cementum and dentine of the root may occur, especially if no movement of the tooth is possible. Eruption is not a uniform, continuous movement in an occlusal direction, but is characterised by intermittent bursts of activity. The pressure effects upon the root of the adjacent tooth is likewise intermittent; a phase of activity is followed by a period of rest. During the active period, the cementum and dentine are resorbed by osteoclastic action; during the period of rest, reparative processes ensue, and cementum is deposited over the resorbed dentine surface. Resorptive processes usually dominate, and pulpal exposure may result,

with devitalisation of the tooth, Kronfeld[102] claimed. Eventually the root may be severed. Stones[13] noted that secondary dentine is laid down in reaction to resorption, and pulpal obliteration may result.

Devitalisation of the resorbed tooth once pulpal exposure is established, is not accepted by all authorities. It is my opinion that even complete destruction of the root does not cause loss of vitality, unless there is a communication between the pulpal tissue and the oral cavity. Several cases illustrate this :-

(i) Masson (1954)[105] found that on extraction of the mandibular second molar, a tooth with a history of intermittent tenderness, a concavity disclosing resorption by an unerupted supernumary bicuspid was visible : the pulp chamber and one root canal were obliterated by secondary dentine, and the remaining pulp tissue proved vital.

(ii) Johnson (1946)[106] reported almost complete destruction of the root of a maxillary central incisor by an embedded canine : despite pulpal exposure, the pulp reacted normally to electrical stimulation

(iii) Angelman (1957)[107] reported pulpal exposure of the mesial root of the mandibular first molar by resorption, caused by an impacted second premolar ; so great was the force exerted, that the first molar was driven distally, into the position of the extracted second molar ; the first molar remained vital and symptom-free.

(iv) Sealey (1955)[108] reported several cases illustrating the rapidity with which resorption of the adjacent roots may occur.

Case 1 : A female aged 21, manifested extensive resorption of the roots of 21/ and both teeth were vital ; an embedded canine resorbed slightly more than half the root of each, resulting in their mobility.

Case 2 : A female aged 12 showed on radiographic examination, the complete severance of the apex of the central incisor from the rest of the tooth. Six months after removal of the cause - the embedded canine - the tooth was vital and firm ; pain was not a feature.

Case 3 : A female aged 12 suffered complete loss of the root of the lateral incisor, with severance and isolation of the apex, caused by an unerupted canine. The lateral incisor was vital, but so mobile that its extraction was unavoidable. The same patient suffered severance and isolation

of the apex of the root of the lower first molar, caused by a distoangular impaction of the second bicuspid.

The above cases clearly show that resorption of the root of a permanent tooth by pressure resorption, is in no way different from loss of root substance, in the process of normal exfoliation of a deciduous tooth. The resorbed tooth remains vital, and becomes progressively more mobile ; pain is generally not a feature, although tenderness is a symptom in some cases - probably through hyperfunction (caused by possible supra-occlusion, and inadequate supporting tissue).

The teeth most commonly affected by pressure resorption by impacted teeth are : the maxillary lateral incisor; the distal root of the mandibular second molar, caused by the impacted third molar ; the maxillary second molar by the third molar ; and the mesial root of the mandibular first molar, caused by an impacted second premolar.

More than one tooth may be resorbed by an impacted tooth. Birtwhistle (1946)[109] reported resorption of the root of the left maxillary lateral incisor (resulting in its spontaneous exfoliation), the apical half of the root of the left central incisor, and deep resorption of the mesial surface of the right central incisor, caused by the maxillary left canine. Osbourne King[110] reported resorption of the root of a maxillary central incisor, caused by a mesial displacement of the lateral incisor, which was undergoing itself, resorption caused by the impacted canine.

Such cases as those quoted above, tend to present a somewhat exaggerated picture of root resorption, which is not a very common sequella of impaction. In most cases, when one tooth impinges on another, it is displaced, without root resorption, and this is a most common finding in the lateral incisor, which is frequently displaced labially by a palatally impacted maxillary canine (and less commonly by

high, labially placed maxillary canine). Hitchin gave an indication of the frequency of resorption by the impacted maxillary canine: in his study of 109 impacted canines, only 5 of 89 standing lateral incisors manifested root resorption.

Discussion.- In view of the severe damage which may be occasioned by the impingement of an impacted tooth on the root of the adjacent tooth, early diagnosis of impaction, and treatment is necessary. Sealey[108] concluded that an impacted maxillary canine could cripple the upper incisors, if ignored; his case reports indicate that resorption may occur at a very early age. Thoma[62] warned : "Portion of a tooth may be resorbed without any neuralgia or tooth displacement, and at times, owing possibly to radiopacity of the impacted tooth crown, the resorption may not show on the radiograph." It is difficult to surgically remove an impacted canine without devitalisation of the pulp of the adjacent tooth when severe resorption is present - immediate root canal therapy may be necessary to prevent its pulp infection.

DISPLACEMENT BY AN IMPACTED TOOTH OF THE ADJACENT TOOTH.

Teeth tend to be displaced, rather than resorbed, by pressure exerted by the impacted tooth.

" Extrusion of the second molar by impacted third molars will create an occlusal imbalance because of the traumatic relation of the second molars. Such chronic distortion of the masticatory neuromuscular balance will contribute significantly toward temporomandibular joint disturbances."[111]

ROOT FRACTURE OF A TOOTH CAUSED BY AN IMPACTED TOOTH.

Ausubel (1945)[112] published a radiograph which clearly demonstrated a transverse fracture of the distal root of a second molar, in relation to a transverse buccolingual impaction of the third molar. The second molar was caries-free, and the patient could report no history of trauma. The second molar showed periapical infection of each root. The bone around the third molar was normal, and therefore an attempted removal of the tooth appears not possible. Belding, editor of "Dental Items of Interest", assumed that the reason for root fracture

was the "biogenetic" force of the third molar in an endeavour to erupt.

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NEUROLOGICAL SYMPTOMS.

One cannot fail to note, on perusal of dental literature, the extraordinary diversity of neurological symptoms attributed to the presence of impacted and unerupted teeth.

Typical Case Histories.- The following case reports from the literature represent a reasonable cross section, in which neurological symptoms have been noted in association with the presence of impacted and unerupted teeth.

1. A man aged 27 years, complained of pain, facial twitching, and drooping of the corner of the mouth. All customary forms of treatment failed to bring relief. The removal of an impacted mandibular third molar was followed by the disappearance of his symptoms, and the restoration of normal facial expression. (Murphy, 1931 [113]).

2. A man aged 63 years, complained of drooping of the left eyelid, loss of the sense of taste, dysphagia, inability to masticate, loss of normal function of the right arm and leg, and melancholia, whose symptoms were relieved by removal of an embedded mandibular third molar. (Engel [114])

3. A girl aged 17, complained of intense pain in the mastoid region, and excruciating tenderness upon pressure. Her physician diagnosed middle ear inflammation, and ordered the removal of her tonsils, which failed to afford relief. An E.N.T. specialist ordered the extraction of an infected maxillary incisor, which was found to be unrelated. Eventual removal of an impacted mandibular third molar brought cessation of pain, and a general improvement in her health. (Nodine, [115], 1948).

4. A girl aged 21, a student nurse, was showing signs of insanity. Medical examination revealed no apparent cause. An impacted mandibular second premolar, impacted against the first molar, which showed root resorption, was revealed by radiographic study. Upon removal of these teeth, her symptoms disappeared. She gained 16 pounds in weight, and later held a secretarial post of high responsibility. (Nodine[116], 1944).

5. A woman aged 40 years, complained of intense pain of the right side of the head, neck and face ; she was "unable to eat or sleep for several months" and appeared weak and emaciated. She had had intense headaches for 24 years, and a facial paralysis on the right side periodically, one attack lasting several weeks. An inflamed swelling on the right palate led to the diagnosis and removal of a horizontally embedded second premolar, the crown of which lay to the palatal of the central incisor. Disappearance of the pain and facial paralysis, with complete recovery of general health, followed. - Thorpe,[117] (1904).

Comment.- Consideration of the above case histories leads me to the following conclusions :-

1. That symptoms disappeared on removal of an otherwise "normal" unerupted tooth, appears to have led the authors to the assumption that therefore the symptoms/were caused by the unerupted tooth. Such an assumption may be correct in many cases, but in others, I feel that disappearance of the symptoms could have been coincidental with surgery, by suggestion, or could have recurred - many such case reports are written after an inadequate follow-up period.

2. Scrutiny of numerous similar case reports gives me the impression that only the successful "cures" are reported ; cases in which removal of the unerupted tooth fails to afford relief, or even augments the pre-operative symptoms, are rarely published. This represents a 'slant on the news', and tends to give the uncritical reader, the impression that the removal of impacted and unerupted teeth is the treatment of choice for all obscure neurological symptoms related to the face and dental tissues. Hasty condemnation of the unerupted tooth should not be considered until an organic cause, and a psychiatric assessment of the patient has been sought.

3. There is an urgent need for a diagnostic method, whereby the relationship, or otherwise, of the symptoms to the unerupted tooth, may be determined : such a method has, fortunately, been published by Haggett (1948)[118] and Griffin (1957)[119], and both have experienced promising results.

A Classification of the Neurological Symptoms Attributed to Impacted and Embedded Teeth.

1. Local pain.- This is not a common finding, unless there is sub-acute, or acute inflammation associated : pericoronitis, denture pressure, the infection of the follicle or cyst around the crown, usually cause local pain.

2. Pain referred to adjacent parts.- Unerupted teeth are rather commonly associated with referred pain, and such pain normally occurs within the distribution of the trigeminal nerve. Earache and pain over the auriculo-temporal region, are quite usual symptoms caused by the impacted mandibular third molar. Such pain is almost always found to be ipsilateral, an exception possibly being caused by the mesiodens, which is reported to set up bilateral referred pain.

3. Pain referred to distant parts.- Pain to the arm, the chest and the pericardium, is possible from the unerupted lower tooth, just as in coronary thrombosis, which sets up pain in these regions, may also cause pain in the mandible.[13] Case reports in the literature sometimes describe pain beyond the distribution of the trigeminal nerve, and, occasionally, bilateral or contralateral referred pain, difficult to explain on anatomical grounds.

4. Obscure headaches.- These are frequent complaints and the ache is at times so ill-defined, that Nodine terms it, the "all-over" pain. The patient not infrequently complains of a feeling of "pressure" which interferes with concentration, and is sometimes associated with insomnia. The headaches are distinct from referred pain in nature, and recent evidence suggests that migraine need not be intra-cranial only ; such headaches are not unlike migraine.

5. Atypical neuralgia.- This is any facial neuralgia which does not conform in distribution, to the trigeminal nerve. The symptoms are definite, the patient is in no doubt as to the distribution, but the anatomical explanation is not yet understood: the sensory component of the 7th cranial nerve, the autonomic system, are the likely explanations.

6. Mental symptoms.- Among the very many mental symptoms "cured"

by removal of unerupted teeth, may be listed the following - irritability, sulkiness, bad temper, insomnia, lack of concentration, aphasia, delinquency, kleptomania, neurasthenia, melancholia, depression, epilepsy, and insanity. The critical observer is balanced between the feeling of scepticism an the one hand, and the realisation that so little is understood of the central nervous system and personality on the other.

7. Motor symptoms.- Paralysis, hypertonicity or muscular guarding, twitching are reported. Involvement of smooth muscle - such as colonic spasm - has been reported[118].

8. Ocular symptoms.- The maxillary canine in particular is reported to cause impaired vision, myopia, choroiditis, optic neuritis, trachoma, sympathetic ophthalmia.

9. Aural symptoms.- Deafness, mastoiditis, otitis, tinnitus are occasionally associated with unerupted teeth, especially of the mandible.

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In my personal experience of impacted and embedded teeth, I cannot claim to have examined patients manifesting even a small proportion of these symptoms : one does not, however, require a vast degree of experience to recognize the frequency with which earache, auriculotemporal pain, perhaps tinnitus, and "deafness" are traceable to partly erupted lower third molars. It is my intention to arrive at an opinion regarding the reasonable assessment of the unerupted tooth : should such teeth routinely be removed as soon after discovery as possible, (suggested by Sealey and Amies), or may a more flexible, more conservative approach be justified according to the circumstances? The accusation that the impacted or unerupted tooth may cause insanity, blindness, and epilepsy, is a grave one, and on no account should it be dismissed lightly. I feel that the following subjects should be well understood, before such an implication may be answered :-

1. The nature of pain.
2. The sensory supply to the facial structures.
3. The neuralgias, typical and atypical.
4. The mechanism of referred pain.

The Nature of Pain.

Medvei[120] states that "pain may be regarded as a sensation in so far as it has a threshold, is localised, can be referred to a stimulus, and has - at least in the skin - specific end-organs as receptors ; it has established pathways, the severance of which can abolish pain sensations completely from the skin and deep somatic tissues, and it has representation in the spinal cord and in the lateral nucleus of the optic thalamus. In contrast to other sensations, however, it is 'much closer to the centre of personality'."

Pain cannot be separated from its emotion, neither as "unpleasantness" nor as "reaction to pain". Pain sensations entering the thalamus, along the trigeminal nerve branches, are there synthesised, and the thalamus adds a synergistic affective tone (Spector, 1949.[121]) Both the unpleasantness of pain, and the reaction to pain, may be modified by conscious control : some individuals react more violently to pain than others, not because the stimulus is greater, but because the nature of pain is more "unpleasant" to them. The emotional response to pain is variable, and the study of factors involved in self-control lie more in the field of psychology than in physiology. Intelligence and memory contribute to the emotional response : fear arises from memory of pain, and the applications of the past experiences to future circumstances ; pain becomes associated with definite objects and situations, e.g., the intelligent child's fear of the white overall. The imbecile and the baby have no memory of pain, therefore they do not suffer from fear, but experience "fright" from their unpreparedness when faced with pain at a future recurrence. Intelligence and memory, depending upon temperament, may contribute to a heightened emotional response in some, but a philosophical stoicism in others. Age, sex, race, upbringing, education, family and community influences, affect the attitude to pain. Every great cause has its martyrs.

The theshold of pain differs : it is raised by excitement (battle, sport, etc), analgesic drugs, hypnosis and suggestion; it is lowered by fatigue, general ill-health, and in persons of psychotic background, it may be very low indeed.

There is ample evidence to suggest that pain may be experienced in a part without actual organic disease. The pain in a phantom limb, the feeling of movement in a paralysed arm of the hemiplegic are cases where sensations cannot arise from peripheral stimuli. This led Medvei[120] to claim that "pain is not necessarily arising out of changes in the peripheral organs where it is felt, nor in the connecting nerve tracts, but in the body perception activities of the cerebral cortex." It is believed that each part of the body has its "image" permanently reflected in the cerebral cortex, the "image" being more "distinct" in those parts of the individual of which he is especially conscious - and the oral cavity is a particular example. The recurrence of tic douloureux following retro-gasserian section is an example of "phantom" pain. Yet, the nerve does not need to be destroyed, before phantom pain is experienced. Carbello (1956)[122] described a case of atypical facial neuralgia which he attributed to conversion, whereby unconscious mental conflicts are transformed into physical debility. It must be emphasised that patients experiencing pain, in a phantom limb, in a crushed nerve, or from conversion, actually feel pain : their pain is, to them, real, and very serious harm may be done, if they are accused of "imagining" it., and such patients always require very careful handling.

Of psychogenic pain, Walshe[120] stated that (i) anatomical mechanisms and ~~psychological~~ physiological processes are not involved, and (ii) it is not a primary sensation, but complex states of mind, emotionally toned ideas, like grief, anguish, and guilt. Keefe (1948)[120] defined psychogenic pain as characterised by (i) irregularity of occurrence with absence of organic cause, (ii) response on several occasions to placebos, such as distilled water ; and (iii) irregular response to potent analgesics. He was severely criticised for these views, as pain with demonstrable organic origin will respond to placebos, suggestion, and hypnosis. Psychogenic pain is, therefore, difficult to define, and equally difficult to diagnose. Innumerable patients have been done a grave injustice, when treatment has been refused, on the grounds that their pain is imaginary.

The Sensory Nerve Supply to the Facial Structures.

Facial sensibility depends chiefly upon the trigeminal nerve, but "as soon as the face is deprived of this source, some other nerve - either the 7th or the sympathetic, or both, take over - or it may be through anastomosis with branches of the cervical plexus. The zero of cutaneous sensibility can be reached not merely through resection of the peripheral nerve supply, but requires deletion of the sympathetic as well." (Stuteville and Levignac[123], 1953).

Facial pain may be classified accordingly :-

1. Trigeminal neuralgia :
   (a) cause known, or symptomatic,
   (b) cause unknown.

2. Pain of central or psychogenic origin.

3. Atypical neuralgias :
   (a) a complex group under the names of sphenopalatine ganglion neurosis, Vidian neuralgia, geniculate neuralgia, all of which suggest involvement of the sensory portion of the facial nerve :
   (b) sympathalgias, vascular headaches, and erythromelalgias, which appear to express the participation of the sympathetic and vascular sensibility.

Atypical neuralgia.- Stuteville and Levignac have published an excellent critical review[123] of the literature pertaining to atypical neuralgias, and have ably discussed what is known of the aetiology and anatomical mechanism of these neuralgias, in addition to which they have indicated the gaps in present day knowledge of the subject. Their conclusions are relevant :-

1. The facial nerve plays an important role in atypical neuralgias, because of its various functions, its many peripheral and central connections, especially with the 5th, 9th, and 10th nerves.

2. They believe that the autonomic system is involved in the pain producing mechanism, but conclude that the sympathetic system has no sensory function proper ; a reflex action from the autonomic system is the probable cause. The nerves of vascular sensibility are the most likely to provoke

a sympathetic or parasympathetic reflex action. The nerves to be considered are the 10th, the upper thoracic, the 7th and 5th nerves.

3. Associated with the pain is vascular change : there is disagreement as to whether vasoconstriction, producing sensitisation of nerve endings by chemicals, is the mechanism, or vasodilatation of arteries, to produce pain by stretching of the walls of the vessels. Migraine is pain with a vascular basis : vasoconstriction is first seen, which produces anoxaemia and pre-headache, followed by vasodilatation of arteries to produce pain. However, atypical neuralgia is observed to be at its height when there is marked, even intense, vasodilatation of the arteries.

4. There are many so-called atypical head and face pains regarded as separate entities, that are probably nothing else but migraine.

5. It has been noted that an injection of 0.3-0.5 mgs histamine will reproduce the migraine headache, which pain ceases with an injection of epinephrine. An allergic basis may be implied.

6. Sympathectomy (superior ganglion) stops some, not all, atypical neuralgias.

Referred Pain.-

1. Head's theory of its mechanism.-[124] Spinal nerves embryologically divide into a dorsal subdivision which supplies an area of skin and muscle and a ventro-lateral subdivision, which again divides to form a somatic and a visceral sensory nerve, the latter supplying an organ ; impulse from the diseased organ though unable to arouse a sensation, set up an "irritable focus" in the cord, with a result that the cells accustomed to receive impulses from the corresponding somatic area, are excited ; new impulses travelling from these cells to the higher centres, are projected by the thalamus to the somatic area, the skin or muscle whichever is accustomed to receive the impulse ; pain is felt in the skin, and muscular guarding is associated, from a diseased organ. Applied to the face, the trigeminal nerve may be regarded as supplying somatic sensory nerves to the skin and mucous membrane, and a "visceral" branch to the teeth. Head's theory does not explain pain felt remote to the afferent endings of the trigeminal nerve, contralateral pain, and the muscular effects reported to result from unerupted teeth.

2. Weisengreen and Winter (1952)[125] recognised the incongruities in the anatomical theories, and disregarding the mechanism of referred pain, plotted the pathways as clinically manifested. Their charts are of considerable practical importance.

3. Hyperactivity of autonomic reflex arcs.- The published work of Haggett(1948)[118] and Griffin (1957, 1958)[119] does much to explain the anomalous behaviour of pain referred from a dental focus. Haggett proved the association between inflamed pulps and distant symptoms, such as colonic spasm, by the temporary disappearance of the symptoms by infiltration of local anaesthetic around the affected tooth, and their permanent disappearance following extraction of the tooth : Haggett discovered thereby, a diagnostic method which can prove, or disprove, the association between symptoms and a suspected dental lesion. Griffin investigated the association more fully. Haggett attributed the mechanism of referred symptoms to "the disturbance of the sympathetico-parasympathetic balance of the autonomic system ," and Griffin termed the disorder "odonto-neuralgia-sympatheticus". Stimulation of afferent nerve endings set up hyperactive reflex arcs, which are capable of producing pain at, or near to, or remote from the peripheral afferent nerve endings of the trigeminal nerve, muscular guarding, rigidity or hypertonicity ; there is usually a psychogenic component.

4. Griffin's findings.- Of the 50 cases he studied, which showed the clinical picture of odonto-neuralgia-sympatheticus, 10% were caused by impacted teeth.

(a) distribution of referred pain:

75% suffered pain referred to the head, neck, shoulder and upper limbs ;

20% to the abdominal and lumbar regions;

5% to the lower extremities.

(b) other symptoms :

17% suffered paraesthesia;

7% malfunction of the gastro-intestinal tract ;

5% severe muscular inco-ordination;

3% anasarca.

Muscular guarding was usually found associated with the painful areas.

(c) By the diagnostic method of Haggett, Griffin was able not only to define the specific source of the symptoms, but also by "control" injections, at a site remote from the suspected focus, was able to assess the presence of the psychogenic component : this he found has an important bearing on the prognosis.

5. Psychiatric implications.- Edwards (1950)[126] stated : "Occasionally, somatic halucinations localised to the buccal cavity, occur as fairly isolated phenomena of a psychotic illness or more frequently, as part of a complex series of symptoms. Inexplicable "neuralgias", foul or sweet tastes, or peculiar sensations referred to the tongue, are of course more likely to be mistaken for manifestations of actual somatic disease...." Edwards claimed also that : " It is not at all infrequent in the study of hysteria to find patients who have courted surgical interference in order to gain the attention and affection that they crave."

How Impacted and Unerupted Teeth May Cause Pain.

It is generally believed that such teeth may cause pain by (i) inflammation, (ii) pressure effects, (iii) temporo-mandibular joint disturbances, (iv) ectopic eruption, and (v) muscle impingement.

1. Inflammation.- The completely bone embedded tooth is sterile (Logan). Pain from the embedded tooth could not be caused by inflammation. The partly erupted tooth may suffer caries, with eventual hyperaemia, pulpitis, and periapical infection ; its investing tissues may undergo inflammation, termed pericoronitis, which may result in pocket formation with bone destruction. The partly erupted tooth may predispose to caries of the adjacent tooth, and there may be periodontitis caused by food packing between their abnormally contacting surfaces. The unerupted tooth may become involved in a dentigerous cyst, which is usually symptom-free unless it becomes infected : the symptoms of acute inflammation then ensue.

2. Pressure effects.- The embedded and impacted tooth may cause pressure to a nerve trunk, or to the periodontal membrane of the adjac-

ent tooth, or the pulp of the adjacent tooth, when exposed by pressure resorption of the root.

(a) Pressure to a nerve trunk.- The most significant example is the pressure of the roots of a lower third molar against the inferior dental nerve. On removal of such a tooth, examination of the socket may reveal pressure resorption of the roof of the canal, and compression of the neurovascular bundle is a feasible deduction. Evidence that such pressure does cause pain, is suggestive rather than conclusive. I have neither seen, nor have I read of a case in which an embedded third molar caused paraesthesia or anaesthesia of the inferior dental nerve through implied compression, whereas paraesthesia is occasionally noted in the case of a rapidly expanding cyst or tumour. Even when the inferior dental nerve passes through a foramen in the root of a deeply placed third molar, the patient is unaware of symptoms in my experience, despite the considerable distortion of the canal caused by intra-osseous migration of the tooth. Thoma[62] believes that the developing third molar may cause an intermittent neuralgia when its roots impinge upon the mandibular canal, and he claims that the intermittent character of the pain is due to the sporadic activity of dentinogenesis. I am at a loss to understand how root development may cause pressure to the nerve trunk - the dentine matrix or predentine is uncalcified and therefore plastic, it conforms to the shape of the canal, rather than distorts it; this is reflected in the not infrequent grooving or bifurcation of the root or roots of the third molar. That neuralgia, involving the mandible and auriculo-temporal region, and at times the ear, does occur in association with an unerupted third molar, is not disputed. The mechanism whereby the pain is caused I feel, has not been conclusively explained.

(b) Resorption of the root of the adjacent tooth.- Thoma[62] quoted Sealey when he emphasised the susceptibility of the periodontal membrane to pain production caused by an impacted tooth; the periodontal membrane possesses a rich ennervation, and pain in the periodontal membrane is usually accurately localised due to the tactile end organs of that tissue. Several authors believe that exposure of the pulp by root resorption leads to pain and even devitalisation of the tooth. Sealey published several case reports in which apical severance occurred by pressure resorption - not only were the patients symptom free, but also

the affected tooth remained vital. It is my firm belief that pain does not arise from exposure of pulpal tissue occasioned by pressure resorption of the root, unless the exposure is in communication with the oral cavity.

(c) Temporo-mandibular joint disturbances.- A traumatic arthritis may result from adoption of a "bite of comfort" : this may follow trauma to the operculum of a partly erupted mandibular third molar, by its extruded antagonist. It has been claimed that a second lower molar may be driven into supra-occlusion by an impacted third molar[127], and this is said to result in eccentric occlusion, and a possible traumatic arthritis. Impingement of an aberrant tooth upon one of the muscles of mastication will likewise cause joint disturbances.

(d) Ectopic eruption.- A tooth may erupt into the antrum, nasal cavity, or through the skin. Almost invariably, infection ensues, which may initially be acute, and usually pursues a chronic suppurative course until the tooth is removed.

(e) Muscle impingement.- Should an aberrant tooth impinge upon a muscle, the latter is inevitably traumatised, and a painful trismus may follow. The upper third molar may drive against the attachment of the lateral pterygoid muscle, and it may become embedded in the body of the temporal muscle, should it erupt ectopically, into the post-zygomatic fossa. The aberrant mandibular third molar has been discovered in all sites of the ramus : due to the thinness of the ramus, the tooth must lie partly outside bone, generally in the region of a muscle insertion. One may glean from published case reports, that infection follows when an aberrant tooth lies embedded in muscle : the reason for infection cannot be due to communication with the oral cavity in most cases, and therefore it must be assumed that infection is of haematogenous origin.

Conclusions.

1. Numerous case reports signify that neurological symptoms may disappear on removal of an unerupted tooth. It may be deduced that (i) there was a causal relationship, (ii) relief of the symptoms was

co-incidental, (iii)an adequate follow-up examination would have shown recurrence of symptoms, and this is likely if the symptoms were of psychogenic origin.

2. Only in the case of inflammation associated with an unerupted tooth, is the causal relationship between the presence of such a tooth and neurological symptoms certain.

3. That neurological symptoms can be caused by "pressure effects" due to an unerupted tooth is difficult to prove, and the basis for such a claim appears to be purely hypothetical. It is significant that paraesthesia and anaesthesia have not been the reported sequellae of unerupted teeth, whereas mental paraesthesia occasionally follows the expansion of a cyst of the body of the mandible. The objective methods of assessment of neurological symptoms advocated by Hackett and Griffin could be used with benefit in the investigation of "pressure effects".

4. If it is suspected that there is a strong "psychogenic component" in the aetiology of neurological symptoms, the surgeon should not remove the unerupted tooth in the absence of obvious pathology. Surgery, always associated with attention, and leading usually to a generous ration of sympathy, is likely to relieve temporarily, pain and other symptoms of central origin, if attention and a "feeling of importance" is that which the patient craves.

5. The writer is of the opinion that many of the case reports claiming a cure of atypical neurological syndromes by the removal of unerupted teeth, should at present, be viewed with scepticism. There are, however, some cases reported (such as case 3, by Nodine, page 91) which strongly suggest that "pressure effects" or parasympathetic-sympathetic reflex arc stimulation due to the unerupted tooth, may be the cause of widespread neurological symptoms.

- PART III -

THE MANDIBULAR THIRD MOLAR.

A. Congenital Absence.

Hellman (1936)[128] estimated that 27.4% of American males, and 35.0% of American females have one or more third molars missing ; that one in twenty males, and one in ten females have all four third molars congenitally absent. Nanda (1954)[129] considered that Hellman's figures were too high, and in a well controlled investigation of 200 American females, found that only 9% lacked one or more third molars, and only one of the group lacked all four third molars. Bjork[10] found that 20% of Swedish males lacked one or more third molars.

Congenital absence of the third molars has been regarded erroneously as the monopoly of the highly civilised races. It has been found in all races, with the lowest incidence reported in Tasmanians and West Africans (Hellman), and the highest incidence in the Mongoloid peoples (Pedersen[19], and Goldstein[19] in the Greenland Eskimo ; Tratman[34] in the Malays ; Saito, Uehara and Ozaki [130]in the Japanese.) 36.6% of Greenland Eskimos of Angmagssalik showed agenesis of one or more third molars ; 34% of the third molars which should be present in 4,347 Japanese were missing.

Tratman, criticising the literature pertaining to agenesis of third molars, stated : "The general argument runs thus : the third molars are absent, the jaws are small, there is not enough space for the third molars, reduction in size of the jaws is a recent development, therefore the teeth are absent because the jaws are small. A non-sequitor argument. An equally valid conclusion would be that ~~small~~ jaws are small, because the teeth are absent." Neither Tratman nor Pedersen found small jaws to be associated with agenesis of the third molars, indeed both reported ample space. Bjork however, found that in no case of agenesis of the third molar, was there sufficient m3 space.

B. Anatomy.

Levin (1946),[131] describing the mandibular third molar, stated : " Unlike its adjacent molars which always have two roots, their roots may be fused and conical with a decided curvature. This tooth cannot be described adequately to apply to every third molar. It varies so in size, shape and number of roots, the contour of its surface, the number of cusps on the crown. The roots may be present as finger-like projections very much like improperly grown carrots, while in other cases, they may be gnarled and twisted, and like as not, the apices will present feet pointing distally."

Winter (1926) [132] found that the crowns of third molars ranged from comparatively small to abnormally large, and noted that "the roots will vary in size from very short to exceedingly long ; they will vary in formation from being fused into a solid mass of a conical shape to consisting of two distinct roots markedly divergent, and the direction will vary from straight to a marked distal inclination. In an occasional case, an extra root may be present, and there may be a bifurcation of the mesial or distal root, or both of these roots may have a bifurcation."

Tratman[34] stressed that " it may, or may not be larger than the first, and when it is large and bulbous in crown, the roots are often small and out of proportion." which description is true for all races. In the Mongoloid race, it often exhibits an extra distolingual root.

Nanda [129] noted that the rudimentary third molar, a common finding in the maxilla, is almost non-existant in the mandible.

Discussion.- It is obvious that each lower third molar must be treated individually, and its very wide variability makes painstaking radiographic, clinical and surgical examination essential. Each third molar removed must be closely scrutinised, to detect fracture of the roots, and if necessary, a check ray taken.

C. Chronology.

1. Hard tissue forms at 8-10 years.
2. Enamel is completed at 12-16 years.
3. Eruption takes place at 17-21 years.

4. Root formation occurs at 18-25 years (Logan and Kronfeld[62,133])

The eruption time is variable : Hellman estimated the average age to be 20.5 years, with a range of 16-27 years.

Mineralisation of the enamel is an important factor in anticipating impaction. Bjork[10] found a statistically significant correlation between retarded mineralisation and impaction : in a group of 20 year olds, he found that only 3.5% of those with fully erupted third molars, showed retarded maturation ; 50% of those without eruption showed extremely delayed maturation. Adamson[134] believed that the maturation time of the third molar could form a basis of prognostication. Delayed maturation indicates that skeletal development is probably in advance of dental development.

D. Frequency of Impaction.

Blum (1923)[135] found that the mandibular third molar was involved in about 50% of all impactions, deciduous, supernumary and permanent. It constitutes 86% of all mandibular impactions.

Montelius[14] found that this tooth was involved in 86% of impactions in the Chinese. Hellman[128] found that 1:10 males, and 1:4 females of U.S.A. suffered impaction of the mandibular third molar.

E. Aetiology.

The main factors in the aetiology of lower third molar impactions will be briefly summarised.

1. Bjork found that in 90% of impactions, the m3 space (shown by cephalometric radiographs) was inadequate. Lack of alveolar space is the most important factor in mandibular third molar impaction.

2. Lack of development of the alveolar process is due to lack of rugged chewing and failure to eliminate cuspal interference by attrition (Begg[21]).

3. Interproximal attrition in the Eskimo and Australian aborigine provides 7-8 mms. space for the accommodation of each lower third molar,

to compensate for an inherited "apical base deficiency".

4. Domestication (civilisation), results in earlier puberty, and a probable early arrest of skeletal growth of the jaws, with negligible effect if any, on dental development(Bjork[29]).

5. Hybridisation predisposes to the inheritance of small jaws and large teeth (and of course vice versa) with alveolar space deficiency in many cases.

6. The third molar is the last tooth to erupt, and consequently suffers most in competition for inadequate alveolar space.

7. Accidental causes are impaction by tumours, cysts, odontomas, and supernumary teeth.

8. Abnormal axial inclination or location of the tooth germ causes occasional non-eruption of the third molar.

9. Any systemic factor (endocrine, nutritionaletc.) which interferes with the eruption force, retards and stunts jaw growth, inhibits bone resorption, will involve the third molar, together with other teeth, in non-eruption.

10. The path of eruption of the mandibular third molar is from a mesiolingual position to the vertical, and any lack of space tends to interrupt the rotation, with impaction of the crown against the second molar.

Bjork's work on mandibular third molar impaction and jaw growth, deserves special mention. The space for the mandibular third molar tends to be reduced when (i) the growth rate in length of the mandible is slight, (ii) the condylar growth direction is vertical, (iii) the direction of eruption of the teeth is retrognathic, and (iv) maturation of the third molar is delayed. Of course, one adverse factor, such as vertical angle of the mandible, may be compensated for by another such as alveolar prognathism. Upon the assessment of these four variables, a prognosis at an early age may be made, in respect of future eruption: this is of importance in "prophylactic odontectomy".

Section G follows directly Section E. Section F is non-existant.

G. Position of the Impacted Third Molar.

Several classifications of the position of the impacted lower third molar have been published. Each omits to include a most important factor - the degree of eruption:-

(i) The third molar may be partly erupted (i.e., in communication with the oral cavity).

(ii) It may be embedded in soft tissue and bone (without clinical communication with the oral cavity).

(iii) It may be completely embedded in bone.

Winter classified the position of the unerupted lower third molars by relating their long axis to that of the second molar :-

Long axis relationship.-
1. vertical
2. mesioangular
3. distoangular
4. horizontal
5. linguoangular
6. buccoangular
7. inverted
8. displaced (aberrant).

Buccal surface alignment.-
1. buccal deflection
2. lingual deflection
3. buccolingual deflection
4. torsion.

Penn and Gregory[136] classified the position of the unerupted mandibular third molar, in relation to the m3 space, i.e., the distance between the distal of the second molar, and the anterior border of the ramus, at the occlusal plane level. The classification is useful as a reflection of the aetiology, and as a prognostic guide to the amount of bone to be removed to the distal and occlusal of the third molar, before the latter may be removed. They classify the third molar impaction as follows :-

Class 1 : Sufficient space for the mesiodistal width of the third molar crown.

Class 2 ; The space is less than the mesiodistal width of the crown,

Class 3 : All or most of the third molar lies within the ramus.

Pell and Gregory also based a classification of position on the depth of the unerupted molar in relation to the second molar, which serves as a prognostic guide to the surgeon, in respect of the amount of bone excision.

Position A : The highest portion of the third molar crown is level with or higher than, the occlusal plane.

Position B : The highest portion of the third molar crown is below the occlusal plane, but above the cervical level of the second molar.

Position C : The highest portion of the third molar crown lies below the cervical level of the second molar.

Nodine [14] considered that a fourth position could be added : the highest portion of the third molar crown is below the apical level of the second molar.

Discussion.- I feel that the importance of the three classifications lies not in a simple definition of position of the unerupted lower third molar, but in the pursuit of a methodical assessment of clinical and radiographic evidence prior to surgical removal of the third molar. The axial relation of the third molar to the second dictates the path of withdrawal of the tooth ; the alignment of the buccal surface in relation to that of t the second molar, suggests the proximity of the third molar to the lingual cortical plate and the thickness of the buccal plate ; the size of the m3 space dictates the amount of bone removal necessary to free the third molar crown from the ramus (the greater the reduction of the ramus, the more the trauma to the temporalis tendons, and therefore the more severe the post-operative trismus) ; the depth of the third molar reflects the amount of buccal plate reduction before the tooth may be lifted from its socket and the trauma which may be expected to the masseter muscle.

H. Clinical Features.

A brief application of the findings covered in Part 11 ("The Pathology of the Unerupted Tooth") to the unerupted mandibular third molar will be presented.

1. Infection.- The completely bone embedded tooth is sterile[66]. The third molar which lies partly within bone, and partly within soft tissue, is, according to Gunter[69], infected, in spite of lack of clinical evidence of oral communication. The partly erupted tooth is a source of infection, and will be described fully under "pericoronal infection".

2. Cyst formation.- The follicle of the third molar may undergo cystic degeneration. A central type of dentigerous cyst will displace the third molar in a contra-eruptive direction, while the lateral type will displace the tooth "sideways". The third molar may be displaced to the angle of the mandible, even as far as the condyle. The dentigerous cyst follows the line of least resistance, and therefore tends to gut the ramus and part of the body of the mandible, before the cortex is "expanded". Due to the late development of facial deformity, the cyst when discovered may be of considerable size. The sequellae of cyst formation are : (i) the danger of pathological and surgical fracture of the mandible; (ii) infection, which usually follows perforation of the cortex, (iii) neoplastic development, which may result in the formation of an adamantinoma, rarely an epithelioma.

3. Pressure effects.- Root resorption of the distal root of the second molar may be caused by the mesioangular or horizontal impaction. The second molar may be driven into supra-occlusion, or it may be tilted backwards, so the the roots of the first and second molar contact, and the distal cusps of the second molar lose occlusal contact. Broadbent[1] considered that crowding of the lower incisors and bicuspids is not due to pressure exerted by the impacted third molar. Neurological symptoms have been attributed to pressure upon the inferior dental nerve by the developing third molar roots.[62]

4. Coronal resorption.- This is not a common finding in the lower third molar[70] but when it does occur, the surgical difficulty in removal is considerably increased, and extensive bone excision is necessary if surgical fracture of the mandible is to be avoided.

5. Predisposition to mandibular fracture.- The presence of an unerupted third molar very considerably increases the chances of traumatic fracture through the angle of the mandible (Fry[137], Ivy[138], Thoma[139]).

6. Neurological symptoms.- The most commonly noted symptoms of the impacted third molar are : (i) pain distributed along the jaw, and over the auriculo-temporal region, (ii) earache and tinnitus. I personally have not seen cases in which remote pain, muscular incoordination, and mental symptoms may be correctly attributed to the unerupted third molar, although a review of the literature gives the impression that such symptoms are of common occurrence.

7. Caries and periodontitis.- The partly erupted third molar almost inevitably suffers caries, and predisposes to caries of the second molar. Food packing between the partly erupted third molar and the second molar is a common cause of destruction of the interdental bone between these teeth.

I. Pericoronitis or Pericoronal Infection. [140, 141, 142]

1. Definition.- Pericoronitis refers to inflammation of the gingival tissues in relation to the crown of an incompletely erupted tooth. It occurs most frequently in the mandibular third molar region. An incompletely erupted mandibular third molar is one which has cut the gum, or at least the crypt is open to the oral cavity. The process of eruption may be at a standstill due to impaction, or active eruption may be proceeding normally ; the mandibular third molar , however, may not be impacted, and the process of eruption may be occurring very slowly, or may be prevented by the ascending ramus, or the gingival tissues, or by an extruded maxillary antagonist. Third molars in such circumstances are susceptible to pericoronal infection, and they have in common, a gum flap, or operculum, or a capuchon, overlying the distal surface of the crown, and a variable portion of the occlusal, buccal and lingual surfaces. One cusp only may be clinically apparent, or the crown may be completely covered, and in contact with the oral cavity only via a sinus, visible, or, as Gunter terms it, microscopic. Once there is communication with the oral cavity, regardless of extent, the entire follicle investing the crown, is open to infection. The second molar may be present, but is not essential to the process. Pericoronitis may occur in the partly erupted third molar, in an otherwise edentulous mandible.

2. Anatomical Considerations.- The gum flap forms the roof of a deep pocket, which is bounded posteriorly by dense, tough fibrous tissue, lined by epithelium of the follicle, anteriorly by the distal surface of the third molar, inferiorly by the attachment of the follicle to the cervical margin of the crown. The buccinator muscle gains attachment to the floor of the retromolar triangle, and the superior constrictor gains attachment lingually to the third molar. Thus the cervical tendinous ring (a term used by Perkins [141]) separates the pericoronal pocket from bone and the adjacent tissue spaces (the buccal, pterygo-mandibular, submasseteric and lateral pharyngeal spaces). The temporalis tendon, lying outside the cervical tendinous ring, is an important relation. A mild infection is usually limited to the pocket, but a virulent infection may break through the anatomical barrier with complivations.

3. Aetiology.- The overlying gum flap, and the pericoronal pocket, offer an ideal nidus for food debris accumulation, and anaerobic conditions for the culture of anaerobes and micro-aerophilic organisms. This pocket is, so to speak, a culture tube for Vincent's organisms, Actinomyces israeli, and Appleton [143] claimed, Clostridium tetani. Even in persons with no clinical symptoms or signs, the gum flap is often chronically inflamed and ulcerated. Acute inflammation is an ever imminent possibility, and is usually initiated by traumatisation of the flap by the extruded maxillary third molar. Poor oral hygeine and lowered general resistance to infection (diabetes, leukaemia, agranulocytosis etc) are of course, important predisposing causes.

4. The course of the infection.-

(a) Chronic pericoronitis may cause no subjective symptoms, apart from an unpleasant taste and an occasional discharge. However, ulcer-

ation of the follicular epithelium and gradual bone destruction to the distal of the crown usually occurs. Bone destruction may involve the interdental septum of the second and third molars. Distal bone destruction is an important prognostic point in surgical removal : reduction of resisting bone is considerably lessened. Other effects of chronic infection are, however, sclerosis of bone and hypercementosis. A chronic pericoronitis is a reflection of the equality between virulence of infection and tissue response : this balance may be disturbed, by an increase in virulence of the infection, or a lowered resistance to the infection, and a subacute or acute attack is likely to follow.

(b) Subacute pericoronitis.- This is probably an acute inflammation quickly aborted by adequate natural drainage of exudate into the mouth. The patient may be aware of tenderness, slight pain, and may temporarily assume a bite of comfort to avoid trauma to the oedematous gum flap. Intermittent recurrence is usual.

(c) Acute pericoronitis.- This is characterised by rapid oedema of the operculum, which becomes raised, red, and tender. The oedematous flap then may become traumatised by the opposing tooth, which greatly aggravates the infection. At this stage the course depends very largely upon whether exudate may drain into the mouth. Regression usually follows adequate drainage. However, the operculum may close the outlet of the pocket when swollen, and pus then accumulates under pressure, and severe complications may follow. If the infection is not too virulent, a pericoronal abscess will form, limited by the cervical tendinous ring, and may rupture into the mouth, with relief. A pericoronal abscess, localised by the cervical tendinous ring, will be accompanied by acute tenderness of the flap, which will interfere with the closure of the teeth, a submaxillary lymphadenitis, and a low-grade pyrexia, with temperature slightly raised (99'-100' F) ; due to localisation of the infection, external swelling, trismus and dysphagia are unlikely to be present. Once the cervical tendinous ring is ruptured, the clinical picture depends upon the direction of spread of infection. It may drive buccally to the pterygo-mandibular raphe : submasseteric infection with severe trismus ensues ; it may pass into the buccal space, and a buccal space cellul itis follows, or a buccal abscess which usually points into the muco-buccal sulcus of the first molar region, anterior to the buccinator attachment;

a lingual spread is limited by the superior constrictor, and so it spreads along the palato-glossus to cause an abscess of the soft palate, which drags the uvula to the affected sideand onto the base of the tongue which makes swallowing of liquids difficult ; peritonsillar abscess and spread to the lateral pharyngeal space is possible, by pushing of pus between the superior constrictor and the buccinator. Once the infection transgresses the anatomical barriers, which tend to confine the infection, a major surgical emergency may ensue, demanding immediate drainage. Pericoronitis is not to be treated lightly, for a neglected case may lead to death. Hyde (1938)[144] reported one such case, which lead to a cellulitis, osteomyelitis, and fatal cavernous sinus thrombosis. Haymaker[145] reported several fatal intracranial complications of pericoronal infections. Jacobs[142] observed that cellulitis is more likely to occur in streptococcal , and abscess formation in staphylococcal infection.

5. Treatment .- This aspect will be discussed in Part V.

J. Periapical Infection of the Third Molar.

The partly erupted lower third molar is a site of stagnation of food debris, due largely to the overlying gum flap, and to lack of functional occlusion. Caries is not an unusual sequella, and, if overlooked, the inevitable result is pulpitis and periapical infection. The lower third molar is said to be particularly susceptible to caries, and is hypoplastic in many cases, with a gnarled, fissured occlusal anatomy.

Periapical infection may present a similar clinical picture to pericoronitis, and it is most important to distinguish between the two conditions, as the principles of treatment are diametrically opposite.

1. The course of periapical infection.- Pulpal infection leads to a periodontitis, then infection of periapical bone. Pus accumulates under pressure, when the infection is acute, and in spreading, adopts the path of least resistance. In the third molar region, the buccal cortex and the lower border are thick and dense, whereas the lingual plate is thin : the likely direction of spread of infection is by perforation of

the thin lingual plate, below the mylohyoid muscle, into the sub-mandibular tissue space. Acute pain is experienced, especially prior to discernible submandibular swelling, due to a subperiosteal abscess. Once infection is in the submandibular space, spread may occur to the pterygo-mandibular space, the lateral pharyngeal space, or the sublingual space. Trismus, dysphagia, external swelling and symptoms of toxaemia are very similar to advanced acute pericoronitis.

2. Treatment.- The establishment and maintenance of drainage with removal of the cause must be immediately instituted. Periapical infection commences in bone, pericoronal infection in the soft tissues about the crown. In periapical infection, extraction of the tooth will establish drainage, unless infection has already spread to the soft tissue spaces : once the soft tissue spaces are involved, fomentation and Hilton's method of drainage are necessary, in addition to extraction. Antibiotic therapy is a helpful, but not necessarily an essential adjunct to surgery. In acute pericoronitis, removal of the tooth during the acute stage, may initiate previously non-existing bone infection.

K. Squamous Cell Carcinoma and the Mandibular Third Molar.

Squamous cell carcinoma may involve the third molar in the following ways :-

(i) Primary carcinoma has been reported to arise as a result of chronic, long-standing pericoronitis (Ausubel[146], Sealey[147]).

(ii) Squamous carcinoma may arise from the epithelium of a dentigerous cyst.

(iii) Metastatic carcinoma may involve the third molar, as it usually arises from the spongiosa of the angle region by haematogenous spread.

(iv) Squamous carcinoma may spread from the floor of the mouth to involve the mandible.

Case Report (Ausubel, 1944[146]).- A male aged 65, complained of pain in the right retromolar area of the mandible. Examination revealed that the right lower third molar was embedded and partly erupted, and the mand-

ible edentulous on the right side except for the premolars ; there was no trismus, oedema or induration of the submaxillary and neck region ; he was a generally weak man who had difficulty in walking. Radiographs revealed an area of bone resorption in relation to the third molar, extending anteriorly as far as the second premolar. Diagnosis - pericoronitis. Treatment - removal of the third molar and curettage of "granulation tissue". Result - pain subsided, the patient could walk, and three weeks later, failed to keep his appointment as he felt fit. Seven months later, he returned with partial trismus, and a history of pain, in the right mandibular region ; he had been in hospital for six months as a result of a car accident. Examination - trismus, visible induration of the masseter which was tender, no lymph gland enlargement, no suppuration, the lip was numb. Radiographic examination revealed bone destruction from the first premolar to the anterior portion of the ramus above the mandibular canal ; the second premolar had been extracted while he was in hospital. Treatment - under general anaesthesia, the area was explored. A deep bone cavity lined with velvety tissue was found ; there was neither degenerated tissue nor suppuration present. Several days later, the patient complained of pain, which was treated by irrigation of the cavity, and topical application of sulphonamides and a crushed anaesthetic tablet. There was no improvement. The first premolar was extracted. It was then realised that the condition might be neoplastic. Biopsy revealed a grade II squamous cell carcinoma.

Discussion.- The above case has been described in detail, as I feel it illustrates many points in connection with the management of carcinoma. A socket which fails to heal normally, especially in a patient over 40 years of age, should be investigated. Osteomyelitis should be considered in the above case : there was no history of acute inflammation, pain, discharging sinuses and sequestration evident, merely a relatively symptom-free resorption of bone. Laboratory aids should be used : a full blood count and a differential white count would reveal leukaemia if present ; urinalysis, diabetes ; serological tests, syphilis ; lung field survey, tuberculosis (and metastatic lung cancer) ; should these tests be negative, a biopsy is indicated. Mental anaesthesia is an important symptom when it develops spontaneously - it is seen im acute osteomyelitis, and carcinoma.

Case Report (Thoma,1954[62]) .- A male aged 70 complained of a swelling of the neck, which resisted treatment for eight months. An Xray revealed a partly erupted third molar, in a horizontal position, and surrounded by osteolytic changes. Diagnosis - pericoronitis and chronic osteomyelitis, with secondary infection of soft tissue. Treatment - removal of the third molar. To the tooth was attached a considerable amount of soft tissue. Pathological examination indicated squamous cell carcinoma, grade II. A previous biopsy of the swelling of the neck had revealed chronic inflammation.

Sealey[147] reported the case of gingival squamous cell carcinoma mistakenly diagnosed as chronic pericoronitis of a partly erupted third molar: he considered that pericoronitis of long standing was the cause. Lymphosarcoma in relation to a partly erupted lower third molar, erroneously diagnosed as pericoronitis, has been reported by Englert and Pasqual (1954)[148]

L. Clinical Examination of the Third Molar.

Winter (1926)[132] stated that : "the successful extraction of an impacted third molar with a minimum amount of trauma, depends on a correct clinical examination and radiographic interpretation in conjunction with an operative technique that is applicable to the type of case presented." All three aspects he covered with illuminating and painstaking thoroughness.

Clinical examination is a necessary adjunct to the interpretation of radiographs, and is carried out digitally, visually, and with the aid of a mirror , probe and a good light. The following points should be noted :-

1. General condition of the patient.- A medical history should be taken, and the patient referred to a physician if considered necessary.

2. General oral condition.- Calculus, Vincent's infection, acute pericoronitis, and any other inflammatory condition should be noted and eliminated before surgery is commenced.

3. The maxillary third molar.- Does it traumatise the operculum,

and if allowed to remain in situ, will it traumatise the mucoperiosteal flap ? Is it infected ? If so, it should be removed before surgical removal of the lower third molar is undertaken.

4. The opposite lower third molar.- If erupted, examination of its occlusal anatomy, its size, and form, will give an indication of the morphology of its unerupted partner, although it should be remembered that symmetry is not necessarily present. If extracted, does the history help ?

5. The exposed part of the crown.- The crown may be completely covered, and only a bulge may suggest its presence - it may of course be congenitally absent, or grossly displaced in position. A sinus may lead to its crown, or a periodontal pocket to the distal of the second molar, may be followed with a probe to the third molar crown. The vertical type impaction will often have the mesio-buccal, or both buccal cusps exposed ; the distoangular type may have the mesio-occlusal part of the crown visible ; the disto-occlusal part of the crown in a mesioangular type, and the distal surface of the crown of the horizontal type, may be revealed. The degree of deflection of the buccal surface should be assessed, and the position of the buccal fissure will reveal torsional deflection, if any.

6. The gum flap.- The degree of inflammation should be noted. By passing a curved probe under the gum flap, to the distal of the crown, the extent of bone destruction may be determined. Vincent's infection if present, must be eliminated before operation. A bacteriological smear should be gained if infection is present, for the detection of organisms and their antibiotic sensitivity.

7. The ascending ramus.- A clinical estimate of the m3 space will suggest the amount of distal bone excision likely.

8. The interproximal space.- The presence of pocket formation should be noted. Overhanging restorations of the second molar should be detected and eliminated. The accessibility of the mesial surface to elevator application should be assessed.

9. Second molar.- Pulp vitality must be checked; resorption of the root or caries may have devitalised the tooth. Should the second molar be non-vital, or badly broken down, it may be extracted and the third

molar retained, if in a position favourable for eruption.

10. Second molar present, first molar missing.- Winter commonly used the second molar as a fulcrum for elevation of the third. He carefully assessed the resistance of the second molar to luxation in these cases. Absence of the first molar considerably weakens the second molar's usefulness as a fulcrum. However, it is my opinion that the second molar ought not to be employed as a fulcrum, for forceful elevation as he suggested.

11. The isolated embedded third molar.- A fistulous opening, an infected gum flap, a denture sore spot, may lead to recognition of its presence. Careful assessment of the related soft tissues should be made especially in the "cancer age group" patient : chronically inflamed, hyperplastic tissue should be suspected, and a biopsy specimen submitted for examination ; examination of the submandibular and cervical regions for enlarged lymph glands should be routine. The degree of mandibular atrophy in the edentulous and especially in the elderly should be noted, and arrangements for a Gunning splint construction made pre-operatively if surgical fracture is thought possible.

12. Attempted extraction of the third molar.- If the case is referred, a routine examination should be made firstly, of the integrity of the mandible (an unadmitted fracture in such cases is a possibility), secondly, of the adjacent soft tissues, thirdly, of the second molar and its restorations, and fourthly, of the inferior dental and lingual nerves. Detailed records should be made of the findings. Infection should be noted, and methods of controlling it immediately instituted.

13. The size of the mouth .- A small oral commissure, a large, poorly controlled tongue, ankylosis of the temporo-mandibular joint, predisposes to difficulty of access.

14. The lymph glands.- These should routinely be palpated.

The treatment plan depends upon the results of the clinical examination. Laboratory aids should be called for if indicated and an examination by a physician is at times advisable.

M. Radiographic Examination.

A standardised radiographic technique is most important, and I am prepared to risk the accusation of dogmatism in stating that the radiographic technique and its interpretation published by Winter in his textbook "The Mandibular Third Molar", [132] has not been surpassed, indeed, if followed correctly, cannot be surpassed.

Winter considered that two views at right angles, of the third and second , and part of the first molars, are essential for localisation of the third molar, displaced aberrant third molars excepted. The films he suggested are : (i) the intra-oral lingual film; and (ii) the intra-oral occlusal film.

To these two views, should be added the lateral jaw film, to show more clearly, the course of the inferior dental canal, which is of importance in many cases. In severe trismus, an intra-oral lingual film initially may be impracticable.

The aberrant third molar may lie anywhere within the ramus and its processes : obviously intra-oral films are unlikely to be of much help, and therefore extra-oral films, such as the P-A, lateral head, and the temporo-mandibular joint films may be found necessary.

1. The Intra-oral Lingual Film.- Winter recommended the following technique :-

(a) Type of film - the dental film, $1\frac{3}{4}$" x $1\frac{1}{4}$" , in size, ultra-fas fast in speed, to minimise radiation dose, and to offset movement.

(b) Position of the patient - mandibular arch horizontal, at the mouth-open posture; sagittal plane of the head vertical.

(c) Position of the film -anterior edge opposite the centre of the first molar ; film depressed as deeply as possible to show the periapical region, and at the same time, to include the enamel caps of the erupted molars ; the upper border of the film parallel to the occlusal plane of the lower teeth; the film should not be bent.

(d) Central ray - should strike the second molar at right angles at the centre of its crown, unless there is linguversion, in which case,

the central ray is horizontal.

Difficulties.- If the patient tends to gag, a topical anaesthetic lozenge should be sucked (decicain, or benzocaine lozenge)[148]. If the floor of the mouth is shallow, or raised by infection, sufficient depth of film may not be possible : by limiting the degree the mouth is opened, the floor may be relaxed and the film may be held in position by artery forceps.

Modifications of the technique.- Ward[149] preferred to superimpose the buccal cusps of the second molar upon the lingual cusps : due to a lingual inclination of the tooth, or uneven attrition of the cusps, I feel little is to be gained, as the central ray will not strike the mandible at a consistent angle, and the comparative value gained by standardisation of technique is lost. Kelsey Fry recommended the use of an xray holder which is a modification of the Spencer-Wells forceps : the film is positioned accurately, and bending is avoided, but I feel that these advantages are outweighed by the fact that the radiographer must hold the film forceps thereby suffering the accumulative effects of radiation. Ultra-fast films are to be recommended because the radiation doseage is reduced, and there is less chance of film movement.

2. The Occlusal Film.- The following technique was recommended by Winter :-

(a) Type of film : The dental film $1\frac{3}{4}$" x $1\frac{1}{4}$" is used.

(b) Position of patient : As the centre ray must be directed through the lower border at right angles to the occlusal plane, the head must be inclined to permit the positioning of the Xray cone.

(c) Position of the film : The film, emulsion side to the occlusal surfaces of the teeth, is placed on the crowns of the teeth, anterior border of the film level with the centre of the first molar, lateral border passing medial to the ascending ramus, and the film is secured in position, by the clenching of the teeth.

(d) The central ray : The central ray must pass through the lower border of the mandible, at right angles to the film, and along the long axis of the second molar.

Modifications.- The Spencer Wells holder, used by Ward and Kelsey Fry, with a metal film holder affixed to one beak, may be used with definite benefit, in the occlusal film technique. The film is slid along the buccal sulcus, and the patient bites against the film and holder, the latter of course, being uppermost. One distinct advantage is seen - the film cannot be distorted by the cusps or bent. The handle remains in the buccal sulcus.

3. The Lateral Jaw Film.- The method is standard technique, and is to be found in Ennis' textbook, "Dental Roentgenology".[150]

4. The Criteria of the Correctly Taken Lingual Film.- The following points should be noted, and if not present, the film should be retaken :-

(a) The contact point between the first and second molars should be a ball-to-ball contact, not overlapped, which indicates incorrect direction of the central ray.

(b) The enamel cap of the first and second molars should be clearly visible, and if the occlusal surface of both molars is thrown onto the film, the central ray is not true in the vertical plane. If, however, the enamel cap of the first molar is distinct, whilst that of the second molar is distorted, clinical examination will reveal linguo-version of the second molar, and such a film is satisfactory.

(c) The root or roots of the third molar, and its crown, in their entirety, must be visible.

Should the film, correct in all other details, fail to define the apical extremity of the third molar, it will, nevertheless define correctly, the exact relationship of the third molar crown to the second molar, and therefore should not be discarded, but supplemented by a somewhat distorted lingual film, with the central ray directed from a more posterior level, to throw the apices onto the film.

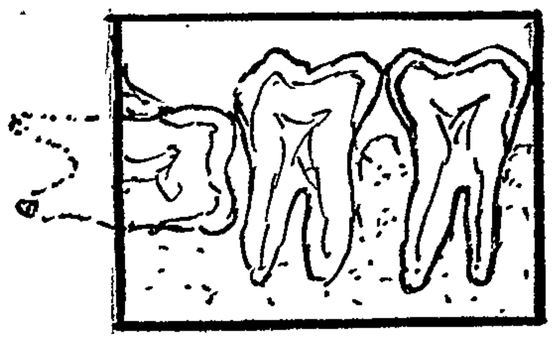
Richards (1952)[151] described a technique which will enable the inclusion of the apices of a horizontal third molar on the normal film, using the normal central ray angulation. The technique is illustrated diagrammatically below.

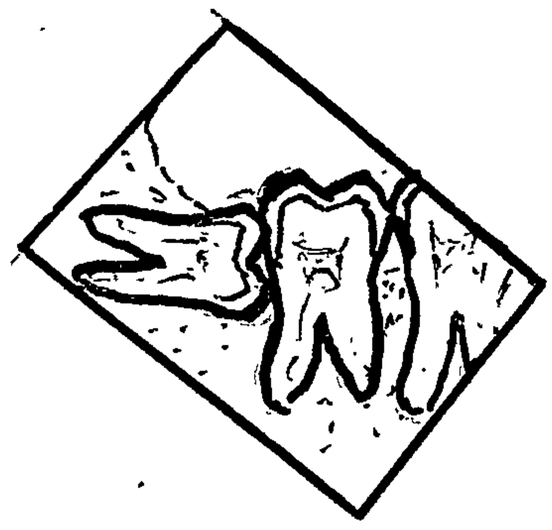
5. The Criteria of the Correctly Taken Occlusal Film.- The following points should be noted, and if not present, the film should be repeated :-

(a) The contact point between the first and second molars should be clear-cut.

(b) The occlusal surface of the first and second molars should be defined, the roots superimposed entirely upon the crown ; the visibility of the roots of the second molar, reflects the linguoversion of the tooth, if the angulation of the central ray is correct.

(c) The third molar region should be included, in entirety, if possible.





Richard's Technique.

N. Radiographic Interpretation of the Lingual Film.

Only the film which conforms to the above criteria is worth interpreting. The following routine of examination is recommended by Winter :-

1. The position of the tooth.- The long axis of the third molar is compared with that of the second molar : it may at once be seen that the third molar impaction is vertical, mesioangular, distoangular, horizontal, or inverted. The diagnosis of buccoangular or linguoangular impaction depends on interpretation of the enamel cap and contact point.

2. The form of the crown.-

(a) size and shape : a wide range of variation is possible;

(b) caries : only partly erupted or erupted teeth suffer caries;

(c) the follicular space : it may be normal in thickness,

wider and suggestive of cystic degeneration, or absent and indicative of coronal resorption and ankylosis ;

(d) fracture of the crown : is noted often after attempted removal, especially by forceps technique.

3. The contact of the crown with the second molar.-

(a) there may be no contact ;

(b) the contact may be clear-cut, without overlapping, which indicates that there is no buccal deflection ;

(c) the contact may be overlapping, which indicates a buccal deflection ;

(d) the contact point if on the distal root of the second molar, may be associated with pressure resorption ;

4. The occlusal enamel.-

(a) if seen in profile as an enamel cap, the third molar is vertical in a mesio-distal plane ;

(b) if the occlusal enamel is visible (i.e., not seen in profile) the tooth is lingually deflected ;

(c) if the occlusal enamel is ivisible, and the contact with the second molar is overlapping, the third molar in in buccolingual deflection ;

(d) if the roots are superimposed wholly upon the occlusal enamel, the third molar is in complete buccoversion, or in complete linguoversion (the latter being the more common) ;

(e) if the occlusal surface is visible, and the roots foreshortened, the impaction is of the linguoangular or buccoangular type.

Discussion.- The following points arise :-

(i) The reliable diagnosis of axial inclination of the impacted third molar, is possible only by use of a standardised technique.

(ii) Indifferent angulation of the central ray may convert a normally erupted third molar into an artifactual impaction (e.g., by superimposing the ascending ramus over the distal portion of the crown.

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(iii) Buccal deflection cannot be diagnosed by the lingual film if there is no contact with the second molar, or when the third molar contacts the second molar root.

(iv) Contact shown on the lingual film between the third and second molars may be artifactual only, a well angulated occlusal film showing actual buccal deflection and absence of contact.

4. The Roots of the Third Molar.

(a) Fused roots : if conical, little resistance to luxation need be anticipated ; if box-like, or hypercementosed, wide bone removal is indicated ; bifurcation grooves may increase resistance to luxation.

(b) Roots inclined distally : a rotational withdrawal is dictated, and is favourable in the vertical, mesioangular, and sometimes in the horizontal impactions, but is an adverse factor in distoangular impaction.

(c) Both roots straight : the septum of bone between the roots increases luxation resistance ; rotation of the tooth favours root fracture ; an uncommon feature of angular impactions ; freeing by bur of the inter-radicular septum, or sectioning of the tooth is indicated.

(d) Distal root curved mesially, mesial root curved distally : great resistance to luxation is exerted by the enclosed inter-radicular septum ; evasion of this resistance is effected by removing the septum by bur, or splitting the tooth longitudinally ; the root bulge may be wider apically than cervically, and this further increases resistance to luxation.

(e) Divergent roots : this is not commonly found ; vertical delivery is impeded, and rotation favours root fracture ; the difficulty may be evaded by sectioning of the tooth longitudinally.

(f) Both roots inclined mesially : not commonly found ; occasionally seen in horizontal or mesioangular impactions, rarely in the vertical or distoangular impactions ; an elevator rotation inevitably causes fracture of the apices - sectioning of the tooth is indicated.

(g) Mesial root distally inclined, distal root straight : Winter considered this the most common combination : the straight root resists rotation of the tooth, the inter-radicular septum resists luxation, and tooth sectioning or wide distal bone excision is necessary.

Note : P. 126 non-existant ; p.127 follows directly p.125.

(h) The mesial root straight, and the distal root mesially inclined : the combination is rarely found.

(i) Hypercementosis : a "dumb-bell" shape may result, which greatly increases the difficulty of removal; bone reduction should be generous, as any attempt to force a passage through bone resilience, is likely to cause either bone contusion and a localised necrosis, or fracture.

(j) Ankylosis : a feature of the later decades, and characterised by the disappearance of the periodontal space ; Thoma indicated that when bone is fused to dentine, cleavage occurs within the bone itself when force is applied - bone excision to the apex, by use of the bur, is necessary if fracture of the mandible is to be avoided.

(k) Torsion : root detail is lost if the tooth is in torsional deflection, which is diagnosed by the pulp chamber morphology on the radiograph; an artifactual root fusion is apparent.

(l) The inferior dental canal : the relationship of roots to canal will be covered in detail, in a separate section.

Discussion.- The pre-operative assessment of the roots of the impacted third molar is essential. Should difficulties in surgical removal of the tooth be anticipated from radiographic interpretation, the patient should be forewarned of complications such as mental anaesthesia, fracture of the mandible and undue post-operative discomfort. Root form influences the site and amount of bone excision, the path of withdrawal of the tooth, and the decision to section the tooth.

5. The Bone Support of the Third Molar.- When the lingual radiograph has been taken in the suggested manner, a very important diagnostic line is seen, viz, the line passing through the interdental septal crest of the second and first molars and its continuation up the ascending ramus. It may safely be assumed that the portion of the third molar which lies above this line, will be visible on retraction of the muco-periosteal flap. The following points should be noted :-

(a) Buccal bone : the above-mentioned diagnostic line will indicate the height of the buccal bone in relation to the crown of the third molar; the buccal plate must be reduced to free the greatest convexity of the molar crown ; the thickness of the buccal plate depends upon the

degree of buccal deflection (when it is thin) and on the degree of lingual deflection, (when it is thick) ; reduction of the buccal plate by use of a bur is more conservative than by a chisel.

(b) Lingual bone : the shadow of the lingual plate is obliterated by the dense shadow of the crown, but it may be assumed to equal the buccal plate in height ; lingual deflection tends to reduce the height of the plate, buccal deflection to increase its thickness ; occasionally the lingual plate covers part of the lingual cusps, and the lip of bone must be reduced.

(c) Interseptum between the third and second molars : it may be thick and wedge-like, especially in the mesioangular impaction ; it may be a thin sliver in the vertical type of impaction ; in the distoangular impaction, it may be obliterated apically, by contact between the roots of the molars, while it may be broad at its crest ; it may be obliterated in the horizontal impaction, when the third molar contacts the roots of the second, but may be broad if there is no contact ; infection may eliminate the bone over the distal surface of the distal root of the second molar.

(d) The inter-radicular septum of the third molar : the septum is a resisting factor to luxation, and must be eliminated by bur reduction, or evaded by tooth sectioning.

(e) The bone on the distal surface : Pell and Gregory's classification is based upon this (the m3 space), and the smaller the space for the third molar eruption, the greater the distal bone reduction required in surgical removal; chronic pericoronitis tends to destroy the distal bone ; the distal bone must be generously excised, when the third molar is to be rotated distally, otherwise mandibular fracture is inevitable.

(f) The distolingual bone : Kelsey Fry and Ward(1958) considered this bone a significant factor in resistance to luxation, and their split bone technique is designed to eliminate it.

(g) Occlusal bone : The deeply embedded tooth is of course,

completely covered on its occlusal surface ; however, a lip of bone may cover part of the buccal or lingual portions of the occlusal surface, depending on the deflection and angulation of the impacted tooth.

(h) Bone on the mesial surface : Winter based his classification of accessibility to the mesial surface, on this ; if a space exists between the interdental septum crest and the mesial surface, he considered the mesial surface accessible to an elevator; when the tooth is buccally deflected, the mesiobuccal bone may be used as a fulcrum.

(i)n Pathological condition of periapical bone : this bone is destroyed by periapical infection ; the lingual cortex too, is often perforated by such a condition, an abscess draining through the lingual plate as a rule ; a fractured apex may be driven into the submaxillary space through this perforation of the lingual plate.

Discussion.- Bone excision is an essential factor in atraumatic removal of the impacted third molar. Bone is excised (i) to expose the greatest crown convexity, (ii) to provide access for an elevator Purchase, (iii) to provide a space into which the tooth may be displaced, and (iv) to provide a bone-free path of withdrawal. Bone excision if correctly carried out, obviates the need for forceful elevation of the tooth, which contuses bone, or causes mandibular fracture when used injudiciously. Greater bone reduction is required in the elderly, as bone becomes more brittle and hard with age. Sectioning of the tooth reduces the necessity of radical bone excision.

6. The Second Molar.- Winter recommended the judicious use of the second molar as an elevator fulcrum, and consequently paid considerable attention to the root formation, and bone support of this tooth in radiographic interpretation. I consider that it is best to avoid using this tooth as a fulcrum, firstly because it is not necessary, and secondly because of the trauma inevitably caused. Adequate bone removal and tooth sectioning has relegated a minor role only to the elevator in modern operative technique. However, the second molar must be carefully examined pre-operatively, and the following points noted in particular :-

(a) The crown of the second molar : overhanging margins of restorations, extensive caries should be noted - overhanging margins should be removed pre-operatively, a badly broken down second molar may be extracted to permit eruption of the third molar in some cases.

(b) The root of the second molar : pressure resorption of the distal root may affect the prognosis of this tooth ; fused, short conical roots give little support - such a tooth may be luxated if used as an elevator fulcrum.

(c) The bone support of the second molar : the distal bone may be destroyed by either a horizontal impaction, or by pathological process - after removal of the third molar, the distal surface of the distal root will probably be exposed and hypersensitive, and fluoride desensitisation may be required ; should periapical infection be evident, the second molar must be removed in advance of the third molar to prevent clot infection .

Discussion.- The second molar must always be very carefully assessed. Its removal is indicated (i) when it is non-vital and infected, (ii) when its distal root will be laid bare to the apex, by removal of the deep third molar impaction, (iii) when access to the third molar is made impracticable by its presence (e.g., when the third molar lies embedded below the second molar apices), (iv) when second molar removal will permit the eruption into functional occlusion of the third molar - before sacrificing the second molar, it must be ascertained that the third molar actually will erupt, (v) when ill-health militates against the more difficult third molar removal. Kelsey Fry [152] suggested that second molars may with benefit, be prophylactically removed to evade the later necessity of surgical removal of the third molar : this controversial topic will be more fully discussed in a later section.

7. Abnormalities Associated with the Impacted Third Molar.

(a) A supernumary tooth : a supernumary tooth, in the form of a diminutive tooth, or a well developed fourth molar is not rare ; the fourth may be fused with the third molar.

(b) A dentinoma is sometimes found overlying the occlusal surface of the unerupted third molar ; in shape it is irregular and radiopaque, and may be surrounded by bone resorption on account of pericoronitis.

(c) A dentigerous cyst is relatively common in this region ; if of the lateral type, the displacement of the tooth is not marked, but if of the central type, the tooth may be displaced as far as the condyle ; an extra-oral film is required to define its margins.

(d) Tumours of the central type may cause the impaction and displacement of the third molar. However, not all tumours cause displacement : a carcinoma of the jaw, primary or metastatic, does not displace, but causes an irregular, ill-defined resorption of bone which may give the radiographic impression of osteomyelitis.

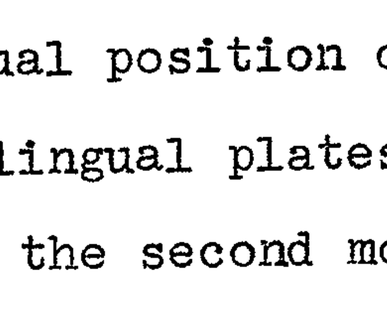
(e) Fracture of the mandible is a possible finding in cases referred following attempted removal of the third molar.

O. Interpretation of the Occlusal film.

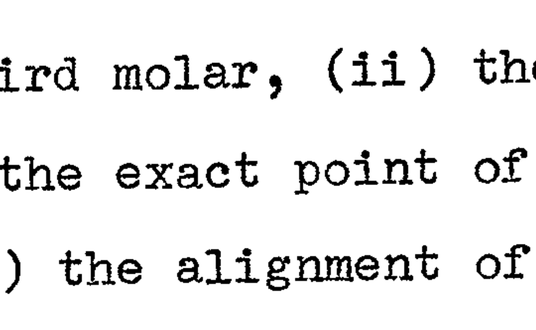
The occlusal film is used to define (i) the bucco-lingual position of the third molar, (ii) the thickness of the buccal and lingual plates, (iii) the exact point of contact of the third with the second molar, (iv) the alignment of the third molar not in contact with the second. An artifactual contact in the lingual film may be an actual separation in the occlusal film.

P. Multiple Impactions involving the Third Molar.

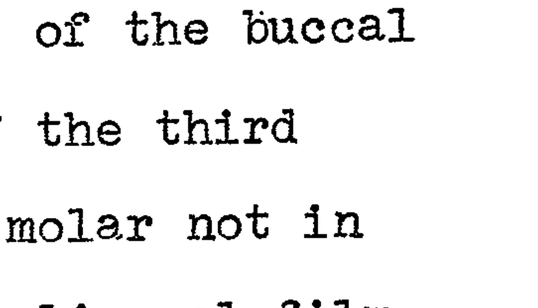
The following diagram illustrates the types of multiple impaction reported in dental literature.



(a)
"Right-angle" type

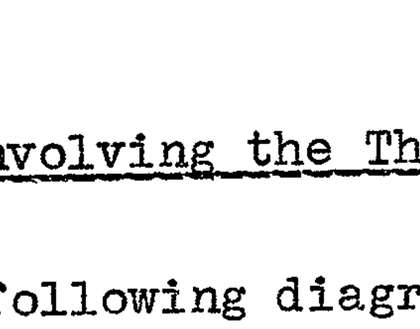


(b)
"Oblique" type



(c)
"Head-on collision"

(d)
"Double berth" type



(a) The right-angle impaction involving the second and third molars has been reported by King (1928)[153], and Anderson (1945)[154]; Winter (1926) published radiographs of four examples of the condition. It is generally noted that in this type od double impaction, the first molar is standing. With loss of the first molar, the positions of the second and third molars change (see below). The initial cause of second molar impaction is probably premature eruption of the second molar before alveolar growth has provided its accommodation.

(b) The "oblique double impaction" of the second and third molars is, according to Sealey[155], the sequella of the right angle double impaction of these teeth following the extraction of the first molar. The pressure of the third molar on the disto-occlusal aspect of the second molar evidently causes the distal rotation of the second molar to produce a more stable result. Fitzgerald[156] reported a case of bilateral double impaction : in the leftmmandible, clinically edentulous distal to the second premolar, there was the oblique double impaction of the second and third molars, whereas the right-angle double impaction was evident in the right mandible, clinically edentulous distal to the first molar.

(c) The "head-om collision" of second and third molars has been reported by Denny (1936)[157] in a 24 year old female, in whom the first molar had been missing from the age of 9 years ; by Barker (1932)[158] in an edentulous female aged 46 ; by Coleman (1930)[159]; by Sawday (1929)[160] and by Blum (1923)[161] who reported the bilateral condition in a 16 year old female. The "head-on collision" is the final stable result gained from the original right-angle double impaction. Most textbooks are able to publish radiographs of an example of this type of impaction.

(d) The "double berth" type of double impaction of the second and third molars is rare. Winter (1926) published a radiograph of the condition. The aetiology of this type of multiple impaction could be (i) the mistiming of eruption of the second molar, with a mesioangular impaction resulting, and (ii) subsequent forceful eruption of the third molar against the disto-occlusal portion of the second, could bring about the more stable double horizontal impaction against the first molar.

(e) The double vertical impaction of the second and third molars appears to be rare : Winter (1926) reported one case.

(f) The horizontal, distally inclined second molar impaction against the vertical unerupted third molar has been reported by Nodine (1941)[162] in an otherwise edentulous mandible. The aetiology of such an impaction is obscure.

(g) Third and fourth molar impaction has been reported by Elliott Smith (1926)[163], and Tait (1933)[164]. A "head-on collision" between the third and fourth molars was reported by French (1928)[165]. Symmons (1938)[166] reported a "double berth" horizontal impaction of the third and fourth molars. Winter (1926) reported a double vertical impaction of the third and fourth molars, and published radiographs of four cases of geminated third and fourth molars.

(h) First, second and third molar impactions are rare. One case has been reported by Moskow (1958)[167], and is diagrammatically illustrated below. The third molar, the crown of which lay under the mucous membrane, alone was removed, to alleviate the pericoronitis caused by the extruded maxillary third molar.

(i) A third molar and second premolar impactions was described by Fitzgerald (1958)[156]. The second premolar lay horizontally with its crown directed distally, and impacted against the vertical, unerupted third molar; it lay near the lower border, under the apices of the second molar. Ennis (1949) published radiographs of similar impactions between the third molar and second premolar.

(j) Impactions of the second molar, third molar and second premolar are apparently rare. Sealey (1952)[155] one case.

Q. Aberrant Mandibular Third Molars.

Nodine (1946)[14] published a collection of twenty eight cases of aberrant mandibular third molars reported in dental literature. He made the following observation : "The third molars have been discovered in all areas of the ascending ramus, from almost the top of the coronoid process to the neck of the articular or condyloid process and the posterior border of the ramus. They have been found in the body of the mandible below the ramus and below the molar teeth, from the cervical margins of the crowns of the molar teeth, downward to the inferior border of the mandible."

Several further cases have been reported since Nodine's collection was published, Jones (1945)[168], Mowatt (1946),[169] McKenzie (1947),[170] Young (1949)[171], Zernov (1949),[172] Balendra (1949)[173], Christianson (1950),[174] Martins Buddery (1954),[175] Moody (1955),[176] Rowe and Broadway (1955),[177] and Stark and Klein (1958).[178]

I have analysed the above 39 cases as follows :-

I. Age of diagnosis : ages were available in only 27 cases.

Diagnosis	16-20 years	3 cases
"	20-29 years	4 cases
"	30-39 years	5 cases
"	40-49 years	6 cases
"	50-59 years	3 cases
"	60-83 years	6 cases

2. Symptoms leading to consultation :

(a) Painful swelling of the face.- 15 cases ; caused by acute infection ; swelling in all but three cases involved the parotid-masseteric region, in two cases, the pre-auricular region, and in one case, the submandibular region.

(b) Painless swelling of the face.- 2 cases only, one due to an adamantinoma, the other due to a cyst.

(c) A discharge.- intra-orally in 4 cases, extra-orally in one case.

(d) Neuralgic pain.- in 11 cases, the chief complaint ; in 4 of these cases only was infection present, which suggests that the neuralgic symptoms in 7 cases arose from pressure effects.

(e) Trismus.- in 2 cases, trismus was the prime complaint ; it was found in most infected cases.

(f) Earache.- found in one case only, this being a woman aged 65, with bilateral earache, bilateral coronoid process impactions of the third molar, and unilateral fourth molar impaction in the condyloid process.

(g) Clicking of the temporo-mandibular joint with limitation.- in one case only, with a coronoid process impaction.

(h) Extra-oral eruption.- in 4 cases ; three erupted in the pre-auricular region, one at the angle ; each case was associated with a purulent infection, and mistakenly diagnosed as sequestration.

Note : there were no symptoms in five cases.

3. Position of the aberrant third molar.

(a) Condyle or condyloid process : 8 cases, including a fourth molar.

(b) Coronoid process : 8 cases.

(c) Angle region : 5 cases.

(d) Lower border of the ramus : 2 cases.

(e) Anterior border of the ramus : 3 cases.

(f) Sigmoid notch : 4 cases.

(g) Posterior border : 2 cases.

(h) Centre of ramus : 5 cases; two erupted lingually, in relation to the mandibular foramen ; one lingually across the mylo-hyoid groove ; neuralgic symptoms were marked in one case, and the inferior dental nerve showed visible signs of neuritis.

4. Cystic involvement.- Dentigerous cyst formation was seen in 8 cases, of which 3 were infected. There was one case of adamantinoma, in which the mandibular third molar lay in relation to the lingula. See discussion.

(5. Infections arising from aberrant teeth.- 20 cases were the cause of infection, 15 of which caused the acute symptoms which led to diagnosis. See discussion.

6. Intra-oral sinus .- 7 cases showed an intra-oral sinus ; 6 appeared in the retromolar region, one in the buccal sulcus opposite the first molar, indicating the tracking of pus externally to the buccinator muscle. The exploration of the sinus with a probe in some cases revealed the presence of the tooth.

7. Extra-oral sinus.- 4 cases showed one or more sinuses to the skin, in three patients, over the ramus, in one, below the angle ; multiple external sinuses formed in one case, and actinomycosis was ruled out by laboratory aids.

8. Extra-oral eruption.- 4 cases showed external eruption , in two additional cases, the third molar was embedded in the masseter muscle.

9. Bilateral cases.- Mowatt [169] reported bilateral aberrant third molar impactions in the anterior border of the ramus, simulating slightly inverted horizontal impactions, well separated from the second molars. Tomes[14] found a specimen with bilateral eruption of the third molars through the sigmoid notch. Stark and Klein[178] discovered bilateral impaction of the third molars in the coronoid process, with a fourth molar impacted in the condyle.

Discussion and Conclusions.

1. Aetiology of aberrant mandibular third molars.- There are four possible causes for the abnormal position of these teeth :-

(a) Cystic displacement.- A dentigerous cyst may displace the tooth involved a considerable distance, especially in the case of a central type cyst ; it is noted that only 8 cases were associated with a cyst, and one case suggests that the tooth, lying in the centre of the ramus, later underwent cystic degeneration ; this implies that not all of these 8 cases were necessarily displaced in the first instance, by cystic expansion.

(b) Abnormal orientation of the tooth germ.- The principle behind this theory is that the tooth germ, for reasons unknown, "points in the wrong direction" and the eruptive force later develoPed, translates the tooth to its aberrant location. No doubt some cases may be satisfactorily explained by this theory : this is confirmed by the inverted teeth, such as the mesiodens erupting into the nasal cavity. However, the explanation is unsatisfactory in those cases in which movement in a contra-eruptive direction is found : Balendra (1949),[173] McKenzie (1947),[170] Tomes (1906)[14] each reported a third molar in the neck of the condyle, apices directed towards the articular surface ; Etchepareborda (1902),[14] Andrew (1909)[14] reported an inverted third molar with apices at the sigmoid notch ; Rounds(1946),[14] Martins Buddery (1954)[175] reported a third molar inverted in the coronoid process - these cases were not associated with dentigerous cyst involvement.

(c) Abnormal position of the tooth germ.- This theory implies the redundancy of the dental lamina, which deposits the third molar tooth germ at a distance from its normal location. This hypothesis would with difficulty apply to some cases, but hardly to the third molars discovered in the condyle or the angle of the mandible.

(d) Intra-osseous migration.- The very considerable mobility of the embedded tooth has been clearly demonstrated (cf Part II - " Migration of the Unerupted Tooth, page 77). Bone is plastic, in that it is ever undergoing reconstruction. In my opinion, most cases of aberrant third molars are explained by an intra-osseous movement of the tooth, unrelated to eruptive force, which may continue throughout life. Those horizontal embedded third molars found to be separated by an interseptum of bone from the distal surface of the second molar in the young patient, may well become the condyloid process impactions in later life. The case of Mowatt (1946) may illustrate the early stage of migration : bilateral horizontal, slightly inverted third molars in a male aged 32 years, with separation of $\frac{1}{4}$" from the second molar roots. The bodily migration in a contra-eruptive direction of the mandibular third molar appears to be probable, but there is little evidence of contra-eruptive migration in other teeth : the second/premolar of the mandible is never found to migrate in such a manner, and migration occurs distally in an eruptive direction throughout life, and this

tooth may, after loss of the first and second molars, finally become impacted against the embedded third molar (Fitzgerald,1958).

2. Infection of aberrant teeth.- 20 embedded, aberrant third molars, were the source of infection. In no case could there have been oral infection, which could only be incurred by (i) a dentigerous cyst, infected by perforation of the alveolar bone and oral muco-periosteum, and (ii) partial eruption in the third molar region, with subsequent submergence, and translation to its aberrant position, and this is, of course, ridiculous. In no case studied, did a dentigerous cyst perforate the bone,and three only of the eight cysts were infected. The only cases of infection readily explained are the four cases of extra-oral eruption. In my opinion, the most likely explanation for infection, is the emergence of part of the crown through the ramus in the region of muscular insertions, with subsequent trauma to the muscle fibres, followed by haematogenous infection. Logan (1937) clearly demonstrated that the pulp and investing tissues of the completely bone-embedded tooth are sterile ; Gunter (1942) warned that infection was likely once the tooth emerged through bone, even though the mucous membrane was clinically intact. There is therefore, every reason to believe that the aberrant tooth while embedded in bone, is sterile. Almost invariably, teeth found in the condyle or the coronoid process were found to be infected, and it is most unlikely, in view of the dimensions of these processes, that the teeth would be completely bone embedded: emergence into the muscle attachments of these muscular processes, would be inevitable. Trauma to the muscles, even in the absence of infection, would be sufficient to cause limitation of movement, and temporo-mandibular joint disturbances, as seen in the case of Jones (1945),[168] who reported these as the only symptoms of a coronoid impaction of the third molar. Several authors reported emergence of the aberrant tooth into the masseter muscle. Infection appears to be the most important sequella of the aberrant third molar.

3. Treatment.- Due to delay in diagnosis, it is probable that several innocent teeth were extracted in an endeavour to detect the cause. Although no specific mention was made, it is probable that several cases suggested acute infection of the parotid gland, after obvious dental causes had been ruled out. In some cases, actinomycosis

was suspected, but the diagnosis was not confirmed, and the infection was eliminated by removal of the aberrant tooth. Surgical approach varied : some successful reports of intra-oral removal were published, and this approach is the most effective for the removal of coronoid process impactions, and of teeth erupting on the medial surface of the ramus ; many aberrant molars were removed by an extra-oral approach, which is the only approach to the condyle, posterior border, and the angle of the mandible. Before undertaking the removal of an aberrant tooth, it must be accurately localised in three planes by extra-oral radiography. The extra-oral films required may include (i) a lateral jaw film to show as much of the ramus as possible, (ii) the P-A film, which, however, may not indicate the presence of the tooth in the condyle, due to superimposition of the shadows of the base of the skull and the mastoid process, (iii) the temporo-mandibular joint films to reveal the condyle and its neck. It should be emphasised that absence of the third molar on the intra-oral radiograph does not always indicate that the third molar is congenitally absent - congenital absence is positively indicated only when exhaustive radiographical exploration of the ramus is negative.

R. Treatment Considerations of the Third Molar

1. How serious is the problem of the unerupted lower third molar ? A study of the incidence and sequellae will provide the basis of a realistic assessment :-

(a) Incidence : 1 in 10 American males, 1 in 4 American females, and 1 in 5 Swedish males will suffer impaction of one or both lower third molars (Hellman,1936 ; Bjork, 1957).

(b) Effects of retention of the embedded third molar : The completely bone embedded tooth may cause no ill-effects ; however, it may destroy the second molar by pressure resorption of its roots, be the seat of diverse neurological symptoms, undergo cystic degeneration, predispose the mandible to fracture, migrate to remote parts of the ramus with the sequellae of infection and temporo-mandibular joint

disturbances. Its removal is often mandatory in the elderly, when it has become exposed through bone resorption, and the surgical procedures are made difficult by the possibility of coronal resorption with ankylosis, hypercementosis, and the brittle, eburnated, atrophic condition of the mandible.

(c) Effects of retention of the partly erupted third molar.- This tooth is rarely trouble-free, even though it may be symptom-free. The second molar may be lost through the caries, periodontitis, or pressure resorption it induces ; occlusal disturbances may result from supra-occlusion of the second molar, with temporo-mandibular joint sequellae. The useful life of the entire dentition is threatened by the Vincent's organisms harboured under the third molar flap, which also forms the ideal nidus for culture of Actinomyces bovis. The partly erupted third molar is the source of infection ranging from a chronic pericoronitis to fatal intra-cranial infection. A fracture through its crypt is not only likely, but also may be complicated by non-union caused by infection and the bone loss incurred in its removal.

(d) Grave complications of the third molar.- Liston (1844) in one of his lectures, warned the dental profession of the disastrous sequellae which may be expected from the impacted lower third molar, and his warning is as true today, as it was over a century ago :

" My friend, Mr Nasmyth of Edinburgh, a most accomplished surgeon and dentist, met with the following case : an extensive abscess of the cheek and great swelling of the face and jaw, the abscess extending down to the clavicle. His mouth could not be opened, the inflammation locked the jaw, and the patient died. On a post-mortem examination, it was found that the cause of the mischief was the wisdom tooth growing forward and horizontally, instead of perpendicularly. This is a rare case, but it shows that much mischief and serious consequences may arise from such a trifling cause."

C.Bowdler Henry (1938),[179] in a study of 456 cases of third molar impaction from private sources and literature, found the following serious sequellae :-

40 cases of osteomyelitis,

35 cases of extensive cellulitis and Ludwig's angina,

8 cases of cavernous sinus thrombosis,

46 cases of fracture (traumatic 18, surgical 21, pathological, 12),

42 cases of cyst formation,

41 cases of operative displacement of the tooth into the soft tissues,

38 patients died,

68 patients experienced severe post-operative complications.

Another feature is that 89 cases received ineffective treatment for some secondary symptoms or lesion, often for long periods, before removal of the third molar was finally effected. There were 46 cases of mistaken diagnosis : one, a doctor, suffered a gasserian ganglion operation, a nurse had her mastoid explored and found normal before symptoms were relieved by removal of the peccant third molar. No doubt bacteriological investigation would have revealed actinomycosis in some of Bowdler Henry's cases of infection.

2. What measures have been suggested to avert lower third molar impaction ?

(a) Kelsey Fry (1933)[180] reminded the dental profession that :

> "Nowadays, when children are placed in the hands of a dentist for advice and attention regarding their teeth, surely it is his duty to take care, not only of immediate needs, but also to consider the probable future conditions. Can it be said that his duty has been well performed if it should be necessary for the patients, on reaching a more mature age, to undergo a difficult surgical operation for the removal of impacted wisdom teeth? "

His plea was for the dentist to take radiographs and study models of the child at 12-15 years of age, and to assess the chances of normal third molar eruption. There are four possible conclusions :

(i) the third molars are congenitally absent ;

(ii) the third molars are correctly positioned for normal eruption

and adequate space is available ;

(iii) the third molars are so positioned that eruption is impossible regardless of space available ;

(iv) the third molars are in correct position for normal eruption but space is lacking.

Obviously in (i) and (ii) no preventive treatment is required. Kelsey Fry suggested that the third molar be removed at 12-15 years if impaction is probable, regardless of space ; he suggested the removal of the first permanent molar to provide space for the third molar in selected cases, and he mentioned caries, and restoration of the first molar as conditions for their removal. Vehement criticism followed these prophylactic suggestions, and his suggestion of first molar removal was treated with derision in America. Prophylactic removal of the third molar was considered surgically difficult, due to the ball-in-socket mobility of the developing, rootless tooth, and unjustified, as prediction of impaction at an early age was regarded as impossible. The substitution of a hypoplastic, caries susceptible third molar for the first molar, so prized by the orthodontists, could not be countenanced by the American dental profession; other objections, more specific and appropriate, included mesial tilt, not mesial drift, of the second molar, and distal migration with contact loss of the premolars. Such a reaction was not unexpected, as the ideal of permanent arch expansion and absolute control of 32 teeth by orthodontic appliances, was still to be proved Utopian. Indeed, disimpaction of the third molar and its alignment was at that stage, considered within orthodontic ability. The controversy subsided, Kelsey Fry gained no support, and the first molar was given a temporary reprieve.

(b) C. Bowdler Henry (1938)[181] revived the discussion by his publication of " Prophylactic Odontectomy of the Developing Mandibular Third Molar " in the American Journal of Orthodontics. He considered that surgical removal of the lower third molar could be averted by the removal of the first or second permanent molars - the sacrifice of sound permanent teeth he regarded as doubtful and inferior, a "masticatory mutilation". He advocated the removal of the third molar, if impaction was inevitable, at an age before root formation had commenced. This practice he termed "prophylactic

odontectomy ". His recommendations were radiographic examination of the child at 7-8 years of age when the crypt is first visible as a bone deficiency, and at intervals thereafter, until the orientation of the third molar and its relation to the developing second molar, could indicate the prognosis. Calcification he claimed, commenced in the third molar at 8-9 years of age, although, as he stated in 1957, maturation may not commence until 12-13 years of age. His criteria for early condemnation of this "delinquent tooth" are well known, and have been frequently quoted. I shall give a brief summary of them only :-

(i) when the developing third molar lies postero-superior to the developing second molar, and appears to be crowding upon it ;

(ii) when the third molar appears inverted, horizontal, or transverse in orientation, or is especially large ;

(iii) when there is a supernumary fourth molar displacing it ;

(iv) when the maxillary third molar is either absent or rudimentary ;

(v) when the first and second molars have been moved distally by an appliance, then third molar impaction is almost certain.

Prophylactic odontectomy Henry claims is simple, and takes a matter of minutes only to perform, and healing is characteristically rapid in the child. His work met with a favourable response; his suggestion is still quoted in the literature, but it has failed to be practised as an approved and recognised technique. The reasons are : Thoma and others believe that the technique is difficult and traumatic, due to the elusiveness of the partly formed molar, which "turns turtle" on application of the elevator, and Thoma believes in delaying removal until the third molar roots are three parts formed ; Lucien Brun[183] and others consider that it is impossible except in isolated cases, to predict whether the eruption position and adequate jaw growth, will be achieved until the adult growth spurt. A commonly held opinion is that the adult growth potential of the jaws cannot be attained, if the third molar is removed before jaw growth is complete. It is my opinion that there is inadequate evidence to support the view that third molar

impaction may be anticipated before the age of 12 years, except in the extreme cases of inversion, fusion between the fourth and third molars, and transverse buccal or lingual placement of the third molar. I feel that the surgical difficulties of prophylactic odontectomy have been exaggerated, or the technique misunderstood, as removal of the developing crown by use of the elevator is unnecessary when the partly formed and partly calcified crown may be so much more simply crushed, and the crypt curetted to eliminate the follicle. However, prophylactic odontectomy, simple operation as it may be, could well be an unfortunate introduction of oral surgery to the young child under local anaesthesia, and a small, but significant risk accompanies every administration of general anaesthesia. The more difficult removal of the third molar at 16 years or so, would be less traumatic psychologically, than the more simple odontectomy at a much earlier age. Brodie[184] showed that the third molar does not stimulate jaw growth in the child.

(c) The pendulum of orthodontic opinion has swung from the ideal of textbook "normal occlusion" of thirty-two teeth, to acceptance of the impracticability of arch expansion, arch lengthening and jaw growth stimulation by myofunctional therapy. Begg's work on the Australian aboriginal dentition, confirms the view that apical base deficiency must be accepted as an immutable state, and may only be corrected by the judicious extraction of permanent teeth if lasting results are to be attained. Today, the suggestion of second molar removal to permit the eruption of the "crowded" third molar, is unlikely to create a stir in the dental profession, or in the orthodontic speciality. Adamson[185], a well known orthodontist, in 1952 admitted he favoured sacrifice of the second molar, if eruption of the third molar could thereby be achieved, and Smith,[186] with impunity, in 1951, stated, as his considered opinion, that:" It is often advisable to decide at about the age of 14-15 whether the third molar will be impacted. This can generally be diagnosed by the general development of the jaws and with the aid of xrays. In my opinion, it is sound practice to remove the second molar at this age. The third molar will then move forwards and upwards into contact with the first molars, and into good functional occlusion. Obviously, a completely horizontal tooth cannot erupt into a good position.

Similarly, if the crown of the third molar is presenting in a buccal or lingual direction, the tooth will not become functional. It is necessary therefore, by careful study of radiographs, to determine the possible position of the third molars, if the second molars are removed. The decision to extract the second molar is a difficult one particularly if they are sound teeth ; however, so much trouble can be avoided that it is worth while. The opinion of an orthodontist should be sought when in doubt."

It should be emphasised that the lower third molar is more likely to erupt in a position of mesiolingual tilt, with failure to contact the first molar satisfactorily in many cases. If such a position is to be accepted, the results are bound to be disappointing : intimate occlusal interdigitation with the opposing tooth will be lacking, and the gnarled occlusal surface of the third molar, rendered non-self-cleansing, will inevitably decay ; ineffectual contact between the third and first molars will render each liable to periodontal disaster. Should the second molar be sacrificed, it must be for a satisfactory substitute : the exchange is not warranted if the third molar possesses a small, fused root, which makes the tooth a poor abutment for a bridge or partial denture. Once the decision has been made to remove the second molar, the responsibility of the dentist does not end with the mere eruption of the third molar- it must be brought into contact with the first molar, be assured a correct axial inclination, and be given reasonable occlusal articulation with its antagonist. Orthodontic opinion is that controlled movement of the third molar by appliance therapy is difficult to gain, and it is probable that the third molar will not be given a position of satisfactory functional occlusion.

3. Is the Mandibular Third Molar Problem Likely to Increase ?

Civilised man is liable to malocclusion, which in Australia, is likely to increase in incidence due to a still greater degree of racial intermixture as a result of post-war immigration. Begg (1954) proved that preponderance of tooth substance over jaw

accommodation, i.e., apical base deficiency, is a normal, not a pathological inheritance, to compensate for tooth substance loss by attrition. There are two principal factors which contribute to modern man's characteristic high malocclusion susceptibility - lack of masticatory function, (the effects of which are negligible attrition and inadequate alveolar arch expansion), and hybridisation, whereby inherited disharmony between tooth size and jaw size may provide a pathological apical base deficiency, which could never be corrected regardless of function attrition. It is therefore obvious that third molar impactions cannot be avoided in a civilised community, but in many cases, are not manifested either because of the more fortunate sequella of hybridisation - small teeth and large jaws - or the self-correction accrueing from premature loss of permanent teeth by caries. Loss of the permanent first molars at the age of 8 or 9 years, often permits the mesial eruption of the second molar, with adequate space for the third (although the chances of "closed bite" are greatly increased). However, an even more significant factor in the promotion of third molar impactions is to be anticipated in Australia and other countries : as high quality conservative dentistry is extended to a greater percentage of school children, by improvement of the school dental service facilities, and perhaps subsidised dental treatment , as fluoridation of water supplies endow the child with relative caries immunity, early loss of the first permanent molar will decrease. As premature loss of the first and second molars by caries decreases, so will the incidence of third molar impactions increase. Third molar impaction will be the inevitable sequellae of socialised dental, and fluoridation schemes, both of which, I hasten to add, are highly laudable schemes when correctly put into effect. But just as the incidence of third molar impaction must rise, the more grave complications are likely to decline due to the advent of antibiotic therapy, and a better understanging of diagnosis and promp treatment.

THE MAXILLARY THIRD MOLAR.

A. Congenital Absence

The maxillary third molar is congenitally missing as frequently as the mandibular antagonist ; the former more frequently is rudimentary in development. Hellman (1936)[128] claimed that 1 in 20 American white males, and 1 in 10 females lacked all four third molars. Nanda (1954)[129] considered his figures too high, and in a study of 200 American white females, found that 9% lacked one or more third molars, and one only of the group lacked all four third molars. Of the 200, 3% showed a rudimentary development of the maxillary third molar, and each suffered agenesis of one or more third molars. This led Nanda to suggest a causal relationship between diminution in size and diminution in number of the third molars.

Discussion.- Intra-oral radiographs may show absence of the third molar in the region of the tuberosity : this may suggest congenital absence of the tooth, but only after an exhaustive search for the tooth, may it positively be declared missing, as third molars if aberrant, may be discovered in almost any site of the maxilla.

B. Anatomy.

Thoma[62] recorded that this tooth has a tendency towards a rudimentary development, but, although sometimes very decidedly undersized, it is distinctly a molar tooth in morphology. Nanda does not agree, and claims that the rudimentary third molar may be peg- or pyramid-shaped : could Nanda be incorrectly considering the peg-like conical paramolar of Bolk - a supernumary tooth - a rudimentary third molar ? The third molar generally has a conical root, often with a distal curvature, which may aid, or impede its removal. Cogswell,[187] however, warned that this tooth shows a wide variation in tooth form, and although they do not often present a marked deviation from the conical type root, a good radiograph is invaluable : when marked enlargement of the root, or when two or three roots exist, the pressure required for the molar's removal is often more than the investing tuberosity can resist, anf fracture of the alveolar ridge may result.

C. Chronology.

Logan and Kronfeld[62] gave the following details for the maxillary third molar development :-

(i) Hard tissue formation commences at 7-9 years.

(ii) Enamel is completed at 12-16 years.

(iii) Eruption occurs at 17-21 years.

(iv) Root formation is complete by 18-25 years.

Hellman[128] concluded that the range of eruption was wider still : he considered that the eruption age ranged from 16-27 years, with a mean eruption time of 20.5 years, in American whites, and he noted no significant sex differences.

Bjork[10] noted the correlation between delayed ,maturation of the enamel and impaction.

D. Frequency of Impaction.

Cramer[14] found that in American whites, 7.8% of males, and 18-20% of females, suffered impacted maxillary third molars.

Blum (1923)[135] found that the maxillary third molar is the third most frequently impacted tooth (the mandibular third molar constitutes 50%, the maxillary canine 22%, and the maxillary third molar 15% of all impactions)/

E. Aetiology.

1. Lack of space is the most frequent cause of impaction.
2. Abnormal axial inclination, or abnormal location of the tooth germ accounts for a small proportion of impactions.
3. A supernumary fourth molar is an unusual cause.
4. An odontoma occasionally causes impaction.
5. A dentigerous cyst is a relatively common association - it is not yet understood whether the cyst causes the non-eruption, or vice versa: the cyst is probably secondary to non-eruption.

Discussion.- The same factors predispose to the impaction of both maxillary and mandibular third molars. Why is the maxillary third molar impacted less frequently than its antagonist ? The maxillary third

molar forms below the posterior part of the floor of the orbit, lateral to the external pterygoid plate. With growth of the alveolar process, the tooth, its crown facing downwards, backwards and outwards, is "wheeled down" into position, with its apex as centre of rotation. Should growth be inadequate, rotation is arrested, and usually there is no obstacle in its path, to prevent eruption into a malaligned position, either distally, or buccally deflected. Arrest of rotation of the mandibular third molar drives the crown against the second molar, and eruption into normal, or abnormal position, is prevented.

F. Position of the Impacted Maxillary Third Molar.

Brody (1954)[188] employed the same classification of position for the maxillary third molar, as Winter, and Pell and Gregory[136] suggested for the mandibular third molar :-

1. The maxillary third molar may be
   - (a) partly erupted,
   - (b) embedded in soft tissue and bone,
   - (c) completely bone embedded,
   - (d) in the antrum, wholly or partly.

2. Its long axis, compared to that of the second molar, may be
   - (a) vertical,
   - (b) mesioangular, and usually tooth-impacted,
   - (c) distoangular,
   - (d) horizontal with the crown directed mesially or /distally,
   - 9e) transversely, either buccally or lingually,
   - (f) inverted.

Each of these axial positions may show buccal, lingual, or torsional deflection. In addition, the tooth may be aberrant, and located in a remote area of the maxilla.

3. It may be impacted at the following levels :-

Position A : The lowest portion of its crown is at, or below, the occlusal plane.

Position B : The lowest portion of its crown is above the occlusal plane, and below the cervical margin of the second molar.

Position C : The lowest portion of its crown may be above the cervical margin of the second molar, but below its apices.

Position D : The lowest portion of its crown may be above the apical level of the second molar.

4. Brodie suggested that its position should be defined in addition, in terms of its relation to the hamular process : proximity to the hamular process creates surgical difficulty.

G. Clinical Features.

1. The bone-embedded tooth is sterile (Logan[66]), therefore symptoms which arise from this tooth cannot be of inflammatory origin, and it may be hypothesised that they arise from pressure on nerve endings or nerve trunk irritation. Such a tooth may itself undergo coronal resorption with ankylosis, cystic degeneration, or hypercementosis with possible fusion to the roots of the second molar. If left in situ, it may cause no symptoms, but eventually it may "erupt" under a denture, or migrate to a more remote region, with antral eruption, or eruption into the soft tissues related to the maxilla.

2. The partly-erupted tooth is not usually the seat of pericoronitis of the acute type, as drainage is dependent, and trauma of the overlying gum by the opposing tooth is rarely possible. It almost invariably suffers caries, as its occlusal surface cannot be freed of debris by mastication, the tongue, or the toothbrush ; it may predispose to caries, the distal surface of the second molar. Occasionally, bone destruction with prejudice to the supporting tissues of the second molar, may follow chronic pericoronitis or food packing. The buccally erupting third molar may cause serious and painful temporomandibular arthritis, on account of its impingement upon the coronoid process, which leads the patient to assume an excursion of convenience.

3. The soft tissue and bone embedded tooth unlike its mandibular antagonist, rarely becomes infected, due to the absence of trauma to the overlying gum. Eventual eruption under a denture leads to its discovery, in the absence of neurological symptoms.

4. The aberrant tooth produces symptoms according to its location. Eruption into the antrum invariably results in a sinusitis which may take the form of a polypoid reaction, a chronic or acute empyaema. If it erupts with impingement upon the temporal muscle, coronoid process, or the lateral pterygoid muscle, an arthritis, or an extra-articular ankylosis may result.

5. Fusion of the roots of the third with those of the second molar is not rare. Fusion occurs by propinquity of the roots of these teeth, with the replacement of intervening supporting structures by cementum. This is a difficult condition to diagnose radiographically, and the surgeon should know the circumstances which lead to concrescence, and thereby be prepared to meet the emergency when it arises. A review of seven cases reported in the literature has revealed the following information :-

(i) Age range.- 23 - 60 years (23, 32, 35 and 60 years, with the age not stated in three cases). Fusion at the age of 23 years in interesting, as hypercementosis is usually seen at a much later age.

(ii) Sex.- Five cases were in females, two in males.

(iii) Third molar position.- Inversion was present in two cases ; horizontal impaction with the crown facing distally, in three cases ; vertical impaction with fusion over the entire root length, in one case ; the position of the third molar in one case was not specified.

The above cases were published by Biggs (1914),[189] Beeson (1945),[190] Feldman (1920),[191] Moffitt (1923),[192] Main (1933),[193] Hyde (1928),[194] and Proctor (1945).[195]

6. Pressure resorption of the second molar roots.- Davy (1923)[196] reported resorption of the root of a second molar by a mesioangular impaction of the upper third molar, in an 18 year old female, who suff-

ered severe neurological symptoms not cured by the removal of both molars. Andre (1930)[197] reported resorption of the apical third of the palatal and distobuccal roots of the second molar by a high impaction of the upper third molar, with severe neuralgia cured by the removal of the second molar. Kidd (1931)[198] reported a case of root resorption of the second molar by the roots of the third molar in a female aged 40; severe neuralgia and neurasthenia were cured by the extraction of the second molar. Kidd's case is of interest, in that root was absorbed by root, and concrescence did not, strangely, occur.

7. Trismus.- The third molar may cause trismus by intra-articular causes, due to arthritic changes ensuing from the "bite of comfort" dictated by buccal maleruption, and by extra-articular causes, such as the impingement of a third molar on the coronoid process, the temporal or the external pterygoid muscle. Sasche (1897)[199] reported trismus relieved by the removal of a maxillary third molar horizontally placed, with its crown directed against the internal pterygoid muscle. Geren (1929)[200] reported a case in which both maxillary third molars erupted into the temporal muscles : the third molars were high behind the malar bone, in the "zygomatic fossa" with apices bone embedded, and the crown and root embedded in the muscle, which had to be dissected from the dentine. Bennett (1931)[201] reported a similar case, in which a third molar erupted into the temporalis muscle, at a level higher than the zygomatic arch. Trismus may also be caused by mechanical obstruction of the coronoid process : Tait (1933)[202] reported such a case, in which the third molar, lying high in the maxilla, penetrated the postero-lateral wall, impinged upon the coronoid process, to produce limitation of movement, with a dragging to the affected side during opening movements. Rowlands (1921)[203] reported a similar case of coronoid process obstruction by an aberrant third molar.

8. Sinusitis.- Nodine noted that sinusitis and maxillary third molar impactions were rarely associated, pre-operatively at least. Thoma remarked that : "It would seem from a search of the literature, that but a small percentage of ectopic teeth are found in the sinuses. This is rather surprising when one considers the proximity of the dental organs to the maxillary sinuses." The impacted third molar may cause

sinusitis by (i) caries, with periapical infection, of the partly erupted tooth, (ii) by ectopic eruption into the antrum, and (iii) cystic displacement of the tooth with destruction of the antral wall, into the antrum, with eventual infection of cyst and antrum. Crolius (1958)[204] reported a case in which the third molar was driven into the antrum by a dentigerous cyst, in a male aged 19 years: intra-oral films showed the absence of the third molar in the tuberosity, with an apparent gross pneumatisation of the antrum ; extra-oral radiographs and puncture tests led to the diagnosis of dentigerous cyst, with the third molar pressed against the roof of the antrum, its roots splayed at right angles, in conformation with the bone, thereby "timing" the displacement of the tooth; histopathological evidence indicated an infected cyst and antrum in communication, with remnants of cystic and antral epithelium amid a mass of necrotic material. Cohen (1954)[205] reported a similar case in which the third molar was lying against the roof of the antrum, which was filled with polypoid tissue : unfortunately, no details of the pathology were mentioned - the third molar could have been displaced by a dentigerous cyst, which later became infected, or it could have been surgically displaced, during attempted removal. Genen (1930)[206] reported a maxillary third molar in the antrum, near the ostium, but again, inadequate details were published to indicate its pathology. Thoma[62] also reported a case, caused by a dentigerous cyst.

9. Neurological symptoms.- It has been shown that acute inflammatory conditions are/unusual in the impacted maxillary third molar: most neurological symptoms, it must be hypothesised, arise from pressure or reflex arc hyperactivity. Pain and neuralgia are the two most frequently mentioned symptoms of which patients harbouring impacted third molars complain. The site of the pain is rarely found in the third molar region, but most frequently in the face, on the affected side ; it may be distributed over the squamous temporal and mastoid region, the forehead, and at times over the affected side of the head. The pain may be of the neuritic or the neuralgic type, and subjectively, is described as chronic, vague, persistant, or paroxysmal, radiating, intense. Two cases of facial paralysis have been described, and attributed to the presence of an impacted third molar (Bacon, 1860[207]; and Cullen, 1945[208]). In each case, however, I consider too little evidence has been presented to incriminate the impacted tooth as the aetiological factor of the facial paralysis.

several cures of eye complaints have been effected by removal of impacted upper third molars, and it is difficult to deny that the impacted teeth were the cause in many cases. Mental symptoms too,have been alleviated by removal of the suspected impacted tooth. A fuller discussion of this controversial topic is presented in Part II ("Neurological Symptoms").

H. The Clinical Examination.

1. Surgical access :- Access to the third molar region during surgical procedures is notoriously difficult in the presence of certain adverse factors. An alveolar ridge of low height is characterised by a low mucobuccal sulcus, and a low, prominent zygomatic process, which factors makes the field of operation, particularly in high impactions, poor in visibility. Should the buccal pad of fat be gross, clinical examination is difficult, and even more difficult, is surgical approach : once the attachment of the buccinator has been raised, the pad of adipose tissue flows through the incision obstructing the field and suffering trauma - adipose tissue has a poor blood supply, and therefore poor reparative powers. The coronoid process further limits accessibility to the region, and, to overcome the impediment, the patient should swing his jaw laterally, to the side bearing the impacted tooth, his mouth "half-open" only. An assessment of the difficulty of the above factors, together with the size of the oral commissure, reflects the prognosis of the surgical procedure.

2. The degree of eruption.- Partial eruption of the third molar will permit a helpful examination of the alignment of the tooth, its relation to the second molar, and its size. An unerupted tooth may be located if superficial, by a swelling of the soft tissue overlying the tuberosity. A deeply embedded tooth often cannot be located visually.

3. The partly erupted.- The presence of pericoronitis, adjacent bone destruction, and caries should be noted. The contact with the second molar is important, and it may be possible to determine whether the third molar crown is locked under the distal surface of the second molar, or whether these two molars are separated. The visual examination of the path of closure into centric occlusion may reveal a bite of comfort, and trauma to the buccal mucosa by cheek biting may be found

associated with tempore-mandibular joint disturbances (best examined clinically by auscultation), when the third molar lies buccally malposed. A partly erupted third molar may be the seat of denture "sore-spot" in the edentulous, and gentle probing of the ulcer bed may reveal the glazed hardness of a cusp. A discharging sinus in the tuberosity region may likewise lead to the third molar.

4. Cystic condition.- A dentigerous cyst involving the unerupted upper third molar usually encroaches on, and expands into the antrum, and enlargement of the maxilla may not be apparent. A tumefaction of the palate is more likely to be due to a palatally unerupted third molar if hard, or an abscess from this tooth if showing the signs of inflammation.

5. The second molar.- The usefulness of this tooth must be carefully estimated : should it be non-vital, badly broken down by caries, mobile on account of periodontitis or root resorption, it may be preferable to extract this tooth, and permit the third molar to erupt, should its alignment, and the age of the patient suggest the possibility. The removal of an infected second molar should always precede the surgical removal of the unerupted third molar when possible, in order that healing of the third molar wound may proceed by first intention, and that the chances of antro-oral fistula are minimised.

6. The maxillary sinus.- The removal of the unerupted or partly erupted third molar is definitely contra-indicated during acute maxillary sinusitis. The antrum may be chronically infected by the third molar, or by other causes. The condition of the antrum should be carefully assessed before undertaking surgery which may possibly perforate its lining : a pergoration of the healthy antral lining may usually be disregarded (providing the principles of asepsis, debridement, and suturing have been adhered to), whereas a perforation of the chronically inflamed antral lining may lead to an acute empyaema, with infection of the third molar wound, and an antro-oral communication.

I. Radiographic Examination.

Clinical absence of the maxillary third molar indicates that (i) it may be congenitally missing, (ii) it may be present, but unerupted, perhaps impacted, or (iii) it may have been

extracted. Should an intra-oral radiograph fail to show the third molar, it may be (i) aberrant, or displaced by a dentigerous cyst, or (ii) congenitally missing. A third molar may be regarded as absent, only after an exhaustive radiographic investigation of the maxilla has proved negative.

Poynton (1958)[209] condidered that a periapical radiograph, and a lateral occlusal radiograph , both intra-oral films, adequately define the third molar.

Extra-oral radiographs are indicated when a maxillary third molar is aberrant, displaced by a cyst, or surgically displaced into the antrum or the soft tissues of the infra-temporal region.

In the edentulous maxilla, localisation of the embedded third molar necessitates the use of localising devices, such as a probe at a known distance from the midline, or a wax base plate bearing wire inserts, included on the radiograph.

Localisation of the surgically displaced tooth may be aided by inclusion on the radiograph, of hypodermic needles inserted into the soft tissues, through a known puncture point, at a readily reproducible angle : a minimum of two extra-oral radiographs at right angles, is required.

1. The intra-oral periapical radiograph.- This film should reveal the third and second molars, the tuberosity and hamular process, and a portion of the first molar. The apices of the teeth should be depicted, and their relationship to the floor of the antrum defined.

Difficulties.- The dense shadow of the zygomatic bone will obliterate the third molar unless it is thrown above or below the tooth. Should the vault of the palate be low, or the alveolar ridge of the edentulous maxilla be groosly resorbed, the dental film will lie too flat : it may be held at the desired angle by placing a cotton roll between the lower border of the film, and the standing teeth or the ridge (Le Master's technique). Difficulties arise in the interpretation of the film : the coronoid process may be superimposed upon the tuberosity ; the maxillary sinus - a radiolucent shadow - obscures periapical "areas" unless the film is very closely scrutinised. The distinction between a cystic cavity

and the antrum is most difficult : the use of lipiodal injection after the apiration of cystic fluid is the method of choice.

Poynton described the radiographic technique as follows :-

(a) Type of film.- An intra-oral film, $1\frac{3}{4}$" x $1\frac{1}{4}$" is used, and the ultra-fast type is recommended to reduce the radiation dose.

(b) Position of the patient.- The sagittal plane should be vertical, and the occlusal plane of the maxillary teeth horizontal.

(c) Position of the film.- The posterior edge of the film should extend slightly beyond the tuberosity if possible, in order that the hamular process may be included. The axis of the film should be at 20'-30' off the vertical. Topical anaesthesia will prevent "gagging".

(d) The central ray.- It should enter at a point 2" anterior to the external auditory meatus, perpendicular to the molar teeth, and at 25' to the horizontal, in order that the shadow of the malar bone may be thrown above the apices of the teeth.

Modification of the technique.- The malar bone shadow may be thrown below the teeth, by placing the film relatively flat, and directing the central ray above the zygomatic arch. Updegrave (1959)[210] in an interesting paper, published a radiographic technique in which the film is placed at an extended distance from, and parallel to, the teeth, in order that "geometric clarity" and "anatomic accuracy" may be gained : he used an instrument to hold the film, and to ensure the correct angulation of the central ray. Updegrave stated that by use of the "paralleling, extended-distance technique", in the maxillary molar region, "The zygomatic shadow is demagnidied and projected well above the roots of the first molar, making positive the interpretation of a periapical roentgenolucency associated not only with the mesiobuccal root, but with the lingual root also."

2. <u>The lateral occlusal film</u>.- This intra-oral film will give a "bird's eye view" of one half of the maxilla, with a somewhat distorted view of the molar roots. It is especially useful if trismus is present, and gives an excellent general representation of the impacted third molar and its relations, and should be used in conjunction with the peri-

apical film. Poynton described the technique as follows :-

(a) Type of film.- The occlusal film, ultra-fast speed.

(b) Position of the patient :- Sagittal plane vertical, maxillary occlusal plane horizontal.

(c) Position of the film.- With its long axis antero-posteriorly, the film is held between the teeth.

(d) Central ray.- It enters the skin, just below the outer canthus of the eye, perpendicular to the molar arch, at 50' to the horizontal.

J. Radiographic Interpretation.

A standardised technique, developed to give consistent results, is necessary for the ready detection of divergencies from the normal. The periapical radiograph should be examined in accordance with the following routine, as suggested by Brody[188] :-

1. Position of the third molar.- The long axis of the third molar is compared with that of the second molar. The level of the lowest portion of the third molar is defined in relation to the second molar. The position of the third molar may thereby be classified,in two planes only.

2. The form of the crown.- The crown varies considerably in size, and has a greater tendency to rudimentary proportions, than the mandibular third molar. The size of the crown should be considered in relation to the space between the second molar and the hamular process, and in relation to the intended path of withdrawal of the third molar. The desirability of sectioning the tooth to facilitate its removal should be considered. The follicular space should be scrutinised in the embedded tooth : if a thin but discernible radiolucent line, the follicle is normal ; if thickened, a cystic condition should be suspected ; if not present, the follicle may either be atrophic, or invaded by vascular connective tissue, with the sequellae of coronal absorption and ankylosis.

3. The form of the root.- Usually the root is single and conical : it may be straight or curved, and will influence the path of withdrawal. There may be two or three roots, which will increase the resistance to luxation. Hypercementosis should be noted if present, and the relation

of the roots of the third to the second molar examined : if concrescence of the roots of these molars is anticipated, the patient should be warned of the possibility of loss of both teeth. Partial development of the roots will significantly reduce the resistance to removal. The roots display a variable relationship to the antrum, and the possibility of antral exposure should be assessed. If in close proximity, the condition of the antrum (whether healthy or otherwise) should be considered.

4. The contact of the third with the second molar.- The partly erupted tooth may suffer caries, and predispose the distal surface of the second molar to caries ; it may contact either the crown or the root of the second molar. The embedded third molar may not contact the second molar, or it may contact the roots of the latter and cause their resorption. The roots of the second and third molars may contact, and this may result in concrescence or root resorption.

5. The interseptal bone.- The vertical type third molar may lie in intimate contact with the second molar, and the interseptal bone may be considerably reduced in width. The interseptal bone may be destroyed by pericoronitis or by food packing, in the case of a partly erupted tooth. The interseptum may be broad, in the distoangular type impaction. A broad interseptum may serve as a fulcrum ; it may resist withdrawal of the tooth and require reduction.

6. The bone support of the third molar.- The bone surrounding the third molar may be weakened in its union with the maxilla, by excessive pneumatisation of the antrum: the tuberosity on no account should be fractured, nor should it be excessively reduced, otherwise intra-oral fistula may result, and a future prosthetic problem is certain. The strength of the tuberosity should be considered in the light of the depth of impaction, the number and shape of the roots, the path of withdrawal of the tooth (as dictated by its position), and the patient's age. The degree of bone reduction, and the degree of force required for luxation of the tooth, may be in some cases, reduced by sectioning the tooth. It must be emphasised that the minimum amount of bone to be removed is that which will overcome the resistance to luxation offered by the roots, and provide a space into which the tooth may be displaced prior to its withdrawal. The hamular process should be closely examined, and the

surgical procedure planned to avoid its injury.

7. The second molar.- Clinical examination will have revealed whether the tooth is vital, or otherwise. Overhanging margins of restorations likely to interfere with third molar delivery, should be noted and corrected. Its alveolar support, and the presence and extent of root resorption should be investigated. Should periapical infection of the second molar be overlooked, there is every likelihood that the third molar socket will become infected. If extraction of the second molar is necessary, on account of pulpal or periodontal pathology, the chances of normal eruption of the third molar should be assessed.

8. Abnormalities associated with the third molar.- The following may be noted :-

(a) The dentigerous cyst .- When a dentigerous cyst associated with the third molar is suspected, the tooth must be localised, and the extent of the cyst determined. It is often difficult to distinguish between cystic cavity and antrum, and then a lipiodal injection into the cyst cavity, after aspiration of its fluid content, will show the clearly demarcated extent of the cyst on a radiograph. The cyst may have become confluent with the antrum as a result of infection, and the antrum will fill with lipiodol.

(b) The supernumary tooth.- Supernumary fourth molars in the maxilla are not a common finding. The paramolar of Bolk, however, is more frequently seen. This tooth is conical, and lies almost invariably to the buccal of the second and third molar, The supernumary tooth will occasionally displace the third molar. The possibility of third molar eruption following removal of the obstructing supernumary , should be considered before condemning the former.

(c) Odontomas.- Not a common association. Their presence may be suspected clinically by a dense enlargement of the tuberosity, and non-eruption of the third molar.

(d) Hyperplasia of the tuberosity.- The tuberosity region may be considerably enlarged by hyperplasia of the subepithelial connective tissue, or bony bony enlargement. Surgery is considerably complicated, and, in the case of the edentulous maxilla, the gingival or bony

hyperplasia should be surgically reduced after the third molar removal is effected.

THE MAXILLARY CANINE.

A. Congenital Absence.

Dolder (1937)[211] found that 3.4% of 10,000 children lacked one or more permanent teeth, excluding third molars, of which only 1.8% were maxillary canines. It may be concluded that agenesis of the maxillary canine is extremely rare.

B. Anatomy.

Stones[13] reported that this tooth may occasionally possess an abnormally large crown. Rudimentary development to my knowledge, has not been reported. The root may be surprisingly long, and Orr[212] found that 50% of unerupted canines if palatally placed, show dilaceration of the root. Hitchin (1951)[213] accounted for dilaceration by the devious path of eruption, during the formative period of root development : he believed that the apical portion lies in the direction of normal eruption, regardless of the degree and direction of diversion of the canine .

C. Chronology.

Logan and Kronfeld[62] noted the following :-

(i) Hard tissue formation commences at 4-5 months.

(ii) The crown is completed by 6-7 years.

(iii) Eruption occurs at 11-12 years.

(iv) Root formation is complete at 13-15 years.

Hurme (1948)[214] found that in girls, eruption takes place between 9 years 7 months and 12 years 4 months, with a mean eruption time of 10.98 years ; and that in boys, eruption occurs between 10 years 4 months and 13 years 1 month, with a mean eruption time of 11.69 years. He recommended that if the maxillary canine has not erupted in girls by 12½ years, in boys by 13 years, it should be regarded as impacted, or absent.

Broadbent [1] considered that the timely extraction of the deciduous canine is of aid in preventing maxillary canine impaction : he recommended serial cephalometric radiographs (P-A and lateral head) to anticipate lingual deflection and the idea therefore loses much of its practical advantage, except in specialist orthodontic practice.

D. Frequency of Impaction.

Blum (1923)[135] found that the maxillary canine is the second most frequently impacted tooth, and constitutes 22% of all impactions. Cramer[14] estimated that 6.12% of American white males and 18-20% of females suffer impaction of this tooth. Rohrer (1929)[14] found that the occurrence of impaction of maxillary canines is three times as frequent in females as in males, and that the maxillary canine is impacted twenty times as frequently as the mandibular canine ; the estimates published by Hitchin (1956)[215] confirm those of Rohrer. It is impacted unilaterally six times as frequently, as bilaterally.

E. Aetiology of Impaction.

A brief summary of the findings recorded in Part I ("Aetiology of Impacted and Unerupted Teeth") will be presented :-

1. Lack of space.- This explains many cases of impaction. Civilised diet and hybridisation are the main factors causing disproportion between tooth substance and its alveolar accommodation. The maxillary canine, erupting after premolars and incisors, inevitably suffers in the competition for space. Labial and "intermediate" impactions are reflections of lack of space. However, palatal impactions cannot be so explained. Dewel (1945)[23] considered contracted arches played no role in impaction, and Norton (1954)[216] found that "in the majority of cases in which unerupted canines are present, arch development is good,while there is adequate space for the unerupted tooth."

2. Palatal deflection.- In some cases, by over-retention of the deciduous canine, in others by labial placement of the lateral incisor, the erupting canine may be palatally deflected. Hitchin [215] stated that

"

"in the normal patient....the permanent second maxillary incisor is well inside the arch of the roots of the maxillary teeth, and only if the canine is deflected sufficiently palatally to become more palatal than the permanent second incisor root, does palatal impaction occur. Once this deflection has happened, it is unlikely, if not impossible, for palatal impaction to be avoided...."

3. Supernumary teeth.- These are rare in the cuspid region, but explained 7 of 109 cases of canine impactions studied by Hitchin[215]. Isokawa (1959)[217] et al. reported duplication of a maxillary permanent canine in a Japanese male. Odontomas are an occasional cause : Rounds (1951)[218] reported such a case.

4. Tumours.- These are an occasional cause. Gorlan and Chaudhry (1958)[219] reported the case of orbital displacement of the canine, by an adeno-ameloblastoma.

5. Aberrant axial inclination and location.- Nodine considered that if the tooth germ is aberrant in location or axial inclination, normal eruption of the canine is not possible.[14] Some cases of impaction may be explained by this theory, such as the unusual impaction in the naso-antral wall.

6. Premature loss of the deciduous canine.- Should the permanent lateral incisor suffer space deficiency, or erupt distally (due to a midline diastema), it may cause resorption of the root of the deciduous canine root and its premature loss. The lateral incisor thereby usurps the space intended for the permanent canine, which consequently becomes impacted.

F. Position of the Unerupted Canine.

Localisation of the unerupted canine is difficult, yet essential to the surgical removal or surgical exposure of the tooth. Standardised radiographic technique and painstaking clinical examination including mounted study models if necessary, alone will determine its exact location.

The unerupted canine is palatally embedded in 75% of cases (Rohrer, 1929 ; Hitchin, 1956). Its long axis may be vertical, which is unusual, more often oblique, sometimes horizontal. Lappin (1951)[220] claimed that the crown is nearly always mesially directed. The oblique position is three times as frequent as the horizontal position (Rounds, 1951 [218]). The unerupted canine is nearly always rotated about its long axis from 60'-90'. Occasionally it may be inverted (Nodine, 1944 [14]). Transposition is occasionally seen : Jackson [221] in 1951, reported the case of a fully erupted canine situated between the maxillary incisors ; Townend (1949)[222] reported the eruption of a canine between the premolars ; Jackson (1951)[223] reported the eruption of a canine in the position of a missing central incisor.

Aberrant canines have been reported in the nasal cavity (Nodine, 1926; [224] Abercrombie, 1925 [225]), the orbit (Carver, 1887 [226]) and the antrum.

Several attempts have been made to classify the position of the unerupted canine. No classification has become traditional, in the sense that it is quoted by all recognised textbooks : it may therefore be implied that no classification yet published has received unanimous acceptance (as Winter's classification of third molar impactions received). Winter (1940), [227] Field and Acherman (1935), [228] and Hitchin (1956) have each proposed a classification of the position of the unerupted canine, each of which are very similar - "variations of a theme" as it were ; Archer (1956) [229] published a classification which appears to be original in basis, but inferior I feel to the former classifications. The above-mentioned classifications possess a common denominator - the unerupted canine has been perceived through the eyes of a surgeon, with a view to its surgical removal. The unerupted canine was classified with regard to its position, from a completely different viewpoint - as seen by the orthodontist - and Adamson (1952)[230] based his classification of the unerupted canine, on the prognosis of disimpaction, which is surgical exposure, with or without, orthodontic traction.

1. Adamson's Classification .-

Group I : The tooth is almost vertical, slightly off normal course, of eruption ; apex lies to the mesial of the mesial surface of the first premolar root, crown no further than the distal surface of the lateral root ; prognosis slightly less if both extremes are present ; eruption after exposure should usually be spontaneous. Should the tooth be so near to the lateral root that bone cannot be relieved, disimpaction is impossible without injury to the lateral. Norton[216] added that the tooth may be either palatal or labial.

Group II : More deeply placed on the palatal side, with apex slightly distal to the first premolar, crown may extend to the mesial of the lateral incisor. Prognosis good if both extremes are not present. However, some will require orthodontic traction after exposure, and all will require orthodontic axial alignment upon eruption.

Group III : More horizontal, and deeply placed. Crown may extend to the midline, apex to the second premolar, even beyond. Often just under the nasal cavity. Will never erupt with surgical exposure alone, but may erupt with orthodontic traction. Morton[216] claims eruption of the horizontal canine is "impracticable".

To the three groups, Adamson further adds the rare case in which the canine is deeply placed, high in the labial bone, above the sulcus : surgical exposure is impossible to maintain, traction too traumatic to the lip.

Discussion.- I consider this classification to be an important contribution. The other classifications are based almost entirely upon the bucco-palatal relationship of the canine, in relation to the arch of the roots: Adamson's classification is based on the mesiodistal relationship to adjacent teeth. Not only is it a valuable guide to the orthodontist and general practitioner interested in the eruption of the tooth into functional occlusion, but also the surgeon, concerned with its removal.

2. <u>The classification of Field and Acherman</u>[228].- This classification has the recommendation of Thoma (1958)[133] for its guidance to surgical approach :-

Labial position	1. Crown in intimate relationship with incisors.
	2. Crown well above apices of incisors.
Palatal position	1. Crown near to surface in close relationship to roots of first and second incisors.
	2. Crown deeply embedded in close relationship with apices of first and second incisors.
Intermediate position	1. Crown between the second incisor and pre first/molar roots fairly close to surface.
	2. Crown above the teeth with the crown either labially placed and root palatally placed, or vice versa.
Unusual positions	In naso-antral wall

<u>Discussion</u>.- This classification is the most helpful for the bucco-lingual position of the unerupted canine, and is an amplification of Winter's original contribution. For simplicity, a sound asset in a classification to be used in teaching and everyday parlance, Hitchin's modification is worthy of mention.

3. <u>Hitchin's classification</u>.-

Type I : Canine palatal near gingival margin

Type 2 : Canine palatal away from gingival margin

Type 3 : Canine labiobuccal

Type 4 : Canine in the arch of the roots

Type 5 : Canine with crown palatal and apex buccal or above the apex of the buccal root of the first premolar

Type 6 : Rare impactions

Type 7 : Edentulous cases.

Hitchin, in his report on 109 cases of canine impaction, found that they occupied the following positions :-

Type 1 ...	18%	Type 5 ...	42%
Type 2 ...	0	Type 6 ...	1%
Type 3 ...	9%	Type 7 ...	18%
Type 4 ...	11%		

It will be seen that the canine lies palatally in approximately 75% of cases (which confirms the observation of Rohrer, 1929), and that about half of the unerupted canines are found with the crown palatal and the apex buccal, or above the apex of the buccal root of the premolar.

4. The degree of eruption.-

The maxillary canine may be (i) unerupted and bone-embedded;
(ii) unerupted and embedded in bone and soft tissue ;
(iii) partly erupted into the oral cavity;
(iv) partly erupted in the nasal or antral cavity;
(v) normally erupted in functional occlusion.

The clinical features vary in accordance with the degree of eruption.

G. Clinical Features.

The term "clinical features" of the unerupted canine refers to the knowledge, accumulated from past experience, of the behaviour of the tooth in given circumstances.

1. The first indication of impaction.- Often the unerupted canine is completely free of symptoms ; its presence is suspected by the parents pr the child when the deciduous canine is over-retained, or exfoliated normally, but not replaced by its successor. Routine dental or radiographic examination often leads to its discovery. Often its absence from the arch appears to have escaped detection, until, in later decades, it "erupts" under the maxillary denture.

The investigation of a swelling of the palatal or facial surface of the maxilla often results in its discovery. Other symptoms include tenderness and mobility of the incisors, the presence of a discharging sinus, occasionally nasal obstruction and discharge, and a variety of neurological symptoms. To the dental surgeon, failure of the canine to erupt in girls by 12½ years, in boys by 13 years, should indicate that its eruption has been impeded, and warrants investigation (Hurme[214]).

2. Infection.- The completely bone-embedded tooth is sterile, and symptoms arising from such a tooth must be due to pressure effects. The partly erupted tooth, however, is the seat of infection, which may be subclinical, chronic or acute ; the nature of the symptoms is largely controlled by its site of eruption - palatal, orbital, nasal, antral, or labial. Pericorenitis, according to Hitchin, is not common, and when it does occur, the notorious symptoms and signs of pericoronitis of the mandibular third molar are not seen. The reason for the mildness of pericoronitis in the maxillary canine region is the dependent drainage possible ; muscle movement does not propagate infection in the palatal impaction, but may disseminate organisms in the case of the labial impaction; in Class II malocclusion, the lower incisors may traumatise the operculum of the partly erupted palatal canine. Hitchin in 1956 asserted : "It is extremely rare for a cellulitis of the face to arise from pericoronitis of the maxillary canine, but it may occur, closing the eye on the affected side. Less rare, but by no means common, is chronic inflammation of the operculum over a partly erupted canine, which may be followed by suppuration and sinus formation. In such circumstances, local tenderness and an unpleasant taste may be the most conspicuous features." Periapical infection is liable to occur in the canine erupting labially, above the gingival level of adjacent teeth : the crown and gum flap become areas of food stagnation, difficult to clean naturally, or artificially, caries ensues, and if untreated, the usual sequellae result. The embedded canine may suffer infection by extension of periapical infection of adjacent teeth : a palatal abscess may follow, with the formation of a sinus,

3. Resorption of the roots of adjacent teeth.- The canine obviously exerts considerable pressure in its frustrated effort to

erupt. Resorption of the root of the lateral incisor, sometimes with its complete severance from the apex, or its complete absorption, has been reported by several authors, including Sealey (1955)[231], Sealey (1955),[108] Thoma (1954),[62] Rounds (1951)[218], Helmore and Norton (1954).[216] The root of the central incisor may be resorbed, even totally destroyed (Johnson,1944;[14] Birtwhistle,1945;[109] Osbourne King,1944[14]). Both lateral and central incisors may be resorbed.[108] Resorption of the roots of adjacent teeth is one of the strongest reasons against the laissez-faire attitude to the embedded canine.

4. Dentigerous cyst formation.- There is a high incidence of dentigerous cyst complication among embedded canines. Hitchin noted that 8 of his 109 cases had developed a cyst.[215] The cyst may arise at any time after the failure of the tooth to erupt, and is often a late development. Stafne and Austin (1945)[70] noted 3 maxillary canines long-embedded and ankylosed by coronal resorption, developed a cyst from the follicular remnants. The cyst on discovery, may range in size from a dilation of the follicle of minor degree, to a huge deforming cyst, with displacement of the canine to the orbit, encroachment of the antrum and nasal cavity, expansion of the palate and facial surface of the maxilla, and an infringement of the midline. The rate of growth may be extremely slow, but may be extraordinarily rapid : Rounds (1951)[218] reported the case of an 18 year old girl, who refused the removal of an embedded canine, at that stage, manifesting a normal follicular space; three years later, she had developed a cyst which already had invaded the nasal and antral cavities, crossed the midline, and completely denuded the root of a sound lateral incisor. Cyst formation is a serious complication which should militate against the tolerance of an embedded canine, regardless of apparent lack of pathology.

5. Coronal resorption.- Stafne and Austin (1945)[70] found that of embedded teeth undergoing coronal resorption and ankylosis, about 50% were canines, compared to the low figure of 10% for embedded mandibular third molars. Thus, the canine is especially liable to eventual coronal resorption, which multiplies the difficulties of surgical removal and increases the chances of complications, such as antral exposure, gross loss of alveolar bone, which creates an unmanageable prosthetic problem. Hitchin [215] found that 8 of his 109 impacted canines

showed coronal resorption, and interpreted in accordance with age, 25% of his "edentulous" cases were ankylosed. Should a canine be partly erupted and the site of purulent chronic infection, coronal resorption is often seen : Carco(1932)[14] reported one such case in a canine erupting into the nostril - he wrongly attributed the pathology of the crown to caries ; the crown invested in an infected dentigerous cyst will also show resorption. It should again be emphasised that coronal resorption and ankylosis is, per se, unrelated to sepsis, as shown by Logan (1937).

6. Neurological symptoms.- "Pain is not always a feature, but when it is present, it may be either local, or more neuralgic in character," commented Hitchin. Nodine (1944)[14] considered pain to be the most common symptom associated with the unerupted canine. Rarely is the pain localised, unless inflammation is a feature, and usually it is referred to the maxilla, eye, mastoid region ; it may be the "all-over" type of pain, and headaches, occipital and temporal, are a common feature. The rheumatic type of pain is sometimes felt in the shoulder with limitation of movement (Thoma, 1958[133] ; Hodes, 1948[232]). Wry neck has been alleviated by the removal of the unerupted canine. Facial paralysis has also been "cured" by removal of the unerupted canine, and such a case was reported by Zemsky (1923).[233] Archer (1956)[229] devoted considerable space in his "Manuel of Oral Surgery" to a case of choroiditis of unknown origin, "probably produced by the impacted, unerupted supernumary rudimentary cuspid".

7. Denture irritation.- That the first symptoms caused by the embedded canine may be its "eruption" under the maxillary denture, indicates that the maxillary canine may lie embedded for decades without giving rise to neurological symptoms.

H. Aberrant Canines.

An unusual position of the unerupted canine is not common, although the number of case reports in the literature would suggest otherwise.

1. Ectopic eruption into the nasal cavity.- Several cases have been reported, including those of Newland-Pedley (1891)[235],

Abercrombie, 1925;[225] Nodine, 1926;[224] Blum, 1931;[234] Caseo, 1932;[14] and Eriesson, 1944.[14] From a study of these case reports, I note that :

(1) the symptoms aroused, included epistaxis over a period of five years; a "tumour" in the nostril; nasal irritation; a foul, generally purulent discharge, with a history of chronic empyaema and nasal obstruction;

(ii) the tip of the canine may penetrate the floor ; the apex of the root may ulcerate the nasal mucous membrane ; the tooth may lie partly within the nasal cavity; partly within the antrum;

(iii) the canine may be inverted, or it may erupt down into the nasal cavity ; it may lie horizontally in the palate, or in the nasa-antral wall;

(iv) penetration of the nasal floor was one inch from the nares, in another case, only 4 mms.

2. Ectopic eruption into the orbit.- Carver (1887)[226] reported the case of a lad aged 6½ years, who complained of a small concrescence on the lower right eyelid where a hard white spot appeared ; gradually the tooth erupted and displayed a fully formed canine crown, with a part-ly formed root. Gorlan and Chaudhry (1958)[219] reported the displacement of a canine into the orbit by an adeno-ameloblastoma. Ocular symptoms do not imply orbital eruption or displacement, however, as they are occasional symptoms caused by embedded canines, remote from the orbit : flickering of the eye was reported by Wharburton-Brown (1900)[235]; lachrymation and inflammation by Luca (1944)[14]; impaired vision by Zemsky (1923)[233]; pain, discharge, and the "gritty" feeling of conjunct-ivitis, with partial loss of sight by Suckling and Martin(1933)[236]; retrobulbal neuritis with sudden loss of vision by Scroggins (1936)[237] who reported a cure by removal of the embedded canine.

3. Eruption into the antrum.- The canine may be embedded in very close proximity to the antrum without giving rise to antral pathol-ogy ; it may erupt into the antrum and an empyaema or polypoid reaction ensues ; it may be displaced into the antrum by a dentigerous cyst, which may rupture with infection, and the impression is gained that the canine lies completely free within the antrum. Field (1931)[238] reported

a chronic empyaema of several years duration, caused by the apex of a canine which lay in the antrum. Walker Downie (1893)[239] reported the case of a girl aged 6, who suffered acute empyaema, caused by a developing permanent canine lying within the antrum, embedded in its roof and anterior wall.

4. Transposition.- This has already been covered.

I. Clinical Examination.-

The results of the clinical examination serve as a cross-check of subsequent radiographic interpretation.

1. Age of the patient.- If eruption of the canine has not occurred by the age of 12½ in girls, and 13 in boys, it may be considered impacted, or agenetic. It has been mentioned above, that congenital absence of the permanent canine is rare, and if present, it is usually associated with the absence of other teeth, and possibly the other manifestations of ectodermal dysplasia (Hallett and Weyman, 1954[240]).

2. Deformity.- A palatal or labial bulge, hard and unyielding, may disclose the position of the crown of the unerupted tooth. Helmore (1954)[216] warned that "such a tumour may be difficult to detect, particularly in an arch where other teeth are malposed." Rounds (1951)[218] pointed out that the absence of swelling may indicate that the tooth lies within the arch of the roots. Small nodular, bone-hard prominences may be noted on palpation, and may disclose the presence of the rare supernumary tooth, or the odontoma. The examination should include a comparison of the one side of the palate with the other for symmetry - slight discrepancies in symmetry are very readily detected on the study model. The canine fossa should be checked for expansion, and if present, may be detected by a flattening or obliteration of the naso-labial fold, or a distortion of the ala naris : such signs betray the presence of a cyst or tumour, A painless, regular bony swelling , a resilient swelling, or a fluctuant swelling are the guises of a dentigerous cyst, and logically interpreted, they reveal the degree of integrity of the bony wall of the cyst. Displacement, mobility, and tenderness of teeth in relation to a cyst, reflect the degree of their involvement. An apical thrill is discernible on percussion of an involved tooth, and the note

is flat or "padded". The globulo-maxillary cyst should be incuded in the tentative diagnosis.

3. Occlusion.- The alignment of teeth is often most revealing. Helmore (1954)[216] stated : "If it be remembered that the crown of an unerupted tooth moves, or perhaps rather tilts, in the reverse direction to the pressure of the unerupted tooth if the pressure is exerted near the apex of the root, and in the same direction if pressure is exerted near the crown, useful information may be obtained and the amount of tilt is a fair indication of the pressure being exerted." However, one cannot be incautious in the interpretation of tilt : labioversion of the lateral incisor may indicate a palatal impaction near the gingiva, driving against the cervical third of the root, or a high, labial impaction, driving against the apical third of the root. Study models are of great help, and certain cases justify a bite analysis : premature occlusal contact may be noted by forceful extrusion of a tooth by the embedded canine, which may result in an excursion of convenience ; the palate of the model may be fruitfully scrutinised for early signsof enlargement, indicating the location of the crown of the canine, or an expanding tumour or cyst.

4. Vitality of adjacent teeth.- It is essential that the pulpless, infected tooth in the vicinity of the impacted canine be recognised pre-operatively : Rounds (1951)[218] confessed to having overlooked such a lateral, which was chronically infected, and a few weeks after the removal of the embedded canine, the wound,apparently healed, broke down by extension of infection ; Helsham (19)[241] however, in a similar case, noted the infected lateral incisor, and combined the surgical removal of the canine with an immediate root canal therapy with apicectomy of the lateral incisor, during the single operation. Vitality tests are unlikely to be negative in teeth undergoing pressure resorption, as they remain vital, regardless of the degree, providing there is no communication with the oral cavity: however, the more specific pulp tester (electric) may glean a variation in response -too subtle to be detected by the "hot" and "cold" tests - which may be of clinical significance.

5. A sinus.- A sinus may be located in the palate; it may be so small as to defy detection during a remission of infection, however, may generally be found if the patient has complained of an intermittent discharge. The sinus should be explored with a silver probe, and it may elicit the feel of the hard, glazed enamel of the canine.

6. Nasal symptoms.- As previously indicated, epistaxis, a foul purulent nasal discharge, nasal obstruction may indicate nasal eruption of the canine. The nasal cavity may be examined by dilating the nostril with a nasal speculum, and illuminating the cavity with a pencil light. The canine usually erupts about an inch from the nares, the mesiodens, about a centimetre from the nares, to judge by the few cases reported.

7. Antral investigation.- The symptoms of chronic and acute sinusitis should be recognised. The transillumination test is an aid, not a means of diagnosis, but nevertheless is helpful in determining when the antra are empty or filled -with polypoid tissue, fluid, a cyst or tumour.

8. Neurological symptoms.- The diagnostic method suggested by Haggett (1948) and Griffin (1957) should be used when neurological symptoms are associated with an unerupted tooth. It must not be assumed that because an unerupted tooth is present, therefore it is the cause of the symptoms.

J. Radiographic Examination.

A radiographic examination must be carefully carried out before a diagnosis is made, and surgical treatment undertaken. The precise position of the unerupted tooth must be visualised radiographically : a canine in a position from which unaided eruption may ensue may be needlessly extracted if the radiographs have been taken in a perfunctory manner to show the tooth in artifactual impaction. The position of the canine must be defined in each dimension- mesiodistal, vertical, and buccolingual - and its relationship to the antrum, nasal cavity, and adjacent teeth defined.

Of fundamental importance is the buccolingual relationship. While it is possible to remove an unerupted mandibular third

molar without an exact realisation of its buccolingual position, it is impossible to remove a palatally embedded canine through a labial approach if standing teeth are present.

Appraisal of the buccolingual position of the unerupted canine is difficult. Several methods have been suggested :

(i) the method of parallax applied to periapical radiographs, called the "tube shift" method ;

(ii) the occlusal radiograph (anterior, true, and vertex) ;

(iii) stereoscopic radiographs (intra-oral and extra-oral).

1. The periapical radiographs.- Helmore (1954)[216] recommended that the first radiograph to be taken should always be of the area which the tooth should occupy, and for this, the usual method of "bissecting angle, short-distance" technique is employed. Usually, in the palatally unerupted canine, its apex is to be found in the approximate position it normally would occupy. To identify the relationship of the canine to the adjacent teeth, periapical views of the incisor and premolar regions are required(Rounds, 1951). The periapical radiographs define the position of the canine in the mesiodiatal and vertical dimensions; by employing the technique described by Lappin (1951)[220], the buccolingual position of the canine may be assessed by parallax. Dilaceration of the root is present in about 50% of palatally impacted canines : this may not be shown in the radiograph, especially if the apex lies in the plane of the central ray (although failure of the shadow of the root canal to reach the end of the root should suggest apical curvature). Hitchin (1951) showed that the apex in the dilacerated root usually lies in the direction of normal eruption.

The follicular space of the canine should be noted : it may reveal a cystic condition, or ankylosis and coronal resorption. The periapical tissues of the erupted teeth should be examined for radiolucent "areas" usually denoting infection. The root of the lateral incisor may show pressure resorption.

2. <u>The occlusal radiograph</u>.- Most authors advocate the use of the occlusal film, which gives a plan of the area under consideration. However, there is a diversity of opinion regarding the technique to be employed. Common to each technique is the positioning of the film between the teeth, in the occlusal plane ; the angulation of the central ray differs. Three types of occlusal radiograph are in common use : the anterior occlusal, the true occlusal, and the vertex occlusal film.

(a) The anterior occlusal film is dependent on the beam directed downwards and backwards, from the cone placed above the nose : the angulation is perpendicular to the bissector of the angle between the long axis of the incisors and the occlusal plane. This film gives the exact length of the incisors, but does not indicate the exact buccolingual relationship of the unerupted canine, the shadow of which is usually superimposed upon the roots of the incisors. Nodine (1941)[242] and Rounds (1951)[218] are amongst those whose publications reflect their use of this view.

(b) The true occlusal film is dependent on the central ray directed vertical to the occlusal film. Whilst this angulation will show the posterior teeth (premolars and molars) in approximate cross-section, it will not show the cross section of the anterior teeth, due to their proclination. Therefore, a foreshortened image of the incisors is given, and it is likely that the canine may be superimposed upon their roots, this obscuring the buccolingual relationship, to such an extent that localisation is not reliable. Albright (1935),[243] Dewel (1945),[244] Thoma (1958),[133] and Helmore (1954) advocate the use of this view.

(c) The vertex type occlusal film is dependent on the central ray directed along the long axes of the incisors. The incisors, in the correctly taken radiograph, appear in exact cross-section with the cross section of the root canal clearly defined. It is obvious that the canine can be exactly localised buccolingually by this technique; Goldsmith (1931),[245] Field and Acherman (1937),[5] Hitchin (1937),[246] have pointed out the importance of the vertex film and its technique has recently been described by Riordan (1957).[247]

<u>Discussion</u>.- In the true occlusal view, and the vertex view, the beam is often in the former, and always in the latter, directed through the

frontal bone, which not only necessitates a long exposure time, but also superimposes the dense radiopaque shadow of the frontal bone over the area to be examined. To overcome the difficulty of long exposure, an ultra-fast film may be used, and intensifying screens in an intra-oral cassette. To overcome the blurring of the image by scattered radiation and to improve the degree of contrast, a grid may be used in conjunction with the intra-oral cassette with its intensifying screens. The anterior occlusal view has no place in the buccolingual localisation of the impacted canine. The true occlusal film is not entirely reliable especially when there is marked proclination of the incisors. The vertex film will define accurately the buccolingual relationship of the canine to the incisor root arch. It should be stressed that the occlusal films of the true and vertex type do not give detail of bone structure of the finer type, but may be employed with benefit, to define the general antero-posterior and lateral limits of a cyst, especially if lipiodol injection is employed. For clear definition of the fine details of bone structure, of the follicular space, and of the roots of standing teeth, the periapical films cannot be surpassed.

3. Stereoscopic films.- In cases of unerupted canines difficult to localise, the use of stereoscopic films is recommended by Dewel (1945),[244] Thoma (1958).[133] The difficulty in technique lies in the placing of two films in replicated position. Hitchin (1937)[246] described a technique to overcome the difficulty. A periapical film holder is constructed from a flanged Spencer Wells forceps, and the holder is held in relation to the teeth by means of a compound impression. Periapical, occlusal, lateral head, and P-A films may be examined stereoscopically when required. Such an examination is beneficial in the case of aberrant canines, in unusual positions such as in the antrum, nasal cavity, or in the naso-antral wall (Hitchin, 1956; Riordan, 1957).

K. Treatment Considerations.

1. Ideally, the canine should be investigated, when it fails to erupt in girls by the age of 12½ years, and in boys, by the age of 13. The investigation may require not only clinical and radiographic examinations, but also mounted study models, and the teamwork of the orthodont-

ist, and oral surgeon is required in most cases. The following possible forms of treatment are usual :-

(i) Non-interference if it is believed that dental development is somewhat retarded, and that the canine will erupt spontaneously, given ample time : adequate space for its accommodation, freedom from impediments of any kind, normal axial position, and a normal eruptive force are pre-requisites of "masterly inactivity".

(ii) Orthodontic provision of space alone may be necessary for its eruption.

(iii) Surgical exposure of the tooth, with or without orthodontic traction. The orthodontist must make the decision as to the practicability and desirability of bringing the canine into occlusion : Adamson's classification of the impacted canine indicates the prognosis for each type of case, and the orthodontist must decide whether provision of space pre-operatively is essential, whether traction on exposure should be arranged, and whether axial correction of the canine on eruption is necessary. The oral surgeon must decide whether surgical exposure is practicable, and he will base his opinion on the relation of the canine to other teeth; whether surgical exposure may be maintained for an adequate period (surgical exposure for instance, is impossible to maintain in the high labial impaction). It is his responsibility to provide a channel through which the tooth may erupt, and this involves the freeing of the greatest diameter of the crown of palatal muco-periosteum, bone, and impediments such as supernumary teeth, the root of a deciduous tooth, or a small cyst.

(iv) Surgical removal of the canine.-

(v) The unerupted canine may be retained in situ, and the patient or the parents of the child must be informed of the possible complications which may follow this course. Periodic examination of the tooth should be made to avert the dangers of retention.

2. What are the damgers of retaining an unerupted canine ? It has been shown that the unerupted canine is capable of undergoing

cystic degeneration at any period after completion of its crown : the sequellae of cyst formation are deformity of the facial, palatal aspects of the maxilla, encroachment of the antrum, nasal and oral cavities, with or without infection ; displacement, loosening, and denudation of the erupted teeth. The canine may destroy by pressure resorption, the roots of erupted teeth ; it may be the cause of diverse neurological symptoms ; it may be the seat of infection if partly erupted, or ectopically erupted. In later life, it may become ankylosed by coronal resorption, and its eventual exposure by alveolar atrophy will cause denture irritation necessitating its removal ; surgical removal of the embedded canine in the elderly patient is complicated by ankylosis, slower healing powers and the advanced degree of pneumatisation of the antrum.

3. When should the unerupted canine be deliberately retained? Surgical removal of the canine may cause the loss of one or more incisors or their devitalisation. Should such a canine show no evident pathology, retention is justified providing that the parents understand the need for periodic follow-up radiographs. Inaccessibility of the unerupted tooth, especially when extensive surgery is contra-indicated for reasons of mental or physical health, may justify the policy of laissez-faire. When retention is deemed advisable, the deciduous canine if present may be retained for cosmetic reasons, as such a tooth may remain functional for four decades.

THE MANDIBULAR SECOND PREMOLAR

A. Congenital Absence.

The mandibular second premolar is the most frequently missing tooth of the permanent series, the third molars excepted. Dolder's figures indicate that approximately one person in every hundred lacks the tooth. 211

B. Anatomy.

The tooth shows considerable variation in crown and root anatomy. Stones[13] noted that the mandibular second premolar displayed an abnormally large crown occasionally. Great variation in form and size is found in this tooth in the West African Negro.

C. Chronology.

1. Hard tissue formation commences at $2\frac{1}{4}$ - $2\frac{1}{2}$ years.
2. Enamel is completed by 6 - 7 years.
3. Eruption takes place at 11 - 12 years.
4. Root formation is complete at 13 - 14 years.

(Logan and Kronfeld,[62])

D. Frequency of Impaction.

It is the fourth in frequency of impaction (Blum, 1921 ; Nodine, 1944). It is impacted seven times as frequently as the first mandibular premolar, twice as frequently as the maxillary second premolar. In U.S.A. studies, it is impacted four times as frequently in males. Bilateral impaction is not uncommon. In this region, impaction of the supernumary third, fourth, and rarely the fifth premolars is an occasional finding, and is commonly found in West African Negroes.

E. Aetiology of Impaction.

1. Lack of space is the most usual cause of the vertical type impaction. Premature loss of the deciduous molars, especially the second, by caries, permits the first permanent molar to tilt and drift forwards.

The sequence of eruption of the mandibular permanent teeth is unfavourable to the eruption of the mandibular second premolar : the second premolar erupts after the first permanent molar, the canine and the first premolar have taken up their position - the second premolar must therefore suffer space deficiency. The mandibular second premolar is more liable to impaction as a result of premature loss of the deciduous molars, than its antagonist, due to the greater propensity of the mandibular first molar to drift mesially and tilt; another reason is that the maxillary first premolar may drift forward to usurp part of the space intended for the later erupting maxillary canine, whereas the mesial drift of the mandibular first premolar is impossible due to the earlier eruption of the canine (and its inherent tendency to drift not mesially, but distally).

2. Premature loss of the permanent first molar before eruption of the second premolar of the mandible, permits the latter to rotate about its apex, intra-osseously, in a distal direction; this permits either the eruption of the second premolar a considerable distance distally, or, more usually, its impaction against the mandibular second molar. Should the second molar also be extracted, the second premolar may contimue its distal rotation, until it lies horizontal : distal migration usually occurs, and the second molar may become impacted against the third molar. The maxillary second premolar is unaffected usually by premature loss of the first permanent molar, as it has a tendency to migrate mesially, not distally.

3. Ankylosis of the second deciduous molar, though not a common occurrence, inevitably causes the impaction of the mandibular second premolar.

4. Supernumary premolars are relatively common in West African Negroes, but rarely seen in the Caucasian. The supernumary premolars rarely cause the impaction of the second premolar, as they commence eruption at a later stage, than the premolars of the normal series, therefore the supernumary premolars themselves are usually impacted.

5. Aberrant axial inclination, or abnormal location of the tooth germ explains only the occasional case of impaction. Examples of these conditions are : the inverted second premolar, and the buccal or

lingual transverse impaction. Occasionally, the partly formed crown of the second premolar is seen to lie distoangularly or distally inclined within its crypt, with the permanent first molar present. This represents a case of the genuine aberrant second premolar.

6. Lack of jaw development, hybridisation, abnormal habits of childhood, and factors influencing adversely the eruptive force, explain some cases of impaction and non-eruption.

Discussion.- The impaction of the second premolar of the mandible may be prevented by the general practitioner in most instances : preservation of the deciduous molars and the first permanent molar, and the early recognition of ankylosis of the deciduous molars will avert the majority of impactions. The premature loss of the deciduous molars is serious, not only from the viewpoint of permanent first molar drift and tilt, but also on account of the healing of their sockets by bone scar, which covers the roof of the premolar crypts with dense impenetrable bone, which must be surgically removed in many cases, before the eruption of the premolars may ensue. Should the tooth germ of the mandibular second premolar be distally inclined, and the first molar standing, early recognition may enable the surgeon to "re-orientate" the partly developed tooth - a practice which is termed "surgical orthodontics" by Holland ~~Biederman~~ (1956)[60].

F. Position of the Impacted Mandibular Second Premolar.

Relatively little has been published in the literature concerning the impaction of this tooth ; the exceptions are the excellent articles by Nodine (1944)[14] and Bluestone (1951).[248] No satisfactory classification of the position of this tooth has been presented.

The impacted second premolar may occupy the following positions :-

1. Vertical (due to "blockage" by premature loss of the deciduous molars, or to ankylosis of the second deciduous molar).

2. Disto-angular (due to premature loss of the first permanent molar before eruption of the second premolar)

3. Horizontal (due to the continuation of rotation of the distoangular type, on loss of the second molar, or due to a primary aberration of the tooth germ).

4. Inverted (a rare position, due to congenital displacement of the tooth germ, or to an acquired displacement - such as may occur in mandibular fracture, or forceps trauma).

5. Mesioangular (a rare position, reported by Thoma[133]).

6. Transverse linguoangular (a case reported by Salter, 1944[14])

Bluestone, in a study of 24 cases of unerupted lower second premolars, published the following data :

Horizontal position	..	15 cases
Vertical position	..	4 cases
Distoangular position	.	2 cases
Lingually malposed	..	3 cases

No conclusions can be drawn from the above report.

The vertical type impaction frequently displays a lingual deflection, and the partly erupted premolar usually penetrates the gingival tissue to the lingual of the adjacent teeth.

McCall (1944)[249] published an interesting radiograph of an aberrant second pre/molar tooth germ : it lay between the distal root of the second deciduous molar, and the mesial root of the first permanent molar. The horizontal impacted premolar displays surprising mobility : it has been found under the first molar (Nodine,1944), below the second molar (Dearden, 1939[250]), below the third molar,and impacted crown to crown against a horizontally embedded third molar (Coleman[14]). Coleman[14] also reported the case of a 50 year old woman, in whom the embedded premolar abutted against the coronoid process.

Inversion is not common. Phillips (1935)[251] reported one case in which the inverted second premolar drove against the mental foramen . Eruption through the lower border has been reported.

G. Clinical Features.

Many unerupted mandibular second premolars cause no symptoms until in later life, they "erupt" under a denture. Retention of a deciduous second molar may lead to its discovery. A hard lingual swelling, which may interfere with the tongue, or a denture or the lingual bar of a denture, may result in its diagnosis.

The following are some of the effects caused by the unerupted second premolar :-

1. Infection is not found in the completely bone embedded tooth. In the partly erupted tooth, a chronic pericoronitis, which may sometimes be acute, but less marked than the mandibular third molar pericoronitis, is seen, and Bluestone reported a long-standing recurrent Vincent's infection caused by a partly erupted second premolar lying under a bridge pontic. A sinus occasionally leads to the discovery of the tooth : Jessop (1898)[14] reported a sinus of the skin of the cheek .

2. Resorption of the roots of adjacent teeth.- Berkshire (1944)[252] reported resorption of the mesial root of the lower first molar.

3. Coronal resorption.- Stafne and Austin (1945)[70] found this tooth to be resorbed in only 7 of 300 cases of coronal resorption.

4. Dentigerous cyst formation.- This is an occasional finding.

5. Neurological symptoms.- Pressure effects are possible due to the intimate relationship to the inferior dental canal and the mental foramen. Pain referred along the jaw to the auriculo-temporal region, with tinnitus of the ear, is sometimes found.

6. A predisposition to fracture of the mandible through thec embedded tooth crypt is a real danger. I have observed several cases of fracture through unerupted premolars of the supernumary series, in West African Negroes. Ivy (1915) [253]reported a traumatic fracture of this type. A surgical fracture is likely, when the embedded premolar must be removed from the frail, excessively resorbed mandible of the elderly patient, especially if the tooth is ankylosed.

H. Radiographic Examination.

Two intra-oral radiographs - the lingual and the occlusal - adequately localise the unerupted lower second premolar in most cases. Should distal migration have occurred, a lateral jaw radiograph should supplement the intra-oral views. The relationship of the premolar to the other teeth, the mental foramen, the inferior dental canal, and the lower border of the mandible, should be clearly demonstrated.

I. Treatment Considerations.

In the child, and often in the young adult, it is usually necessary to gain an orthodontic opinion, before the fate of the unerupted second premolar may be decided.

1. Preventive measures to promote unaided eruption.

(a) The deciduous molars should be preserved ; space retention is required should premature loss be unavoidable.

(b) The ankylosed second deciduous molar must be diagnosed early, and immediately removed ; a space retainer must be inserted until the premolar eruption is imminent.

(c) The permanent first molar must be preserved ; if its early loss in unavoidable, every effort should be made to delay its extraction until the second premolar has erupted ; distal migration of the erupted premolars may the prevented by a suitable appliance after the first molar is extracted.

(d) Abnormal childhood habits predisposing to "collapsed" arches, should be diagnosed early, and corrective treatment instituted, to prevent lingual maleruption or impaction of the second premolar.

2. Surgical exposure is indicated when (i) eruption is delayed, (ii) the eruption of the premolar is desirable, (iii) the tooth is in a satisfactory position for eruption, (iv) adequate space exists, or can be provided by orthodontic appliance therapy, (v) bone overlies the greatest diameter of the crown.

3. "Surgical orthodontics" - the practice of re-orientating an aberrant tooth germ - has been applied with success by Holland[60] to selected cases (vide p. 310). The unerupted, impacted, partly formed premolar is surgically displaced from an angular, to a vertical axis.

4. Surgical removal is the treatment of choice when the eruption of the second premolar is either impossible, or undesirable. To retain an unerupted second premolar is not without risk : cyst formation, pressure resorption of the roots of adjacent teeth, a predisposition to mandibular fracture are among the sequellae ; should removal be postponed until the patient is edentulous, and the mandible atrophic, mental anaesthesia and surgical fracture of the mandible are likely complications. Orr[254] advocated the removal of the first premolar, with exposure and traction of the second premolar in young children in order to avoid mental anaesthesia and a difficult surgical procedure when space cannot be provided for both premolars.

THE MAXILLARY SECOND PREMOLAR.

A. Congenital absence.

Dolder (1937)[211] found that this tooth constituted 25.3% of congenitally missing teeth, third molars excluded.

B. Frequency of impaction.

Blum (1923)[135] found that the maxillary second premolar totalled only 1% of the 467 impacted teeth in his collection. Nodine (1944)[14] found it to be impacted only half as frequently as the mandibular second premolar.

C. Aetiology.

Lack of space due to premature loss of the deciduous molar, collapsed arches and extreme apical base deficiency is the principal cause of impaction. Ankylosis of the deciduous predecessor is much less common in the maxilla. Occasionally, the tooth germ may be aberrant in axis.

D. Position.

The impacted and unerupted maxillary second premolar is usually vertical, and palatally deflected. Maleruption is not an uncommon finding, and it may erupt in the palate, between the first molar and the first premolar, in torsional deflection. Inverted maxillary second premolars have been reported by Coleman (1944)[14] and Hughes (1931),[255] and the crown may be directed mesially or distally. Morrison (1935)[256] reported a horizontal impaction lying with its crown distally, below the occlusal surface of an unerupted maxillary third molar.

E. Radiographic Examination.

The periapical film will usually reveal the position of the impacted premolar, in a mesiodistal and vertical dimensions. Palpation may reveal the crown lying under the palatal mucops membrane. Should palpation be negative, a vertex or true occlusal film is helpful. Extra-oral radiographs (P-A and lateral head) aid in the localisation of the premolar in an unusual position.

F. Treatment Considerations.

1. Preventive measures.

(a) Preservation of the deciduous molars prevents the mesial drift of the maxillary first permanent molar. A space retainer should be provided on loss of deciduous molars by caries.

(b) Ankylosis of the maxillary second deciduous molar should be diagnosed early, the tooth removed and a space retainer substituted.

2. Surgical exposure, combined with orthodontic provision of space and traction if necessary, is the treatment of choice in selected cases. However, orthodontic opinion in recent years has veered towards the acceptance of apical base deficiency, and its correction by the sacrifice of the second premolars.

3. Surgical removal is the treatment of choice when eruption is impossible or not desired.

THE MANDIBULAR CANINE.

A. Congenital Absence.

This tooth is very rarely absent, except in cases of partial anodontia or agenisis of the permanent dentition.

B. Anatomy.

The crown veryrarely shows variations in form. The root is variable, and occasionally the canine is dual-rooted.

C. Chronology.

(1) Hard tissue forms at 4-5 months.

(ii) Enamel formation is complete by 6-7 years.

(iii) Eruption occurs at 9-10 years.

(iv) Root formation is complete by 12-14 years.

(Logan and Kronfeld[62]).

D. Frequency of Impaction.

Impaction of this tooth is not common. Blum[135] found that it constituted 2% of the impactions he studied. Rohrer[14] found that the maxillary canine was impacted twenty times as frequently as the mandibular canine.

E. Aetiology.

The sequence of eruption of the permanent teeth of the mandible (6,1,2,3,4,5,7,8) explains why impaction of the canine is uncommon. Space deficiency is suffered by the mandibular second premolar not the canine. The lower incisors are small, weak teeth, and tend to overlap when space is deficient, and, unlike their antagonists, do not effect premature resorption of the deciduous canine.

Buccal maleruption is the usual sequella of gross space deficiency.

Aberration of the tooth germ in axis explains some cases of impaction.

Supernumary teeth are rare in the lower canine region.

F. Position of the Unerupted Canine.

The most common position is the horizontal, with the crown directed towards the symphysis. It develops deeply towards the lower border of the mandible : therefore, it may be found in the body of the mandible (Thoma,1954[62]). The horizontal canine, with its apex under the premolars, and its crown level with the incisor roots, has been reported by Lodge (1909),[14] Lamb (1912),[14] and Brindlye (1928).[14]

The mandibular canine, when horizontally embedded in the body, shows a remarkable tendency towards migration :-

(i) Hopewell-Smith (1911)[14] reported the finding of a congenitally missing left mandibular canine, with a supernumary impacted horizontally, its crown directed distally against the partly erupted right canine. What in fact Hopewell-Smith reported, was the first published case of the migration of a canine across the midline of the mandible.

(ii) Fitzgerald (1958)[156] reported the case of a woman in her late twenties, whose mandibular canines had failed to erupt. A radiograph of the left mandible revealed absence of the left canine. However, a radiograph of the right mandible, revealed the left canine, horizontally embedded, with its crown beneath the first and second right premolars, and the right canine horizontally and labially embedded in relation to the right incisors.

(iii) Thoma (1954)[62] reported a similar case in which a horizontally embedded canine had migrated to the opposite side of the mandible, its distally directed crown level with and below the apex of the mesial root of the opposite side first molar. That the tooth had migrated across the midline was proved on surgical removal, by Rose, who found that bilateral regional anaesthesia was necessary : the canine had carried with it, its nerve supply.

(iv) Bruzst (1958)[257] reported a case of the same type.

Not all canines which fail to erupt are horizontal. The vertical impaction is sometimes seen, lying between the first premolar and the lateral incisor. The ~~access~~ axis is usually mesially inclined, and,

as a result, the lateral and central incisors are displaced by pressure, which causes their mobility, tenderness and malalignment: resorption does not occur when a tooth is more easily displaced. Charyn (reported by Nodine,1944[14]) described such a case.

Ectopic eruption has been reported. Truman (1891)[14] Whittles (1901),[14] and Middleton Shaw (1935)[14] reported eruption of the inverted canine through the skin of the chin ; infection by skin organisms leading to sinus formation and sequestration, occurred in the cases of Truman and Whittles. Colyer (1891)[14] reported eruption of a mandibular canine under the tongue. Mason (1874)[14] reported a horizontally embedded canine with its apex buried in the lower lip, causing a swelling of the buccal sulcus.

Bluestone (1951)[248] found six impacted mandibular canines in 40,000 army veterans. Three were horizontal, two vertical, and one oblique.

In the elderly, the usual position found is horizontal, and such teeth may only be noted late in life, after loss of all alveolar bone has exposed them.

G. Clinical Features.

1. Infection.- The bone-embedded tooth is sterile. The partly erupted canine is the seat of pericoronitis, which is generally mild: the crypt when exposed, may be the focus of Vincent's infection. Infection may occur when an associated dentigerous cyst perforates the cortical plate.

2. Coronal resorption.- There is a high incidence of coronal resorption with ankylosis in the embedded canine. Stafne and Austin (1945)[70] found that 17 of 300 teeth so affected, were mandibular canines.

3. Dentigerous cyst formation.- As with other impacted teeth, cyst formation is possible.

4. Pressure effects.- Displacement of the lower incisors appears to be the only pressure effects exerted - neurological symptoms and root resorption being rare findings.

5. Retention of the deciduous canine.- The deciduous canine may remain in function until the age of 40-45 years ; root resorption may, or may not occur, and often is arrested by loss of vitality of the cementum with ankylosis resulting.

6. Swellings of the jaw.- Palpation of the buccal and lingual surfaces of the mandible may reveal swelling in association with the unerupted tooth. It may be due to the emergence of the crown through the bone, or to a dentigerous cyst.

H. Radiographic Examination.

When a mandibular canine is missing, it may be regarded almost certainly, as unerupted, for congenital absence is rare. Radiographic investigation should not be confined to the canine region, as migration across the midline may have occurred.

The buccolingual relationship of the horizontal canine is important, as it may be to the lingual of the apices of the lower incisors. Therefore the occlusal film is obligatory. The method of parallax may be employed to determine the buccolingual relationship.

When the horizontal canine has migrated across the midline, its relationship to the mental foramin and the mandibular canal is important : it is more likely to lie beneath the canal than above it.

I. Treatment Considerations.

The horizontal canine may be extremely difficult to remove, especially when deep, and lingual to the lower incisors. Trauma to the apices of the incisors and premolars is possible during removal, and there is the possibility of mandibular fracture. The advantages of removing the deeply embedded canine must be carefully weighed against the possible dangers : there is a good case for "masterly inactivity", but when this policy is pursued, periodic radiographic examinations are essential.

SUPERNUMARY TEETH.

Blum (1923) [135] found that supernumary teeth constituted 4% of all unerupted teeth in his study. The incidence of supernumary teeth in American whites Stafne (1932)[258] found to be 0.91%, and his survey indicated that 441 persons of 48,550 examined, possessed 500 supernumary teeth.

The distribution of supernumary teeth.-

(i) Central incisor region of the maxilla 46%

(ii) Third molar region of the maxilla 38%

(iii) Premolar region of the mandible 6%

It should be stressed that Stafne's figures as shown, apply to the Caucasian of America. There are variations in incidence and distribution from race to race.

THE SUPERNUMARY CENTRAL INCISOR.

A. Anatomy.

The supernumary tooth which is found between the central incisors is termed the "mesiodens". It is shorter than the adjacent permanent teeth and peg-shaped.

B. Clinical Features.

1. Number.- Stafne found that single supernumary incisors were eight times as common as bilateral ones.[258] Orr (1959)[254] reported four supernumary incisors in the midline of the maxilla, and warned that the discovery of one supernumary incisor, should lead one to suspect the presence of others. Fastlicht (1943)[259] reported a central incisor obstructed by six midline supernumaries.

2. Eruption.- Stafne[260] found that only 10% of supernumary incisors erupted, some palatally, some between the central incisors, and some in place of the central incisors, which were consequently malposed.

3. Nasal eruption.- Nasal eruption is not a common finding, but such cases have been reported by Charleroi (1911),[14] Long (1924)[14] and

Boval (1911)[14]. That more do not erupt into the nose is surprising, as inversion of the mesiodens is common. Eruption into the nasal cavity occurs approximately 1 cm. from the nares. The nasally erupting canine penetrates the floor approximately one inch from the nares.

4. Inversion.- 50% of the supernumary incisors reported by Stafne[260] were inverted. Orr (1959)[254] doubted the wisdom of removing inverted supernumaries, due to their deep position, and the possibility of injury to the central incisors, unless periodic radiographs revealed pathology.

5. Resorption.- Stafne and Austin (1945)[70] found that of 300 embedded teeth showing coronal resorption, 20% were supernumary incisors, and noted that almost complete elimination by resorption occurred in some cases by 35 years of age.

6. Cyst formation.- Stafne (1932)[260] found that 5.5% of 200 supernumary incisors were associated with dentigerous cyst formation. Nasal obstruction, and rupture of the cyst into the nasal cavity are possible sequellae.

7. Effects upon adjacent teeth.- The most common cause of impaction of the central incisor, is the presence of a supernumary incisor. Delayed eruption of the central incisor shouls suggest an embedded supernumary. A diastema between the central incisors is often caused by a mesiodens, and its presence prevents all orthodontic measures to eliminate the space, which could secondarily cause impaction of the canines. Malposition (e.g., palatal eruption of the central incisors) is often a sequella. Resorption of the roots of adjacent teeth is an uncommon finding.

B. Clinical Examination.

1. The occlusion.- Delayed eruption, malposition (labial, palatal, or torsional) of the central incisors, the presence of a diastema, especially in the absence of a pronounced labial frenum, suggests that a supernumary tooth may be the cause. The central incisors and the supernumaries may be erupted : the eruption of one supernumary should lead to a thorough investigation of the possibility of others.

2. Palpation.- The presence of a supernumary may be reflected by a high labial swelling, and such a finding can aid the localisation of the tooth. However, the majority of supernumary incisors are located palatally to the central incisors (Orr,1959).

C. Radiographic Examination.

The periapical type radiograph will reveal the presence of the supernumary incisor. It will not, however, reveal its exact position. It is often difficult to discern the supernumary when it impacts the central incisor, as the supernumary or supernumaries, are usually superimposed upon the crown of the latter and lie against the concavity of its lingual surface. The buccolingual relationship of the supernumary cannot therefore, be determined by the "tube shift" or parallax method.

A vertex occlusal film, which should reveal the erupted incisors in true cross section, is invaluable in determining the buccolingual position of the supernumaries: A lateral head film is recommended by Cranin and Cranin (1958)[261] to determine the axial inclination of the supernumary in the P-A plane, and its absolute depth of impaction.

D. Treatment Considerations.

The erupted supernumary should be extracted, as it causes speach disabilities when palatally placed, and is unsightly when lying in the diastema between the erupted central incisors. Pericoronitis is to be expected in the partly erupted , palatally placed supernumary, especially when a class II div. I malocclusion permits palate biting by the lower incisors.

The embedded supernumary causing occlusal disharmony should be removed in order that orthodontic repositioning of the teeth is possible.

The embedded supernumary causing impaction of the central incisor or incisors, should be surgically removed, and the latter surgically exposed to promote their eruption. The operation

should be performed as soon as possible after diagnosis.

The symptom-free embedded supernumary incisor should be removed, unless the surgical dangers or the general condition of the patient suggest otherwise. If retention of the supernumary is decided upon, or the patient refuses surgery, a periodic radiographic examination is advisable, in view of the high incidence of cyst formation. Neurological symptoms have been reported, and may be bilateral in distribution. If the supernumary is proved to be the cause, its removal is, of course, indicated.

OTHER SUPERNUMARY TEETH.

A. The Supernumary Mandibular Premolars.

These teeth usually resemble the premolars of the normal series in anatomy. Third, fourth, and occasionally fifth premolars are discovered. Rarely do they interfere with the eruption of the normal series premolars; either they remain embedded in the mandible, or they erupt in lingual malposition. The embedded premolar may cause resorption of the roots of the erupted teeth. The lingually erupted supernumary premolars almost invariably give rise to complications ; the supernumary premolars, lying to the lingual of the first and second premolars, form a "cluster" of teeth, between which food packs, causing severe periodontal abscesses and caries.

Rubin (1958)[262] reported an interesting solution to the problem of three embedded supernumary mandibular premolars : two were vertical, one lay horizontally close to the mandibular canal. The first molar and second premolar were non-vital. He extracted the two non-vital teeth, surgically exposed the supernumaries. The two vertical premolars rapidly erupted into occlusion, and the horizontal premolar rotated about its apex, until its crown emerged, when it was extracted easily, without trauma to the mandibular canal. This case emphasises the fact that embedded teeth should be examined as indicidual cases, and treated as the circumstances demand.

B. The Paramolar of Bolk.

This supernumary tooth, a common finding in the

Negro, is almost invariably conical in crown, with a single, short, conical root. Usually it erupts into the buccal embrasure, rarely the palatal embrasure, between the maxillary second and third molars. Should the maxillary third molar be congenitally absent, it replaces it. The buccally erupting paramolar predisposes to caries, and periodontal abscess formation, due to food packing and calculus accumulation. There is no reason to justify retention of the paramolar once it has erupted.

C. The Fourth Molars.

The fourth molar may be fused to the third molar, or it may cause the impaction of the latter. In clinical features, clinical and radiographic examination, it does not differ appreciably from the third molar.

THE MAXILLARY CENTRAL INCISOR.

A. Congenital Absence.

Dolder (1937) found no case of agenesis of this tooth in 10,000 Swiss children.

B. Anatomy.

This tooth shows uniformity of anatomy, although it may be abnormally large of crown.[13]

C. Chronology.

1. Hard tissue formation commences at 3-4 months in utero.
2. The enamel is completed by 4-5 years.
3. Eruption occurs at 7-8 years.
4. The root is complete by 10 years.

(Logan and Kronfeld[62])

D. Frequency of Impaction.

Blum (1923)[135] found that 2% of all impacted teeth studied were maxillary central incisors. Nodine[14] claimed that the

tooth is fifth in frequency of impaction. Though not of frequent occurrence, impaction of the central incisor produces a most conspicuous deformity.

E. Aetiology.

1. Supernumary tooth.- This is the most frequent cause, and has already been discussed.

2. Odontoma.- This is a relatively common cause. A nodular, bone-hard swelling of the labial surface of the alveolar process, associated with non-eruption of the central incisor, is almost pathognomonic. The odontoma may range in size from a small cherry, to a large walnut, and it usually contains twenty or thirty denticles, shaped like a miniature conical mesiodens.

3. Dilaceration of the root.- This may be caused by trauma ; if the deciduous incisor is driven by a blow into the alveolar process, the epithelial sheath of Hertwig is likely to be distorted, with the result that the root of the developing tooth forms at an angle to the axis of the tooth. Eruption is impossible in most cases of dilaceration, despite surgical exposure and orthodontic traction.

4. Scar tissue.- Should the deciduous central incisor be lost prematurely by caries or trauma, the socket fills with bone, through which the permanent tooth must later erupt. Should the lower incisors occlude on the gum, it will become fibrous and resistant to eruption of the permanent tooth.

5. Alveolar clefts.-

F. Position.

Nodine[14] noted that "they are found in all angles in relation to the vertical axis. They are not uncommonly discovered in a horizontal position with the incisal edge of the crown directed labially or palatally." Occasionally a central incisor may cause destruction of some part of the nasal fossa, rarely have they been discovered inverted.

Ectopic eruption is a very occasional finding. The central incisor has been found buried in the upper lip, erupting through the upper lip(and in these cases violence at an early age is generally the cause) and erupting into the nasal cavity. The unerupted incisor may be found in relation to an alveolar cleft. A case of the central incisor buried in the hard palate has been reported.

G. Clinical Features.

1. Sex distribution.- The female is more commonly affected.

2. First symptoms.- Retention of the deciduous incisor, and delayed eruption usually lead to a diagnosis by the age of 9-10 years.

3. Complications.- Should the patient not seek treatment and correction, cyst formation, neurological symptoms (pain, local and referred to the face, eye, or head) and eventually, denture irritation may be suffered. Coronal resorption does occur and Stafne and Austin (1945)[70] found that 4 of 300 embedded teeth undergoing resorption were central incisors of the maxilla.

H. Clinical examination.

1. Occlusion.- Note the alignment of the adjacent erupted teeth : rotation or palatal maleruption may suggest the presence of a supernumary. The degree of overbite should be observed : the greater the overbite, the more likely the possibility of traumatic loss of the deciduous central incisor, which may suggest dilaceration of the permanent central incisor ; early loss of the deciduous tooth by caries or trauma may lead to scar tissue formation of the gingival tissue, by its occlusion with the lower incisors.

2. History.- The following factors may lead to a sound tentative diagnosis : (i) early loss of the deciduous incisor by caries or trauma; (ii) a severe infection (such as infantile osteomyelitis) may cause sequestration with loss of the permanent tooth germ ; (iii) a family history of supernumary teeth (Hitchin, 1959,[263] in twin studies, noted the genetic factor in supernumary tooth formation).

3. Palpation.- A labial swelling may reveal the crown of the central incisor ; a smaller, hard swelling, the unusual labially placed supernumary ; a hard, nodular swelling, an odontoma ; a broad bony expansion of the labial or palatal surface of the alveolar process, the dentigerous cyst.

I. Radiographic Examination.

The periapical film of the region will define the crown of the embedded incisor, the superimposed or adjacent shadows of the supernumary tooth or teeth, an odontoma, or a cyst. The alveolar cleft repair, which should be obvious in a clinical examination, may be mistaken otherwise for a cyst.

Periapical films of adjacent teeth should be taken to define the relation of the embedded tooth, or teeth, to their apices.

The vertex film and lateral head film will determine the axis of the incisor and supernumary, their relationship to the floor of the nasal cavity, and the amount of bone coverage.

J. Treatment Considerations.

The unerupted central incisor is of prime importance to the orthodontist, who should be consulted. The obstacle preventing eruption must be noted, the axis of the incisor, and the surgical accessibility assessed.

In most cases, removal of the obstacle (odontoma, supernumary, fibrous scar tissue, or dentigerous cyst) can be accomplished, the incisor surgically exposed, and its eruption brought about, with or without orthodontic traction.

Removal of the unerupted central incisor is indicated when its eruption is not desired, as in the following cases : the otherwise edentulous patient; when space does not exist and cannot be provided ; when the central is geminated with the lateral incisor, or is in the condition of dilated odontoma ; when its axial relationship makes eruption impracticable or impossible (e.g., the rare inverted case) ;

when its root is dilacerated sharply, and makes eruption impossible.

The unerupted incisor may be retained in the absence of pathology (such as a cyst, neurological symptoms), when its surgical removal is contra-indicated because of ill-health, likelihood of damage to adjacent teeth, or the refusal of the patient to submit to surgery. The possibility of cyst development (which may destroy the hard palate, obstruct and infect the nasal cavity, in the brief period of a few years) should be recognised, and the patient should receive a periodic radiographic examination.

THE FIRST PERMANENT MOLARS.

A. Congenital Absence.

Exceedingly rare, and one case only has been reported in which all permanent first molars were congenitally absent.

B. Anatomy.

The first molars show little variation in anatomy. The mandibular first molar may show three roots, the distal root being occasionally bifurcated. When three roots are present, resistance to luxation is greatly increased, and root fracture likely.

C. Chronology.

1. Hard tissue formation commences at birth.
2. The enamel is completed by $2\frac{1}{2}$ - 3 years.
3. Eruption occurs at 6-7 years.
4. Root formation is completed by 9-10 years.

(Logan and Kronfeld[62])

D. Frequency of Impaction.

Blum (1923)[135] found no case of maxillary first molar impaction among 457 impacted teeth, and 2 cases only of mandibular first molar impaction. Dixon (1959)[264] reported 9 cases, 2 in the maxilla,

7 in the mandible. However, it must be mentioned that impaction of the first permanent molars is far more common in mixed dentitions, the maxillary molar being affected twenty-five times as frequently as the mandibular first molar : it is likely that impaction of the mandibular first permanent molar in the mixed dentition will persist as impaction in the permanent dentition, whereas most maxillary impactions of the first permanent molar in the mixed dentitions are self-correcting. (Dixon, 1959).

E. Aetiology of First Molar Impaction.

1. Impaction in the mixed dentition.- Impaction of the first permanent molar against the second deciduous molar appears to be not related to predominance of tooth substance over jaw accommodation, as Dixon in several of his cases noted spacing of the deciduous teeth co-occurring. He concluded that impaction reflected disharmony between dental development and jaw growth; the first molar appears to erupt prematurely, before alveolar growth provides adequate space. The disimpaction of the maxillary first molar occurs more readily than of its antagonist, as the maxillary molar is free to move buccally, so releasing its mesial marginal ridge from the convexity of the deciduous molar with subsequent jaw growth. In the mandible, impaction generally persists, as the ascending ramus prevents buccal eruption.

2. Impaction in the permanent dentition.- Loss of the deciduous second molar surprisingly does not permit the eruption of the first permanent molar in the mandible, due to the very rapid eruption of the premolars. The mandibular first molar appears to lose eruptive force, and even orthodontic traction is usually not successful. Although Dixon considers that ankylosis is not the aetiology, it is the generally accepted explanation - the radiographs which he published, seem to confirm ankylosis as the likely aetiology, on the following grounds :-

(i) Spacing occurs between the first molar and the premolars, which drift distally, with diastema formation. It may be implied that mesial migration of the alveolar process and the teeth anterior to the

first molar has proceeded, whilst the latter remains stationary, due to ankylosis.

(ii) Continuous eruption of the first molar does not occur, and therefore it drops below the occlusal plane. There is a deficient vertical growth of the alveolar process in relation to the first molar.

(iii) The second molar tilts forward over its occlusal surface, and the second premolar tilts over its mesial aspect, which gives the erroneous impression that the first molar is being "submerged" by the teeth overlying its crown. A radiograph was published in recent years which clearly showed a small occlusal amalgam restoration in a first molar , the crown of which was covered with gingival tissue.

F. Clinical Features.

1. Impaction of the second premolar does not occur : indeed there is often a diastema between the first molar and the second premolar.

2. Impaction of the second molar against the first molar does occur, and Dixon (1959) published such a case. The possible explanation is failure of mesial migration of the mandibular first molar, on loss of the second deciduous molar, and its replacement by the narrower successor.

3. Submergence (sic) of the mandibular first molar beneath the occlusal plane permits food stagnation and caries; as the occlusal surface becomes covered with gingival tissue, pericoronitis may occur, together with a smouldering Vincent's infection.

4. The mandible becomes weakened at the first molar site, due to arrested vertical growth, and to the deep placement of the tooth. An intimate relationship to the inferior dental canal may be noted.

G. Treatment Considerations.

Should impaction of the maxillary first permanent molar be noticed in the mixed dentition, the distal bulge of the second deciduous molar should be removed by a diamond disc - this will permit normal eruption to occur.

On the first indication of mandibular first permanent molar impaction in the mixed dentition, the second deciduous molar should be extracted forthwith, to permit the normal eruption of the former. A space maintainer should be inserted to avert the impaction of the second premolar.

An impacted first molar of the permanent dentition should be removed as early as possible, to prevent second molar impaction, and to evade the surgical complications invited by delay. The surgical complications arise from ankylosis (necessitating wide bone removal), an intimate relationship to the inferior dental canal (especially on completion of root formation), and the possibility of mandibular fracture - traumatic or surgical.

THE PERMANENT SECOND MOLARS

Impaction of these teeth is uncommon. Stafne and Gardner (1929)[265] found only ten cases among 1,885 cases of impacted teeth. Blum (1923)[135] in his series of 457 impacted teeth, found one case of second molar impaction, of the mandible only. Nodine (1945)[14] could find only 40 cases of second molar impaction, 27 in the mandible, 13 in the maxilla, in his review of dental literature: 17 of these cases were in patients with endocrine dysfunction, and multiple impactions were seen.

A. The Mandibular Second Molar.

Nodine reported 23 cases of single impaction, one case of bilateral impaction, and one case of bilateral impaction of the second and third molars. Of these 27 cases, 10 were associated with third molar impaction, and one with first molar impaction.

1. Aetiology.- Systemic factors seem important in the aetiology : the second molar is involved in impaction with numerous other teeth (as in cleidocranial dysostosis, osteopetrosis, and endocrine dysfunction). The imprisonment of the second molar may be caused by impaction against the developing third molar, if this tooth lies over and above

its distal aspect. First molar ankylosis occasionally causes impaction of the second molar. Other cases may be attributed to traumatic injury (fracture of the mandible) in childhood, or to infections of the mandible. Occasionally a cyst is associated, and this is probably secondary to impaction, or a neoplasm, which causes the displacement and the impaction of the developing tooth (e.g., ossifying fibroma).

2. Position.- In the cases Nodine collected, the following positions were given :-

Horizontal : ten cases, of which seven were in "head-on" collision with the unerupted third molar, two were horizontally placed with the crown facing mesially.

Oblique : three cases of which two were in distoangular position, one in mesioangular impaction against a retained mandibular first molar root.

Vertical : five cases.

3. Symptoms.- These vary with the position, and conform to the usual rules : frequently, the second molar lies in contact with the inferior dental canal, and occasionally, the root is grooved, or canalised, and contains the mandibular bundle, therefore neurological symptoms may be expected. Denture irritation is often the first symptom, and this is usually a late manifestation. When the tooth is partly erupted, infection occurs. Cystic degeneration was reported in one case of Nodine's collection.

4. Treatment Considerations.- As Sealey (1952)[155] noted, when the second molar is impacted against the third molar, with loss of the first molar, a rotation ensues, to produce the "head-on" collision. Surgical removal is then difficult, due to the amount of dead space following operation, and to the probability of fracture when removal is undertaken in the elderly. Inferior dental nerve involvement is likely. There is every reason to remove the impacted second molar as soon after diagnosis as possible, preferably during the teens in order that the third molar may erupt into functional occlusion.

B. The Maxillary Second Molar.

Impaction of this tooth is less common than of the mandibular second molar. Aetiological factors in its impaction are similar to those of impaction of its antagonist.

The position of the impacted maxillary second molar as indicated by analysis of Nodine's collection of case reports, may be

Horizontal : two cases reported.
Inverted : two cases, one erupted into the antrum.
Vertical : one case.
Oblique : one case, in which the crown lay between the hard palate, and the mucous membrane.
Aberrant : one case was found in the zygomatic fossa.

The pathology, signs and symptoms of the impacted second molar do not differ greatly from those of the impacted maxillary third molar.

RETAINED TEETH in the EDENTULOUS JAWS.

The following radiographic surveys of edentulous jaws have been published :-

1. Logan (1921)[266] examined 35 edentulous jaws and discovered 2 embedded teeth.

2. Easterman (1921)[267] examined 89 edentulous jaws, and found 13 embedded teeth.

3. Cook (1927)[268] examined 120 edentulous mouths with 5 unerupted teeth ; 305 edentulous maxillae with 6 unerupted teeth ; 75 edentulous mandibles, with 2 unerupted teeth.

4. Cheope (1936)[269] examined 190 edentulous patients, with 9 unerupted teeth.

5. Wagner and Austin (1941)[270] examined 568 edentulous patients with 43 unerupted teeth, of which the maxillary third molar was the most

common finding. 30 were maxillary teeth, 14 were mandibular. The total included 8 supernumary teeth.

6. Smith (1946)[271] examined 1,000 edentulous patients, and discovered 38 unerupted teeth. He noted that 1 in 4 patients had residual roots, unerupted teeth, cysts, residual infection, and foreign bodies.

7. Ennis (1949)[272] studied 1,952 edentulous jaws, and found that 27 patients showed 32 embedded teeth, 25 being maxillary, 7 being mandibular. He also found that the maxillary third molar was the most frequently retained tooth.

Discussion.- These studies emphasise that radiographic examination of the edentulous jaws is essential before the provision of dentures is commenced : this is a practice which has been customary without exception, at the United Dental Hospital, Sydney. It is obvious from the above studies, that not all unerupted teeth give rise to pathology and symptoms. This is no reason, however, to tolerate the presence of unerupted teeth in younger individuals, unless there are good reasons for avoiding surgery.

- PART IV -

PRE-OPERATIVE AND POST-OPERATIVE CONSIDERATIONS.

Pre-operative Considerations.

Before the oral surgeon embarks on the removal of unerupted and impacted teeth, he must ask himself two vital questions:

(i) "If I were the patient, would I subject myself to this treatment ?" (Cash, 1951[273]).

(ii) "Is this extraction or surgical procedure definitely contra-indicated for the patient at this time, by systemic or local conditions ?" (Archer[229])

If the answer to the former question is in the negative, how can he recommend the treatment to one of his patients? Amies (1946)[274] considered that "every unerupted tooth (whether partially or completely unerupted) is a liability to the patient, and sooner or later, will become infected." Amies therefore favoured the rule that all impacted teeth should be removed. However, while I agree that most impacted and unerupted teeth do eventually give rise to serious sequellae, such considerations as age, general mental and physical health, and the position of the unerupted tooth often suggests that the wiser course is to leave the tooth in situ ; to advise an elderly patient to undergo the surgical removal of an ankylosed unerupted lower third molar, is, in my opinion, unjustified in most cases. Should the removal of an unerupted tooth be deemed inadvisable, it is the surgeon's responsibility to his patient, to stress the necessity of periodic clinical and radiographic examination.

A. Systemic contra-indications to the surgical removal of unerupted teeth.

A medical examination of the patient is obligatory. Laboratory aids and consultation with the patient's physician are often

necessary. The following systemic conditions are contra-indications, before consultation with the patient's physician :-

1. Cardiac disease.
2. Rheumatic disease.
3. Diabetes.
4. Toxic Goitre.
5. Pregnancy.
6. Jaundice
7. Liver damage.
8. Nephritis.
9. Haemorrhagic diatheses.

1. Cardiac Disease.

Further investigations are indicated by the following symptoms : breathlessness; chronic fatigue; palpitation of recent origin; sleep which is disturbed unless the head is elevated; and, a tendency towards vertigo. The physical signs of cardiac disease are : cyanosis of lips, tongue, and fingernails; dyspnoea on mild exertion; inability to hold the breath more than 15 seconds; engorged cervical veins; tachycardia; elevated blood pressure; and oedema of the ankles. Drugs which may induce ill-effects are: carbon dioxide, atropine, and adrenaline ; xylocaine without adrenaline is the local anaesthetic of choice. Premedication with a sedative such as phenobarbitone on the night before operation, and seconal half an hour before operation is recommended. This will minimise apprehension and the likelihood of struggling.

2. Rheumatic Disease.

The patient must be asked directly, whether he has suffered from rheumatic fever : a positive answer indicates immediate consultation with his physician. Removal of teeth results in a transient bacteraemia, the predominant organism being Str. viridans (O'Kell and Elliott, 1935;[275] Coffin, 1957[276] and others). Such a bacteraemia may precipitate a sub-acute bacterial endocarditis. Burket (1957)[277]

considered that local is preferable to general anaesthesia. If valvular lesions are known to exist, a prophylactic course of penicillin (or aureomycin, if allergy to the former is known) is essential: Burket recommended that 600,000 Oxford units/day of procaine penicillin be administered to attain a therapeutic blood level at the time of extraction, and for 24-36 hours thereafter.

3. Diabetes Mellitus.

The symptoms suggesting diabetes mellitus are : general weakness and loss of weight; excessive hunger and thirst, and polyuria; generalised pruritis, drowsiness after a heavy meal; cramps or pains in the extremities; and a history of pyogenic infections - boils, styes, carbuncles, paronychiae, alveolar abscesses and a rapid progression of periodontal disease. It is a simple procedure to test the urine for sugar and acetone bodies, and may be carried out in the dental surgery. The suspected diabetic should be referred to his physician, and oral surgery carried out only with the co-operation of the latter. The anaesthetic of choice is 2% xylocaine without adrenaline: adrenaline increases the blood sugar level, and by causing local ischaemia, predisposes to sloughing (Burket). If a general anaesthetic is indicated, the patient should be admitted 24 hours before operation, and carefully balanced (Crombie, 1952[278]). Operative trauma must be reduced to a minimum, and the administration of vitamins B complex and C and antibiotics is recommended (Burket).

4. Toxic Goitre.

The symptoms are : nervousness, tremors, and emotional instability; tachycardia and palpitations; excessive perspiration; a diffuse enlargement of the thyroid gland (occasionally absent); generally exophthalmos; loss of weight; excessive appetite; early fatigue, and muscular weakness; and occasionally, pressure symptoms- hoarseness, dysphagia, and dyspnoea. Archer[229] warned that a thyroid crisis may be precipitated by oral surgery, leading to cardiac embarrassment, and definite heart failure; oral surgery may be undertaken only

after treatment of the condition. Burket considered that general anaesthesia is preferable to local anaesthesia, as this lessens the psychic trauma. Procaine with adrenaline is definitely contra-indicated, and the local anaesthetic of choice is xylocaine without adrenaline.

5. Pregnancy.

Archer [229] treated thoroughly the advisability of performing surgery during pregnancy ; he presented the following suggestions : (i) emergency oral surgical procedures may be carried out at any time; (ii) necessary, but non-emergency work should be carried out in the middle trimester; (iii) elective work is best delayed until after pregnancy. In the first trimester, nausea and vomiting may be present, and 90% of abortions ~~may~~ occur at this stage : as in the lay opinion, extractions are the "cause" of many abortions, it is better to postpone until the middle trimester, all but emergency work. It is the opinion of obstetricians and pathologists that the abortions following extractions would have occurred regardless of the extraction. The third trimester is unsuited to difficult oral surgical procedures, because the patient is uncomfortable and cannot sit for a protracted period. Local anaesthesia is the method of choice.[229,277] The foetal brain is more susceptible to narcotics and barbiturates, and readily suffers hypoxia : the general anaesthetic of choice is 30% cyclopropane, 70% oxygen mixture. Only with the obstetrician's consent, may N_2O/O_2 anaesthesia be used.

6. Jaundice.

Jaundice is not a disease, but a symptoms of obstructive jaundice, haemolytic jaundice and infective hepatitis. The symptoms include : yellowish skin, and sclera; yellow-green mucous membranes (well illustrated at the junction of hard and soft palates); putty-coloured stools; and a dark urine. Of significance to the oral surgeon, is the danger of prolonged haemorrhage, due to prothrombin deficiency. Surgery should be postponed until the condition has been treated, unless emergenct surgery is required. Thoma stated that the

patient should receive vitamin K, 4 mgs. t.d.s., bile salts 0.3 Gm t.d.s. to assure the absorption of this fat-soluble vitamin; a transfusion of 250 ml. of blood containing prothrombin, and 0.5ml. of synthetic water-soluble vitamin K intravenously or intramuscularly b.d. to bring the prothrombin time to normal.[133]

7. Liver Damage.

Two aspects affect the oral surgeon : there is often a haemorrhagic tendency, due to failure of synthesis of prothrombin in the liver ; and barbiturates, morphine, vinyl ether, and ether, all being detoxified by the liver, are contra-indicated. Liver damage may be caused by carcinoma, and cirrhosis. Such patients must be referred to their physician, and preferably treated in a hospital when oral surgery is indicated, in order that the diet may be controlled, fresh blood transfusions and vitamin K therapy be administrated.

8. Nephritis.

The symptoms include dysuria, haematuria, albuminuria, lumbar pain, xerostomia and burining of the mouth, a generalised stomatitis in uraemia, and a urinous odour of the patient's breath. The effects of oral surgery may be the precipitation of an acute nephritis.

9. The Haemorrhagic Diatheses.

As these conditions are of extreme importance to the oral surgeon, they will be covered in detail in a subsequent section.

B. Local contra-indications to the surgical removal of unerupted and impacted teeth.

The following local conditions oftem render surgical procedures of the jaws hazardous :-

1. Irradiation of the jaws.
2. Acute Vincent's infection.
3. Streptococcal infection.
4. Acute maxillary sinusitis.
5. Acute pericoronal infection.
6. Osteosclerosis.

1. Irradiation of the Jaws.

The removal of teeth from the jaws following irradiation in the treatment of carcinoma, may lead to radionecrosis, to which the mandible is especially susceptible.

Many feel that it is best to remove all teeth prior to radiotherapy, in order to avert this disaster. Thoma[133] warned that "if possible, the patient should be hospitalised and given penicillin therapy, and all teeth should be removed at one time so that there will be no delay in the treatment of malignancy." He stated also that "the treatment of carcinoma should be delayed for a week to ten days."

Sleeper (1950)[279] concluded that if the teeth and their supporting tissues are healthy, only those teeth in the line of radiation need be removed, but that a full mouth clearance ought to be effected when the mouth shows neglect throughout.

The authorities of the American Oncologic Hospital recommended, as a result of their vast experience that : "All patients who are to undergo intensive irradiation through the mandible and/or maxilla, and have the prospect of a long term survival, when large

portals are used (underlined words in italics), as in most general hospitals, should have all their teeth removed prior to irradiation." They recommended also that "When teeth are extracted in preparation for irradiation, the sockets should be protected from irradiation until healing of the gum lacerations is complete. There is no set time - at least 10-14 days."

A distinct reversal of previous concepts of the proper management of teeth in the path of radiation was published by Wildermuth and Cantril (reported by Archer, 1956[229]) who maintained that they found radionecrosis of the mandible only in the group managed by extraction of the teeth before roentgen therapy ; failure of resorption of the bony spicules of the alveoli caused general mucosal ulceration with subsequent exposure of the mandible to infection. I have been unable to trace the reference, and therefore am in no position to criticise the validilty of their findings. I feel that the views of Wildermuth and Cantril may make an oblique contribution to the management of patients in need of radiotherapy : it is my belief that when a long term survival is expected, a full mouth clearance of teeth should be carried out, with an alveolectomy, in order that all bony spicules be removed, and a regular denture base be gained, prior to the institution of radiotherapy. It is particularly important that the ridges of the jaws are smooth, and free of bony undercuts : irradiated soft tissue is left with a lowered resistance to trauma and infection, and a denture ulcer is sufficient to launch a radionecrosis of the mandible in particular.

A partly erupted tooth, and the superficially embedded tooth should be removed prior to radiotherapy of the oral region. However, I consider that the removal of the very deeply embedded tooth is not justified, unless it is thought that bone resorption is sufficient to expose the tooth at a later period.

2. Vincent's Infection.

Oral surgical procedures must not be performed in the presence of the acute bacterial phase of the disease. Amies (1946)

stated that : "Some of the worst pain which can be experienced, follows a dry socket associated with Vincent's infection after the removal of a lower third molar ; ordinary doses of morphia fail to relieve the pain." The acute infection should be eradicated by vigorous local and systemic therapy, before an unerupted or impacted tooth is considered ready for removal.

3. Streptococcal Infection.

This is usually seen in an acute form, in the mucous membrane of the oro-pharynx (e.g., acute tonsillitis).

4. Acute Maxillary Sinusitis.

The removal of maxillary premolars and molars is contra-indicated during the acute phase, due to the inevitability of infection of the field of operation, and the likelihood of antro-oral fistula.

5. Acute Pericoronal Infection.

A review of the literature pertaining to acute pericoronitis of the lower third molar region, reveals a divergence of opinion in the treatment of this condition. The following attitudes have been taken : (i) the pericoronitis should be allowed to subside (Frew,1937[280]) ; (ii) the infection should be treated, by removal of the maxillary third molar, if it traumatises the gum flap, or by other methods, and then the lower third molar may be extracted if necessary (Parfitt,1938;[281] Kent,1942;[282] Miller,1944;[283] Cook,1944[284]); (iii) the third molar may be removed during the acute phase of pericoronitis, regardless of the presence or absence of Vincent's infection, (Krogh, 1951[285]).

It is malpractice to "wait until the swelling subsides" before extracting the mandibular third molar, if no measures are taken to bring the infection under control ; infection from a pericoronotis, if ignored, will extend to the lateral pharyngeal, the pterygomandibular, the submasseteric and the buccal spaces, often with fatal

sequellae. To remove the impacted third molar in the presence of acute pericoronitis, exposes sterile bone to infection, and this is the objection generally raised against the removal of the tooth at that stage. It is the opinion of the writer that when acute pericoronitis is localised to the gingival tissues only, it should be treated as a gingival infection - drainage should be established by intra-oral hot packs, gentle irrigation under the gum flap, and incision - and the tooth removed if necessary, after the acute phase has been controlled; should there be considerable bone destruction to the distal og the crown, an acute infection cannot necessarily be controlled by the above methods, and then the removal of the tooth, under an antibiotic cover, is justified, if it is the only method of drainage available, and providing the removal is simple; should the infection have spread to the soft tissues, causing trismus, external swelling, dysphagia, the infection must be localised by intra-oral and extra-oral heat, and drainage established and maintained by Hilton's method, before the removal of the tooth is justified. Whenever antibiotics are to be used, it must be established that the causative organisms are sensitive to the drug chosen, and that the patient is not allergic to that drug.

6. Osteosclerosis.

Various conditions lead to osteosclerosis, among which are : Marble bone disease, Paget's disease, and low grade chronic infection of bone. Surgical procedures in relation to areas of osteosclerosis usually lead to sequestration, due to the defective blood supply of such bone.

Discussion.- Each unerupted or impacted tooth must be regarded individually ; each patient must be regarded individually. The surgeon must decide what is the best treatment for the patient, under the given conditions. Unerupted teeth may be divided into the completely bone-embedded type, which shows no local pathology, the embedded tooth lying immediately beneath an apparently intact gingiva, and the partly erupted tooth, which may, or may not be, impacted against an erupted tooth. The completely embedded tooth may be retained, in the absence of

pathology, especially when systemic conditions contra-indicate surgery. The partly erupted tooth is a hazard, and eventually may lead to emergency surgery if ignored. The gum-covered unerupted tooth often becomes infected, and this is especially likely in the mandibular third molar, but unusual in the maxillary third molar. Patients with the systemic conditions contra-indicating surgery, previously discussed, often do not tolerate emergency surgery well, due to lack of time for medical treatment to control their condition ; however, once medical treatment has been arranged, they tolerate elective surgery satisfactorily. It should be the principle of the oral surgeon to decide which impacted and unerupted teeth are likely to precipitate emergency surgery if retained, and in young patients, such teeth should be electively removed ; in elderly patients, a less radical approach may be adopted.

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Post-operative Considerations.

That post-operative considerations in this critical review of literature precede the description of technique for oral surgical procedures is intentional. Surgery must not be undertaken without prior comprehension of the nature of the injury to be sustained by the tissues, and of the complications that may possibly arise. Only by such forethought may an elastic surgical technique be evolved - a technique which may be modified to meet all emergencies. Haddon Kemp (1951)[286] drew attention to this generally overlooked fundamental principle in his observation that "descriptions of technique for oral surgical procedures are comparatively plentiful when considered in relation to post-operative treatment for patients having oral surgical operations performed in the mouth...." and advised that " there must of necessity be a balance between surgical technique, and post-operative treatment, and the former should be performed only with a complete realisation of the responsibilities of the latter."

Post-operative complications will be considered in accordance with the following plan :-

A. Inflammation and repair.

B. The avoidance of wound infection.

C. Infections complicating unerupted teeth and their removal.

1. pyogenic,
2. actinomycotic,

D. The control of infection.

E. Haemorrhage

1. haemostasis,
2. bleeding diatheses,
3. control of haemorrhage.

A. <u>Inflammation and Repair</u>.

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Hunter (1812) was the first to recognise that inflammation is a tissue response to cellular injury ; he considered it as a "salutary operation". However, although the effects of inflammation are generally beneficial, measures today are being investigated to minimise and control the inflammatory response to oral surgical procedures. If the inflammation is unduly prolonged, the separate biological process of repair is retarded as a result of circulatory stasis and consequent tissue anoxaemia. Anti-inflammatory therapy includes the use of anti-histamines, hydrocortisone, streptokinase, hyaluronidase and trypsin, which will be discussed fully at a later stage. It must be recognised that the purpose of inflammation in surgical wounds is to oppose bacterial invasion, and to absorb or exfoliate debris such as non-vital tissue and foreign bodies - which would strongly suggest that anti-inflammatory agents should be used only when aseptic procedure ensures against bacterial contamination, and surgical technique is sufficiently painstaking and gentle, to ensure the minimum tissue trauma. The question posed by this new concept is : can the surgeon be justifiably confident that bacterial contamination

may be precluded, and tissue resistance to infection maintained by gentle tissue handling and debridement? It is certain that any deliberate inhibition of inflammatory response should only be entertained after full realisation of the attendant dangers involved.

The repair of oral surgical wounds -such as those occasioned by the removal of unerupted teeth - will be considered under the following :-

1. The healing of an incised wound.
2. The healing of a bone cavity.
3. The mucoperiosteal flap reattachment.

1. Healing of the Incised Wound. Such a wound, caused by the blade of a scalpel, is essentially a wound without loss of substance. Into the slit-like gap made by the knife, blood plasma and whole blood are poured. On coagulation, the lips of the wound are sealed by fibrin, into which grow new capillaries and fibroblasts. The temporary clot is replaced by granulation tissue, and epithelium covers the wound. The granulation tissue is finally converted to dense avascular scar tissue. It is not possible to give an accurate "time-table" of these events. Boyd[288], however, quoted the following estimates:-

End of 12 hours	: Vascular and connective tissue reaction begins;
End of 2nd day	: Gramulation tissue begins.
End of 4th day	: Temporary clot has been replaced by gramulation tissue.
End of 5th day	: Epithelium covers narrow wound. Definite fibrils appear.
End of 3 weeks	: Dense non-vascular scar tissue is formed.

Should infection supervene, the clot is destroyed, and healing by second intention follows : healing is thereby delayed, and scarring is increased. The exclusion of infection from a wound, especially of the skin, is of paramount importance. Moynihan[289] defined healing by first intension thus :-

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"The wound which heals by first intention is one which heals within a few days, leaving a thin straight narrow line of palest pink. Around the line and the stitch-marks, everything appears cold. There is no redness, no swelling, no stiffness or induration, and at the line itself the most accurate apposition of the skin edges is seen. There is no discharge from the wound. There has been neither local nor constitutional reaction following the operation."

Anything that falls short of such healing, even in the absence of active suppuration, must be looked upon as a type of infection. Moynihan's ideal, though rarely if ever seen, is a useful yard-stick for the assessment of surgical technique.

2. Healing of the Bone Cavity. Such a wound is one with loss of substance. The following stages in healing are noted :

(i) The defect fills with a blood clot.

(ii) The blood clot is organised : fibrin and blood pigment are removed by phagocytes, and replaced by proliferating young vascular connective tissue.

(iii) Gradual replacement of the young connective tissue by coarse fibrillar bone.

(iv) Reconstruction of the alveolar process by resorptive activity on the one side, and the replacement of immature bone by mature bone on the other.

(v) Epithelialisation on the wound surface occurs simultaneously with the other processes.

Simpson (1958)[290] found that the healing of extraction sockets in rhesus monkeys, occurred at the following rate, which is three times as rapid as in man : after one week, bone trabeculae are present at the base of the socket ; by four weeks, the socket is filled with immature bone ; the defect is obliterated by eight weeks. The rate of epithelialisation he found to be hastened by the falling in of the gingival margins over the defect. When pieces of necrotic bone lie within the socket, they are either resorbed, if sterile, by osteoclasts,

or surrounded by epithelial downgrowths and exfoliated. Should infection of the clot occur, it is destroyed and healing by primary intention is not possible : the socket is lined with granulation tissue, and this gradually fills the socket, finally being replaced by bone. This type of healing is seen in the so-called "dry socket", and the infected bone of the socket must be eliminated by undermining resorption, before healing may commence. When a bone cavity is packed with either vaseline gauze or periodontal pack (as recommended in some instance by Linghorne and Thompson, 1958[291]), healing by second intention is deliberately induced.

3. <u>The Mucoperiosteal Flap Reattachment</u>. This aspect of healing has been thoroughly investigated by Simpson (1959)[293] and Dedolph and Clark (1958).[292] That reattachment to the bone occurs, without difficulty, has long been recognised. The reattachment of the gingival tissues to the teeth has been the subject of controversy : periodontists and surgeons have been unable to agree as to whether the epithelial attachment to teeth is restored to its pre-operative height or not. Dedolph and Clark published the following evidence :

" Microscopic study of 13 specimens of teeth and surrounding tissues revealed that mucoperiosteal flaps which are raised from about the teeth, will return to their pre-operative health level in approximately three weeks. There are, therefore, no contra-indications to outlining and reflecting mucoperiosteal flaps of sufficient size to provide optimum visibility and access for a given surgical procedure."

Their histological serial studies revealed that : (i) at 48 hours, the gingival epithelium is separated from tooth and bone by acute inflammatory exudate and haemorrhage ; (ii) at one week, the acute inflammatory phase has given way to a scene of generalised repair. "Epithelium had proliferated, covering rather immature granulation tissue beneath, and had extended towards the tooth surface. Unmistakable evidence of regeneration and lining-up of the gingival periodontal membrane fibres were apparent." ; (iii) at 3 weeks, epithelial reattachment is complete, and the attachment of periodontal fibres and other connective tissue

elements is restored. Simpson (1959) found that reattachment of the flap to bone occurs in two phases : during the first week, capillaries and fibroblasts grow out of foramina,and,through the agency of the organising exudate between the flap and bone, anchor the flap in position; during the second week, the new connective tissue between the flap and bone becomes attached to new bone forming on the resorbed areas of the alveolar process. Should bone fragments be caught between the flap and unoperated bone, they are almost invariably retained, and histological studies showed them to be functioning as centres of ossification. When dislodged, and found in the bone cavity, however, they become infected and must be exfoliated,

B. The Avoidance of Wound Infection.

It has been shown that healing by first intention is rapid, and results in the minimum deformity, and incurs a minimum of post-operative inconvenience. The oral surgeon must aim for healing by first intention, and therefore he requires a thorough knowledge of the factors opposing healing by clot organisation and of the methods to avoid them.

The measures employed to prevent wound contamination are :-

1. Prophylactic exclusion of bacteria from the wound.
2. Maintenance of normal tissue resistance to bacterial contamination.

1. Prophylactic exclusion of bacteria from the wound. The surgical wound may be contaminated by bacteria from

(a) the oral flora,

(b) adjacent sites of infection, and

(c) cross-infection.

(a) The oral flora. All intra-oral surgical procedures are carried out in a field laden with bacteria of the oral flora. While it is poss-

ible to reduce the number of micro-organisms in the mouth by pre-operative precautions, sterility of the oral cavity is impoosible to attain. To reduce the number of organisms temporarily, a prophylactic scaling, pre-operative removal of debris, and vigorous mouthrinsing just prior to the operation, will reduce the absolute numbers of bacteria, without altering their relationship to each other. The oral flora "live in a happy state of symbiosis, both in relation to one another and to the tissues of the mouth" as Wallace (1951)[294] pointed out, and it is most important not to upset this symbiosis. Should penicillin lozenges, for example, be given in order to reduce the oral flora, penicillin-sensitive organisms will be reduced, whereas the penicillin-resistant organisms flourish, to the extent that they may precipitate super-infection, which may bring about catastrophic results in wound healing. Measures to reduce the degree of salivation during the operation will help in excluding oral bacteria from the wound. Preparation of the field of operation is important, and this is carried out by drying the mucous membrane with sterile gauze and applying an antiseptic solution : it is recognised that sterilisation of the membrane is not gained, but fixation of the bacteria does aid in reducing their implantation into the tissues. Wallace (1951) recommended : "Tincture of iodine in 2½% solution in 70% alcohol is the general choice, and it is claimed that the organisms not actually destroyed are fixed to the epithelial surface. Tincture of metaphen in another commonly used agent,and while slower in action than tincture of iodine, it has the advantage of not producing sensitisation.....Alcohol in 70% dilution is also serviceable..."

(b) Contamination from adjacent sites of infection. The danger of blood clot infection incurred in the removal of the partly erupted lower third molar during the acute phase of pericoronitis, has already been covered. Vincent's infection, especially when acute, should receive vigorous local and systemic treatment pre-operatively. Surgery should not be undertaken electively, in the presence of stomatitis, tonsillitis, and adjacent periapical infection.

(c) Cross-infection. Wallace[294] felt that "the real danger of wound infection does not lie so much in possible contamination from the usual inhabitants of the mouth as in the introduction of extraneous organisms from without by the surgeon in the course of the operation."

It is only by the most careful adherence to aseptic theatre technique that cross-infection can be avoided. The technique comprises :-

(i) The use of an operating theatre. Considerable attention in recent literature has been paid to the design of the theatre : the main factors of significance in the prevention of cross-infection are correct ventilation, easily washable floor coverings, soiled-linen and refuse shutes, scrub-rooms outside the theatre. "Clean" surgical cases should be treated prior to "septic" ones.

(ii) All instruments, swab sticks, gauze swabs, cotton wool, and suture material etc. must be sterilised and maintained in a sterile condition prior to use. Corbett (1951)[294] treated very thoroughly, the correct methods of sterilisation, and the ideals in his publication should be observed. There is only one satisfactory method of sterilisation - by autoclaving, which involves the use of saturated steam. Reddish(1954)[295] stated that: "There is little justification for maintaining steam temperatures higher than 121-123'C, equivilent to 15-17 lbs pressure . Exposures to temperatures beyond this point hastens destruction of fabric and rubber goods." He further claimed that: "The oft-quoted statement that 'no living thing can survive ten minutes direct exposure to saturated steam at 121'C ' would appear a reasonably accurate standard.... Although some thermophilic spores have survived longer, none of the pathogens have been shown to be resistant to an exposure of even 3 minutes." The exposures generally used are 10-30 minutes. The boiling of instruments for 20 minutes has been considered adequate by many authorities. Recently, some questions have been raised as to the capacity of boiling water to destroy the viruses of homologous serum hepatitis and infective hepatitis. Evidence indicates that these pathogens are unusually hardy and more resistant to destruction than most organisms. It has been established conclusively that the principal

mode of transmission of viral hepatitis - particularly the homologous serum strain of the disease - has been through the parenteral route by means of contaminated syringes, needles, lancets, surgical instruments and by blood and other products (Maurice[295a]). Foley and Gutheim,[296] Eisenbud[297] and Maurice[295a] recommended that instruments puncturing the skin or mucous membrane must be autoclaved, if transmission of viral hepatitis is to be avoided.

(iii) Full theatre attire (sterile gown, rubber gloves, impermeable face mask and cap) must be combined with the proper scrubbing-up ritual and applies to the surgeon and his assistants.

(iv) The draping of the patient, and preparation of the exposed skin of the face.

2. <u>The Maintenance of Normal Tissue Resistance to Infection</u>.

Fomon (1939)[298] stated that :

"Even after the most rigid aseptic care, a certain amount of bacterial contamination is inescapable. This minute contamination, however, does not interfere with wound healing, provided the bactericidal properties of the tissues are left unimpaired. When tissues are compromised by mechanical and chemical trauma, they are deprived of exerting their germicidal powers and the few organisms which they would under normal circumstances destroy, assume a greater importance, and set up infection."

If the details of an atraumatic technique are strictly observed, the tissues will be left in the best possible condition to cope with the unavoidable contamination. This implies protection of the raw tissues against undue exposure, avoidance of mechanical injury in the handling of tissues, notably rough manipulation, crushing with clamps, tight sutures and prolonged retraction, limitation of the amount of foreign material in the wound, and avoidance of chemical and thermal damage by strong antiseptics or too hot applications.

The steps in the surgical technique relevant to the removal of unerupted and impacted teeth, will be discussed from the viewpoint of the maintenance of hormal tissue resistance to infection.

Speed of Operation. The operation should be performed as expeditiously as is consistent with thoroughness and gentleness. The longer the operation, the greater the blood loss, and the longer the exposure of raw tissue surfaces to drying, which interferes with healing and predisposes to infection. This calls for adherence to a well thought out plan based on a thorough knowledge of surgical anatomy and surgical pathology, and by the execution of a step-by-step technique aimed at a pre-conceived goal. As Komon[298] mentioned, there is truth in the saying that "an endless amount of trauma can be accomplished if a surgeon formes the habit of mauling the tissues while he is trying to collect his sluggish thoughts." Timorous, indecisive surgery - the scratching of soft tissues with a scalpel, and the pecking at bone with a chisel - is traumatic surgery in the disguise of gentleness.

The Incision. Neat, decisive incisions with a sharp scalpel, held at the correct angle throughout, cause the least trauma. The blade must be perpendicular to the mucoperiosteum ; should it be at an angle, the mucous membrane is reduced to a feather edge, which, deprived of its underlying corium, and therefore its nutrition, necroses and sloughs. While the incision must be made to bone, so dividing the periosteum. the blade must not be firm against the bone, as its sharpness is reduced. A jagged incision, caused by the pinching of tissues through use of the point, rather than the belly of the blade, forms recesses for bacteria, and tags of non-vital tissue.

The Mucoperiosteal Flap. The specifications for flap design are :

(i) It must give a wide view of subperiosteal landmarks.

(ii) It must give good visibility and access to the field, without necessitating traumatic retraction.

(iii) It must have an adequate blood supply, and therefore its base must be broad and directed towards the nutrient artery.

(iv) It must rest on a broad shelf of undisturbed bone when replaced.

(v) It must be so designed, that extension is possible during the operation if found necessary.

A small, inadequate flap increases the trauma sustained by the tissues, due to poor visibility and accessibility ; it is likely to suffer tension and tearing at its margins by the retractor and to be injured by chisels, burs or elevators. As Dedolph and Clark demonstrated, there are no contra-indications to outlining flaps of sufficient size to give optimum visibility and access.

Elevation of the Flap. Much of the post-operative pain and swelling arises from this procedure, if poorly carried out. Before an attempt is made to raise the flap, the surgeon must be certain that the periosteum is incised to the bone. In areas free of muscle attachments, the mucoperiosteum separates cleanly and easily, and this may be effected with a periosteal elevator. However, in areas of muscle insertion, the periosteum is firmly adherent due to Sharpey's fibres, and a sharp chisel may prove more gentle than the blunter periosteal elevator. The periosteum possesses a rich nerve supply, and unnecessary trauma leads to severe post-operative pain.

Retraction of the Flap. Toothed retractors crush tissue, and thereby supply a non-vital tissue culture medium for pathogens. The flap if well designed, should require no tension at its base for adequate visibility. The retractor should rest firmly on bone, and not on soft tissue which would be bruised. Much tissue trauma can be avoided by careful attention to chair height, and patient posture ; the head should be so placed that the overhead light can be focused on the operative field ; skilful use should be made of the cheek, flap, and tongue retractors ; a good aspirator is invaluable in increasing the visibility ; restlessness on the part of the patient can be reduced to a minimum by premedication, adequate anaesthesia, and occasional reassurance by the surgeon.

Haemostasis. Blood is best aspirated. Persistent "bleeders" should be clamped and twisted with fine beaked artery forceps, and ligatured

only if necessary. A minimum amount of tissue beyond the ligature should be included. Broad-beaked artery forceps crush adjacent tissue and increase the inflammatory response. Ligatures should be sparingly used, as they incite a foreign body inflammatory reaction. The practice of vigorous rubbing and mopping with gauze is to be condemned - "gauze has a tooth that can bite and tear and scratch"- and in addition, tends to leave in the wound, threads which must be exfoliated or absorbed, and offer a nidus for bacteria. Should it be necessary to use gauze for the purpose of haemostasis, it should be moistened with normal saline, and held without rubbing, against the bleeding tissue. Absolute haemostasis is not necessary before replacing the flap, as a certain amount of haemorrhage is necessary for the gluing of the flap to bone. However, a haematoma must at all costs, be avoided, as it causes pressure (and thereby induces ischaemia), tension to the sutures, and is the ideal culture medium for pathogens.

Bone Removal. The purpose, extent and method of bone excision will be fully discussed in a later section. The effects of bone excision on the inflammatory response, and repair will be considered.

Ward (1956)[299] believed that the more radical the bone removal in the lower third molar surgery, the more likely is healing to be by first intention - his reason is that "dead space" must be reduced to a minimum, by saucerisation of the bone cavity, in order that the blood clot ~~should~~ may occupy as little volume as possible, thereby possessing enhanced powers of organisation. Others consider that bone removal should be as conservative as possible, and they attain this end by sectioning embedded teeth prior to their removal.

The principles to be observed in bone removal are : (i) avoidance of thermal necrosis (incurred when burs are injudiciously employed) ; (ii) avoidance of crushing by the use of blunt chisels ; (iii) adequate bone reduction to permit the luxation and withdrawal of the unerupted tooth without contusion of adjacent bone.

Chisels have the advantage of being heatless; however, the chisel cut leaves a ragged, crushed edge. Burs if not water-

cooled, rapidly overheat bone, which undergoes necrosis; they cause also, fragmentation of bone, and it is important that the fragments are removed by irrigation, if undue inflammatory response is to be avoided. Simpson (1958) on the topic of tissue response to bone removal, stated : "The part of the bone which has been cut, shows a few empty lacunae near the operated surface, but proceeds to repair without delay, so that the actual method of bone removal is not of direct importance." However, if wide areas of bone are devitalised, and infection supervenes, healing is protracted, and is not completed until the necrotic bone is separated by undermining osteoclastic activity and exfoliated as a sequestrum. Hall (1959)[300] reported promising results with the high-speed air turbine drill, using a tungston carbide bur under a distilled, sterile water coolant : bone, he reported, is "wiped away" at speeds of 200,000 - 350,000 r.p.m., and operating time and trauma is very considerably reduced.

The Use of the Elevator. No instrument used in the removal of unerupted teeth can cause more damage than the injudiciously used elevator. It cannot be overemphasised that forceful use of this instrument is to be condemned. No tooth should be submitted to elevation until a space has been created for displacement of the tooth, otherwise the bone of the fulcrum is crushed, and the tooth itself compresses bone of its crypt, so that wide zones of bone may be devitalised. If the tooth is not displaced by gentle elevation, it is best to desist, and to seek the cause (root resistance, inadequate bone removal, or tooth impaction).

Wound Debridement. Before tissue regeneration may proceed, foreign material must be absorbed, encapsulated, or expelled. Foreign material includes all non-vital tissue (bone, enamel, tags of fat, muscle, tooth follicle and gingival tissue) and extraneous matter (such as gauze threads and amalgam fragments). Gauze and non-vital tissue in particular, harbour infection, and isolate organisms from the action of phagocytes. The wound must be clearly visualised, and by gentle syringing, foreign material removed ; bone fragments isolated from their nutrient supply, bone inadvertantly crushed (such as that used as an elevator fulcrum) should be eliminated. The follicle should be curetted free of bone, and removed

for two reasons - its cystic potentiality, and its low viability. Fat, often noted to be abundant in the maxillary third molar field of operation, has a poor blood supply, and if it extrudes through the flap, it must be cleanly excised. Sharp bone spicules may ulcerate the flap, and are best removed by a bone file. A reasonable degree of haemostasis should be gained before the flap is repositioned. Should the flap be excessive, as a result of bone loss, it should be cleanly trimmed, and its margins should rest in good apposition without tension, on a shelf of vital bone.

Suturing. Sutures are required only to hold the tissues in correct relation until they are sealed in position by a coagulum. Therefore, in most cases, they are removed after 48 hours. Sutures should not be too tight, as this indusees ischaemia of the tissues, and sometimes necrosis. A rubber dam or rubber tube (suitably prepared), should be inserted if there is danger of persistant haemorrhage, otherwise a haematoma will form. Catgut is an absorbable suture material, and its main use lies in ligaturing of vessels. To increase the period of usefulness, gut may be chromatised, which delays its absorption. Its disadvantages include; difficulty of sterilisation ; contamination with chemicals used in its sterilisation ; allergic reactions are occasionally reported ; knots become untied due to its slippery nature ; its thickness demands the use of heavier, more traumatic needles. Silk, cotton, and nylon suture materials are useful, and have the advantage of being boilable. Nylon is indicated when the sutures are to remain for 5-8 days, and are especially indicated in cases of antral exposure. Stainless steel is a benign material, which may be used in the place og nylon, when the antrum is involved.

The Pressure Pack. The wound is immediately covered with a gauze pad, and the patient instructed to bite firmly on it. This permits the protection of the wound from contamination, enhances early haemostasis, and reduces the degree of oedema. A pressure pack of sterile gauze, in an autoclaved container of latex, has been used with success, placed in the buccal sulcus and combined with a barrel bandage for the first 24 hours, by

C. Infections Complicating Unerupted Teeth and their Removal.

1. Pyogenic Infections.

Fatalities occurring as a result of oral pathology and oral surgical procedures, are dominated by the extension of infection. Occasionally death may be due to haemorrhage in patients afflicted with haemophilia and other blood dyscrasias, occasionally to asphyxia. Anaesthetic deaths are not included, as the administration of a general anaesthetic should be regarded as the field of the skilled anaesthetist.

Haymaker (1945)[302] in an excellent report of autopsy cases, published comprehensive histories, the clinical course, and the post-mortem findings of 28 cases in which fatal intra-cranial or spinal lesions occurred after tooth extraction. To all who believe that the gravity of dental infections is over-emphasised in undergraduate teaching, Haymaker's paper will bestow perspective. His description of the course of events is worthy of mention, indeed of memorisation :

"Direct spread of infection from the site of extracted teeth to the intracranial cavity usually occurs as follows :- One or more teeth are extracted, regional suppurative cellulitis ensues, the pus burrows along fascial planes to the base of the skull, and en route, it may invade paranasal sinuses and an orbit ; sooner or later the cranial wall is penetrated either by means of veins leading to sinus thrombosis, or via the bone itself, causing subdural empyaema, leptomeningitis, or brain abscess, singly or combined. The extra-cranial collection of pus may be so deeply situated that it is either not recognised or not reached by surgical means."

An Analysis of Haymaker's Report.

(a) Multiplicity of extractions.- Not a necessary adjunct to fatal complications : a single tooth was extracted in 19 of 28 cases.

(b) The site of extraction.- Maxillary teeth were more frequently involved in spread by direct extension ; mandibular teeth in spread by

the general circulation (the arterial route).

(c) Bacteriology.- The streptococcus predominated in haematogenous spread ; the staphylococcus in spread by direct extension.

(d) Haematogenous spread.- Bacteraemia as the immediate sequella of extraction is relatively frequent. After routine extraction, the organism may be cultured from the blood stream during the period from 10-60 minutes after extraction. In the vast majority of cases, the bacteraemia is inconsequental ; in 14 cases reported by Haymaker, there was evidence of bacteraemia ; in 3 cases, the bacteraemia involved the viscera only; brain abscesses were the result in 7 cases.

(e) Paranasal sinuses.- In 5 cases, the sphenoidal sinus only was purulent ; in one case, the sphenoidal and maxillary sinus ; in one case, the maxillary and frontal ; in one case, all sinuses. Sphenoidal sinusitis in 5 of 7 cases was secondary to osteomyelitis of the greater wing of the sphenoid bone, and in the other two cases, it was secondary to cavernous sinus thrombosis.

(f) Intra-orbital abscesses .- In 6 of 28 cases, the orbital contents were involved . In three cases only, was it the determining factor in the outcome, one case resulting in spread via the frontal bone to the frontal lobe,two cases resulting in cavernous sinus thrombosis. Haymaker stated :

"Alveolar infections associated with progressive suppurative cellulitis may spread to the orbit by various routes, the most common apparently, being through the infra-orbital fissure. From cuspid and bicuspid teeth, an infection may gain the orbit subperiosteally (Thoma) Another route is through the lateral walls of the sphenoidal and ethmoidal sinuses. Both are in relation to the medial wall of orbit - the sphenoidal to the apical part of the orbit, and to the optic nerve in the optic foramen."

Other reports of fatal complications following infection.

Bowdler Henry (1935)[302a] reported a fatal termination in 38 cases involving the mandibular third molar. Childs and Courville (quoted by Haymaker) reviewed 74 cases of cavernous sinus thrombosis of dental origin.

Osteomyelitis.

The term when applied to the jaws is a misnomer : there is very little marrow in the jaws. It does not refer to a localised osteitis as seen in the dry socket, nor to a limited necrosis, but to a rapidly spreading inflammation which is followed by necrosis and sequestration.

Aetiology.- Osteomyelitis of the maxilla following extraction is almost non-existant ; the blood supply to the bone is vertical and segmental and incapable of complete occlusion in its periphery (Mowlem[303]). The maxilla is not invested in a dense cortical sheath, and the alveolar bone is thin and porous. Drainage is dependent, and may take place into the oral cavity, antral and nasal cavities.

Mowlem[303] in 1945, reported 45 cases of osteomyelitis of the mandible associated with teeth. He explained the aetiology of osteomyelitis of this bone as follows :- In every extraction, a thin lamina of bone is damaged and thrombosis of its vessels results - if very thin, it is covered by granulation tissue and resorbed without complication, with a leucocytic barrier preventing extension of the infection ; should a thicker piece of bone be damaged, the leucocytic barrier will be more distant, the bone too thick to be resorbed, and the "egg-shell" sequestrum is formed ; should the degree of damage involve the inferior dental artery, this will thrombose ahd deprive a wide zone of bone of its blood supply, the leucocytic barrier is too far distant, and the infarcted bone is open to infection. In only one of Mowlem's cases, did osteomyelitis follow extraction of the teeth anterior to the second premolar. The roots of the molars are in intimate relation to the inferior dental artery, and especially those of the impacted mandibular third molar. The extraction of the more anterior teeth

which have no intimate contact with the inferior dental canal, is less likely to cause osteomyelitis.

It is reasonable to assume that thrombosis of the inderior dental artery probably occurs as frequently as mental paraesthesia is experienced, following the surgical removal of the lower third molar. And yet, osteomyelitis is, in comparison to the incidence of mental paraesthesia and anaesthesia, a rare complication. It would appear that the collateral circulation of the mandible is greater than has been generally assumed, and that the degree of anastomosis between the inferior dental artery and the periosteal nutrient arteries is of significance. Craddock Henry (1946)[304] considered that, for osteomyelitis to supervene, not only must the inferior dental artery thrombose, but also the periosteal arteries must be involved ; the condition most likely to cause this co-occurrence, would be traumatic extraction followed by a subperiosteal abscess, osteomyelitis supervening to the point at which collateral circulation is established.

Diagnosis.- "Such cases generally start with the teeth becoming loose and sensitive to percussion, so that the patient is unable to masticate. In acute osteomyelitis, the gingivae are 'soggy' and pus wells out from the gingival margins. The jaw becomes extremely sore owing to the periostitis which generally sets in a little later. The patient suffers severe pain which is often referred to the ears. There may be marked trismus, and numbness of the lip has been observed. The leucocytic count may increase to 12,000 or 20,000. The temperature is elevated ; sometimes a high fever develops with malaise. There may be marked swelling of the face associated with acute lymphadenitis leading to general and local complications. The condition may progress rapidly, occasionally with a fatal termination." (Thoma, 1954[62]).

Mowlem[303] noted that it is necessary to distinguish between the flare-up and gemuine osteomyelitis after extraction. Early diagnosis is essential, and the earliest diagnosis in his cases was five weeks, and excluding those cases diagnosed after 100 weeks, the average delay was 10 weeks. In the early stages, the radiograph shows no indication : Craddock Henry[304] found that after three weeks have elapsed, a radiographic diagnosis may be made, if fortunate.

C (2) Actinomycosis.

Actinomycosis is a chronic inflammatory process of the jaws, but more frequently of their surrounding soft tissues, when the term cervico-facial actinomycosis is used.

Aetiology.- The disease is caused by the "ray fungus" more correctly termed the Actinomyces bovis, israeli, or hominis, and Bacillus actinomyces bovis. Goldsworthy (1947)[305] stated that it appears as diphtheroid rods which occur in clusters, although some may show rudimentary branching, and is strictly a parasite ; that no tissue is immune and connective tissue is particularly susceptible. In vivo the organism forms "sulphur granules" often about the size of a pin's head : these consist of a felt-like web of branching filaments that are gram-positive. In histological sections, these colonies are surrounded by club-like bodies, gram-negative and radially arranged: the bodies are entirely an in vivo feature, and are not seen in laboratory cultures. Its pleomorphic character, however, defies description ; the organism from an old sinus differs in morphology from that from freshly draining pus; the presence of secondary infection alters considerably its morphology, and the most reliable specimens are from an unopened abscess; the morphology varies with the choice of culture medium.[306] Ludwig (1955)[307] published a detailed account of the bacteriological methods used to diagnose actinomycosis, which is an important contribution, as the isolation of the organism and its culture presents very considerable difficulties. A single negative bacteriological examination of pus does not rule out actinomycosis : several attempts should be made to isolate the organism and despite repeated negative results, the condition should be regarded as actinomycosis, if the clinical features of the disease are evident (Rosh and Seldin,1948[306]). MacGregor (1945)[307] recommended in vitro sensitivity tests as he noted considerable variations in sensitivity of the organism to antibiotics. In the examination of pus, it should be swilled around the sides of the test tube, and the adherence of the sulphur granule to the side, noted. A granule may be selected, squashed between glass slides, stained with H & E, and examined microscopically.

The organism is microaerophilic, and when found in the oral cavity, it can exist only in sites of reduced oxygen tension, and of especial importance is the gingival flap overlying the partly erupted mandibular third molar. Thoma (1916) found it in the root canal of a mandibular premolar; Parsch (1932) published a microphotograph illustrating its presence in the root canal of a tooth; Goldsworthy considers it to form part of the normal oral flora.

Pathology of Actinomycosis.- Thoma (1954)[62] stated that actinomycosis may occur in the jaws as a central lesion, or it may involve the soft tissue, especially the connective tissues around the jaws. The mandibular region is most frequently involved, although cases involving the maxillary region have been reported by Cook (1957)[308] and Ludwig (1955).[309]

An injury to the soft tissues surrounding the jaw is necessary, and actinomycosis sometimes follows mandibular fracture, and in the majority of cases, mandibular third molar removal. There is an incubation period following injury, which may be several weeks, often after the healing of the initial injury.[304] MacGregor[307] stated that

"It is one of the more curious features of the disease that an organism that apparently gains entrance through in many cases, a tooth socket, should cause abscesses in the cervico-facial region, leaving no sequellae in the original portal of entry. Bone involvement is rare."

Spread is via the cellular planes and the blood stream. There is no lymphatic spread, but should the lymph glands be involved, this is due to secondary infection.[309]

Rosh and Seldin[306] noted that

"Actinomycosis is often complicated by what has been termed secondary infection. It is probable that such infection by pyogenic organisms may precede the actinomycotic infection and may result from the presence in the tissues of a foreign body or an infected or impacted tooth. Such a suppurative process is known to lower the oxygen tension in the tissues involved and consequently may produce conditions ideal for propogation of the anaerobic Actinomyces."

Axhausen (1935)[62] pointed out the co-existance of pyogenic or sub-pyogen-

ic infection, with actinomycosis, and believed that the clinical picture - chronic, subacute or acute, varied accordingly.

More usually, the process is of the chronic type, and the clinical picture is dominated by a slow suppuration, with a fibroblastic reaction (Ritchie and Arnott, 1949[310]). A painless swelling appears, with extension to the temporal region and down the meck. The swelling becomes indurated and brawny, and develops a purplish hue, without fluctuation. Trismus generally occurs, and settles into a painless locking of the mandible. Lymph nodes are usually not enlarged. The general health is not affected, and the temperature may be normal or only slightly elevated. Eventually the swelling becomes nodular, and there may be a focal fluctuation with abscess formation, followed by a discharge of pus, and the establishment of a sinus. The process continues, and multiple sinuses form, and the mass becomes nodular and irregular due to fibrosis. Thus multiple isolated pockets of infection are walled off by dense fibrous, avascular tissue ; therein lies the difficulty of treatment, as surgical drainage,is difficult, and antibiotics are walled off from the infection. Mowlem therefore considered that the most effective method of treatment is deep incision of the abscesses and open packing with tulle gras, accompanied by a long term cover of the antibiotic of choice. So difficult is eradication that Rosh and Seldin [306] and Cook[308] considered that a cure cannot be confirmed until six months elapse without recurrence after treatment ceases.

The acute type of actinomycosis has only recently been recognised. Ludwig described the condition as rapidly progressive with suppuration predominating over fibrosis; it was his opinion in 1955, that most of the cervico-facial infections follow the acute course, and have not been recognised as actinomycosis, as the bacteriological investigations ~~were~~ are not specifically requested. Of MacGregor's ten cases, six were of the single abscess, acute variety.

Diagnosis of actinomycosis.- Diagnosis is certain when the sulphur granules are recognised. When pus is obtained, a specific request must be made for examination for Actinomyces bovis, which is a time consuming

process of anaerobic culture.

In the chronic type, a detailed history is essential; the disease manifests itself often after a long incubation period, weeks, at times months, after the initial soft tissue trauma which may well be overlooked.

Differential diagnosis must include the consideration of pericoronitis, cellulitis (non-specific), osteomyelitis (radiographic study is usually negative in cervico-facial actinomycosis), a tuberculous lesion (a Mantoux test, lung field survey, sputum study, and a biopsy may be required), malignancies (osteogenic sarcoma, even squamous cell carcinoma involving the lymph glands), salivary gland infection, and the rare fungus infections.

Conclusions re actinomycosis.- Actinomycosis has been covered in considerable detail intentionally, as the partly erupted tooth, especially the mandibular third molar, is the most usual origin of the Process. The following recommendations may be made :-

(i) The partly erupted mandibular third molar, especially when chronic pericoronitis is a feature, should never be removed without recognising this disease as a possible late sequella. This implies a follow-up period long after the wound has apparently healed.

(ii) Actinomycosis is a disfiguring disease, and multiple puckered sinuses may be its result. Early diagnosis is essential, and bacteriological examination of pus for Act. bovis specifically, should be arranged : a negative finding does not rule out the disease, and the tests must be repeated, whenever pus is obtained.

(iii) Actinomycosis spreads by the blood stream, and possible by implantation in the respiratory tree. A follow-up lung field survey should be carried out to confirm that respiratory actinomycosis has not complicated the cervico-facial lesion.

C (3) The Bacterial Flora in Odontogenic Infections.

Alin and Agren (1954)[311] and Ludwig (1957)[312] found that the majority of suppurative lesions of the dental tissues and related oral structures are mixed infections. The composition of infecting organisms generally resemble that of the normal flora of the mouth. Both investigations showed that Staph. albus and Str. viridans were the most commonly found organisms in the oral infections they studied. Ludwig found that when Staph. aureus or beta haemolytic streptococci were the organisms associated with acute infections, they were low-grade pathogens because the former were more often non-secretors of coagulase, the latter more often not of Lancefield's Group A. Alin and Agren studied also the sensitivity of bacterial specimens from odontogenic infections, and noted that in such mixed infections, the organisms frequently displayed different chemoresistance; they found that the organisms were most susceptible to chloromycetin and terramycin, penicillin and aureomycin occupying an intermediate place, and streptomycin and sulphonamides in most cases proving ineffective.

D. The Control of Infection - Antibiotics.

Today, the oral surgeon has at his disposal, a wide range of antibiotics with which to combat infection. The advantages gained, however, have been tempered by several hazards, which may prove more serious in nature than the original infection :

1. Failure to make a diagnosis.
2. Failure to do good surgery.
3. Toxic and allergic reactions, anaphylactic shock included.
4. The emergence of strains resistant to antibiotics.
5. Superinfections, which may prove fatal.

1. Failure to make a diagnosis.

Kramer (1956)[313] illustrated the abuses of the antibiotics by the following cases :

Case I : A female aged 60 years, was found to have ulcerations of the lip, palate and gum, associated with a submandibular swelling. She was treated unsuccessfully by a medical practitioner firstly with penicillin, then with aureomycin, after which all the antibiotics in turn, and all forms of topical medication were tried. When eventually referred, she was found to have acute leukaemia.

Case II : A patient was unsuccessfully treated for an ulcer of the palate, with penicillin, ascorbic acid, Taubman's iodine without success. When eventually referred, after very considerable delay, a biopsy showed a tuberculous ulcer.

2. Failure to do good surgery.

There is reason to believe that slovenly, careless surgery is sometimes carried out under the protection of an antibiotic cover. The scope of surgery has considerably been broadened by the judicious use of the antibiotic cover, on the other hand. Reliance on the antibiotics is no substitute for surgery - the same principles apply to the treatment of infections (removal of the cause, the establishment and maintenance of drainage) when antibiotics are used, as applied before the antibiotic era.

3. Anaphylactic shock due to Penicillin.

In the first nine years of penicillin, two deaths only were reported from anaphylactic shock ; in the following eighteen months, fifteen deaths, and the number has increased ever since (Bell, 1956[314]). Becker (1958)[315] reported that the yearly death rate is between 200 and 300. The rate of life-threatening and non-fatal reactions is much higher, and constitutes a serious therapeutic problem. The results of a nation wide survey in America on the frequency of severe reactions to antibiotics disclosed that 2,995 reactions were recorded of which 1070 were adjudged to be severe (actually life - threatening), whilst 1925 (largely angio-neurotic oedema) were adjudged to be non-life-threatening. In the severe group, 809 were of the anaphylactoid type, 793 being associated with preparations of penicillin of which only 49 of these reactions followed oral administration,

(Welch et al 1958[316]). Bell[314] quoted two cases of anaphylactic shock due to oral penicillin.

There are two severe reactions to penicillin : first, the anaphylactic type, characterised by severe circulatory collapse, bronchiolar spasm, and angio-neurotic oedema ; second, the acute emotional upset, which usually is typified by a fear of impending death, and follows within seconds of the injection of procaine penicillin, and apparently unrelated to allergy.

The acute emotional upset is thought to be due to the accidental injection of procaine penicillin into a vein, and the basis of the reaction is the toxicity of the procaine moiety. Buff (1950) described a procaine syndrome very similar to the procaine penicillin reaction and he found that flushing, dizziness, and generalised numbness ensues on the intravenous injection of procaine at the rate of 8 mg. per minute. The procaine moiety of procaine penicillin (600,000 units) is 240 mg. and the intravenous injection would cause severe toxic reactions in the absence of hypersensitivity to the drug, and allergy to penicillin. The procaine syndrome includes tachycardia, tremor, irregular respiration, blurring of the vision, sweating, paraesthesia, dilated pupils, intense anxiety with angor animi, and closely resembles the reactions reported with procaine penicillin.

The anaphylactic type of reaction is characterised by coughing and gasping, tonic spasms, cyanosis, a weak thready pulse with a profound drop in blood pressure. It may follow soon after the injection, whilst the patient is in the dental surgery, or it may occur fifteen minutes later, when the patient is generally on the way home.

Treatment of anaphylactic shock.- At the first sign of the acute reaction, adrenaline hydrochloride (1:1000), 0.5 ml. is injected intravenously or subcutaneously ; the injection is contimued at the rate of 0.1 ml per minute until the attack shows signs of terminating. Positive oxygen should be administered if available. Intravenous injection of an antihistamine drug may be tried, but this prolongs the drowsiness. Lewis (1957) [317] recommended an intravenous barbiturate

if procaine toxicity is suspected.

Worthy of mention is the suggestion of Spiegel (1959)[318] who stated that :

"If the patient with an anaphylactic reaction to penicillin receives penicillinase within 24-48 hours after the reaction starts, the circulating penicillin will be inactivated and all acute symptoms will be relieved. There are no specific contra-indications to the use of penicillinase, which is non-toxic and completely safe for parenteral injection."

Spiegel is obviously describing a mild allergic reaction, which was characterised by the appearance of urticaria, pruritus, a generalised angio-neurotic oedema, difficulty in breathing, profuse sweating and a temperature of 101'F. It must be emphasised that penicillinase is of little use if any, in the life-threating acute anaphylactic shock, and the injection of adrenaline hydrochloride is alone likely to be life saving. Penicillinase is, however, promising in the milder reactions, as a supportive measure.

Conclusions.- Before prescribing penicillin in any form, a personal history and family history of allergy must be investigated, remembering that 1 in 7 persons is an allergic subject. A negative history of penicillin experience should not be accepted, as the patient will remember injections, and overlook lozenges, ointments, eye-drops etc. When in doubt, the intra-dermal skin test should be carried out; 0.05-0.1 ml. of a solution containing 25,000-50,000 units per ml. penicillin should be injected into the skin of the flexor surface of the fore-arm, to form a bleb : if after 15-30 minutes, pseudopods have formed,(there is an increase in size of the bleb with an irregular outline) with a zone of pallour or erythema, the patient is allergic to penicillin (Thoma[133]).

When procaine penicillin is injected, the plunger should be withdrawn once the needle is positioned, in order to determine whether the latter has been inserted into a vein : if blood is aspirated, the needle should be repositioned.

4. <u>The Emergence of Resistant Strains</u>.

The "Hospital Staphylococcus" is the offspring of the Antibiotic Era. It is familiarly known by the public as the "Golden Staph". This organism - the coagulase positive Staph. aureus, resistant to penicillin and most, often all,antibiotics -was formerly considered to be a hospital resident, and now is recognised to have extended its habitat to the general community. Its danger lies in hospital cross-infection.

Hospital epidemics caused by staphylococcal cross-infections are rightly regarded as grave emergencies : Beaven and Burry (1956)[319] reported an epidemic of staphylococcal pneumonia in the new-born with eight fatalities, and correlated the epidemic with the free use of broad-spectrum antibiotics since restriction of their use by general practitioners had been lifted in New Zealand. Gray (1956)[320] reported a similar epidemic in a maternity hospital ward, caused by beta-haemolytic streptococcus, which was clinically resistant to penicillin, although sensitive in vitro ; Rountree (1955)[321] found that if penicillin-resistant staphylococci are harboured in the nares of patients with a streptococcal infection, the penicillinase elaborated by the former is sufficient to nullify the effects of penicillin on the sensitive streptococci.

The hospital staphylococcus is thought to be disseminated by hospital staff : when a member of a surgical unit is a carrier, the results -surgical wound infection- may be most unfortunate. Brodie (1956)[322] emphasised the importance of the faecal carrier state, and found active growth of the organism on the skin of the perineum, which subsequently causes contamination of clothing, and subsequent dissemination of the organism throughout the hospital. The nasal carrier state has been investigated by Rountree.[323] Goldsworthy et al. (1959)[324] drew attention to the possible significance of the oral carrier state : a small proportion of children in Hopewood House, Bowral were oral carriers of coagulase-positive, penicillin-resistant Staph. aureus.

5. Superinfection.

Minor intestinal symptoms following the use of oral antibiotics are not uncommon : most cease on completion of the course, but some are serious. They may range from sore tongue and mouth, vomiting, nausea and abdominal distention, to ano-rectal syndrome which may persist for weeks after the antibiotic is stopped. The incidence of gastro-intestinal symptoms is about 30% of cases. and its causes are attributed to :-

(i) Breadth of the spectrum. The tetracyclines are the worst offenders, oral penicillin is less dangerous, as it leaves the coliform bacilli untouched.

(ii) Chemical irritation. The spectra of the tetracyclines is the same, tetracycline is more likely to cause complications than chlortetracycline and oxytetracycline.

(iii) Doseage and duration of the course.

Most disturbances are due to upset in the bacterial ecology, and to irritant effects on the intestinal wall.

The above conditions are distinct from superinfection. Superinfection with resistant organisms, such as monilia and staphylococci, may follow the administration of broad spectrum antibiotics, indeed, have followed intra-muscular penicillin. It is unlikely that intra-muscular penicillin and streptomycin could sterilise the gut. More likely is the sterilisation in part, of the nose and mouth, with unrestricted growth of resistant organisms, and their subsequent injestion (Binns, 1956[325]). Organisms generally responsible for superinfection, are :-

(i) Candida albicans. This fungus increases rapidly in stools after antibiotic administration. Rarely does it cause harm, yet it has caused alimentary moniliasis and septicaemia, and fatal cases have been reported. Stenderup et al.(1956)[326] reported good results in the treatment of moniliasis using intra-muscular injections of 200 mg. of pentamidine isethionate once or twice daily.

(ii) Proteus. The organism is harmless until it becomes the predominant organism of the bowel. It may respond to neomycin.

(iii) Pseudomonas pyocyaneus. This is the least common cause of superinfection. It will respond to polymyxin B which produces no toxic effects as it is not absorbed.

(iv) Staphylococcus aureus. Staphylococcal enteritis is a hospital disease and it is of grave prognosis. "An overwhelming infection results -profuse diarrhoea, shock-like state, dehydration, high temperature and pulse, hypotension and oliguria- running a fulminating course with fatal outcome in 3-11 days. Erythromycin is of value in combatting this infection, known as staphylococcal pseudomembranous enterocolitis."[327]

The Dental Significance of Antibiotic Hazards.

It may reasonably be asked wherein lies the justification for inclusion of the above dissertation on antibiotic hazards in a review of literature pertaining to unerupted teeth. The oral surgeon is permitted to prescribe and administer antibiotic drugs, and for this privilege, he must be aware of his deep responsibilities not only to his patient, but also to the community : to his patient in that administration of antibiotics may cause life-threatening sequellae such as anaphylactic shock, aplastic anaemia, and superinfection ; to the community, in that abuse of antibiotics will increase the incidence of the "hospital staphylococcus", which today, is even becoming an out-patient problem.

The following are the opinions of the writer regarding the use of antibiotics in dental and oral surgery :-

(i) Prophylactic use of antibiotics is considered obligatory in oral surgical procedures in patients suffering from valvular lesions of the heart, to avert subacute bacterial endocarditis, and in patients with very low healing propensities.

(ii) Antibiotic therapy is justified in the treatment of

(a) infections of tissue spaces containing veins which lead directly to the cavernous sinus;

(b) acute osteomyelitis, as the infection is thereby aborted, and the degree of sequestration very considerably reduced;

(c) acute infections not readily controllable by surgery and supportive measures alone (such as those having extended to the base of the skull) : the cardinal signs of poor patient resistance should be sought and noted - a high temperature, especially of the rising and swinging type, a poor leucocytic response, toxaemia and anorexia which precludes the raising of resistance by proper dietary measures- and when present, they serve as a guide for the need of antibiotic therapy;

(d) displacement of teeth into the tissue spaces around the mandible, the displacement of the maxillary third molar into the antrum, infra-temporal or buccal space;

(e) infections associated with leucopoenia (agranulocytosis, aplastic anaemia) and leukaemia;

(f) infection associated with poor resistance states (uncontrolled diabetes, the anaemias, and malnutrition etc.);

(g) actinomycosis.

(iii) The use of penicillin lozenges, poly antibiotic root canal pastes, dental cones, penicillin dusting powders, ointment and chewing gum, cannot too emphatically, be condemned, as inviting allergy to the drug, and resistance of the organisms.

(iv) A searching enquiry in respect of allergy to penicillin, and allergic tendencies (asthma, hay-fever etc) before prescribing penicillin, should be carried out ; routine skin scratch tests are fully justified.

(v) In the treatment of infections, a specimen of pus should be gained as early as possible, in order that the organisms, and their antibiotic sensitivities may be determined. Should facilities for the carrying out of these tests not be available, penicillin may be administered, and changed if there is no clinical response in 36-48 hours.

(vi) In order to delay the development of allergy to antibiotics, and to reduce the opportunities for bacterial mutation, antibiotics should be restricted to carefully selected cases : the routine use of an antibiotic cover in oral surgical procedures and in the control of infections is to be condemned. It should be remembered that severe infections were ably treated and cured in the pre-antibiotic period, by the wise interpretation of clinical signs and symptoms, and by well-timed surgical intervention.

(vii) Diagnosis is the most important part of treatment, and the tendency to evade diagnostic procedure, by reliance upon antibiotics, should vigorously be opposed.

E. The Control of Haemorrhage.

Johnson (1959)[328] found that in a group of 175 patients who underwent oral surgical procedures performed by eight different oral surgeons, the average blood loss was 233 ml. and the range of blood loss for the group was 35 to 912 ml. Blood loss in the more extensive oral surgical procedures compares with that of major surgery. It is essential that the oral surgeon anticipates post-operative bleeding before embarking upon oral surgical procedures.

1. Haemostasis.

Haemostasis, or cessation of bleeding, is effected by three mechanisms :

(i) outside pressure upon the vessel wall ;

(ii) retraction of the vessel wall ;

(iii) blood coagulation.

2. The Mechanism of Blood Coagulation.

The classical theory of blood coagulation, although made more complex by subsequent research, is fundamentally correct :

$$\text{PROTHROMBIN} + \text{CALCIUM} + \text{THROMBOPLASTIN} \longrightarrow \text{THROMBIN}$$

$$\text{FIRBRINOGEN} + \text{THROMBIN} \longrightarrow \text{FIBRIN}$$

Walker (1959)[329] stated that :

"The researches of the last ten years have added a great deal to our understanding of the clotting mechanism and its disorders, but in so doing, have so amplified and elaborated the classical theory of coagulation, and produced so profuse and confusing a nomenclature as to be the despair at times of the devotees as well as the uninitiated."

A comprehensive tabulation of the current theory is unlikely to be exact, nor is it likely to be helpful to the oral surgeon. A simplified version is therefore presented.

Scheme of Blood Clotting (modified from Walker,[329] Kranz and Ruff,[330] and Antioch.[331]

PLATELETS ⟶ serotonin ⟶ vasoconstriction following injury

Platelet factor (thromboplastinogenase)

+

A.H.G. (thromboplastinogen) (VIII)

+

Christmas factor (P.T.C) (IX)

+ ⟶ BLOOD THROMBOPLASTIN

Calcium (IV)

+

Plasma labile factor (V)

+

Plasma stable factor (VII)

BLOOD THROMBOPLASTIN + PROTHROMBIN + CALCIUM ⟶ THROMBIN

THROMBIN + FIBRINOGEN ⟶ FIBRIN

Retractoenzyme ⟶ retraction of the blood clot

Fibrinolysin (From the circulation) ⟶ solution of the clot.

Comment.- Thromboplastin has two sources : from tissue cells (commercial thromboplastin is an isotonic saline extract of cattle brain) and from blood itself, sparked off by lysis of platelets. Tissue thromboplastin is the equivilent of a mixture of AHG, Christmas factor, and platelet factor, but it requires factor VII (stable factor) as a co-factor (Walker[329]). Tissue thromboplastin is the basis of the prothrombin time test : tissue thromboplastin when added to blood, accelerates the clotting time to twelve seconds, instead of the normal several minutes. Russel's viper venom is thromboplastin.

So confusing is the nomenclature used by different workers and accepted by different countries, that an International Committee of Nomenclature has been set up, to correlate the different

terminologies. The report of this committee has been published in the British Medical Journal.[332]

3. The Bleeding Diatheses.

Bleeding diatheses are those conditions, congenital or acquired, in which protracted bleeding, localised or general, spontaneous or traumatic, is the presenting, most important, and perhaps the only symptom. Any surgical procedure in these patients will cause bleeding which may be dangerous and is always troublesome.

The bleeding diatheses have various causes, which may be classified thus :

(a) Lack of tissue support.
(b) Abnormal vessels.
(c) Platelet deficiencies (purpuras).
(d) Blood coagulation disorders.

(a) Lack of tissue support. This is a local factor, not a disorder. It has little oral significance. The bed of an ulcer, the antral lining are sites where tissue support may be regarded as negligible.

(b) Abnormal vessels. Vessels may be abnormal in form, they may be unduly fragile, or they may lack retractile properties.

(i) Abnormality of form.- Hereditary haemorrhagic telangiectasia is a congenital condition, characterised by bright red lesions of the skin and oral mucosa, which are capillary haemangiomas. The lesions must be carefully avoided in oral surgical procedures. The angiomata may be present on the tongue, palate, gingivae, and the lips, especially at the muco-cutaneous junction. They may be pin-point or larger, and in the elderly, are often spidery. Unlike petechiae, angiomata are red, and blanch on pressure. Haemorrhage is on the surface, and is never interstitial. It may be spontaneous, or the effect of injury. Haemorrhage may be severe and persistant. Diagnosis is by appearance, the family history (simple dominant transmission in both sexes), no abnormality in bleeding and coagulation times, in platelet count, and in clot retraction. Haemorrhage may be controlled by cautery - electric or

chemical (e.g., 50% trichloracetic acid). Oestrogen therapy is effective, but not suitable for males.

(ii) Capillary fragility.- Hereditary fragility is seen in von Willebrand's disease, a rare condition, which has been called "vascular pseudo-haemophilia" and conservative, prophylactic treatment is important. Scurvy causes capillary fragility, due not to ascorbic acid deficiency, but to vitamin P or citrin deficiency (Thoma[133]) Capillary fragility is acquired by irradiation - Hiroshima disease - and is found to result in blast injuries. Capillary fragility increases before the commencement of the menstrual cycle. Capillary fragility is easily recognised by the tourniquet test, but must be differentiated from the purpuras, which also give a positive tourniquet test result.

(iii) Lack of vascular retractility.- This is especially seen in arteriosclerosis : rigid vascular walls, high blood pressure, and the contra-indication of adrenaline containing local anaesthetic solutions, makes oral surgery difficult. Diabetes and certain collogen diseases affecting the arterioles, predispose to post-operative haemorrhage.

(c) The Platelet deficiency diseases.

These are characterised by petechiae or "flea-bite" haemorrhages, which appear beneath the skin and mucous membrane ; the capillary fragility disorders, it must be remembered, produce the same flea-bite haemorrhages. Thrombocytopoenic purpura is of three types :

(i) Monosymptomatic , cause known.

(ii) Polysymptomatic, cause known.

(iii) Idiopathic (Cause unknown).

All are characterised by a reduced platelet count, the normal platelet count being 250,000 per c.mm.

(i) Monosymptomatic purpura is, as the name implies, characterised only by the bleeding disorder caused by platelet deficiency. It is a reaction to a drug. Any drug may cause it - quinine, benzene derivatives and sedormid (a proprietary sleeping drug on the market) are examples. Levine (1959) [333]reported a case caused by an abortifacient containing "quinine and steel" which was the cause of nasal and gingival bleeding,

petechiae of the skin and oral mucous membrane, and haematuria. The drug induced purpura may, however, be part of a polysymptomatic syndrome.

(ii) Polysymptomatic.- Purpura may be secondary to carcinomatosis, leukaemia, aplastic anaemia, and various antigen-antibody reactions - Henoch-Schonlein disease (joint pains, renal and alimentary symptoms) and collogen diseases.

(iii) Idiopathic purpura.- This forms the largest group - which reflects present day ignorance of the aetiology of most cases of purpura.

Diagnosis of purpura.- Diagnosis depends on a positive tourniquet test, a prolonged bleeding time (due to lack of serotonin), a normal clotting time, a low platelet count, failure of clot retraction, and the clinical finding of petechiae. Once purpura is diagnosed, it must be decided whether it is a monosymptomatic disorder (when it falls into either the idiopathic group, or the sensitisation group), or polysymptomatic, when leukaemia, carcinomatosis, or sensitisation may be suspected. Polysymptomatic purpura must be differentiated from the polysymptomatic petechiae-producing disorders caused by scurvy or irradiation, and this is readily effected by the platelet count or clot retraction time.

Treatment of purpura.- Idiopathic purpura responds favourably to splenectomy. The sensitisation type may disappear on withdrawal of the drug causing it, if known; if sensitisation is suspected, and the drug or substance causing it is unknown, cortisone therapy, which inhibits the antibody-antigen reaction, may prove successful, although it must be realised that the systemic effects of cortisone (the symptoms of Cushing's syndrome) are the inevitable sequellae when the course is prolonged. Oral surgical procedures should be postponed until the platelet count is normal ; haemorrhage responds to pressure, with the aid of biological haemostatics - fibrin foam, Gelatin sponge, or absorbable cellulose (Burket, 1957).

(d) Blood coagulation disorders. These conditions produce ecchymosis which is a subepithelial haemorrhage covering a considerable area, and quite distinct from the pin-point haemorrhages called

petechiae. Ecchymosis is a characteristic reaction to trauma in the blood coagulation disorders. The blood coagulation disorders which will be considered are :

(i) Haemophilaa,
(ii) Christmas disease,
(iii) Prothrombin deficiency,
(iv) Heparin therapy,
(v) Fibrinolysis
(vi) Afibrinogenaemia.

(i) Haemophilia. This is a sex-linked hereditary disease, confined to the male, and transmitted by the female to about two-thirds of her male offspring. Although a rare disease, the haemophiliacs of Sydney number about 200. The aetiology of the disease is not completely known, but it is associated with a deficiency of A.H.G. or anti-haemophilic globulin, and consequently there is no plasma thromboplastinogen for conversion to thromboplastin.

Diagnosis is usually made at an early age ; haemorrhage is noted to be persistent on circumcision, and on eruption of the deciduous teeth ; the slightest injury will produce widespread ecchymosis, and the slightest scratch, persistent bleeding ; haemarthrosis is a common occurrence, and is liable to lead to disability. The blood clot when formed, is soft and cyst-like. The coagulation time is greatly delayed (1-2 hours), but all other tests are normal. Haemophilia must be distinguished from hereditary haemorrhagic telangiectasia, Christmas disease, hereditary thrombocytopoenic purpura, von Willebrand's disease (pseudo-haemophilia), and familial nose-bleeding.

A knowledge of the treatment is essential to the oral surgeon. In emergency, or elective surgery, the patient should be admitted, and FRESH human plasma (A.H.G. is labile), or freeze-dried material of normal plasma administered, pre-operatively, operatively, and post-operatively. As Archer (1956) suggested, 500 ccs of fresh whole blood (no more than 24 hours old) may be transfused the day preceding surgery; 500 ccs. of fresh whole blood the day of surgery and blood to be available during the operation ; 500 ccs. of fresh whole blood daily until bleeding stops.

In addition to the blood transfusion, Archer recommended the usual precautions - bed rest, sedation with phenobarbital or paraldehyde, and periodic blood counts and prothrombin times. Concentrated A.H.G. of animal origin may be used in 10 day courses, globulin of a different animal species being used each time to prevent anaphylactic shock ; a 10-day course in Sydney costs, according to Professor Blackburn of the University of Sydney, approximately £300, as the concentrate must be flown in deep-freeze from the United Kingdom.[334] Orr (1959)[335] recommended the use of local anaesthesia when extractions are to be carried out, combined with the use of a gutta percha lined acrylic splint. Walker (1959) disagrees with Orr however, and prefers to use general anaesthesia to avoid the needlepuncture, which predisposes to haematoma formation. Walker (1959) recommended the packing of a socket under positive pressure for at least five minutes, with oxidised cellulose gauze, soaked in Thrombin Topical (Parke Davis) diluted to 100 units per cc to prevent lysis of the clot caused by more concentrated solutions, and he warned against the suturing of the gingivae over the socket, as he has seen asphyxia caused by compression of the trachea by a haematoma so formed. It should be added that if oxidised cellulose is to be used in combination with thrombin, the acidity of the former will inactivate the thrombin, unless the thrombin solution is used with oxidised cellulose neutralised beforehand with sodium bicarbonate solution.

(ii) Christmas disease. Walker (1959) stated that this disease is clinically and genetically indistinguishable from haemophilia. In American literature, Christmas disease is termed "thromboplastin component deficiency" (P.T.C. Deficiency) The male suffers the disorder while the female shows only laboratory signs; the affected female has a 50% chance of female, and 50% chance of male offspring with the condition (Blackburn,1959). The disorder is diagnosed by the family history, and is distinguished from haemophilia by the thromboplastin generation test (Burket,1957). Treatment is similar to that of haemophilia, except that stored serum is satisfactory as Christmas factor is very stable, unlike A.H.G.

(iii) Prothrombin deficiency. Prothrombin is produced in the liver from the fat-soluble vitamin K. Vitamin K may be injested from food sources, and is elaborated by intestinal micro-organisms. Prothrombin may, therefore, be deficient when the diet is deficient in vitamin K, or when the intestine is sterilised by broad-spectrum oral antibiotics, when absorption from the intestine is defective (in sprue, biliary deficiency, and in intestinal hurry), and when the liver is incapable of its manufacture (cirrhosis, carcinoma of the liver, hepatitis). Antioch (1959)[331] reported that prolonged salicylate therapy can depress the prothrombin level, but Walker(1959)[329] considered that this ~~was~~ is unlikely except in renal impairment, as the alarming symptoms of salicylism - ringing in the ears, headache, disturbances of vision, loss of apetite - would precede incoagulability. The prothrombin level may be depressed by dicoumarol, an anti-coagulant deliberately used in thrombo-embolic disorders, and causes prolonged haemorrhage by the lowered prothrombin level, and involvement of Christmas factor, and factor VII.a

In cases of hypoprothrombinaemia caused by faulty absorption of vitamin K, oral administration of vitamin K is useless ; Blackburn recommended menaphthone 1-5 mg. intramuscularly. Oral synthetic vitamin K with bile salts may be given, if the only defect is biliary deficiency. When the liver is incapable of synthesising prothrombin, vitamin K administration is, of course, useless, and transfusion alone is effective. To counteract the depressant effects of dicoumarol, it is useless to cure haemorrhage by withdrawal of the drug, as its effects may persist for 96 hours after discontinuance of therapy[327]; intravenous injection of vitamin K_1 should be given by the patient's medical attendant, or a haematologist, for it the full coagulation propensity is restored, the patient may be precipitated toward the thrombo-embolic risk.

(v) Heparin. Heparin is another commonly used anticoagulant. It inhibits thrombin - therefore it is not reversed by vitamin K injection. It may be reversed by the injection of no more than 50 mg protamine sulphate.

(v) Fibrinolysis. This is a rare condition, which gives a typical post-operative history following extraction : normal haemostasis occurs ; in 3-4 days, there is haemorrhage ; haemorrhage recurs on the 8-9th day. The reason is clot lysis. Macanch (1959)[336] reported a case. To counteract fibrinolysin, Blackburn (1959) recommended a transfusion of serum albumin IV, with anti-fibrinolysin attached to the albumin molecule.

(vi) Afibrinogenaemia. Fibrinogen is a labile plasma protein. It is formed in the liver. Acquired deficiency of fibrinogen is not uncommon, and it may result in excessive bleeding : it occurs in liver disease. Congenital absence of fibrinogen, on the other hand, is rare, and Kranz and Ruff (1959)[330] reported the twenty-fourth case in literature. Congenital afibrinogenaemia is thought to be related to consanguinity, and appears to be genetic. It is similar to haemophilia clinically, yet haemarthrosis appears to be less prominent in afibrinogenaemia. Complete absence of clot formation in a test-tube, as well as in the one-stage prothrombin time, is suggestive of the condition. A simple chemical test for fibrinogen clinches the diagnosis. The treatment is the same as in haemophilia, and in general, consists of the transfusion of fresh plasma or whole blood, or thawed plasma which was frozen when fresh. The use of commercial fibrinogen is not recommended by Kranz and Ruff.

Screening Tests for Bleeding Problems.

1. Tourniquet test if positive, indicated capillary fragility, or platelet deficiency.

2. Bleeding time if prolonged, indicates hereditary pseudohaemophilia (the rare von Willebrand's disease), or thrombocytopoenic purpura haemorrhagica. Coagulation time for each disorder is normal.

3. Coagulation time if delayed, indicates diminished prothrombin, or diminished thromboplastin, or diminished fibrinogen, or anticoagulants in the blood. In such disorders the bleeding time is normal.

4. One stage prothrombin time if delayed, indicates prothrombin deficiency, or afibrinogenaemia.

5. Clot retraction if absent, reflects platelet deficiency, or disfunction.

Conclusions.

1. All patients for whom oral surgical procedures are intended, should be asked whether they are "bleeders" : if they claim they are, further questioning is necessary concerning past extractions, proneness to bruising, the effects of minor cuts and scratches, and family history.

2. A medical history is necessary - the patient may be under anti-coagulant therapy for thrombo-embolic disorders. As the patient may be unaware of the relevance of this medical treatment, it should be routine on the part of the dental surgeon, to question a patient deliberately regarding medical treatment.

3. Should a bleeding diathesis be suspected, the patient should be referred to a pathologist for screening tests.

4. If a bleeding diathesis is confirmed, conservative and prophylactic treatment is preferable to surgery. Careful restorative work, caries control (such as the regime recommended by the Institute of Research, United Dental Hospital, Sydney), root canal therapy rather than extraction, should be undertaken. If surgery is unavoidable, the patient should be admitted to hospital, where the necessary arrangements may be made for the administration of transfusions. Acrylic splints with gutta percha linings should be arranged pre-operatively.

5. Unerupted teeth should be left in situ, unless pathological conditions militate removal. If a simple extraction will act as a substitute for surgical removal of the impacted tooth, this course is justified.

4. The Treatment of Haemorrhage.

The treatment of haemorrhage may be divided into two parts : the arrest of bleeding, and the restoration of the normal blood volume and cellular content.

Methods of controlling haemorrhage.

(i) Pressure.- The principle of pressure may be applied in several ways : digital pressure of tissue against a bony support ; gauze pressure packs; artery forceps ; the plugging of a cavity with gauze ; the deep-buried suture ; acrylic splints or gutta percha or compound bungs.

(ii) Posture.- The erect posture to a certain extent, reduces blood pressure to the head, and this posture may with benefit be adopted in oral bleeding, unless cerebral anoxia is present. The use of the semi-erect posture, combined with hypotensive drugs, is employed in operations under general anaesthesia, such as osteotomies, when haematoma formation is to be avoided.

(iii) Thermal.- Heat coagulates blood, and the principle is exploited in the use of hot saline packs. Cold applications cause a vasoconstriction, and are used to limit haematoma formation.

(iv) Cautery.- Tissue is coagulated by dull-red heat, or by chemical protein coagulants (tannic acid, ferric perchloride).

(v) Biological preparations.- The following are in current use :

(a) Adrenaline hydrochloride of strength 1:1000, now synthesised, causes vasoconstriction for half to one hour.

(b) Ephedrine hydrochloride is used in 1% solution. So prolonged are its vasoconstricting effects, that sloughing may result. (Croker, 1952[337])

(c) Cobefrine hydrochloride is a synthetic product similar to adrenaline, but of little use as a haemostatic, as its vasoconstricting effects are considerably less.

(d) Thromboplastin is gained by the isotonic saline extract of cattle brain. It is used as a local haemostatic especially in haemorrhage from oozing surfaces, in epistaxis, and in surgery of bones. Its effects are less important than those of thrombin.

(e) Thrombin Topical (Parke, Davis) is a standardised, sterile haemostatic powder obtained from bovine plasma. It is dispensed in 5,000 N.I.H. units (2.5 N.I.H. units equals 1 clotting dose), with a 5-ml vial of diluent. Walker (1959) prefers to dilute it to a strength of 100 N.I.H. units per ml. to avoid lysis of the clot. It is best applied on gelatine sponge ; oxidised cellulose, unless neutralised by sodium bicarbonate solution, is an unsatisfactory vehicle for thrombin, which is inactivated by an acid medium.

(f) Human fibrin foam (B.P.) is an insoluble material made from human fibrinogen. It is prepared as a fine white sponge of firm texture. "It is possible to cut and shape the material with a scalpel. The pores rapidly fill when liquid thrombin is applied, causing a slight shrinkage, and it becomes soft and plastic." (Croker 1952). It is of course absorbable, and is useful in capillary haemorrhage arrest.

(g) Plasma and whole blood. These are used to restore blood volume, and missing coagulating factors (such as AHG, Christmas factor prothrombin and fibrinogen). It must be used fresh for AHG and fibrinogen. Before a full blood transfusion, grouping and cross-matching tests must be carried out, and the Rh grouping should also be investigated, especially in transfusions for a woman of child-bearing age.

(h) Snake venoms. Stypven (Burroughs Welcome) is dried,purified and sterile venom of the Russell viper, available with a solvent (a sterile aqueous 0.5% solution of phenol) in sets of 1 ml. and 5 ml. for topical application. It is recommended by Walker(1959) and Helmore (1958)

(vi) Absorbable materials. There are four in common use :

(a) Catgut, which is an absorbable suture material, is used in the ligaturing of vessels ; when chromatised, its absorption is considerably delayed.

(b) Oxidised Cellulose (B.P.C., U.S.P.) is a sterile white or cream-white gauze or cotton, of feint odour and acid taste. It is prepared by the oxidation of surgical gauze by nitrogen peroxide. It is packed in sealed containers, and its contents must be used completely or discarded, as the material cannot be resterilised. It is absorbable, swells on contact with blood, and its action depends on the formation of an artificial clot. Small amounts absorb in 2-7 days, but large quantities in up to six weeks. It cannot be used as a surface dressing, as it inhibits epithelialisation. It is contra-indicated in bone surgery, as it delays callus formation. However Helmore (1958)[338] considers this drawback of delayed healing, clinically insignificant. Oxidised cellulose inactivates by its acidity, penicillin and thrombin.

(c) Gelatine sponge absorbable (B.P.C., U.S.P.) is a light, off-white, non-elastic, tough, porous material, and may be cut into any shape and size, and does not disintegrate rapidly even with rough handling. It may be wetted rapidly, and worked vigorously in the dampened fingers or gloves. It will then absorb liquid rapidly, and may be squeezed out again if necessary, and used repeatedly (Croker, 1952). Gelatin sponge is the ideal carrier for thrombin. Haddon Kemp[286] noted that : "The use of an absorbable gelatin sponge combined with thrombin in solution 100 units per cc. appears to have great possibilities in situations where the dressing can be closed in the wound with the aid of sutures, and left in situ." It does not inactivate antibiotics.

5. Haemostatic Methods Used in Oral Surgical Procedures.

(a) Soft tissue haemorrhage. Haemorrhage from intra-oral incisions is usually mild and readily controllable, the correctly executed incision severing no vessels larger than the arteriole.

Durbeck (1957) claimed that : "....the scalpel may be used with perfect impunity from the tip of one coronoid process, down the ramus and external oblique ridge, around the buccal and labial surface of the mandible, to the tip of the coronoid process of the other side." without severing any important artery or nerve. The

severance of an artery in intra-oral surgery is generally accidental. The facial artery may be severed by a scalpel, when a buccal sulcus abscess is incised incorrectly : haemorrhage can readily be arrested by compression of the artery at the point where it crosses the lower border of the mandible ; both ends of the artery should be secured by artery forceps, and ligatured, as collateral circulation via the superior and inferior labial arteries, causes haemorrhage from the distal end. The greater palatine artery, if severed, is a source of serious haemorrhage ; "a stopping of the haemorrhage by local clamping of the artery or by tamponade is almost impossible and recourse has sometimes to be taken to ligation of the external carotid artery."(Sicher[355]). Nevertheless, an attempt must be made immediately to control it by pressure, and a splint, made by compound impression, can be held against the point by masticatory pressure, and a Barrel bandage if necessary. The sublingual artery can be severed by a slip of an instrument, and in the molar region is a serious accident ; the artery is deep, and an attempt at local clamping may not be successful, and the parent artery must be exposed and ligated ; Sicher[355] pointed out that the sublingual artery usually arises from the lingual artery, but sometimes from the submental artery.

In extra-oral surgical procedures, the deliberate severance of an artery is often necessary. The artery, when exposed, is severed between two artery forceps, and both proximal and distal ends ligated. Ricker (1949)[340] advised that :

"Vessels should be ligated at both the distal and proximal ends, so that when collateral circulation is established, there will be no additional bleeding from the region. Troublesome venous bleeding may be handled in the same way. Ligatures of silk, cotton or catgut may be used. They should be large enough so that increases in pressure will not cause rupture, but not so large as to create unnecessary foreign body reaction. Ligatures should not be tied so tightly that they will cut the intima of the vessels."

Thoma[133] recommended the use of catgut 00 or 000, or No.5 silk or linen thread, for the ligation of vessels. Not all vessels requiring artery forceps pressure need be ligated : skin incisions cause a number of small vessels to bleed on either side of the wound - these should be grasped with haemostats, twisted, and released after a few minutes.

Capillary bleeding from soft tissue usually ceases spontaneously, but, if persistant, hot saline, or adrenaline packs applied with positive pressure for a few minutes, is generally successful. Better still, fibrin foam, saturated in thrombin, may be placed over the wound, and left in situ (Thoma[133]). Haddon Kemp(1952)[286] noted :

"Cautery is sometimes valuable for the control of capillary bleeding from soft tissue.....Precautions should be taken to keep the instrument at a dull-red heat, otherwise it may sear through the tissues, cause unnecessary damage, and leave carbonised tissue in the wound to act as a foreign body."

Helsham (1959)[341] considered that the suturing of dry gauze tightly in the wound, may be used as a last resort.

(b) Haemorrhage from bone. Helsham[341] recommended that the bone cavity be cleaned and plugged with, preferably, a wet swab, so that the cavity is filled under pressure for about three minutes ; the process may be repeated if necessary, using a swab soaked in hot saline. Cortical bone may be crushed or burnished to arrest haemorrhage - the crushing of cancellous bone serves only to increase the haemorrhage. An artery or vein, when severed in bone, may, if accessible, be closed by the use of a pair of rongeur forceps (Haddon Kemp[286]). Should the inferior dental artery be damaged during surgery, bleeding may be arrested by a hot saline swab, plugged to overflowing, in the socket, and held under pressure by a bite pack, or digitally, or by a bite pack and barrel bandage in the hysterical patient. Horsely's bone wax (yellow beeswax 7 parts, phenol crystals 1 part, olive oil 2 parts) applied to bleeding bone, is sometimes helpful. Persistant bleeding from a socket is in most cases, satisfactorily arrested by clearing the socket of all debris, placing into it gelatine sponge saturated with thrombin, which is held under pressure by moist gauze; the gauze may be removed after a few minutes, and the tissues sutured tightly over the cavity. Helsham[341] recommended as a last resort, the packing of the cavity with dry gauze. In very rare cases, ligation of the external carotid artery is necessary.

(c) Haemorrhage from the nose or antrum. The blood clot is cleaned out as quickly as possible, and the cavity packed tightly with swabs (a count must be kept) or ribbon gauze soaked in adrenaline, and left some mimutes. This should be repeated if necessary, and hot saline swabs tried. If the bleeding is much reduced, but not stopped, vaseline gauze packed in lightly is useful. If not successful, dry gauze tape may be packed in and removed the next day. Antral packing forceps are useful for this purpose. - Helsham.[341]

(d) Haemorrhage following tooth extractions. This is a frequently met complication, of even the simplest extraction. Haddon Kemp[286] exhorted the dental profession to give its closest attention to this complication:

"On some occasions, the patient will require assistance at his or her home, since the haemorrhage may have occurred after usual surgery hours. Therefore, an emergency kit must at all times be readily available. The contents of such a kit should include :-

(1) An efficient hand-torch.
(2) A lip retractor.
(3) Two pairs of dressing forceps - one curved and one straight.
(4) Two or three pairs of artery forceps.
(5) A pair of scissors.
(6) A generous supply of sterile gauze "throw-away" swabs, and sterile cotton wool.
(7) Narrow ¼"in. to ½ in. gauze (with selvedge).
(8) Adrenaline hydrochloride - concentration of 1 : 1000.

This armamentarium should suffice for most emergency treatments, but in the event of a haemorrhage not being brought under control, within a reasonable time, the facilities of a hospital should be sought, where an aspirator, a good light, and trained assistants are available."

The method of treating a bleeding tooth socket is comprehensively described by Arnott (1948)[342]:

" 1. Remove the blood clot from the oral cavity and the tooth socket.

2. Obtain a clear view of the bleeding spot (bleeding may occur from a wound close to the socket).
3. Pack half inch ribbon gauze against the bleeding point and then complete the packing of the socket with the remainder of the ribbon gauze, using positive pressure.
4. Pack additional gauze pack securely and firmly on top of the gauze in the socket.
5. Instruct the patient to close the jaws together firmly biting into the gauze packs.
6. Apply a firm barrel bandage.
7. After a few minutes elapse, examine the clour of the saliva within the oral cavity without disturbing the pressure maintained.

By this method, bleeding from tooth sockets can be quickly controlled. As a styptic a couple of drops of liquor ferri perchloridi can be used to moisten only the end of the gauze tape which comes in contact with the bleeding point. Excessive use of perchloride of iron is contra-indicated. When bleeding is not evident for some minutes, the barrel bandage and the gauze packs over the socket may be removed leaving the socket still packed for 12 to 24 hours. Then the ribbon gauze is removed slowly and carefully. The basis of the treatment is the application of pressure. "

(e) <u>The haematoma</u>. This is internal bleeding, and may be ecchymosis or haemorrhage under the epithelium, or it may be interstitial, spreading along the tissue planes. Walker (1959) witnessed the death of a haemophiliac, caused by the obstruction of the trachea by a haematoma resulting from the suturing of the gingival tissues tightly over a socket. Archer (1956) gave a detailed account of death by pharyngeal collapse with respiratory embarrassment in a haemophiliac, as a result of a haematoma, following mandibular regional anaesthesia. Haematomas may occur from any soft tissue injury, and especially from accidental venipuncture, occasionally incurred as a complication of maxillary regional anaesthesia. Arrest of haemorrhage is usually possible by the application of ice packs over the affected region. However, surg-

ical exposure of the vessel and its ligation may be necessary. Sequellae of haematomas include blood loss, swelling, skin discoloration, pressure effects, and infection. Antibiotics should be given to keep the haematoma sterile, a counterirritant applied to hasten its absorption. Ossification of the haematoma is occasionally seen (there is a case of ossification of an intra-cranial haematoma in the records of the United Dental Hospital, Sydney[343]), and if a haematoma collects under a buccal flap raised in lower third molar removal, it may ossify and be responsible for a bony enlargement of the region.

6. Manifestations of Severe Blood Loss.

Bennett, Dow, Leader and Wright (1938)[344] found that in blood loss, "the clinical sequellae are slight when the loss is less than 20% of the patient's estimated blood volume, moderate when between 20 and 50%, and serious, with a large proportion of deaths, when over 50%." The circulating blood volume is about 5 litres. It would be unusual for a patient undergoing oral surgical procedures to lose 20% (1 litre) of his blood volume, although Johnson [328]reported the loss of 912 ml in an oral surgical procedure.

The symptoms and signs of haemorrhage are important. The local sign of bleeding is familiar to all, yet, internal bleeding is not so simple to diagnose, and then, the systemic symptoms and signs should be carefully sought and assessed. Bailey and Love (1949)[345] list the signs as follows :-

1. Increasing pallour, especially affecting the mucous membranes such as the lips and eyelid.
2. Pulse - becoming increasingly rapid, small, and compressible.
3. Dry lips, mouth, and throat. The patient continually complains of thirst.
4. Air hunger, associated with deep inspirations.
5. Sweating.
6. Failing vision, the patient may complain that "the light has gone out." The retina is a very specialised structure, and its function is impaired if the blood supply is inadequate.

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- PART V -

OPERATIVE PROCEDURES IN THE REMOVAL OF UNERUPTED AND IMPACTED TEETH

Reference has been made to the clinical and radiographic examination and interpretation of unerupted and impacted teeth in Part III. Pre-operative and post-operative considerations have been discussed in Part IV.

A. Surgical Removal of the Mandibular Third Molar.

1. History.

The removal of unerupted and impacted lower third molars by forceps technique has for a long time, been recognised as ineffective : failure to remove the tooth, generally with decoronation, soft tissue trauma, the crushing of bone, and even fracture of the mandible, are the sequellae.

Winter (1926)[346] in his comprehensive publication, "The Impacted Mandibular Third Molar", made the first significant contribution to operative technique. In the able hands of Winter, the method he advocated would, undoubtedly, be safe and effective. However, his technique is open to criticism, in that the techniques he taught, in the opinion of the writer, could be dangerous in the hands of the inexperienced, and the less able. He relied upon mucoperiosteal flaps which today are unanimously regarded as inadequate - often they were mere cruciate incisions over the third molar crown. He used no flap retractors, but simply held the 'flap' aside with the ossisector or elevator he was using, reliance being placed not on good visibility, but on touch and, it would seem, an intuition bred by very considerable experience. He designed the cross-bar handled bone chisels or ossisectors (which are designed to be used either with a cutting action, or a scraping action) and these could be used to "channel" the bone around the tooth, by a vertical bone reduction, to give a result similar to that gained with a fissure bur. He employed what may be regarded as forceful leverage in luxation of the tooth, applied by cross-bar elevators, which are today condemned as dangerous. Thoma[133] advised :

"I should like to warn against cross-handle bars ; for the uninitiated they allow too much force to be applied and may cause fracture of the jaw if the tooth resists luxation. At any rate, I believe in so preparing for the act of luxation that very little force need be used."

Winter, however, did remove bone in accordance with the principles of mechanics, and in his book, devotes considerable time to the diagnosis of the bone which resists luxation and withdrawal of the tooth. The lasting contributions of Winter include : His technique of radiographic examination which has not been surpassed ; his classification of mandibular third molar impactions ; and his insistance on a planned operative technique, based on painstaking clinical and radiographic interpretation.

Winter believed in removing the third molar in toto. One gains the impression, on reading his book, that he regards sectioning of the mandibular third molar somewhat in the same light as "shooting a sitting pheasant". Kells (1918)[133] advocated the sectioning of the tooth to facilitate its disimpaction and withdrawal. Pell and Gregory (1942)[346a] listed the advantages of sectioning, which, summarised, include :

1. the use of less force as the tooth need not be forced or sprung over the bulbous distal surface of the second molar to gain disimpaction;

2. fine bladed elevators and light picks may be used instead of heavy elevators - trauma to bone, second molar, and temporomandibular joint is thereby obviated ;

3. bone reduction is minimal - space for withdrawal is gained at the expense not of bone, but of tooth substance ;

4. divergent and convergent roots present no difficulty, as each is removed separately ;

5. sectioning of a tooth permits its withdrawal in the direction of its long axis - damage to the inferior dental canal is averted, as the tooth need not be rotated, this rotation being responsible for the

depressing of the root apices through the roof of the mandibular canal. The tooth may be sectioned either by splitting with a chisel along anatomical planes, or by use of a bur. Whilst it is possible to split a tooth cleanly with a single blow of the chisel, the method has several disadvantages :

i. a tooth with a fused, conical root lying close to the lingual plate, may be displaced through it into the submandibular space ;

ii. the practice is liable to cause fracture of the mandible in the elderly ;

iii. the direction of split is diffocult to control.

In the opinion of the writer, a tooth can be split with more control, by making a deep slot in the crown with a bur, and twisting in the slot, an elevator blade ; or, the tooth may be sectioned entirely with a bur.

Bone removal by the chisel amd mallet has the following advantages :

i. it is heatless,

ii. it is 'clean', not leaving bone fragments and dust in the wound, as may occur with a bur,

iii. bone may be cleaved neatly along anatomical planes.

However, the chisel has certain disadvantages :

i. the dense cortical bone of the mandibular third molar region cannot be cut readily with a chisel,

ii. a mallet blow may cause fracture of the mandible in the elderly,

iii. as bone can be cut only in the direction of the chisel shaft, it tends to be radical,

iv. the procedure is upsetting to the apprehensive patient.

Kelsey Fry and Ward (1958) [347] prefer radical bone reduction in lower third molar removal : they achieve this in their "split bone technique". They utilise the grain of the bone, and by using a chisel and mallet, they make starting and stop cuts ; by twisting the chisel in the starting cuts, bone may be cleaved cleanly, in a controlled direction. The technique is very well described by Ward, and

his publication is a worthy contribution to the literature. However, it is a technique better taught by demonstration. Ward's justification for the radical bone reduction he recommends, is that "dead space" is reduced, by saucerisation of the cavity, and healing is thereby promoted.

Durbeck (1958) recommended the use of the automatic (spring recoil) chisel for bone removal. The technique has many adherents. Fine blades of various designs are used in the instrument, and bone may be removed with efficiency, control, and speed, without upsetting the patient.

Bone removal by bur is the technique which offers the greatest scope. The amount of bone reduction may be controlled with great accuracy. There is no danger of fracture of the mandible, and the patient tolerates the light touch of the surgical bur better than a chisel of any type. Dense cortical and eburnated bone may be removed without difficulty. The main danger in the use of the bur is thermal necrosis. The bur and bone must be kept cool by a saline coolant. Perhaps the simplest method of bone removal is by the use of the tungston carbide bur driven at 250,000 r.p.m. and over, by the air turbine, using a sterile distilled water coolant spray : bone in the words of Hall(1959) is "wiped away"; although Hall reported no case of emphysema, it is the opinion of the writer that emphysema,in using the air turbine, is a definite danger.

Flap design has become less timorous as research into flap reattachmemt, not only to bone, but to tooth, has progressed. Winter never extended his flaps to involve the buccal of the second molar. Nodine[14] and Thoma[133] are among those who make their buccal sulcus incision to the distal of the second molar. Cogswell (1932) included the second molar in flap design if he considered the tooth "doubtful" ; Durbeck (1958) did not make a buccal sulcus incision, but raised a buccal flap as far mesial as the canine; Archer (1956) raised ca flap to include the second molar, if he considered it advantageous. It has been the practice at the United Dental Hospital, Sydney, to gain good access and clear visibility : a broad buccal flap, to include if necessary, not only the second, but also the first molar,

is used with advantage. Warwick James (1940)[348], in a method which is largely of historic interest only, raised a lingual flap in order that he could remove the thin lingual plate of bone, and thereby gain a low drainage point with saucerisation of the bone cavity ; the disadvantages of the method include : difficulty of access to fractured apices buccally deflected, lingual nerve vulnerability, exposure of the lateral pharyngeal, pterygomandibular, and submandibular spaces to infection, dysphagia due to trauma suffered by the superior constrictor and mylohyoid muscles. Warwick James claimed, nevertheless, a significant reduction in post-operative infection, in those pre-antibiotic days.

Primary closure , or open packing ? Both methods have their place in lower third molar surgery, and both methods have their followers. Should healing by first intention or clot organisation be attempted, or should healing by epithelialisation of the cavity be gained by packing the socket??Chapman (1948)[349] advocated packing of the socket, in view of the high incidence he noted, of clot breakdown when suturing was employed ; he quoted Amies who prefers to pack the socket " to see how things are going", thereby gaining an objective assessment of post-operative progress. In cases in which haemorrhage from the cavity is persistant, open packing, using vaseline gauze, is considered advantageous, sutures being used only for the buccal incision. Thoma[133] considered that when clot breakdown is anticipated in cases of poor oral hygeine and local infection, the buccal incision only should be sutured, the retromolar incision being left open. As an alternative,Thoma recommended that such a cavity should be packed with gelatine sponge saturated with thrombin and penicillin solution, and the mucoperiosteum sutured firmly over the socket, unless the danger of infection was great. The writer is of the opinion that local use of penicillin without the administration of parenteral penicillin, is not justified; that primary closure by suturing is the method of choice in most cases. Healing by epithelialisation is slow, the patient is inconvenienced for a considerable period by food packing and requires the use of a syringe to irrigate the cavity ; there is usually failure to achieve the same full ridge contour when open packing is used, and the patient is sometimes left permanently with a depression

in the retro-molar area ; when the bone overlying the distal surface of the second molar root has been surgically removed, open packing tends to leave the root permanently exposed (resulting in hypersensitivity and impairing the support of the tooth), whereas healing by clot organisation usually permits regeneration of the bone.

Anti-oedematous measures have received considerable attention in recent years, to overcome the three post-operative develOpments so frequently seen after lower third molar removals - trismus, swelling, and pain. Many consider that an exaggerated inflammatory response is the reason for the post-operative discomfort, whereas others with, I feel, some justification, believe that the reduction of this discomfort lies in technique.

The overall purpose of the inflammatory response is beneficial, and serves to supply to the injured field, antibodies and agglutinins to oppose infection, and fibrinogen, to prevent its spread. The factors delaying healing, and promoting post-operative discomfort are :

1. failure to remove non-vital tissue,
2. lack of haemostasis,
3. inadequate wound margin blood supply,
4. introduction of infection,
5. foreign bodies in the wound.

The first effort to be made is to ensure that the above factors are not permitted to complicate healing. If they are present, it is obvious that the institution of measures deliberately designed to inhibit the inflammatory response, invites disaster.

Anti-oedematous measures include : the application of hot and/or cold compresses; antihistamine therapy ; enzyme therapy (hyaluronidase, streptokinase, and trypsin) ; and cortisone.

Cold applications.- Ice packs applied to the face is the method used. Thoma[133] redommends ice packs for 15 minutes of every hour after operation : this causes vasoconstriction, a decrease in metabolic rate, is more penetrating than heat, reduces the degree of oedema, and is

contra-indicated in the presence of infection.

Hot applications.- There is little justification in regarding the application of heat as an anti-oedematous measure. It is generally considered that it has little influence on the rate of healing, or oedema. A superficial vasodilatation, and a local rise in metabolic rate are its effects.

Antihistamines.- Silverman (1953)[350] reported an increased rate of healing, diminished oedema and trismus using antihistamine therapy consisting of 50 mg taken on three successive nights after the operation. Szmyd (1956)[351] found that antihistamines did not significantly change the post-operative picture except in so far as post-operative nausea was reduced, with drowsiness increased. Keesling and Hinds (1957)[352] in a double-blind technique, revealed that there is no improvement in post-operative results to be gained. Hinds (1958)[353] considered that antihistamines have little or no value in oral surgical procedures.

Enzymes.- Hyaluronidase converts ground substance, which is chondroitin sulphate, hyaluronic acid, and heparin bound to protein, from a barrier to a conductor. The aim in hyaluronidase therapy is to institute it 24 hours after surgery (by which time agglutinins, antibodies, and fibrinogen have been delivered to the site of injury) in order that the oedematous fluid - the mere vehicle for these localising factors - may be dissipated. Hinds [353] commented : "The theory of dispersion of fluids by hyaluronidase seems to have been adequately proven. Its application in oral surgery, however, needs further investigation." Hinds contimued : "Hyaluronidase must be contra-indicated in the presence of obvious infection, unless combined with an effective antibiotic." Greater success in oedema dispersion is gained when a pressure bandage is used, as suggested by Benzer and Schaffer (1952).[354] The enzymes streptokinase and trypsin have been used : it is thought that these drugs activate a series of enzyme reactions which serve to dissipate the fibrin clots; enzymes are proteins, and an allergic reaction should not be overlooked as a possibility of this form of treatment, if repeated.

Cortisone.- This drug , claims Hinds,[353] has no effect upon a haematoma or a suppurative wound. It has, however, side effects, such as pain

and erythema at the site of injection, a rise in temperature, and urticaria.

The writer agrees with the assessment of Hinds[353] who concluded : ".... none of the newer drugs mentioned is a substitute for careful surgical technique and judgment."

2. Surgical Anatomy of the third molar region.

Certain aspects only of the anatomy of the mandible will be stressed.

The mandibular third molar lies at the level of the junction of the ascending ramus and the body. The ascending ramus does not meet the alveolar process at its posterior extremity, due to the medial divergence of the latter, but sweeps laterally to it, to become continuous with the external oblique ridge. The external oblique ridge is therefore superimposed upon the alveolar bone of the third molar. Lateral to the alveolus of the third molar, the massive bone forms either a near horizontal ledge, of considerable width generally, or a groove, of variable depth and width, lateral to which is the external oblique ridge : the ascending ramus may lie at a level posterior to the third molar when the ledge is seen, or it may flank the posterior portion of the third molar, when the groove is seen. In vertical, and distoangular impactions, the distal portion of the crown may be buried within the ascending ramus.

The other teeth are invested in thin bone of the alveolar type, and the cortical covering of their alveoli is poorly developed. The dense, thick cortical bone to the buccal of the third molar influences the pathology and surgery of the tooth as follows :-

i. Pus from a dento-alveolar abscess, in its pursuit of the line of least resistance, will penetrate the thin lingual plate rather than the dense buccal plate.

ii. In surgical removal of the lower third molar, the dense buccal cortex must be considerably reduced in most cases, which is a formidable task ; the thick cortical bone, being relatively avascular, has poor reparative powers, and may sequestrate if traumatised, heated

by burs, or compressed by elevators. It is essential that the muco-periosteal flap is so designed that it covers and protects this vulnerable bone.

iii. The overlapping of the third molar by the ascending ramus complicates third molar removal, by reducing visibility, rendering access difficult, and bringing into the field of operation, the tendon of the temporalis.

The relation of the third molar to the lingual plate varies in accordance with the degree of mesial divergence of the alveolar process from the body, the lingual deflection of the third molar in relation to the dental arch, and the depth of impaction. The socket of the third molar projects on the medial surface of the mandible "somewhate like a balcony" according to Sicher (1949)[355] and it may be shifted so far medially, that it lies entirely within the arch formed by the body. The mandible itself is undercut in this region by the submaxillary fossa, below the mylohyoid ridge. Thus, the third molar may beetle over the contents of the submandibular space, separated only by the thin lingual plate of bone. Wheeler (1958)[356] stated that "if a specimen of mandible from which the third molar has been removed, is held up to the light, the bone at the bottom of the sockets is so thin that light will penetrate it." Especially thin is the bone covering the distal apex, and the disto-lingual supernumary root if present. Winter (1926) illustrated this point by a specimen of mandible showing actual perforation of the apices through the lingual plate. It follows that the removal of apices fractured and retained, can be a most hazardous undertaking : the danger of their displacement into the submandibular space should be carefully weighed against the possible advantage to be gained by their successful removal; should the third molar be vital before its removal, the writer feels that retention of a fractured apex in relation to a thin lingual plate is the wiser course, unless the surgeon is particularly well experienced. Displacement of the entire third molar into the submandibular space has been reported. Warwick James (1940) was so impressed with the lingual relationship of the third molar that he used the lingual approach and withdrawal in surgical removal.

The relationship of the third molar to the inferior dental canal is important. Stockdale (1959)[357] found that in the series of third molars he studied, 100% of the unerupted teeth were related to the inferior dental canal, 55% of the partially erupted teeth were related, while only 5% of the fully erupted teeth were related. He classified the relationship of the roots of the third molar to the inferior dental canal as follows :

(1) No relationship : this is seen in the normal case.

(2) Apices lying on the roof of the canal : In cases of this type, root formation ceases as the apices reach the roof of the canal Some grooving is usually seen on the inferior surface of the root.

(3) Apices lying to the buccal or lingual side of the canal : This is seen when the developing roots reach the canal before their normal growth is completed. The roots are then deflected by the canal, and grooving is found on that side of the root in contact with the canal. Examination reveals that grooving is most commonly found on the buccal side of the roots, indicating that the inferior dental canal lies lateral to the roots of the mandibular third molar.

(4) Partial or complete encirclement of the canal by the roots : In these cases the roots grow straight toward the canal. When they reach it, they continue developing to either side of the canal and rarely may encircle the canal, fusing below it. If no fusion occurs below the canal, an artificial bifurcation is produced.

(5) Other relationships : When the mandibular third molar is lying in a horizontal position the tooth may lie on the canal, and thus part or all of the mesial surface of the root may be in relation to the canal.

When there is an intimate relationship between the canal and the root apices, the cortical bone which surround the canal may be deficient and then the periapical connective tissue of the mandibular third molar is in direct contact with the contents of the mandibular canal. Stockdale found that : "In the majority of cases .....the canal has been placed lateral to the roots of the mandibular third molar, and only in cases where there has been marked lingual inclination of the tooth has the canal been found on the medial aspect of the roots as indicated by

grooving on the lingual surface of the root."

3. The Vertical Impaction.

Clinical and radiographic findings.- The tooth may be partly erupted with its occlusal surface covered in part by an operculum, without occlusal bone coverage. It may be impacted against the ascending ramus which may cover the entire occlusal surface or its distal cusps only. Pathologic bone destruction may be noted. The crown may be out of contact with the second molar, or it may be impacted under the distal convexity of the second molar, or the overhanging margin of a restoration. The roots may be fused, and bulbous or conical ; they may be bifurcated, and curved distally, straight, divergent, convergent apically, or, rarely, inclined mesially. Hypercementosis of the roots, ankylosis of the crown, intimate relation of the roots to the canal may be noted.

Treatment Plan .- This is based on the clinical and radiographic findings, and the surgeon's experience. It is readily seen that each impacted tooth must be considered carefully, and the technique modified to suit the particular problems posed by the case under consideration. The difficulty of the operation is assessed generally, by the depth of impaction, the size of the m3 space, local pathology, and the age of the patient. Other factors influence the prognosis, such as the size of the oral commissure, the presence of trismus, the amount of salivation, the degree of patient co-operation etc.

Instruments and materials required.-

Towels and towel clips.
Aspirator and reamer.
Cheek and tongue retractors.
Scalpel, with No 12 and 15 B.P. blades
McIndoe forceps
Periosteal elevator
Flap retractor, non-toothed
Bone removing armamentarium of choice
Metal water syringe
Surgical pliers
Elevator, fine, straight
Elevator, binangle (Apexo type)
Elevator, ploughshare (R & L)
Picks, fine binangle
Rongeur forceps
Bone file
Curette, straight and binangle
Needle holders

Instruments and materials required (continued)

Needles, small half circle, with cutting edge.

Scissors

Haemostats.

Forceps, for securing the tooth once luxated.

The following should be available :- sterile cotton wool applicators, throw-away gauze swabs, ½ in. ribbon gauze, pressure packs ; suture material, catgut and nylon ; sterile saline, hot and cold ; gelatine sponge ; dettol ; vaseline; metaphen tincture ; gentian violet and sterile pen.

Operative Procedure.

(a) The buccal flap. The external and internal oblique ridges are palpated, and the retromolar triangle defined. The retromolar incision bissects the triangle, passing forward from its apex, to the distal surface of the second molar at its mid-point. The incision is swept around the second molar to the buccal, the gingival attachment being severed ; the gingival incision passes mesially to the interproximal space between the second and first molar. From this point, the incision passes to the buccal sulcus, at 45° to the vertical, and should not be carried beyond the level of the apex of the first molar.

Greater access may be gained by including the first molar, even the second premolar, in the flap design. The retromolar incision may be carried further, up the ascending ramus along its anterior border. There are no contra-indications to the outlining of generous flaps, providing these principles are followed.

(b) Flap elevation. The flap, lightly held by the McIndoe forceps, is cleanly raised from the bone, commencing with the bucco-mesial section. It may be necessary to dissect the flap free of the scar tissue around the third molar crown, if there has been pericoronitis. Access may be required by elevation of the lingual mucoperiosteum ; the lingual nerve should be visualised and avoided. The buccal flap may be retracted by the assistant, using a flap retractor (non-toothed), or by the surgeon, using the periosteal elevator.

(d) Bone removal.- The greatest convexity of the crown must be freed of bone in the first instance ; this may incur bone excision occlusally, distally, buccally, and sometimes lingually, if the tooth is lingually inclined. The path of withdrawal must be anticipated, and further bone reduction planned accordingly , allowance being made for the sectioning of the tooth if it is thought advisable. The form of the roots determines the path of withdrawal : if they are distally curved, the tooth may be rotated distally and upwards, and bone must be removed to the distal of the crown to permit this movement ; should the roots be straight, and the second molar offer no interference, a vertical path of withdrawal is indicated. Sectioning is indicated in the rare case in which both roots curve mesially, and when the roots are widely divergent; when the roots converge in their apical portion, the crown may be severed and the roots removed individually, or, the bifurcation should be exposed, the inter-radicular septum excised, to overcome its resistance. It is seen therefore, that bone removal must conform to the following principles :

(i) the greatest convexity of the crown must be exposed;

(ii) an application point for an elevator blade must be provided;

(iii) a space must be provided, into which to displace the tooth in luxating it;

(iv) a bone-free path of withdrawal must be created.

The buccal plate is best reduced by outliningthe portion to be excised with bur holes, which may be linked with either a fissure bur, or chisel; the bone so isolated, may be lifted out en bloc, by an elevator, chisel or osteotome. Bone is most simply reduced distally by bur, and a channel to the distal of the crown can by this means be provided with the minimum bone wastage.

(e) Luxation. This should be effected with the minimum of pressure, using an elevator. If the tooth is to be removed vertically, the blade of a ploughshare, or a binangle elevator may be applied to the bifurcation of the root, using the buccal plate as a fulcrum. If the tooth is to be rotated distally, a fine straight elevator may be applied to the mesiobuccal surface of the crown. If the tooth does not move as a result of light pressure, it reflects the action of a resisting factor : this factor may be bone resistance, which should be overcome, by further bone

excision ; it may be unfavourable root form, which can be overcome either by judicious bone reduction, or sectioning of the tooth; in the vertical impaction, the second molar is not likely to be the resisting factor. Forceful elevation is dangerous - it may result in bone crushing (at the fulcrum, and in expansion of the socket), decoronation, fracture of the elevator point, luxation of the second molar (if injudiciously employed as a fulcrum), and in fracture of the mandible. Once the tooth has been freed of its periodontal attachment, it may be withdrawn either by the elevator, or by forceps.

(f) Debridement. The cavity should be carefully syringed, and aspirated to remove small bone fragments. Bone fragments, deprived of their blood supply, should be removed by surgical pliers. The follicle and granulation tissue should be curetted ; granulation tissue should be preserved for histological examination, in view of the cases of epithelioma reported.[62,] Bone traumatised by instruments should be excised, and the bone margins smoothed with a bone file, to eliminate spicules which could ulcerate the flap. Haemostasis is gained, gelatine sponge moistened with either saline or thrombin, inserted into the cavity if required, and the flap replaced.

(g) Suturing. The flap should be checked for apposition. Should it be excessive, or its margins traumatised, trimming with scissors is indicated. The first suture to be inserted, should be at the mesiogingival angle, to assure correct flap alignment. Further sutures (interrupted catgut) are inserted as required. A pressure pack is placed over the wound, and held there for thirty minutes.

4. The Mesioangular Impaction.

Clinical and radiographic assessment. The tooth may be partly erupted, and associated with caries or bone destruction. It may be unerupted, and the mesial cusps may contact either the crown, or the distal root of the second molar ; pressure resorption should be noted if present. The root size, and form should be studied, as the path of withdrawal is influenced by the roots. The contact with the second molar should be scrutinised - is a rotation distally and vertically resisted by the

crown of the second molar ? The depth of impaction, the angle of impaction, the size of the m3 space, the relation of the roots to the mandibular canal, the roots reflect the prognosis.

Operative procedure.

(a) The buccal flap. As for the vertical type. The more difficult the case, the greater should be the flap extention.

(b) Bone removal. It must be decided whether tooth sectioning is indicated or not. Sectioning of the crown from the roots is advantageous when : (i) the crown of the second molar engages the occlusal surface of the third molar, to such a degree that rotation about the root apex of the latter is prevented ; (ii) the root form is unfavourable and militates against distal rotation ; (iii) the ascending ramus overlies the crown of the third molar, and the degree of distal bone removal to permit distal rotation, impracticable. When sectioning is indicated, either the mesial cusps may be excised (to permit rotation) or the crown may be severed. When the crown is severed, the space between crown and root should be widened by a fissure bur, in order that the crown may be disengaged from the second molar. Bone removal follows the principles laid down for the vertical impaction. If distal rotation is indicated, space must be generously provided to the distal of the tooth, if fracture of the mandible is to be avoided.

(c) Luxation. When the crown is not locked by the second molar, when the root form permits distal rotation, and when bone removal to the distal of the tooth has been adequately effected, a light straight elevator may be inserted to the mesiobuccal of the crown, and the rotational force applied, using the buccal cortex, not the second molar, as a fulcrum. If light pressure does not luxate the tooth, the resisting factor must be sought, and eliminated. If the crown is severed, the roots may either be removed individually, or if fused, by the aid of a traction slot cut into the buccal surface with a bur.

(d) Debridement, and suturing as above.

5. Horizontal Impaction.

Treatment plan. The second molar must be carefully assessed : caries caused by a superficial horizontal impaction, root resorption from a deep impaction, may indicate loss of the second molar. If the third molar lies in contact with the apical third of the root, care must be taken to avoid injury to the second molar. Archer (1956) stated that:

"the extraction of the second molar for the purpose of access to the third molar, is a clear-cut case of malpractice." The writer feels, however, that the case does arise when removal of the second molar is justified to reduce the severity of surgical intervention, especially in elderly persons, and those in poor health. The depth of impaction, the size of the m3 space, the presence or otherwise of hypercementosis and ankylosis, and the age of the patient are the main factors influencing the prognosis.

Instruments. The same as described for the vertical type impaction. A right angle handpiece with a long-shanked fissure bur, is a useful addition to the armamentarium, for the severance of crown from root in a particularly deep impaction.

Operative procedure.

(a) Buccal flap. The more difficult the case, the more extensive the flap design. In deep impactions, the second premolar may be included in the flap.

(b) Bone removal. The greatest convexity of the crown must first be freed of bone. The degree of bone removal then, will depend on whether the tooth is sectioned or not. Thoma[133] stated : "Most third molars in horizontal position require sectioning. Even when in high occlusal position, their removal is greatly facilitated if they are bisected at the neck just below the enamel, or where the enamel is very thin. When in low position, division of the tooth is almost always imperative."
If the third molar is to be removed in toto, bone must be removed to expose at least half the buccal surface of the crown, and the distal root must be freed of bone at least half-way to the apex, on its distal, buccal, and lingual sides ; bone reduction is best carried out with a fissure bur in a straight handpiece. If gentle leverage under the

crown fails to move the tooth, the crown is best severed. When sectioning is the method chosen, the crown after its greatest convexity is exposed, should be severed with a fissure bur, and the bur cut widened considerably to enhance the mobility of the crown. The crown is removed and the roots either removed individually, or, if fused, drawn forward by an elevator blade in a traction slot, into the space left by the crown. The angle of the mandible is considerably weakened by the buccal bone reduction, and every effort must be made to avoid force : it is advisable to relieve the roots of bone by a fissure bur, before their luxation is attempted. The horizontal third molar, if deeply positioned, may lie on the roof of the inferior dental canal, and care must be taken in severing the crown with a bur, if damage to the neurovascular bundle is to be avoided.

6. The Distoangular Impaction.

Clinical and radiographic features. This type of impaction is less frequently met, but usually is more difficult. The tooth may be partly erupted, with the mesial cusps, or the mesial surface partly visible. The distal portion of the crown usually lies buried under the ascending ramus. The tooth may be unerupted, and then it lies very deeply buried in the ramus. There may be contact between the crown of the third molar and the crown or root of the second molar ; often, the crown lies out of contact, and the roots of the molars are in contact. Winter (1926) fpund that in most cases the distal root is straight, and the mesial root is distally inclined; both roots inclined distally is a common, and unfavourable finding.

Operative Procedure.

(a) The buccal flap. The extention up the ascending ramus is of advantage.

(b) Bone removal. Any attempt to elevate the tooth distally without distal bone removal, must lead to fracture of the mandible. Distal elevation in most cases, is impracticable, as the amount of disto-occlusal bone reduction required, is excessive. The best procedure is to expose the crown by bone reduction occlusally, buccally, and lingually if

necessary ; the crown is then severed from the root, and the gap between the fragments widened; the crown is removed ; the roots, freed of bone to an extent determined by size, shape, hypercementosis etc., may be elevated into the space left by the crown. Archer (1956) advocated the splitting of the tooth longitudinally with a chisel; the writer finds it difficult to understand how access may be gained for a chisel. Nor could the tooth be split longitudinally using a bur, as the ascending ramus interferes with the alignment of the handpiece. Horizontal severance, recommended by Thoma[133] appears to be the only practicable method of tooth sectioning.

7.Unusual Positions of the Mandibular Third Molar.

The buccoangular and linguoangular impactions are unusual, the linguoangular being the more common. Complete lingual and buccal positions are rare. The principle involved in their removal are no differemt from other types previously described : ample crown exposure, severance of the crown and its removal, elevation of the roots into the space provided. In the complete lingual impaction, retraction of the lingual mucoperiosteum may be necessary, with protection of the lingual nerve by means of a periosteal elevator.

Aberrant third molars must be treated on their merits. The main factor in their removal, is accurate localisation by radiographic examination, and localising devices. It should then be decided whether removal extra-orally is the method of choice, or intra-oral removal is practicable. Third molars located in the coronoid process may be removed intra-orally ; if located in the condyle or posterior border of the ramus, an extra-oral approach is indicated.

Christianson (1950)[174] described the extra-oral removal of an aberrant third molar lying in the ramus. He used a long incision 1 cm. under the lower border of the mandible, through the skin and deep cervical fascia. The facial artery was located, ligated, and severed. The masseter was divided along its fibre direction and parted, to expose the periosteum of the ramus. The tooth was located by protractor measurements from the radiograph, the overlying bone removed, and the tooth luxated, without difficulty, due to the chronic

infection which involves most third molars located in the ramus. The granulation tissue was curetted, a drain inserted, ahd the wound closed by sutures.

Multiple impacted teeth involving third molars must be treated on their merits. The danger of surgical, and post-operative fracture, must not be overlooked, and if the complication is likely, splints should be arranged before the operation. Chisels in such cases are contra-indicated, gentle bone reduction with the bur, sectioning of teeth, again with the bur, and the lightest of elevation, alone will reduce the chances of fracture. Archer (1956) described in detail, the method of removal he advocated for the "head-on collision" between the unerupted third and second molars.

Third molars in the edentulous mandible are treated in accordance with the principles outlined above. The buccal flap is similar; the angle between the external and internal oblique ridges is bisected, and the incision carried along the crest of the ridge for a suitable distance, and then is swept into the buccal sulcus. Bone is removed by bur, not chisel ; the tooth is sectioned if necessary, by bur, not chisel. Especially in the elderly, is fracture of the mandible a complication, if leverage, or impact with a chisel, is employed. In the elderly, coronal resorption with ankylosis, is common, and necessitates the reduction of bone around the tooth, to its apex.

8. Complications Peculiar to Mandibular Third Molar Removal.

(a) Trismus. Trismus, usually associated with post-operative swelling, is a common sequella. It is usually due to trauma to the tendon of the temporal muscle, and to the masseter muscle. If traumatic in origin, it should resolve in 3-5 days ; if due to post-operative infection, it is usually due to infection of the pterygo-mandibular or submasseteric spaces. Submucosal ecchymosis is a common finding. A haematoma may extend into the buccal space, and cause external discolouration. Dysphagia may follow the operation, due to trauma experienced by the superior constrictor muscle, and the mylohoid, both of which gain attachment in the vicinity of the third molar socket. Post-operative oedema and trismus must be differentiated from the acute "flare-up",

which is of infectious origin. The temperature, pulse rate, and white cell count are aids to the diagnosis.

2. Inferior Dental Nerve Involvement. The inferior dental nerve may suffer damage either by mechanical compression (by the third molar root, or an elevator), by haemorrhage within the canal (revealed by the appearance of "bruising" in the region of the mental foramen some days later), and by severance (necessitated when the nerve passes through a foramen in the third molar root, and accidental incurred when severed by a bur). The symptoms of inferior dental nerve damage is mental anaesthesia. When the nerve is severed, mental anaesthesia usually persists for 6-12 months ; if the nerve ends are located, and sutured with human hair, regeneration is hastened, and sensation may return in 3-4 months. The patient is inconvenienced in shaving, applying lip stick, and occasionally may lip bite ; the reaction to anaesthesia is more tolerant if the patient is warned pre-operatively of the possibility. The annoying state of hyperaesthesia may precede the return of normal sensation, and the patient suffers a burning, pin-pricking sensation ; should hyperaesthesia persist, and upset the patient, it may be necessary to avulse the mental nerve - this decision should not be carried out until a trial alcohol nerve block has been found preferable to hyperaesthesia by the patient. When the neurovascular bundle is seen to pass through a third molar root foramen, Ward (1958) considers it best to cleanly sever the bundle, as regeneration is more rapid ; others prefer to twist and thereby snap the bundle, as haemorrhage is considerably less. Clean division, in the writer's opinion, is the method of choice, as haemorrhage presents no great problem, and regeneration of the nerve is most important.

The transitory mental anaesthesia, which persists for 3-4 weeks, is thought by Amies (1946) to be due to an inflammatory oedema in the canal, caused by damage to the canal roof ; the condition is relieved when the oedema resolves.

Stockdale (1959) noted that " Very occasionally, foramina have been observed in that part of the root grooved by the inferior dental canal. The pulpal blood vessels here are connected to the inferio dental vessels by extremely short apical vessels which,

when the tooth is extracted, may be ruptured close to the parent vessel within the canal. Haemorrhage thus occurs within the canal. Consequently there is a slow rise of pressure in the canal, and some hours after the extraction, the patient becomes aware of the onset of paraesthesia of the peripheral distribution of the nerve in question. A day or two later, traces of this haemorrhage may escape from the mental foramen, producing an ecchymosis of the overlying skin."

3. <u>The fractured apex</u>. The danger of displacing the lingual placed fractured apex into the submandibular space has already been discussed. An apex near the inferior dental canal may be depressed into the canal : if near the canal, Ward (1958) prefers to leave it in situ if vital ; if driven into the canal, he believes that it must be removed at all costs. Helsham (1959) is sceptical of the belief that the apex is depressed into the canal and he stated that : " it is actually through the lamina dura into the cancellous bone."; he recommended that a large block of buccal bone be removed mesially and distally of the socket to permit good visibility ; cancellous bone should be curetted out until the mandibular bundle is exposed and the root located ; a curette should be passed behind the root and it is lifted out.

4. <u>Displacement of the third molar into the lingual soft tissues</u>. When a third molar has been displaced through the thin lingual plate of bone into the lingual soft tissues, it must be recovered without delay, before infection supervenes, and the tooth is driven by muscle contraction, along the tissue planes to inaccessible regions. Localisation of the tooth is important, and hypodermic needles inserted into the tissues at known angles, may be used as localising devices.

Several such cases have been reported. McKenzie Ross (1946)[358] reported a displaced third molar in the bed of the tonsil, found during tonsillectomy : the patient's history revealed that the extraction had been attempted eighteen months previously. Knutson (1946)[359] reported the displacement of the crown of a third molar (fractured by an elevator) into the "soft tissues of the throat". Harpman (1954)[360] reported that during a lower third molar removal, the tooth "turned turtle" during elevation, and disappeared into the soft tissues medial

to the mandible; an xray taken immediately revealed that the tooth was medial to the angle ; an unsuccessful attempt was made on the following day to recover the tooth; three days later, radiographs revealed that it lay at a higher level, and more medially; it was finally removed and it was found to be deeply embedded on the medial aspect of the internal pterygoid muscle. Harpman described the surgical approaches possible, and his article is a useful contribution to the subject. The case illustrates the mobility of the displaced tooth, and the need for immediate action. Sullivan (1957)[361] reported the case of a conical rooted, mesioangular type third molar displaced into the pterygomandibular space, during an attempt to split the tooth with a chisel. Howe (1958)[362] reported the surgical removal of a third molar displaced into the submaxillary space; the technique he described in detail, a summary of which is as follows :

i. Endotracheal anaesthesia
ii. Incision along the ascending ramus, crest of the ridge, to the premolar region.
iii. Assistant applies digital pressure under the angle to prevent the tooth from slipping into the lateral pharyngeal space, and the operator should place his finger orally, distal to the tooth.
iv. Lingual mucoperiosteal flap is retracted, the large lingual cortical bone fragment removed; the mylohyoid muscle is separated from the ridge, and the tooth is revealed.
v. The tooth should be milked to an accessible position, secured by a suitable instrument, and removed.
vi. Socket is curetted, sharp bone margins smoothed, the soft tissues sutured. Care should be taken throughout, to avoid lingual nerve damage.

A submandibular approach through a skin incision may be necessary.

5. Fracture of the mandible. This is a likely complication in the elderly patient, especially when ankylosis of the tooth necessitates wide bone removal..It is possible when the mandible has been gutted by a dentigerous cyst, when multiple impacted teeth occupy the body of the jaw, and is likely when elevators are used with force combined with inadequate bone removal.Should fracture be anticipated, a splint should be constructed pre-operatively, and the patient should be advis-

ed of the risk. When fracture does occur, the posterior fragment which usually contains the tooth, should be stabilised, the tooth dissected out by bone reduction with a surgical bur, and upper border wiring carried out. Intermaxillary fixation must be combined with intra-osseous wiring.

6. Emphysema. Emphysema is the presence in the meshes of the subcutaneous tissues of air or other gases in a quantity sufficient to cause a diffuse swelling, which gives a crackling sensation. Brown (1949)[363] reported emphysema following lower third molar removal caused by the patient blowing out saliva and blood. Penney (1949)[364] reported emphysema following four hours after lower third molar removal ; he attributed the cause to a gaping incision, which had been made too far lingually. Scher and Scher (1954)[365] reported emphysema caused by the use of compressed air to clear the field of blood during third molar removal.

The air in the soft tissues gradually spreads along tissue space planes : it may cause the patient to loosen his collar ; it may interfere with speach and respiration by spread to the larynx; it may spread to the mediastinum. Respiratory embarrassment is the greatest danger and the patient should be admitted until the danger has passed. There is a gradual absorption of oxygen, which results in reduction of the swelling in 24 hours ; the residual gas, mainly nitrogen, must dissolve in the tissue fluids - a slow process. The swelling is much reduced after 48 hours. Brown recommended the use of antibiotics, as infection is undoubtedly introduced into the tissue spaces.

7. Traumatic aneurysm of the inferior dental artery. Gunter and Meloy (1941)[366] reported such a case, following lower third molar removal. After luxation of the tooth, the normal welling was noted ; haemorrhage was readily controlled. On the 5th day, the patient complained of a throbbing pain in the jaw, and paraesthesia; the wound was bulging, and when reopened, an infected clot was found, and arterial haemorrhage followed, which was controlled after an hour. On the 12th day, a similar picture was seen, persistant arterial haemorrhage was again controlled by packing. On the 15th day, when the packing was

removed, a systolic surge occurred, which was again controlled by packing, which was daily reduced. On the 25th day, a pulsating bluish mass was noted in the socket, and it readily ruptured, to cause arterial haemorrhage. A traumatic aneurysm was diagnosed, and it was presumedly incurred by rupture of the tunica externa and media during the operation; the balooning of the vessel wall caused paraesthesia of the inferior dental nerve distribution, and bone destruction. The condition was finally corrected by ligation of the external carotid artery.

8. Infection.- Post-operative infection has been covered in Part IV.

B. Surgical Removal of the Maxillary Third molar.

1. Factors complicating the operative procedure.

Access to the region is difficult, due to the presence of the coronoid process , and is further limited by a narrow orbicularis oris, and a low attachment of the buccinator muscle. Should the buccal sulcus be low, and the patient of the adipose type, severance of the buccinator permits the extrusion of fat, which limits visibility, and, being a tissue of low regenerative power, becomes infected readily if traumatised ; fat may, with advantage, be excised if its appears to be traumatised. Surgery is further complicated by the intimate relation of the tooth to the antrum and the pterygopalatine fossa. The third molar may be accidentally displaced into the antrum, the infratemporal fossa, and the pterygopalatine fossa, during surgery. Fusion between the roots of the third and second molars can occur by hypercementosis. Stafne and Austin drew attention to the high incidence of coronal resorption with ankylosis. Gross cystic displacement of the tooth is an occasional finding.

Operative Procedures

(a) Instruments. Those instruments and materials listed for mandibular third molar removal may be used for the maxillary third molar. Forceps of the surgeon's choice should be available.

(b) The Flap Design.- Using a No. 12 Bard Parker scalpel blade, the

mucoperiosteum overlying the tuberosity is severed from the hamular notch to the midpoint of the distal surface of the second molar. The incision should not be made palatal to the crest of the tuberosity as the flap becomes thick and unwieldy, and haemorrhage is invited. The incision is passed around the buccal of the second molar through the gingival sulcus, and may be continued to the interproximal space between the second and first molars, or between the first molar and the second premolar. From the interproximal space chosen, an incision is made forward and vertically, at 45' to the occlusal plane, to the buccal sulcus. Thoma (1958)[133] favours a more conservative flap design, and he prefers to avoid entirely, the gingival attachment of the second molar ; to gain additional exposure, he sometimes carries a transverse incision palatally for a distance of $\frac{3}{8}$ in. from the midpoint of the distal surface of the second molar. The writer finds it difficult to understand why the second molar should not be included, in view of the findings of Dedolph concerning flap reattachment to tooth and bone. The design of a flap must afford good accessibility and visibility, if the degree of trauma is to be reduced to a minimum.

Gogswell pointed out that when the mucobuccal fold is high, the zygomatic process is also high, and an excellent view of the tuberosity may be achieved on retraction of the flap ; he noted that "very often, however, the retraction of the buccinator discloses none of the form of the tuberosity, but instead the distal soft tissues dropping sharply from the distal gingival margins of the second molar. In these cases we have a highly vascular tissue deeply covering the underlying third molar and alveolar tissue."

(c) <u>Bone removal</u>. The following principles must be applied :

i. Overlying bone must be removed to expose the third molar;

ii. Further bone must be removed to free the greatest convexity of the crown.

iii. The purchase point of the elevator blade must be provided.

iv. The path of withdrawal of the tooth must be anticipated, and adequate bone removed to ensure that the path of withdrawal is bone free.

The path of withdrawal is decided by (i) the axial inclination of the tooth, (ii) the root shape, (iii) the relation of the distal surface of the second molar, and the hamular process. If the crown of the third molar lies beneath the second molar distal curvature, disimpaction must be achieved either by a distal rotation (into a space provided by bone removal), and if this is impracticable, the mesial portion of the third molar crown may be severed, or the tooth may be decoronated by a surgical bur. Bone in the tuberosity region is generally soft, and may be pared away with a hand chisel. A mallet driven chisel is contra-indicated, as the third molar may be displaced accidentally into the antrum. At times, the tuberosity is bulbous and a heavy, dense nodule of bone may be present; the bur-elevator combination is indicated, and bone may be thereby removed en bloc. Buccal deflection is usual, and therefore the buccal plate is thin, indeed often penetrated by the third molar crown.

The vertical impaction. Removal of this tooth is generally not difficult, unless the tooth lies hard against the second molar. Usually after ample bone removal, it may be displaced buccally, by an elevator applied between the teeth, using a gentle rotary movement with a fine blade only (Thoma[133]). Nodine[14] suggested that a bone-free channel should be cut on the palatal surface of the tooth, in order that either the crown can be graspedabove its convexity, with appropriate forceps and displaced buccally, or an elevator may be passed between the tooth and the palatal bone, and rotated "with a wiggling, apple-coring movement."

The mesioangular impaction. The crown of the third molar is often buccoverted, and its removal is thereby facilitated. However, its crown may be locked under the convexity of the second molar ; to disimpact such a tooth, three methods are in general use :

i. Cogswell recommended that the mesial portion of the crown be severed, a traction slot cut in the root, and the tooth withdrawn vertically.

ii. After extensive buccal bone reduction, the third molar may be displaced laterally.

iii. After considerable buccal and distal bone has been removed, it

may forcefully be elevated free of the second molar crown ; this technique is recommended by Thoma[133] who warned, however, that the possibility of tuberosity fracture should not be overlooked. The writer feels that if the tooth possessed a conical fused root, with a distal curvature, the likelihood of tuberosity fracture is not great ; if three separate roots are present, the root resistance offered could cause fracture - severance of the crown would then be the safest procedure.

The distoangular impaction. When in high position, the third molar may be pushed into the antrum more easily than other types of impaction. Brodie (1954) emphasised that the distal cusps may overly the hamular process, which may be fractured inadvertently. Thoma[133] recommended the use of forceps properly angulated, to engage the crown above its greatest convexity, in order that its displacement into the antrum can be averted. Cogswell favoured the severance of the crown by a straight fissure bur ; the removal of the roots is aided by a bur-traction hole ; Cogswell claimed that :" This use of a bur-traction hole and a small round point elevator gives amazing results in all cases where access to the tooth substance is limited." Traction he prefers to the use of bone or tooth as a fulcrum.

The horizontal impaction. When the crown faces distally, the tooth is best exposed along its full length, and bone to the buccal and palatal reduced to its greatest convexity ; the tooth can then be luxated by a rotary movement, using suitable forceps, such as the "cow-horm" type. Severance of the crown is necessary, when its occlusal surface faces the second molar.

2. Maxillary Third Molars in Unusual Positions

The aberrant third molar may lie in the antrum, at times as high as the orbit: if localising radiographs indicate that the tooth is in the antrum or its walls, the approach of choice is through the canine fossa (Caldwall Luc operation). The third molar may be behind the malar bone, erupting into the zygomatic fossa, its crown embedded in the temporal muscle.

3. Complications Peculiar to Maxillary Third Molar Removal.

(i) Antral exposure. In view of the intimate relation of the third molar roots to the antrum, it is not surprising that the antral lining is not infrequently exposed, and indeed, sometimes perforated. The socket should be cleared of debris and non-vital tissue, packed with gelatine sponge saturated with thrombin, and the mucoperiosteal flap sutured securely with interrupted nylon sutures, which may be removed on the 5th day. A pressure pack must be held firmly over the wound for at least thirty minutes, and the patient must receive specific instructions to avoid forceful inflation of the antrum. Antibiotic therapy is necessary only if post-operative infection is considered likely.

(ii) Prolapse of the antral lining. This appears to be an unusual sequella of third molar removal. Cases have been reported by Stoy (1949);[367] Hall Felton (1949);[368] and Moules (1949).[369] Each case followed routine extraction of the third and / or second molar, not surgical removal. The cases of Stoy and Felton are similar : in each, the prolapse was described as a soft, whitish swelling, which fluctuated with an increase in intra-nasal pressure ; in each case the antral lining was returned , the gingival tissues approximated, and sutured ; on the following day, the prolapse had recurred, bursting the sutures, and the condition was treated successfully by excising the prolapse, insufflating penicillin, and carefully suturing the gingival tissues over the socket. In Moules' case, the prolapse was associated with a polypoidal thickening of the antral lining ; the condition was treated by a radical antrostomy, curettage of the socket with removal of sharp bony edges; no attempt was made to suture the gingival tissues ; healing of the socket was uneventful.

(iii) Displacement of the third molar into the antrum. Thoma[133] stated that this is more likely to occur in the young patient. To attempt to remove the tooth from the antrum through the socket is to invite disaster. The socket should be carefully debrided, gelatine sponge saturated with thrombin inserted, and the flap secured in good apposition, by interrupted nylon sutures which should be retained for at least 5 days. It is important to determine whether the tooth is in fact within the antrum, between the antral wall and the lining, or displaced into the

infra-temporal fossa, and to localise the third molar, extra-oral and intra-oral radiographs at various angles are necessary. If the tooth lies within the antrum, or under the antral lining, the approach is via the canine fossa (after Caldwall Luc) : if in the antrum, recovery is simple ; if under the lining, a bulge must be sought, and the lining incised . Davidson (1956)[370] reported the case of a third molar which "retreated into the soft tissues surrounding the maxillary tuberosity"; this tooth was recovered from the antrum, which illustrates the importance of localisation of the tooth ; Salzman (19)[371] reported a similar case.

(iv) Displacement of the third molar into the infra-temporal space. Salzman (1958)[372] reported the case of a third molar "lost" in the soft tissues of the face, during attempted removal the previous day ; trismus and swelling over the malar bone and malar arch; radiographs (P-A and profile) revealed that the tooth lay in the infra-temporal fossa; it was found to lie deep to the coronoid process, and was removed through an incision high in the muco-buccal fold, from the first molar to the tuberosity. Armer (1947)[373] further stressed the importance of radiographic investigation immediately after a third molar disappears ; during the removal of a second molar and impacted third molar, the third molar was found to have been accidentally displaced ; after removal of the second molar, it was observed that the antral lining was intact therefore it was assumed that the tooth had been displaced into the infratemporal space; radiographs confirmed that it was in the antrum at orbital level; Caldwall Luc operation confirmed that it lay between the lining and the posterior wall of the antrum. Uhler (1951)[374] warned that forceful elevation between the third and second molar may drive the former into the infratemporal fossa, and he advised that a finger always be placed behind the third molar, during its luxation, to detect early movement.

(v) Fracture of the tuberosity. Cogswell warned that :
"When marked enlargement of the roots, or when two or three roots exist, the pressure required for the molars' removal is often more than the investing tuberosity can resist, and fracture of this portion of the ridge is apt to result."

Thoma considered that it could occur when forceful leverage was exerted on the mesioangular impaction to disengage it from the second molar crown. The tooth should be removed by careful dissection, and the tuberosity preserved ; the mucoperiosteal flap should be sutured tightly into place, using nylon thread, and the sutures removed after five days. Loss of the tuberosity is a future prosthetic disaster, and should be averted if possible. Should the fractured bone be displaced with the tooth - that is, isolated from its blood supply - no attempt should be made to replace the fractured bone, which should be separated from the soft tissue by blunt dissection. A complication of tuberosity fracture is haemorrhage from the greater palatine artery : it may be arrested by tightly suturing the wound, after packing it with gelatine sponge and thrombin ; a gutta percha or compound splint, held under biting pressure, is a useful aid.

C. The Maxillary Canine.

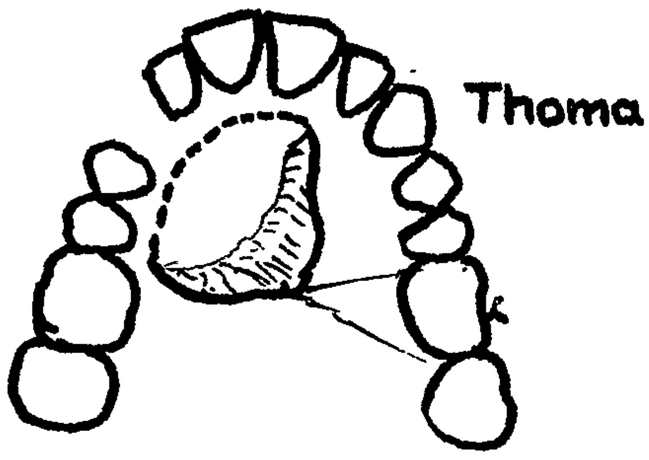
It has been emphasised in Part III that operative techniques should be commenced only after the most careful clinical and radiographic investigation of the position of the unerupted canine.

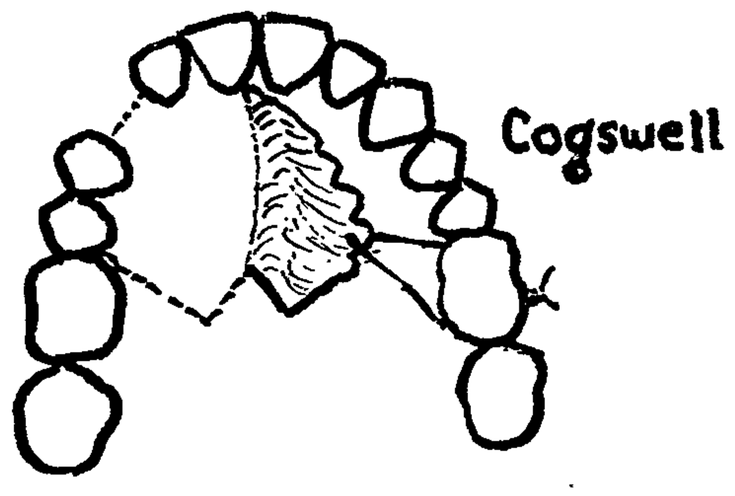
1. Canine Palatal and Near the Gingival Margin.

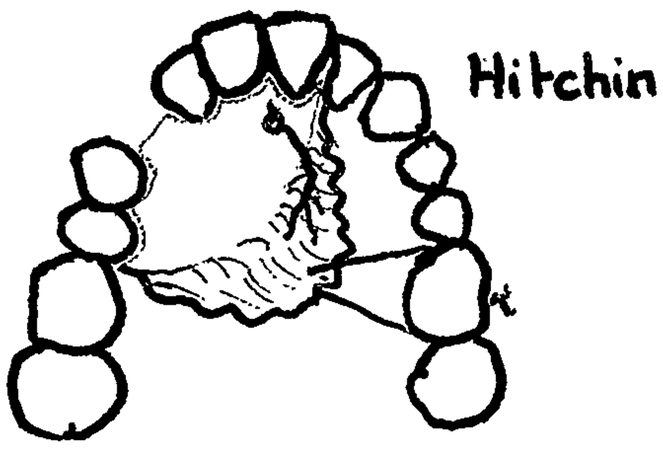
The instruments for surgical removal of the unerupted maxillary canine are the same as those listed for removal of the mandibular third molar. A contra-angle or right-angle handpiece is helpful, and the appropriate forceps should be available.

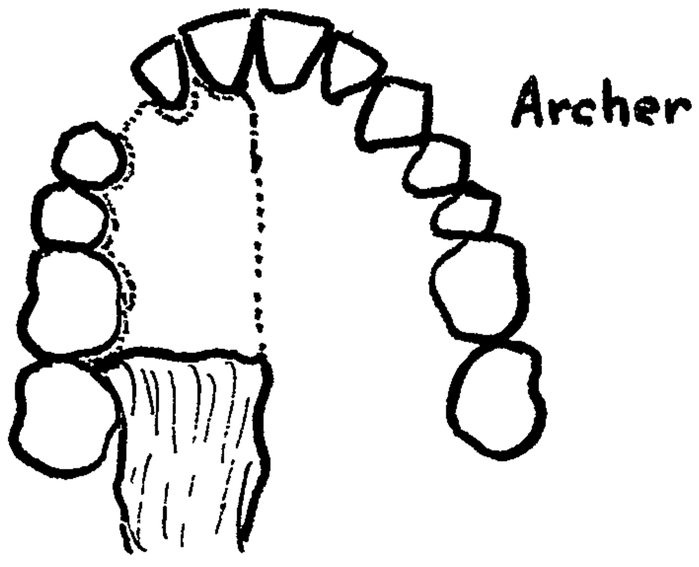
(a) The palatal flap. There is little agreement among the authorities on the ideal palatal flap design. To the writer's way of thinking, a

reasonably straight forward problem has been unnecessarily complicated by the authors of textbooks of oral surgery, no two of whom use the same design. The following line drawings represent the palatal flap design recommended by various writers :









Thoma (1958)[133] shows his customary respect for the gingival attachment of standing teeth . He carries his incision from the first molar level to the distal of the opposite central incisor, and stresses the importance of not injuring the nerves and vessels emerging from the incisive foramen. Thoma's flap design has the following disadvantages :

i. visibility is restricted;

ii. the flap when replaced, does not lie on a shelf of intact bone;

iii. the mucoperiosteum of the margin may be traumatised by instruments, and it may be undermined should bone reduction near the necks of the standing teeth prove necessary.

Cogswell (1932) designed his palatal flap to coincide with the distribution of the nasopalatine artery, emerging from the incisive foramen. It is the writer's opinion that Cogswell has overestimated the importance of the nasopalatine artery (which rarely causes noticeable haemorrhage when severed), and has underestimated the distribution of the greater palatine artery.

Hitchin (1956) severs the gingival attachment from the mesial of the first molar, to the midline anteriorly, and to the distal of the contralateral central incisor. The flap may be replaced on an unoperated ledge of bone, the sutures knotted on the labial and buccal aspects away from tongue-worry. Hitchin takes steps to preserve the vessels and nerve of the incisive foramen.

Archer (1956) uses a Bard Parker No 12 blade to sever the lingual gingival attachments around the necks of the teeth, beginning to the distal of the second bicuspid, and extending to the mesial of the central incisor. He extends the incision along the midline of the palate for 1½ in. Archer thereby ignores the neurovascular bundle issueing from the incisive foramen, and gains good visibility of the palate. There are certain disadvantages in this design : the patient is bound to be worried by the suture knots on the palate ; it is no mean feat raising such a flap, as the mucoperiosteum is particularly adherent to the palate at the midline, and this margin of the flap is bound to suffer trauma.

It is surprising that the gingival attachment to the teeth exerts such a limitation to flap design. Clinical evidence for decades has indicated that reattachment of the flap to the teeth takes place without complication. It is the writer's opinion that the integrity of the nerve and vessels issuing from the incisive foramen may be severed without ill-effect, if flap design should be extensive : haemorrhage is insignificant and readily controlled, the area of distribution of the nasopalatine artery is confined to a very small area of the palate, and patients never complain of the temporary anaesthesia of the incisive papilla region.

Hitchin's flap design is the most satisfactory : it may be extended beyond the second premolar if necessary, and beyond the contralateral incisor during the operation. Such a flap may be retracted by a periosteal elevator, held in place by the surgeon, or it may be sutured to the contralateral first or second molar.

(b) Bone removal. On retraction of the flap, the follicle of the canine may be visible; the presence of the canine may be betrayed by a bulge of the bone ; or, if the tooth lies deeply embedded, there may be no sign of the crown, which is best located by sinking a bur into the palate, and "feeling for" the enamel. Once located, the crown is freed of overlying bone, and part of the root uncovered. Bone is readily removed by a fine round bur, using a stroking motion. Care must be taken in bone removal to avoid the roots of standing teeth. If the tip of the crown lies near the roots of the incisors, elevation of the tooth is not indicated, and the crown should be severed at the neck. If the crown is distant to the roots, a straight elevator may be placed under the crown after adequate bone has been removed, and gentle elevation applied : should it rise from its socket, the tooth may be grasped with the appropriate forceps, and removed ; should resistance be felt, despite adequate bone reduction, the resisting factor is probably a dilacerated root, and severance of the crown is advisable. Once the crown has been severed, the gap should be widened until the crown can be disengaged from its bed without trauma to adjacent teeth. The root may be elevated from its socket by an elevator inserted into a slot cut in the root. Throughout the operation, the standing teeth must be tested for movement digitally whenever elevation is attempted.

Operative complications.- Adjacent teeth may be loosened : to prevent, firm digital pressure as mentioned above, is helpful. Devitalisation is unlikely. If loose, they may be splinted. The nasal cavity or the antrum may be exposed : providing infection is not introduced, the replacement of the mucoperiosteal flap will prevent fistula formation. Fracture of the root apex is not likely due to the bulk of the root : if the apex is fractured, its removal may endanger the vitality of the adjacent teeth, or it may be dislodged into the antrum; Thoma[133] considered that the inexperienced surgeon should leave the apex in situ, as an embedded tooth is sterile. Fracture of the alveolar process may occur ; it should be repositioned, the flap securely sutured, and a cast open ferrule splint inserted as soon as possible, until union occurs.

(c) Debridement. As for the mandibular third molar.

(d) Suturing.- Sutures should be knotted on the buccal aspect, to prevent tongue worry.

2. The Labiobuccal Canine.

(a) The Labial Flap. Thoma[133] prefers to use the apicectomy type flap, to avoid invovement of the gingival margins : visibility is thereby reduced, and there is the possibility that the flap margins may be deprived of their bone support. The writer prefers the flap used by Archer (1956) who raises a broad buccal flap to include the gingival margins. Deliberate gentleness must be exercised in the raising and retraction of the labiobuccal flap, to avoid, or reduce to a minimum, post-operative oedema and the infra-orbital haematoma.

(b) Bone removal and luxation. A window is cut in the bone using either a chisel or bur, over the prominence which marks the position of the crown. Bone is carefully stroked away with a fine rose-head bur, to expose completely, the entire crown, and particular care must be exercised to avoid the roots of the adjacent teeth. The root of the tooth usually lies in the broad base of the nasо-antral partition, and at times it is separated from the antrum, only by the antral lining. An elevator such as the Warwick James type, may be used to luxate the tooth with light pressure : should resistance be felt, dilaceration of the root is the cause, assuming that adequate bone has been removed; further bone should be removed, and if the root is generously invested in bone, a channel may be cut around the root. Further elevation may be attempted. Thoma[133] recommended the use of fine root forceps should sufficient space be available for application of the beaks, and a gentle rotary and buccal movement is employed. Bone margins are smoothed, the cavity debrided, and the flap sutured. A pressure pack should be applied immediately, and ice-packs, applied 15 minutes every hour to the cheek, is advisable, in order that haematoma formation can be reduced to a minimum. So frequent is the occurrence of the "black eye", that the patient should be warned Pre-operatively of the possibility.

3. Canine in the arch of the roots.

Thoma[133] considers that the removal of the canine in the "intermediate" position is more difficult than in the labial or palatal position. It lies between the roots of the lateral incisor and

first premolar, and may be near the gingival margin or in a high position. Frequently the deciduous tooth is present, and Thoma considered it advisable to extract the deciduous canine, and explore the socket, in order that the incisal tip may be located if possible.

(a) Flap design. Thoma[133] stated : "Generally it is necessary to obtain access from both the buccal and palatal sides." Hitchin concurred with Thoma, but considered it best to reflect the labial flap in the first instance, as he believed that "it should be borne in mind that the concavity of the canine fossa means that the canine is more superficial on the facial side than at first sight appears to be the case from an examination of the vertex occlusal view in which the shadow of the crowns obliterate the shadows of the roots." A labial flap is raised to include the gingival attachment of the lateral incisor, the deciduous canine if present, and the first premolar. The deciduous canine should be removed, and the socket explored for the crown of the canine. Once located, the crown is exposed by bone removal using for preference, a fine round bur with a stroking motion, and every precaution is taken to avoid injury to the roots of adjacent teeth. It may be possible to grasp the crown with fine beaked forceps, especially if the crown is superficial and near the gingival level. When deeper, bone must be removed along the mesial of the root, and into this space, the tooth is elevated, by application of pressure to its distal side; should the proximity of the tooth to adjacent roots make this impracticable, the crown should be severed, the gap between the fragments widened, and the crown removed. Once the crown has been removed, the root can usually be elevated into the space provided. Should the relation of the embedded canine to the roots indicate the advisability of a palatal flap, this should be raised, the bone overlying the crown removed, and the tooth, severed if necessary, withdrawn palatally. Hitchin believes that it is sometimes preferable to remove either the first premolar or the lateral incisor, especially if affected by root resorption : there must be reasonable certainty that eruption of the canine will follow (orthodontic traction may help, unless there is sharp dilaceration of the root).

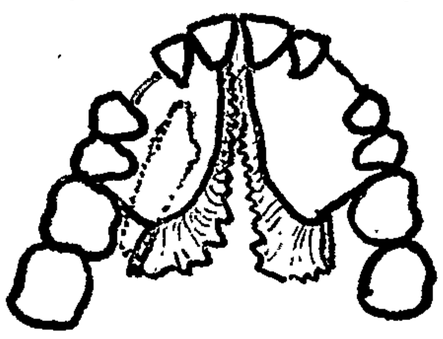
4. Crown of the Canine Palatal and its Apex Buccal or above the Buccal Root of the First Premolar.

Providing that the root is reasonably straight, such a tooth may be removed by the palatal approach only, using an elevator after ample bone reduction, or, as Thoma[133] suggested, forceps. When the tooth is almost horizontal, or its root sharply dilacerated, persistance with either forceps or elevators, is likely to fracture the apex and perforate the antrum. A two-flap operation is then advisable : (i) a buccal apicectomy-type flap is raised over the region which the canine apex would normally occupy, the apex is exposed by bone reduction and removed, (ii) a palatal flap is raised, the canine crown exposed, and generously freed of bone, and the crown displaced through the palatal window, by tapping the root face with a suitable mallet-driven instrument.

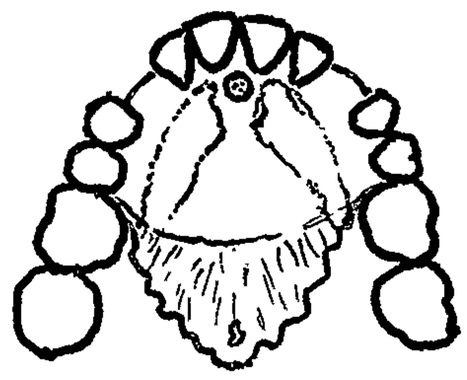
5. Removal of Bilaterally Impacted Canines in the Palate.

Should both canines be removed at the one operation? This depends upon the skill of the surgeon, the difficulties presented, and, if carried out under local anaesthesia, the attitude of the patient.

Flap Design.- Archer (1956) is reluctant to sever deliberately, the nasopalatine neurovascular bundle. He employs a double-flap technique "to preserve the nasopalatine artery and nerve, and is applicable where the cuspids do not come too close to the midline." It is noted that he does not employ the midline incisions in the double flap design, and instead, increases the flap mobility by short transverse incisions distal to the bicuspid. (see diagram). He employs a single flap for exposure of bilaterally impacted cuspids, "where the cuspids are close to the midline, because if tissue is left in the midline, as in the double-flap technique, it is traumatised and usually sloughs."



Double Flap (Archer)



Single Flap (Archer)

Thoma [133] is in agreement with Archer, and prefers to preserve the nasopalatine artery. The writer feels that there are no contra-indications to the outlining of the single, bilateral palatal flap ; the advantages are a better exposure of the palate, less soft tissue trauma due to freedom from tension experienced in the retraction of small, "stiff" palatal flaps.

6. Edentulous Cases.

Most embedded canines in the later decades, are found to be ankylosed following coronal resorption. The bone in elderly patients is hard but brittle, and there is a danger of extensive alveolar process fractures, if luxation force is applied to the embedded canine before sufficient bone has been excised. Fracture of the alveolar process is to be regarded as a prosthetic disaster.

Flap design. An incision is made just buccal to the crest of the ridge for a length sufficient to permit visualisation of the entire length of the tooth ; buccal incisions are swept up to the buccal sulcus, to permit a broader base to the flap. Should it be found necessary to gain greater exposure, short relieving incisions may be made palatally. Hitchin (1956) prefers the above flap design. Cogswell (1932) prefers the palatal approach in order that the crest of the ridge and the buccal slope of the ridge are not prejudiced, in view of the prosthetic problem offered by these patients. Archer (1956) agrees with Cogswell's approach. However, each case should be treated on its merits. The canine in the dentulous cases, in 75%, lies palatally ; in edentulous cases, it usually lies near or along the ridge crest, with its crown near or perforating the buccal bone. Every effort should be made to conserve the alveolar ridge, and loss by excision, or fracture, avoided.

7. Canines in Unusual Positions.

Each case must be treated as an individual problem. Localisation by radiographs, intra-oral and extra-oral, must be exact. This aspect has been discussed in Part III.

The aberrant canine is usually found to lie in the naso-antral wall, and the approach according to Thoma, is through the maxillary sinus which must be opened : "An incision is made across the canine fossa extending to the pyriform aperture of the nose. The tooth can generally be exposed by cutting away the anterior part of the naso-antral wall. The nasal membrane should be detached and retracted, as perforation into the nose is to be avoided." [133]

The canine may lie high up in the facial wall of the maxilla, often just below the infra-orbital ridge. Thoma recommended a buccal sulcus incision and retraction of the soft tissues; the crown usually perforates the bone, and after slight reduction, the tooth may be luxated by forceps.[133]

D. Surgical Removal of the Mandibular Second Premolar.

The tooth may lie within the arch of the roots, its crown jammed between the mesially tilted first molar, and the first premolar. Without damage to the roots of these teeth, the tooth cannot be removed buccally. Severence of the crown is the method of choice, and sufficient reduction of the root is effected (by a wide fissure bur) to permit the crown to be removed ; removal of the root is then a simple procedure, it being elevated into the space provided by the crown.

Should the premolar be vertical but linguo-verted, Archer (1956) recommended the use of the lingual flap ; the crown is exposed and freed of bone. A buccal flap is raised, and a hole is drilled between the roots of the first molar and first premolar, until the crown of the second premolar is met (the hole should be inclined slightly upwards). A blunt instrument is passed through the hole and the

tooth is tapped out of its socket through the lingual window.

The second premolar is often found to be in a disto-angular position, if the first molar has been lost. Access is usually simple in such a case and the tooth may be elevated after adequate bone removal. Should the crown lie under the mesial surface of the crown of the tilted second molar, the crown of the second premolar should be severed, the gap widened, and the crown removed ; it is a simple procedure to remove the root.

In the edentulous mandible, the embedded second premolar lies horizontal, with its crown usually facing distally. Ankylosis may be a complicating factor. The removal of the tooth is made difficult by (i) possibly extreme fragility of the atrophic mandible, (ii) the close relationship to the mental nerve, which may lie at the crest of the ridge, and (iii) the relation of the tooth to the inferior dental canal. Splints (of the Gunning type) should be arranged pre-operatively and the patient warned, if fracture of the mandible is thought likely. The patient should be warned of the possibility of mental anaesthesia or paraesthesia. If the mandible is surgically fractured, the tooth should be dissected carefully from the fragment, the minimum damage possible meted out to the mucoperiosteum, the splinting action of which must not be impaired, and the fragments should be immobilised by upper border wiring, and intermaxillary fixation (by Gunning splints or full dentures, combined with a barrel bandage).

When the second molar lies horizontally, and between the apices of the second molar and the inferior dental canal (a rare position, but one which has been reported), sacrifice of the second molar , surgical exposure of the second premolar with orthodontic traction(if possible), is the method of choice in the writer's opinion. Once the second premolar has assumed a more superficial position, it may be removed without damage to the inferior dental nerve and artery.

Extra-oral removal is the method of choice in the case of the deeply positioned inverted second premolar.

E. The Surgical Removal of the Mandibular Canine.

The mandibular canine may be embedded in the vertical position, and will then lie between the roots of the first premolar and the lateral incisor, somewhat to the buccal of the root arch. The technique of removal is similar to that employed in the case of the vertically impacted second premolar.

Thoma[133] stated that the unerupted canine is generally found embedded in the substance of the mandible on the outer aspect of the jaw ; the axis may be either oblique or horizontal. It is generally necessary to raise a buccal flap to include all teeth mesial to the second premolars. At least two-thirds of the tooth must be exposed before an elevator should be used to lift it from its socket. Severance of the crown and its removal, from the root is the method of choice in some cases.

F. The Surgical Removal of the Mesiodens.

The supernumary maxillary incisors may lie to the labial, or to the palatal of the arch formed by the roots. They may be single, or multiple - Orr (1959) reported four supernumaries in the midline. The surgical removal of supernumary incisors is generally combined with the surgical exposure of the permanent central incisors they impact.

Thoma[133] stressed the importance of exact localisation of the mesiodens before commencing the operation. This is not always possible, as he implied in the following statement :-

"In many cases an incision should be made on both sides : first, to determine the location of the tooth, and the easiest approach, and second, in order to push the tooth from one incision into the other rather than to attack it with elevators that may injure the closely packed roots of the incisors."

Details of the operative procedure.- Thoma recommended the wide U-shaped incision on the labial side, wide enough for the entire maxilla up to the anterior nasal spine to be exposed if necessary. On the palate a flap may be raised to the first molar level on each side, the incisive foramen neurovascular bundle being ignored. If placed labially, the tooth will come into view after removal of bone near the median suture. If located in the nasal spine, bone may be removed there, and inverted supernumaries erupting towards or into the nasal cavity may be reached by nibbling the bone of the anterior margin of the pyriform fossa.

If palatally placed, the supernumary incisor is generally apparent at the base of the alveolar process, immediately on raising the palatal flap. Thoma warned against the over-confident use of elevators on the palatally placed mesiodens, due to the close proximity of the tooth to the roots of the incisors ; he prefers to raise a buccal flap in addition, cut a bone window to expose the supernumary, and knock it through palatally by a suitable instrument tapped by a mallet.

Should the supernumary lie within the lingual concavity of the crown of the embedded central incisor, the supernumary should be carefully removed, and a bone-free path arranged for the eruption of the permanent tooth.

G. Other Unerupted and Impacted Teeth.

Other unerupted and impacted teeth, not specifically mentioned in this section, can be readily removed by application of the principles outlined above. In the removal of any unerupted tooth, the following principles should be observed :-

i. Exact localisation of the tooth by painstaking clinical and radiographic examination;

ii. Consideration must be given to the anatomical relations of the tooth to be removed, before the operation is commenced.

iii. The flap must be designed to give a generous exposure of the field of operation, and should conform to the specifications listed above.

iv. Bone removal should expose the crown of the embedded tooth and free its greatest convexity ; a bone-free path of withdrawal should be provided ; before an attempt is made to luxate the tooth, a space must be provided into which to displace the tooth. If the tooth cannot be removed in toto without damage to adjacent structures, it is usually preferable to sever the crown of the tooth at the neck, widen the gap between root and crown, in order that the latter may be lifted from its bed ; the elevation of the root into the space provided by the crown, is generally a simple matter.

PART VI

SURGICAL POSITIONING OF TEETH

A. Surgical Exposure of the Unerupted Tooth.

The aim of surgical exposure of the unerupted tooth is to create an artificial path of eruption, free of bone and soft tissue, in order that the tooth will erupt, either by its own inherent eruptive force, or by the aid of orthodontic traction. It is the responsibility of the orthodontist to consider whether eruption (as against surgical removal) is desirable, and to provide the necessary space for its accommodation in the arch; it is the surgeon's responsibility to determine pre-operatively, whether surgical exposure is practicable. Exact radiographic localisation of the unerupted tooth, and its relationship to the adjacent teeth, is essential - localisation is particularly important in the case of the unerupted canine.

The decision to surgically expose the unerupted tooth to promote its eruption, depends on the following factors, listed by Salzman (1943) :-

1. The degree of deviation of the unerupted tooth from its usual position in correlation to the general dental developmental state of the patient.

2. The distance of the unerupted tooth from the site into which it is to erupt.

3. The length of time past the normal eruption period of the unerupted tooth.

4. The adequacy of space into which the tooth is to erupt.

5. The presence of osteogenic or cystic disturbances, anomalies of the tooth itself or interference from the roots of adjacent teeth.

Norton (I954)[216] and Adamson (1952)[185] have published a useful classification of the positions of the unerupted maxillary canine, from the viewpoint of surgical exposure.

Helmore (1954)[216] and Strock (1938)[375] have published excellent material on the surgical techniques used to promote eruption.

Three main surgical methods have been used :

(1) Simple excision of overlying tissue.

(2) Raising of a muco-periosteal flap, removal of bone, stimulation of the tooth by slight instrumentation, and replacement of the flap. This technique is referred to as the "Stimulation Technique".

(3) Raising of a muco-periosteal flap, removal of the coronal sac, channelling of the bone, cutting a window in the flap, and, after re-suturing, packing to prevent healing over of the soft tissues. Thus, a cone-shaped opening is formed at the end of which the crown of the cuspid may be clearly seen.[216]

The first method fails because it does not allow access to the crown to ensure its freedom ; re-growth of the excised soft tissue occurs - with added impenetrability through scar formation - before eruption.

The second method is more useful, in that bone is freed, the position of the canine can be visualised (but necessitates the presence of the orthodontist at the operation), but fails in that soft tissue is not relieved, and orthodontic traction cannot be applied.

The third method is the technique of choice, as both bone and soft tissue are freed in the path of eruption, orthodontic appliances may be inserted at leisure after healing has occurred, and regrowth of soft tissue is prevented. Helmore and Strock both employ this technique, with slight individual modifications.

Operative Procedure (after Helmore)

1. A palatal flap is raised, from the distal of the ipsilateral second premolar, to the distal of the contralateral central incisor.

2. Bone is removed (preferably by chisel[216]) to expose the crown. The follicle is secured, and removed by scalpel and curette. Bone is removed to create a channel wider than the maximum diameter of the crown. The tooth is "stimulated", or wiggled gently with an elevator. The crown should be visible to the cemento-enamel junction.

3. The flap is replaced, the position of the crown in relation to the flap is determined, and a window is excised from the flap to ensure complete clearance of the crown of the cuspid. It is usually necessary to include in the window, the palatal gingival margins of the central and lateral incisors.

4. The flap is then replaced, and firmly sutured into position.

5. The channel around the canine crown in packed with a putty-like paste of zinc oxide and eugenol, reinforced with cotton wool fibres. A suture over the pack will ensure its retention; Helmore recommends the use of 00 catgut, which is left in situ for 6 or 7 days, when the pack is removed. By 10 days, the cone-shaped opening is epithelialised, after which orthodontic appliances may be employed (a platinum wire loop cemented into the crown, attached to an arch-wire by a finger spring).

Strock did not raise a flap, but excised adequate tissue overlying the crown, removed bone, and maintained the opening by cementing over the crown, a celluloid form, using an obtundant and antiseptic paste (camphor 6.5 parts, metacresol, 3.5 parts, petrolatum, 50 parts, white wax, 10 parts, and hydrous wool fat(lanolin), 30 parts).

The raising of a wide flap, in my opinion, is preferable to the mucoperiosteal excision recommended by Strock and Thoma, as better exposure is gained, bone can be removed adequately Without trauma to the soft tissue, and the exact relation of the canine to the incisors is better visualised.

This technique can be modified to suit teeth other than the maxillary canine. It is frequently necessary to expose unerupted premolars, which are impacted by the bone scar incurred by premature loss of the deciduous molars. A buccal flap is raised, the lingual mucoperiosteum retracted, in order that the alveolar ridge can be visualised. The greatest diameter of the crown is exposed (care in particular, being taken to free the lingual cusps of the lower premolars) and a bone-free channel furnished. The tooth is stimulated, and the buccal and lingual flaps trimmed to leave the crown exposed. The opening may be maintained until epithelialisation occurs, by either a zinc oxide pack, or tulle gras sutured into position.

B. Surgical Positioning of Unerupted and Impacted teeth.

This practice has been referred to in recent literature as "Surgical Orthodontics". The principle involved is the correction of the angulation of the impacted tooth, by bone removal and tilting of the developing tooth, without destroying its blood supply in the process. Holland (1956)[60] presented reports of several successful cases treated in this manner. The operation is applicable to unerupted canines which would not erupt by surgical exposure alone, and in particular to the disto-angular unerupted and impacted lower second premolar.

It is essential to select cases for surgical correction of axis carefully. The age of the patient, the stage of development of the tooth, and the displacement necessary to position it correctly must be considered. A tooth with incomplete apex and large apical foramen may be removed from its crypt and transplanted because such teeth may be revascularised if detached from their blood supply[133]; it is obvious therefore, that repositioning of the tooth within its crypt is unlikely to damage is blood supply significantly. On the other hand, if the apex is fully formed, only slight rotation is permissable without damage to its blood supply ; with apex almost formed, very little traction of the apical base is tolerated. Gentle handling is essential : should the epithelial sheath of Hertwig be distorted, dilaceration of the root follows, and may prevent vertical eruption.

The technique is diagrammatically illustrated below :-



Pre-operative radiograph.



Extraction of deciduous molar



The unerupted tooth is freed of mesial bone



The premolar surgically positioned

C. Transplantation of the Third Molar.

Tam (1956)[376] stated : "Since the third molar often is of no functional value in young adults, its use in selected cases as a transplant to replace the first molar, which is commonly lost due to caries, appears logical. This is especially true for this age group when crown and bridge restoration for missing teeth is generally not advisable." Apfel[377] felt transplantation of the prefunctional third molar to be the answer to early first molar loss, with its serious sequella of arch collapse. The usefulness of the procedure, I feel, is limited to the rare case, as it depends on the co-incidence of carious destruction of the first molar at an age when the third molar has reached a specific stage of root development.

The principles of the operative technique are :

(1) The third molar to be transplanted must be narrower than the first molar it is to replace ; the crown must be completed ; the root must be developed to the bifurcation in the lower molar, to the trifurcation in the upper molar. The recipient site must be free of infection, of bone and of soft tissue.

(2) The third molar tooth germ must be removed without trauma. This is no mean task, as the developing third molar lies deeply embedded in bone, which must be generously excised, before the tooth can be lifted from its crypt. The follicle must not be damaged, and it is essential that the dentine papilla, which contains the delicate epithelial sheath of Hertwig (the blueprint of the future root), must not be distorted. Apfel[377] advocated the inclusion, in the transplant, of the overlying mucosa, with the gubernaculum and the perifollicular tissues. Tam[376], however, found that the wedge of oral mucosa underwent necrosis in his cases, and on these grounds questioned the advisability of this procedure. In my opinion, two advantages accrue from inclusion of the small triangular wedge of oral mucosa : (i) the superior surface of the transplant is marked, and this precludes the possibility of inverting the tooth in the first molar socket (such occurrences have been reported) and (ii) the oral mucosa overlying the third molar can be sutured to the mucoperiosteum of the first molar region.

(3) The first molar is extracted, and the inter-radicular septum is excised to the base of the sockets. The third molar is placed in the prepared socket, and care must be taken that the buccal aspect of the third molar is correctly positioned. Gelatine sponge may be packed over the tooth, the buccal and lingual gum flaps closely approximated and sutured. To free the mucoperiosteal flaps, buccal and lingual alveolar plates should be reduced before the third molar is inserted.

Agnew and Fong (1956) published an excellent report of their histological studies of experimental transplantation of teeth in rhesus monkeys.[378] At four days, the pulp showed significant changes : the tissue remote from the apices showed signs of degeneration (oedema, fine fibrillar zones, areas of degeneration, thrombosed vessels, odontoblasts atypical and few); near the apices, pulpal tissue was viable (murtured by diffusion), and the odontoblastic layer was intact and correctly aligned. Beneath the apices, healthy granulation tissue had appeared. New bone formation was seen in progress on the bony walls. At 4 weeks post-operatively, the prepared crypt showed new trabeculae of bone; periodontal membrane was normal and cellular, and attaching cementum to bone; pulp showed revascularisation, the fibrillar zones having been converted to fibrous tissue; new dentine formed remote from the apices was non-tubular (osteodentine), but relatively normal at the apical region. At 4 months, little increase in root length could be noted; the pulp chamber was filled with calcified tissue - osteodentine, bone, cementum, and tubular dentine; the pulpal face of the roots showed a gradation from normal pre-operative dentine, to osteodentine, irregular dentine, tubular dentine, and a healthy odontoblastic layer; eruption was proceding normally despite significant root length increase.

Fong (1953)[133] listed the criteria for estimating the success of transplantation of teeth : (1) gingival tissues should be normally attached to the tooth; (ii) the tooth should be of normal colour, without areas of hypoplasia, or deficient calcification of the enamel; (iii) the tooth should have normal articulation and should be free of abnormal mobility; (iv) pulpal behaviour should be within normal limits ; a radiograph should show normal root formation, a discernible periodontal space and a lamina dura, as well as a normal alveolar crest. Thoma[133] pointed out that while the vitality may be

normal, the neurility may not be, depending on whether the nerves have grown into the pulp ; the obliteration of the pulp chamber by osteodentine, will of course, dull the effects of heat and cold. If the tooth fails to erupt, it should be removed, as it probably is non-viable, or ankylosis has occurred. Dilaceration of one or more roots can follow distortion of the epithelial sheaths of Hertwig.

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