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Contributors

Colyer, Frank, 1866-1954.
Colyer, Stanley (Stanley William Randolph)

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DENTAL DISEASE
IN ITS RELATION TO
GENERAL MEDICINE

J. F. COLYER AND STANLEY COLYER

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IN ITS RELATION TO GENERAL MEDICINE.

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IN ITS RELATION TO

GENERAL MEDICINE

BY

J. F. COLYER, L.R.C.P., M.R.C.S., L.D.S.,

Dental Surgeon to Charing Cross Hospital and the Royal Dental Hospital; Member of the Board of Examiners in Dental Surgery of the Royal College of Surgeons, England;

WITH THE ASSISTANCE OF

STANLEY COLYER, M.D.Lond., M.R.C.P., D.P.H.

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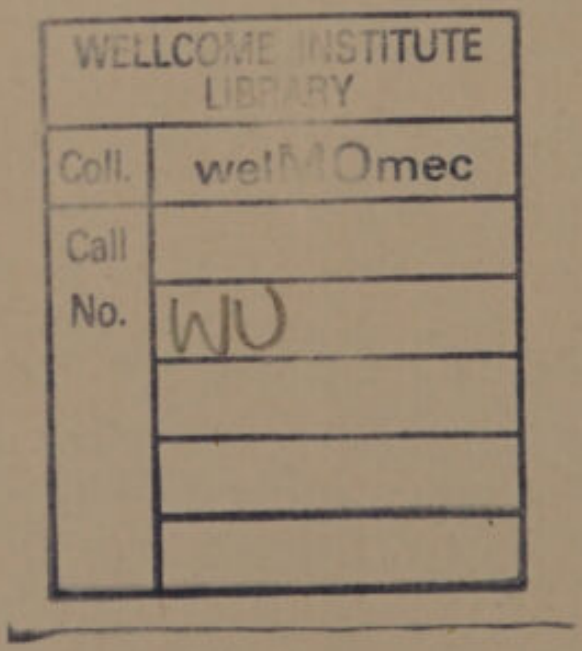


PREFACE.

THIS little book has been published with the view of placing before practitioners of medicine those portions of odontology which have a direct bearing upon general medicine. In compiling it I have drawn largely upon the recent edition of my text-book on Dental Surgery and Pathology.

The two most important subjects, dental caries and chronic general periodontitis, have been treated in considerable detail. As regards dental caries, the recent advances made in the knowledge of its etiology and a more intimate acquaintance with its clinical aspects have convinced me that it can now be regarded as a preventible disease.

A list of papers for reference has been added to the chapters dealing with the more important subjects.

I have to acknowledge my indebtedness to Dr. Stanley Colyer for his valuable assistance, more especially in the chapters dealing with dental caries and oral sepsis.

J. F. COLYER.

CONTENTS.

CHAPTER I.

CHAPTER II.

CHAPTER III.

CHAPTER IV.

CHAPTER V.

CHAPTER VI.

CHAPTER VII.

CHAPTER VIII.

CHAPTER IX.

CHAPTER X.

Dental Disease in its Relation to General Medicine.

CHAPTER I.

Dentition—Normal and Pathological.

(A) THE ANATOMY OF DENTITION.

In the human subject the earliest manifestations of teeth are the enamel organs for the deciduous teeth, which appear during the seventh week of fœtal life, followed during the ninth week by the dentine bulbs. At the twentieth week calcification commences with the central and lateral incisors, and at the twenty-fourth week the canines and deciduous molars begin to show signs of calcification.

At birth calcification of these teeth has advanced to the stage shown in fig. 1, and has also commenced in one of the anterior cusps of the first mandibular permanent molar.

At the age of eight months the growth of the deciduous incisors and canines has progressed to the stages shown in fig. 2. The roots of the first molar are commencing to form and the crown of the second molar is almost complete. In the permanent teeth calcification has commenced in the incisors and the canines and is well advanced in the cusps of the first molars.

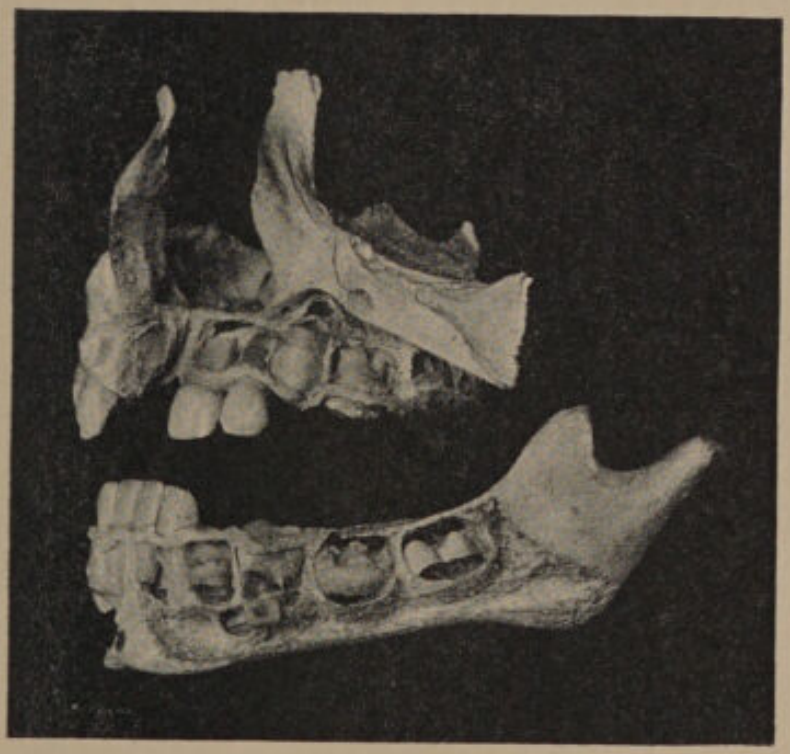
Birth.

FIG. 1.

Eight months.

FIG. 2.

At the age of twelve months (fig. 3), the root of the deciduous canine has commenced to form, growth in the first deciduous molar has progressed beyond the division of the roots, and in the second molar almost up to the division. The deciduous incisors are fully erupted, and eruption of the first molars has commenced. In the permanent teeth, about half of the crown of the first molar is formed, the upper third of the crown of the central incisor, about one-fifth of the lateral, and the tip of the canine.

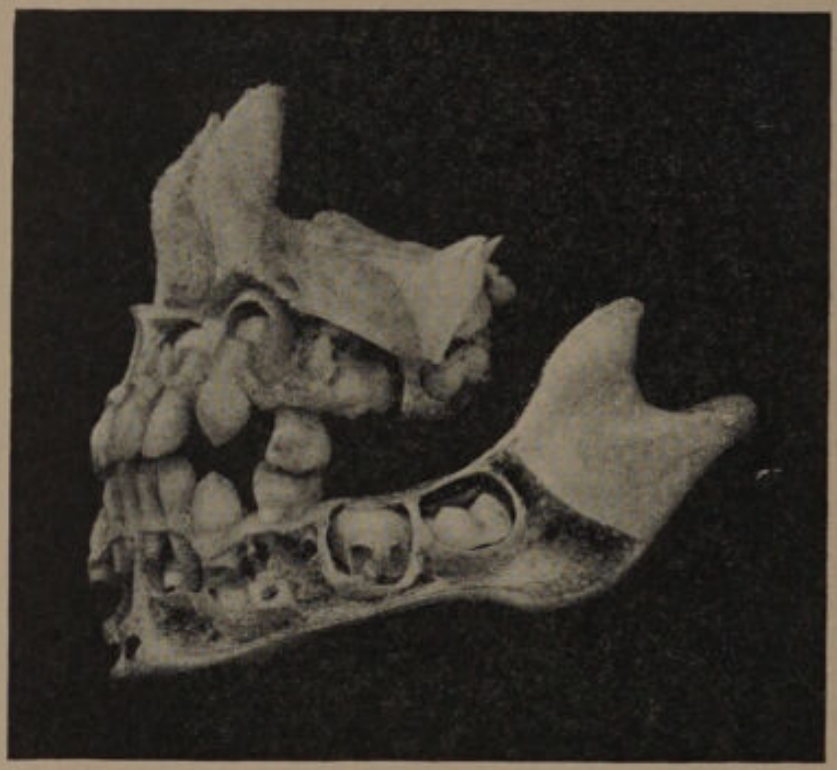


Twelve months.
FIG. 3.

Fig. 4 shows the jaws of a child aged *fourteen months.* The first molars are in place, and the canines are partially erupted.

At the age of eighteen months the roots of the central and lateral incisors of the deciduous set are almost

complete; about two-thirds of the roots of the canines, practically the whole of the roots of the first molars, and about half the roots of the second molars are calcified. In the permanent teeth calcification has advanced, as shown below:—



Fourteen months.
FIG. 4.

The central incisors about $\frac{3}{4}$ths of the crown.

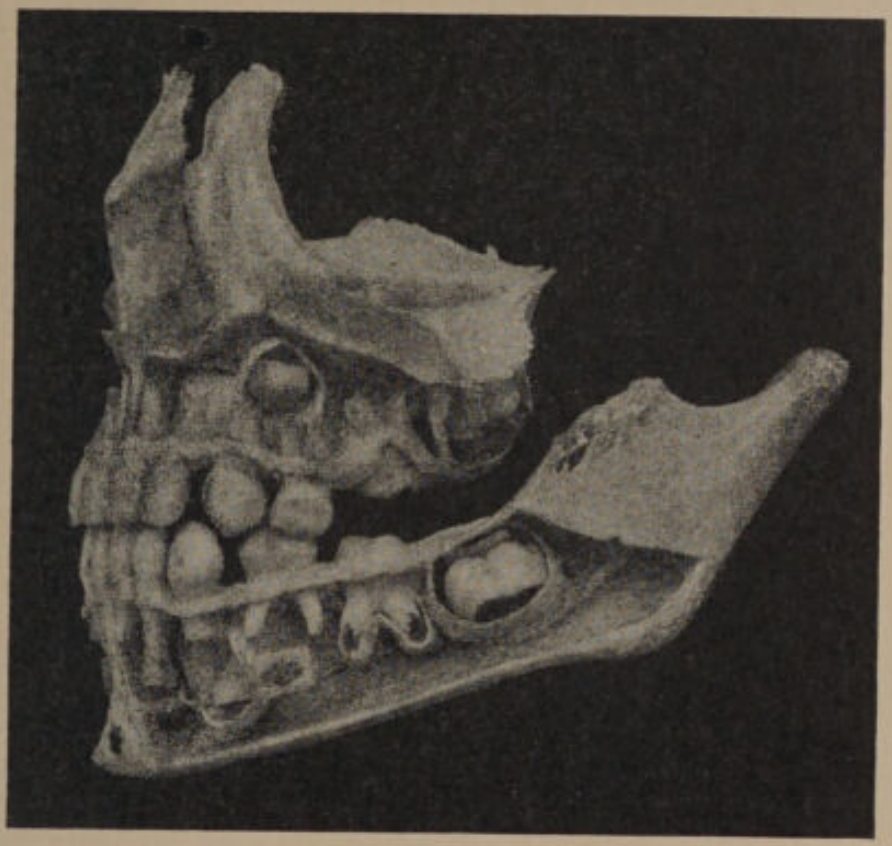
The lateral incisors (maxillary) about $\frac{1}{4}$th of the crown.

The lateral incisors (mandibular) about $\frac{2}{3}$rds of the crown.

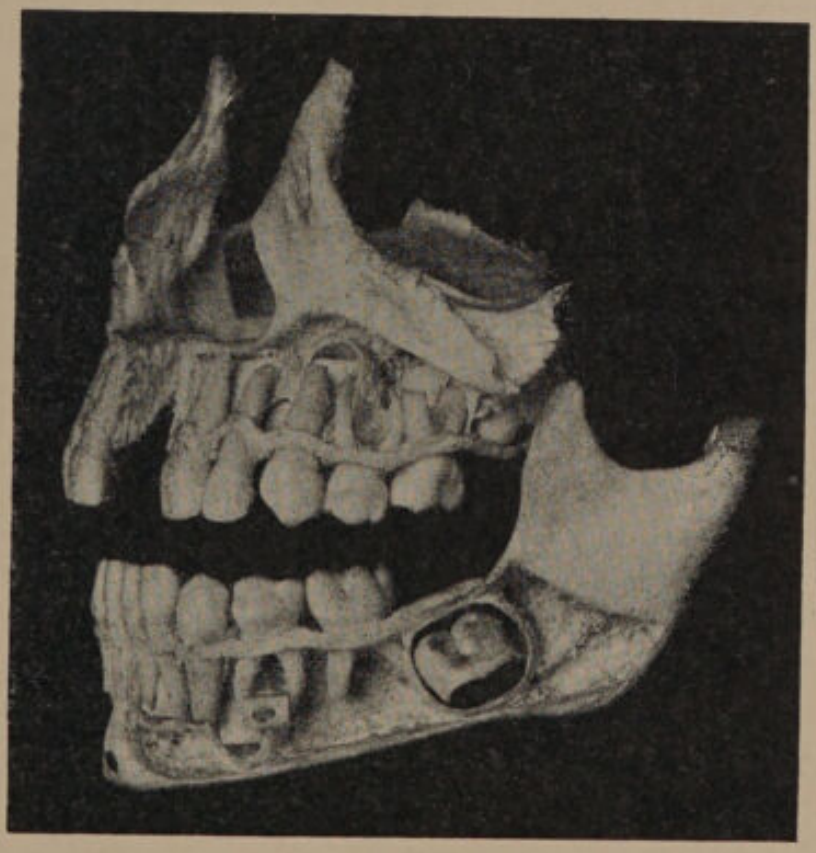
The canines about $\frac{1}{2}$ of the crown.

The first molars a little over $\frac{3}{4}$ of the crown.

At two years of age (fig. 5), the formation of the deciduous teeth is complete, with the exception of the terminal portions of the canines and second molars. In



Two years.
FIG. 5.



Three years.
FIG. 6.

the permanent series the crown of the first molar is fully formed, and calcification has advanced in the incisors and canines, but the crowns are not complete.

At three years of age (figs. 6 and 7), the deciduous dentition is complete and fully erupted. Of the per-

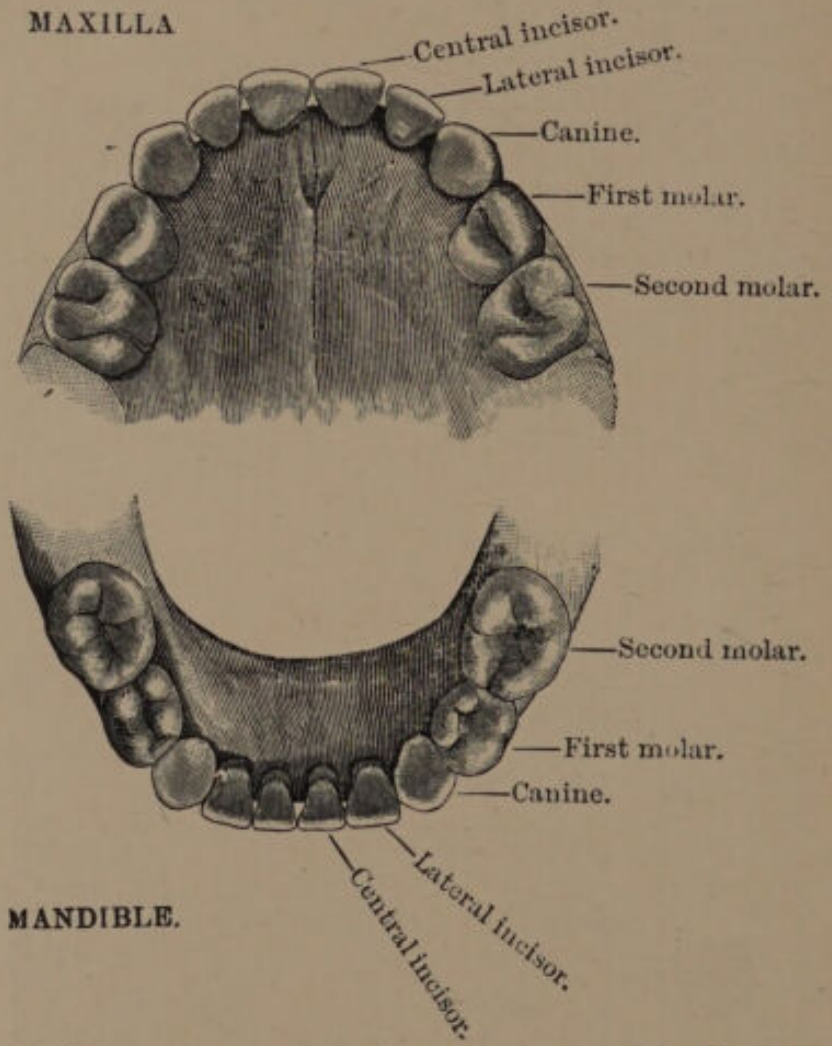
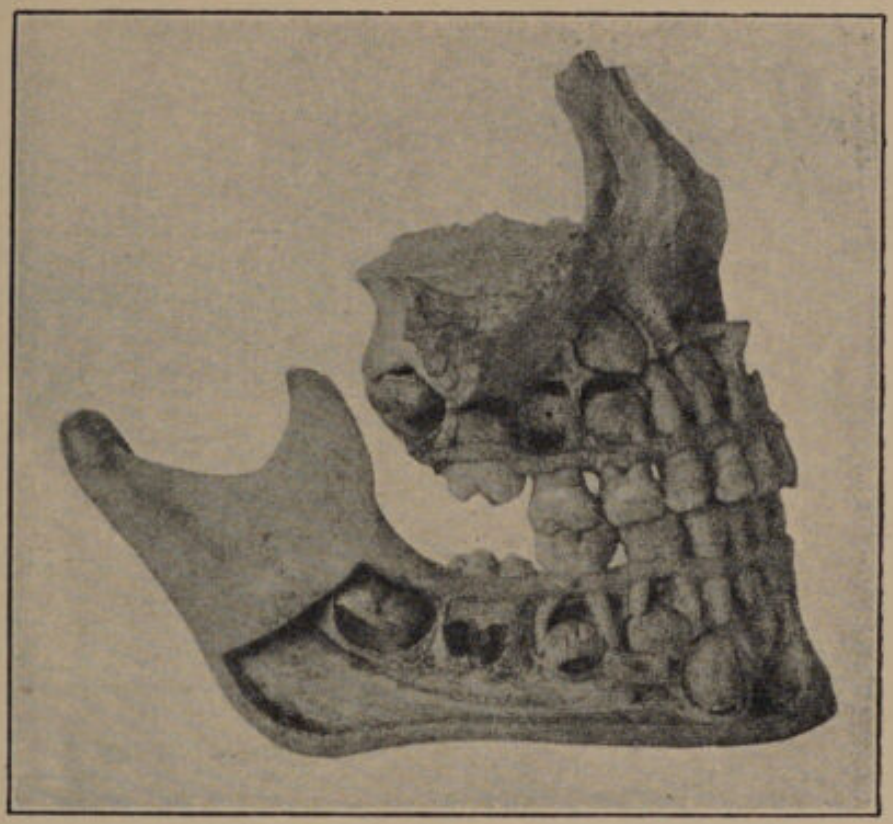


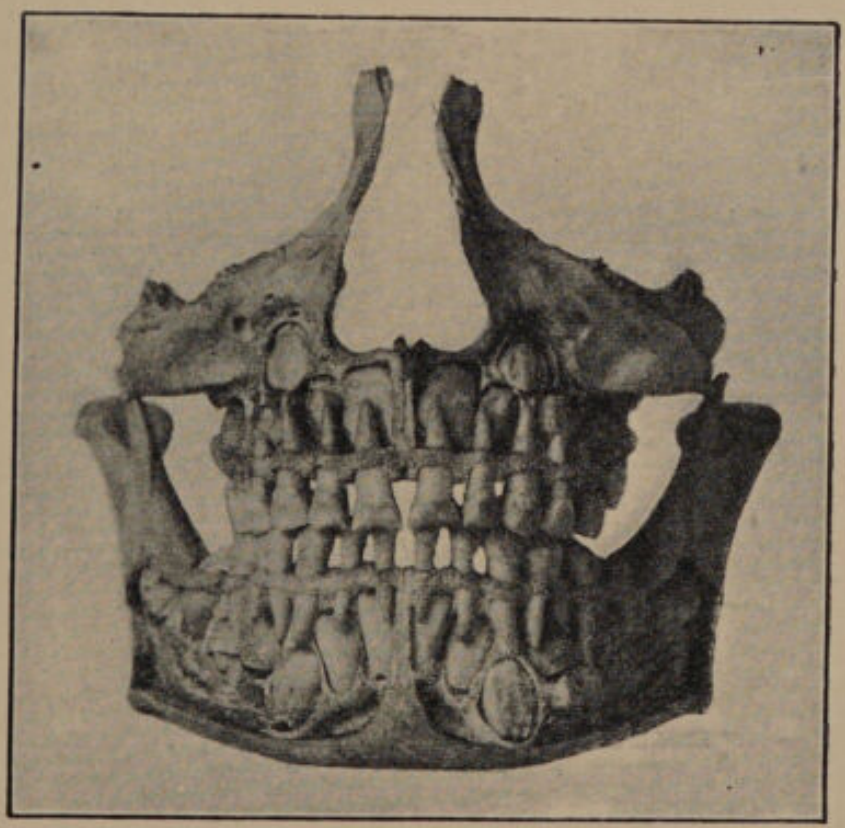
FIG. 7.—Represents a fully-developed set of deciduous teeth.

manent teeth the crowns of the first molars, and the incisors are fully formed. Calcification has commenced in the first mandibular premolar, and is beginning in the corresponding maxillary tooth.

At four years of age calcification has commenced in the second premolars and in the second permanent molars.



Six years.
FIG. 8.



Six years.
FIG. 9.

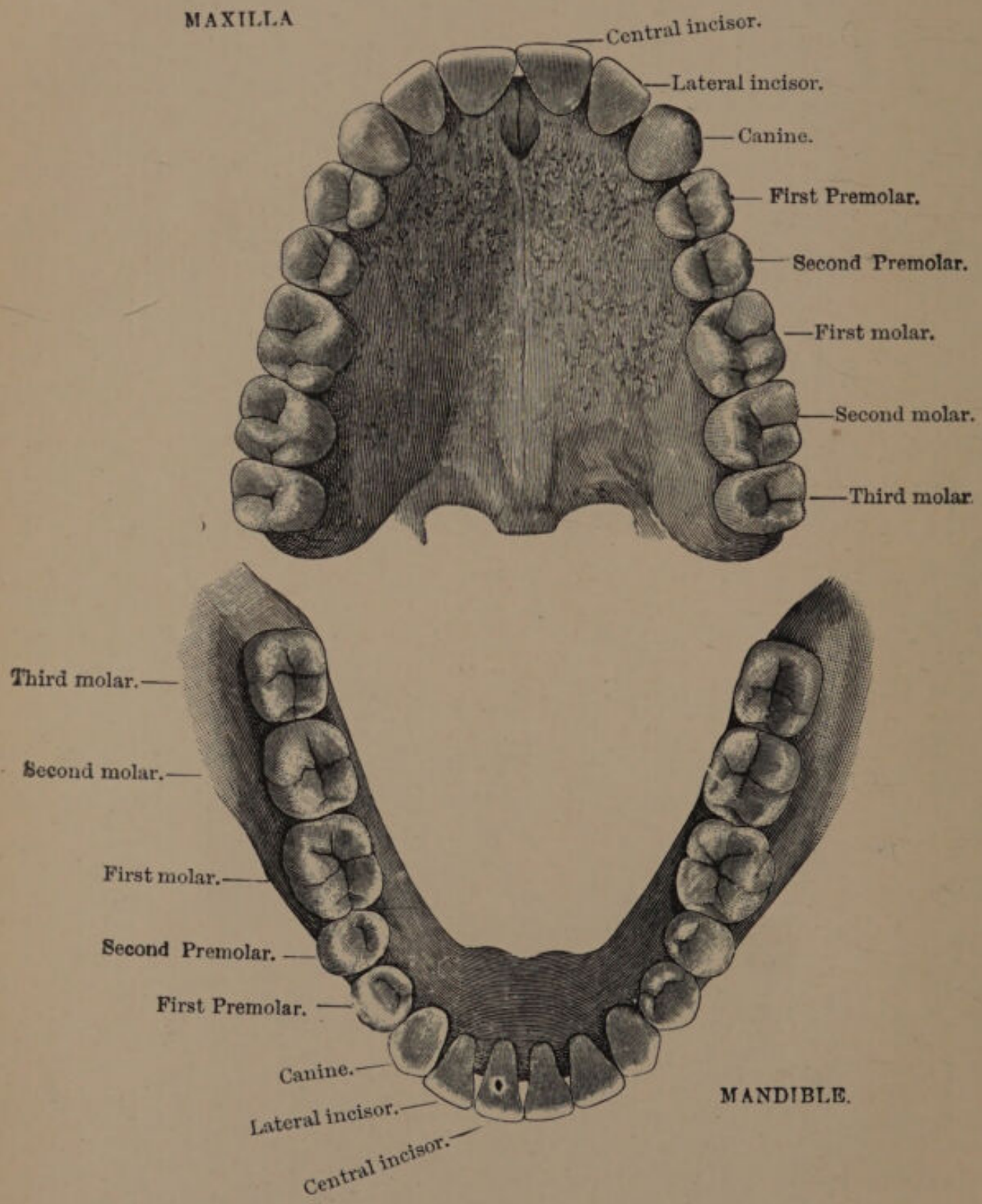


FIG. 10.—Represents a fully-developed set of permanent teeth.

At six years of age the permanent dentition is in the state shown in figs. 8 and 9. The crowns of the premolars are not yet fully formed, but the calcification is nearer completion in the first premolar than in the second.

The roots of the first permanent molars are also partially formed, and the crowns of the second molars

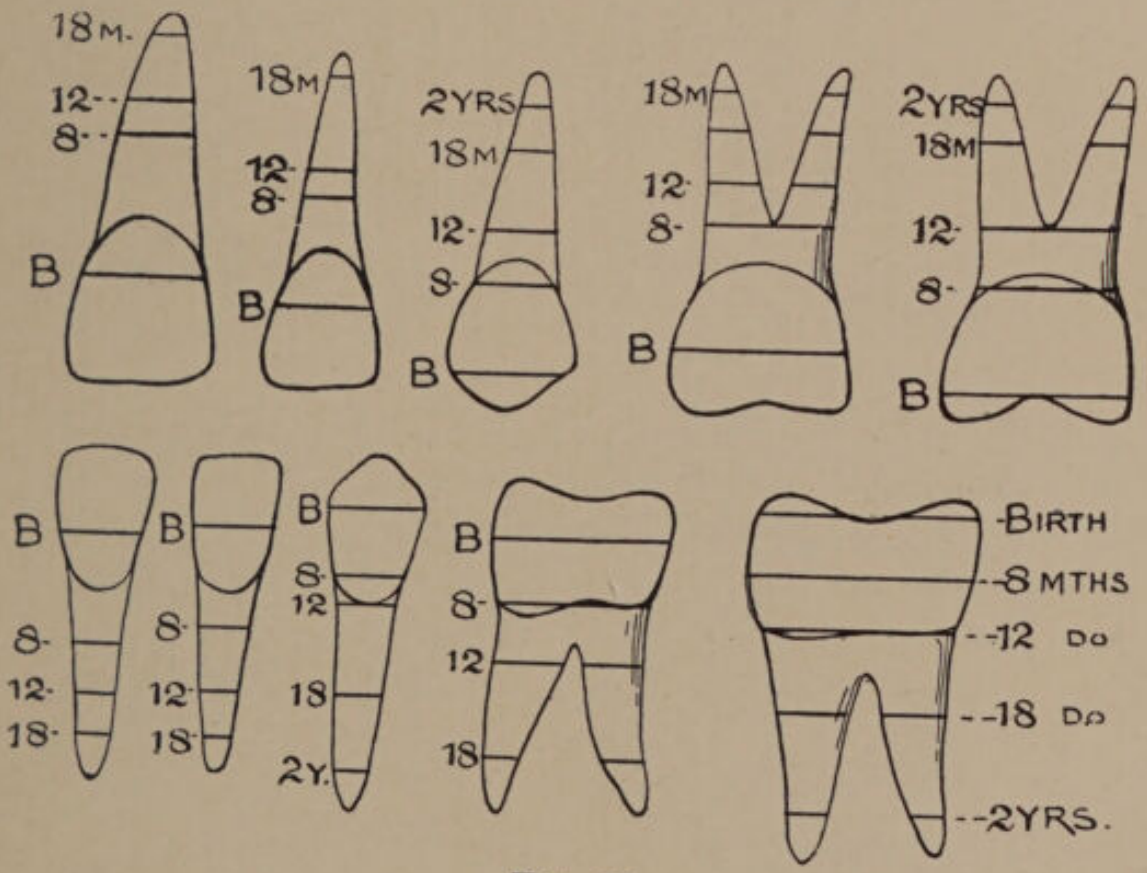
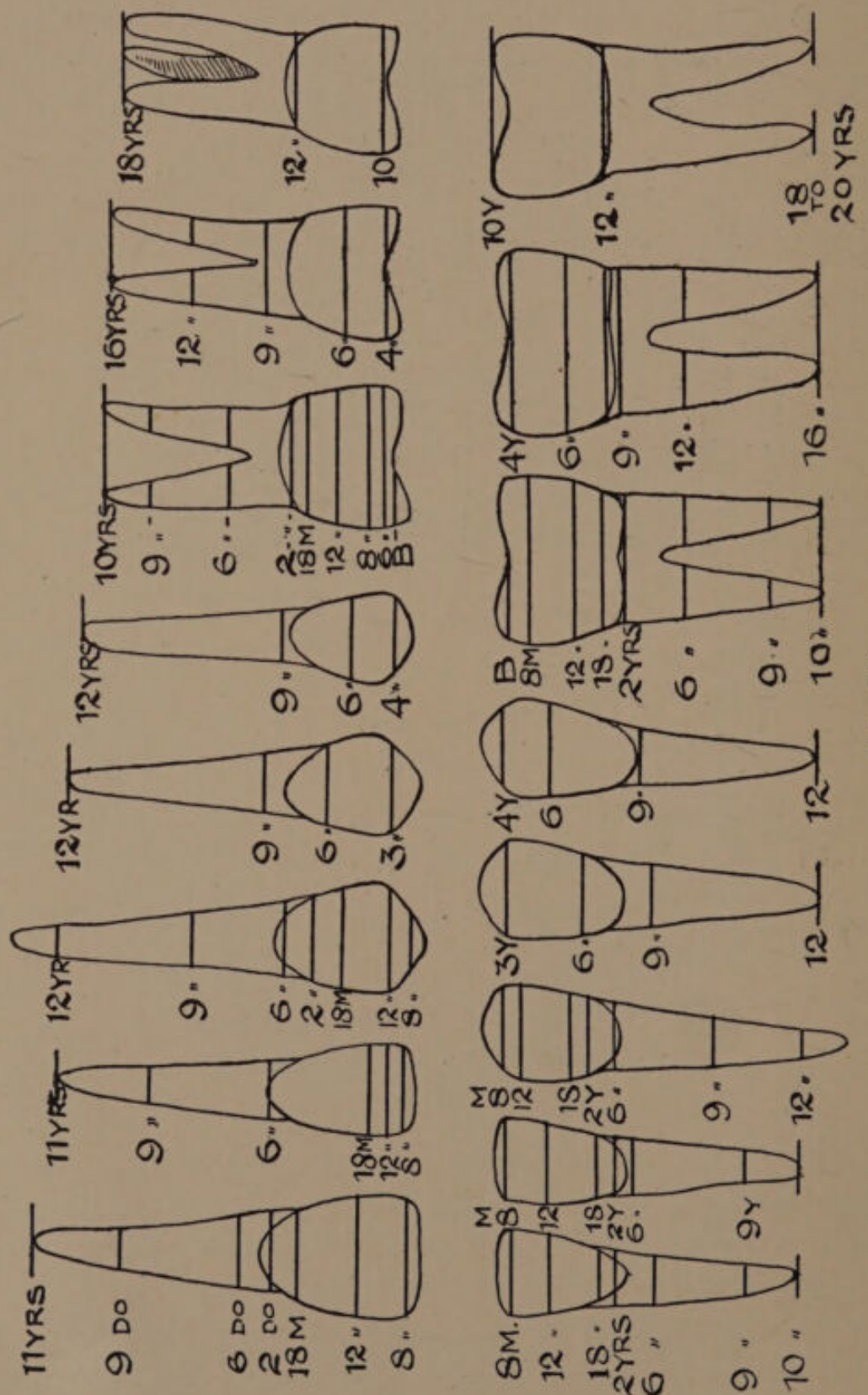


FIG. 11.

are about half formed. The roots of the incisors have commenced to form, but the crown of the canine is barely complete. The roots of the deciduous incisors show signs of absorption on the posterior aspects.

At about the age of six years, the first permanent molars make their appearance, and during the following six years the deciduous teeth are gradually replaced by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6"
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12"
16"
10Y
12"
18
TO
20YRS

FIG. 12.

their permanent successors. The second permanent molars erupt at about the age of twelve years, and with the appearance of the third molars the second dentition is complete, and should present the appearance shown in fig. 10.

In the process of dentition the periods of greatest activity are during the early years of childhood. This fact cannot be too strongly impressed upon parents by those responsible for the medical care of children. The diagrams shown in figs. 11 and 12 may be useful to illustrate the amount of calcification of the deciduous and permanent teeth at various age periods.

(B) THE ERUPTION OF THE TEETH.

The normal signs of approaching eruption are an increased flow of saliva, and a tendency on the part of the child to bite at any tangible object. A healthy child will often cut its teeth without the slightest sign of discomfort, but more often the gums become a little tumid, tense, and shining over the erupting tooth, the local condition being accompanied by slight irritability, restlessness, and a rise of temperature.

The order and date of eruption vary considerably within normal limits. Reliable statistics as to the order and time of eruption of the deciduous teeth are not available. The following dates, however, may be taken as approximately correct:—

Mandibular central incisors—fifth to eighth month.

Maxillary central and lateral incisors—seventh to tenth month.

Mandibular lateral incisor—tenth to twelfth month.

First molars—twelfth to fourteenth month.

Canines—fourteenth to twentieth month.

Second molars—twentieth to thirtieth month.

The teeth erupt as a rule in pairs or groups, and between the eruption of each group there is an interval varying from two to five months, the interval becoming greater between the later groups.

Eruption of teeth before birth is extremely rare. When this occurs the teeth are usually imperfectly developed and loosely attached to the muco-periosteum, but occasionally they are well formed and attached to normal alveoli.

With the permanent teeth, as with their predecessors, there is considerable variation as regards both the order and the time of eruption.

From clinical experience it would seem that the first permanent molars and the incisors erupt in somewhat rapid succession, and that their eruption is followed by a distinct period of rest often extending over two years. After this rest another period of eruption occurs, and the premolars, canines and second molars appear. The approximate order and date of eruption of the permanent teeth are shown below.

Mandibular first molars Maxillary ,, ,, Mandibular central incisors Maxillary ,, ,, Mandibular lateral ,, Maxillary ,, ,,	6 to 8 years.
Maxillary first premolars ..	10 to 11 years.
Mandibular ,, ,, Maxillary second ,, Mandibular canines	11 to 12 ,,
Mandibular second premolars Maxillary canines Mandibular second molars Maxillary ,, ,,	12 to 13 ,,
Third molars	20 to 25 ,,

The time of eruption of the third molars depends, to a great extent, upon the amount of space in the arch. The early removal of second molars will accelerate the appearance of the third molars, which may erupt as early as the fifteenth year, and early removal of the first molars may produce the same effect.

In children suffering from rickets the eruption of the teeth is sometimes considerably retarded, and it is not uncommon for the advent of dentition to be delayed until the commencement of the second year. Retarded eruption of the permanent teeth is also met with in those who have suffered from rickets in infancy.

The general effect of syphilis on eruption is to delay the process, but in a few instances syphilis may accelerate eruption.[1]

Idiocy.—The teeth of microcephalics and cretins are usually small, and eruption is considerably delayed. In cretins the eruption of both the deciduous and permanent dentition is often delayed, and may also commence irregularly, though in the subsequent growth of the permanent series the trend is toward eventual perfection of the dentition.

It is interesting to note that the administration of thyroid extract, which assists growth in general, also promotes the eruption of the teeth. The administration of thyroid extract has no effect on the dentition in cases where it effects no general improvement.[2]

---

[1] See article by Dr. Cavallaro, on "Syphilis in its Relation to Dentition," *Cosmos*, November, 1908, to February, 1909.

[2] "Teeth of Microcephalics and Cretins," by J. G. Turner *Trans. Odonto. Soc.*, vol. xxxiv., p. 1.

(C) DISORDERS ASSOCIATED WITH THE PROCESS OF TEETHING.

(1) General.

Certain general disorders frequently occur about the time of teething, and considerable diversity of opinion exists as to exact relationship between these disorders and the teeth. There can be little doubt that formerly illnesses at this period were too readily regarded as a natural sequence to the process of dentition, while there now seems to be a tendency to ignore altogether the possibility that dentition and general disorders are related to each other as cause and effect.

In endeavouring to arrive at the actual facts of the case it is well to remember that weakly children, and more especially those that have been hand-fed, are prone to general disturbances, and that it is probable that local trouble in the mouth would tend to aggravate such conditions. Again, general disturbance occurring during teething is often wholly traceable to other causes —for example, gastro-intestinal troubles and convulsions may arise from defective feeding, and respiratory affections and otitis media may be due to the presence of adenoids. But after eliminating these cases there still seem to be instances in practice where the general disturbance is coincident with dentition, and there is no known cause other than dentition with which to associate it. There is naturally extreme difficulty in placing dentition in causal relationship with such disorders, because we have to rely mainly on clinical observation; but it seems only reasonable to assume that such a relationship does exist when the occurrence of a disorder not only coincides wlth dentition but has a special tendency to coincide repeatedly with the eruption of successive teeth.

In referring to this question Dr. Still[1] expresses the opinion that there is too great a tendency to assume that, as dentition is a physiological process, it is therefore incapable of causing disturbance of health. He points out that pregnancy is also a physiological process, and no one would deny that pregnancy may disturb the health in many ways. Dr. Still states that with increasing experience he is inclined to think that the *rôle* which dentition plays in the production of disturbances of various kinds in infancy is greater than is generally supposed.

Clinical observation clearly establishes the fact that irritation often exists in the mouth during the period of eruption. In most cases the irritation is due to direct pressure of the tooth upon the superincumbent tissue. Dr. White, however, points out that the aperture at the end of the growing tooth is large, and undue resistance of the gum tissue would lead to pressure on the nerve trunks entering the pulp. Dr. White supports his view by clinical evidence afforded by cases in which there are no local signs of inflammation and in which constitutional disturbance disappears when the gum is lanced.

The following general disorders may, in Dr. Still's opinion, *be traceable to dentition* :—

(*a*) Loss of appetite.

(*b*) Sleeplessness.

(*c*) Instability of the nervous system. Infants troubled with coming teeth often show a tendency to convulsive twitchings, upward rolling of the eyes, slight strabismus, although not losing consciousness. In children of neuropathic heredity, or, through rickets,

[1] In compiling this section I have been greatly assisted by Dr. G. F. Still's work on "Common Disorders and Diseases of Childhood."

predisposed to infantile convulsions or epilepsy, dentition appears to be distinctly a time of peril. Dr. Still has seen attacks of petit mal which were much aggravated by dentition and ceased when the last tooth was cut.

(*d*) Rise of temperature. This may occur without the presence of an inflamed condition of the gums, and in most cases is probably purely reflex and analogous to not uncommon cases in which neurotic children will show a rise of temperature with any excitement, such as a visit to a pantomime; the rise of temperature with the worry of an erupting tooth is thus part of the induced instability of the nervous system.

(*e*) Attacks of vomiting sometimes occur, with little or no disturbance of the bowels, just before the appearance of a tooth.

(*f*) Bronchitis and diarrhœa. Although children are prone to these conditions, there are cases, nevertheless, which seem to be distinctly nervous and traceable to reflex influence from the teeth. That such a condition could exist is quite conceivable, seeing that the bronchial catarrh of the asthmatic child and the lienteric diarrhœa (the nervous diarrhœa of Trousseau) are admittedly dependent upon nervous influence, often quite remote from the organs affected.

(*g*) Violent screaming attacks without obvious cause, independent of any digestive disorder and not apparently due to any pain, are sometimes associated with dentition. This condition is, however, uncommon.

(*h*) A rare disorder which is closely related to dentition is spasmus nutans, or head-nodding with nystagmus. This disorder is so distinctly coincident with dentition in its onset and cessation, and is so definitely aggravated by the eruption of a fresh tooth, that there would seem to be a causal relationship between the two.

(2) LOCAL.

Simple Stomatitis.—The stomatitis is usually limited to the neighbourhood of the erupting tooth, and is characterized by intense redness and swelling, the mouth at the same time being hot, the child fractious, restless, and in evident pain. The temperature may reach 104° or 105° F.; but it should be remembered that pyrexia in children readily supervenes upon slight causes.

Ulcerative Stomatitis.—In these cases the gums become hot, swollen, and painful, and these symptoms are especially marked over a certain tooth. Ulceration supervenes, and may extend to the gum around any other tooth already erupted. The ulcers thus formed have a sloughy appearance, the breath is fœtid and hot, the flow of saliva is increased, and the child rejects its food. In addition there is marked pyrexia and at times gastro-intestinal disturbance. Ulcerative stomatitis is due to local infection and may often be traced to dirty feeding bottles or neglect of oral hygiene. It is interesting to note that as greater care in such matters is exercised in dealing with oral hygiene the local disturbances associated with dentition decrease.

Treatment.—Treatment of the local condition consists in giving strict attention to the hygiene of the mouth and the application to the gums of hydrogen peroxide; a brisk purge should also be given. In cases where the ulceration shows signs of spreading, the application of an escharotic is advisable. The patient should be isolated.

Superficial Cysts.—Small cysts are occasionally met with over erupting teeth. They do not in any way obstruct eruption, and, on being punctured, emit a small amount of clear fluid.

The local disturbances accompanying eruption of the

permanent teeth are mainly confined to the third molars, especially the mandibular. When a molar tooth erupts the anterior cusps appear first, and the small portion of gum which lies over the posterior part of the tooth occasionally ulcerates from constant pressure of the antagonizing tooth. The ulceration may become extremely painful and the adjacent tissues may become involved. The patient complains of pain in the region of the fauces, but perhaps the most tender point is where the mucous membrane of the gum becomes continuous with that of the cheek. This condition is best relieved by free incisions, care being taken that the knife divides all the tissues overlying the buccal surface of the tooth. Should this treatment not bring relief, it is advisable to remove the portion of gum that is covering the tooth; this can be accomplished with the scalpel and forceps. In addition, fomentation of the mouth inside should be advised, and hot water at a temperature just bearable is perhaps as comforting an application as any, although decoction of poppy-heads is often to be recommended, the opium contained in the poppies being supposed to act as an anodyne. The ulcerated surface should be freely swabbed with hydrogen peroxide. Suppuration may supervene, and in that event the offending tooth must be removed, otherwise trismus may occur. The trismus is said to be produced in most instances by spasm of the masseter muscle, due to reflex irritation; but it is more than probable that it is generally caused by spread of inflammation to adjacent tissues.

In connection with the subject of pathological dentition the discussion on "Teething and its Alleged Troubles" (*Brit. Med. Journ.*, August 22, 1908) is well worthy of perusal.

CHAPTER II.

Conditions which Influence the Growth of the Jaws and the Formation of the Teeth.

(A) THE NORMAL GROWTH OF THE JAWS.

BEFORE entering into a description of the conditions which interfere with normal growth, a brief reference may with advantage be made to the manner in which the jaws develop.

At birth the tooth germs in the maxilla lie in close relationship to the orbital plate, the antrum being represented by a slight depression internal to, and immediately above, the follicle of the second deciduous molar. The crypts of the incisors and canines are complete, but between the first and second molars the septum is still imperfect. The lateral incisor lies slightly posterior to the central incisor, and the crypt of the first molar is in close proximity to it, the canine lying between and anterior.

As the time for the eruption of the incisors approaches, the space between the lateral incisor and the first molar increases, the growth of bone at this point continuing until the eruption of the canine. There seems to be some reason for supposing that room is made in the arch for the canine by growth of bone at the suture between the premaxilla and the maxilla.

With the eruption of the second molars the deciduous

dentition is complete. An examination of the mouth at this stage will show an interval between the first molar and the end of the alveolar ridge. This interval increases in size until the advent of the first permanent molar. A skull examined at this period, namely, about six years of age, would show the following conditions: In the maxilla the lateral incisors are placed slightly posterior to the central incisors, and are directed more vertically. The premolars are embraced by the roots of the deciduous molars and their crowns are directed inwards, the second more than the first. The first premolar is normally situated close to the lateral incisor. The canine lies above and external to the arch of the incisors and premolars, and is directed slightly outwards. The first permanent molar will be in the process of erupting, and the occluding surface will be directed outwards to a slight extent. The second permanent molar is situated high up in the tuberosity of the bone, with its occluding surface directed downwards, outwards, and well backwards.

In the mandible the lateral incisors lie in a plane posterior to the centrals, and the canines are placed near the lower border of the bone and lie in a plane anterior to the lateral incisors, with a slight tilt towards the median line. The premolars are embraced by the roots of the deciduous molars, and their crowns are directed inwards. The first permanent molars are directed upwards and forwards, the second being under the base of the coronoid process with the occluding surface directed upwards, forwards, and slightly inwards.

Now, in order that a regular arch may result from this somewhat chaotic arrangement of the developing teeth, it is necessary that the growth of the jaw should continue without interference.

Let us consider, first of all, the replacement of the deciduous teeth by their permanent successors. The permanent teeth are broader than the deciduous teeth, and they are accommodated in the arch partly by assuming a more sloping direction than their deciduous predecessors, and partly by fresh growth of bone at the suture between the premaxilla and the maxilla.

The permanent canine is larger than the deciduous canine, but the premolars are smaller than the deciduous molars, the increase in size of the permanent canine being counterbalanced by the decrease in the premolars. The growth of the maxilla backwards provides room for the permanent molars. The growth of the maxilla has been shown by Keith to be dependent on the expansion[1] of the maxillary antrum. At birth the antrum consists of a slight depression just above the follicle of the second deciduous molar. This depression spreads towards the orbital plate, and at the end of the first year has developed forwards over the first deciduous molar, and backwards over the first permanent molar, an extension of the sinus rapidly taking place as the remaining molars are formed.

The effect of the growth of the antrum on the eruption of the teeth can be studied by examining the relation of the teeth to the antrum in a sixth-year skull. The first permanent molar lies in close relationship to the sinus, with the occluding surfaces looking downwards and backwards. "As the tooth moves into position, it rotates so as to bring its crown downwards and backwards, while the sinus spreads downwards and backwards between the roots of the teeth. The growth of the antrum

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[1] "The Expansion of the Maxillary Antrum," A. Keith, *Brit. Journ. Dent. Sci.*, June 16, 1902.

wheels, as it were, the tooth into position. An exactly similar condition occurs with the second and third molars."

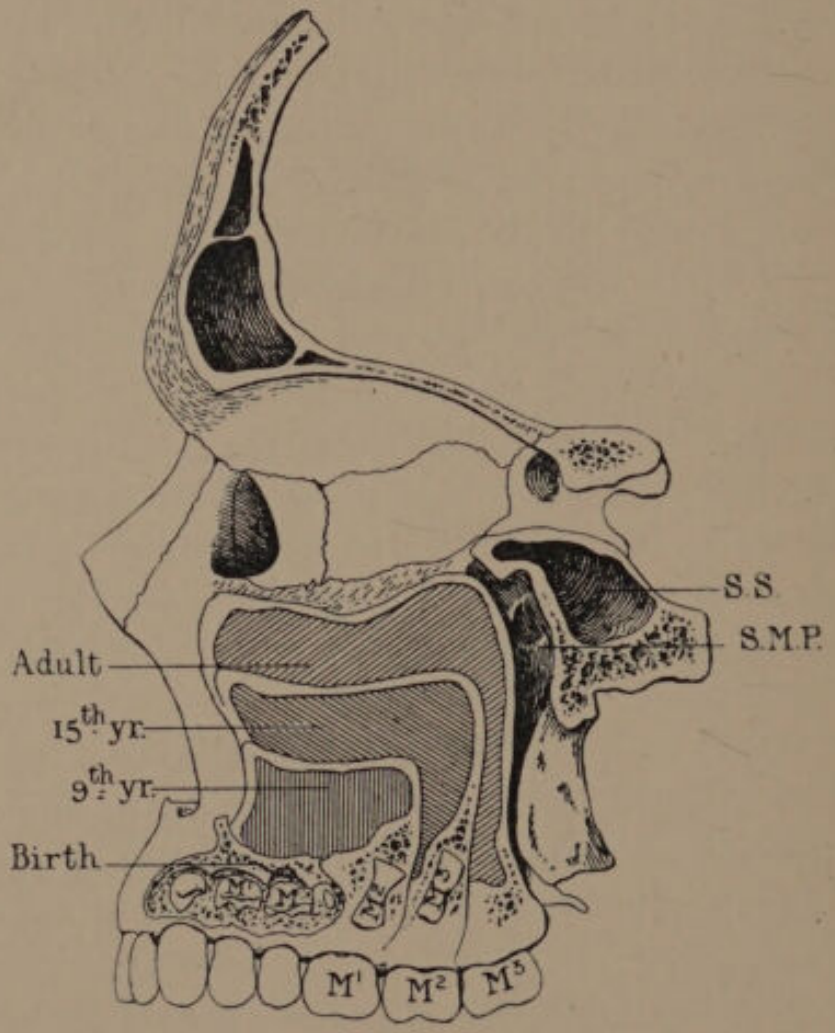


FIG. 13.—The above drawing by Arthur Keith, M.D., F.R.C.S., illustrates the position of the developing molars and the relation of the maxilla to the pterygoid processes. "The diagram is drawn to scale and is founded on material in the museums of the Royal College of Surgeons and of the London Hospital. Four stages are shown: (1) at birth, (2) at the 9th year, (3) at 15th year, and (4) in the adult. . . . While the diagram demonstrates clearly the growth changes which occur in the jaw, it must not be forgotten that, in the body, the postero-superior border of the sinus is the fixed, and not the moving point as represented in the diagram."

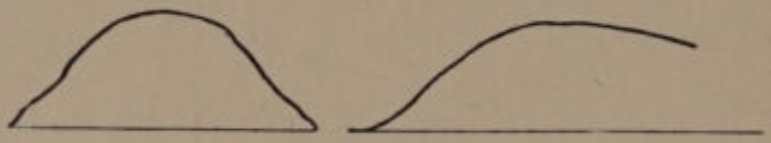
"The fulcrum on which this rotation of the bone takes place is formed by the body of the sphenoid, the anterior surface of its great wing, and the pterygoid plates, mainly the internal" (see fig. 13).

The mandible is developed in membrane as a single element, and its growth is largely dependent on the growth of the maxilla. At birth, the mandible consists of two halves joined by fibro-cartilage, and at about the sixth month the two segments unite. It is possible that before they unite the segments grow to some extent. The deciduous teeth in the mandible are replaced by the permanent teeth in the same manner as in the maxilla, the space for the molars being obtained by a backward growth of the bone, the growth being correlated with the growth of the maxilla consequent on the expansion of the antrum. A recognition of this fact is necessary in studying the pathology of certain types of irregularities of the teeth.

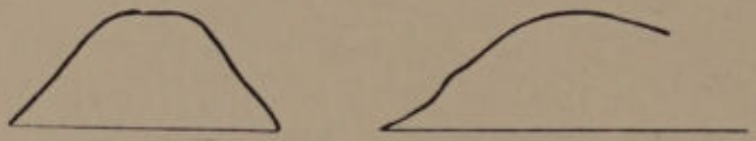
(B) FACTORS WHICH INFLUENCE THE GROWTH OF THE JAWS.

(i) *Modern Civilization.*—There is a general consensus of opinion that the jaws and teeth of modern races are becoming smaller, but that the diminution in size is greater in the jaws than in the teeth. The cause of the progressive diminution in the size of the jaws has not yet been clearly demonstrated, but in view of all the ascertained facts bearing on the subject the change would appear to be mainly attributable to the influences of civilization, and particularly to diet and the methods of preparing food. Dr. Sim Wallace, who has given considerable attention to the subject, considers that the diminution is not due to heredity, but is a "characteristic developed in each generation, as the result of the action of the environment"—*i.e.*, insufficient mastication deprives the jaws of the stimulus necessary for their full development, the character of the foodstuffs of the present day being responsible for the insufficient mastication.

WITHOUT ADENOIDS.



Hand-fed.



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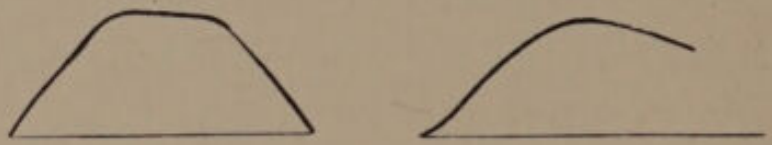

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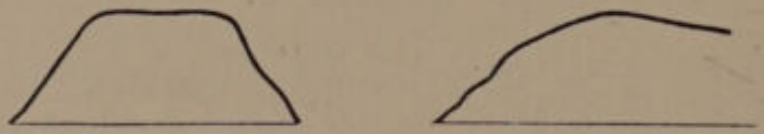
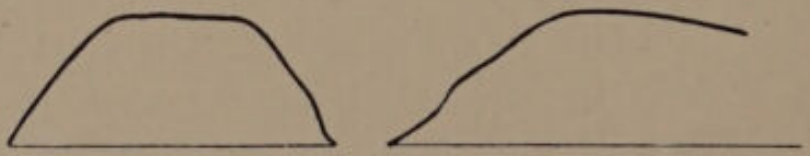
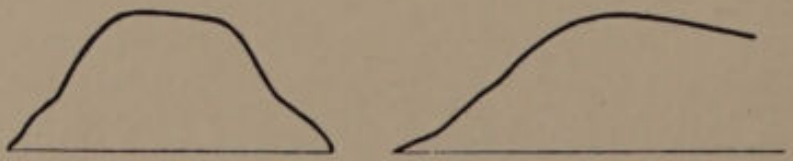


Breast-fed.



Breast- and hand-fed.



Breast-fed.

FIG. 14.[1]

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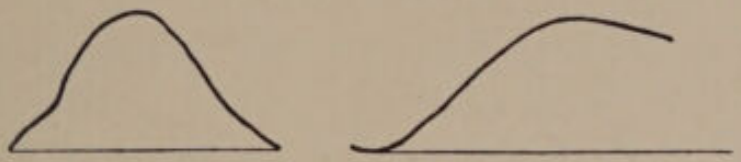
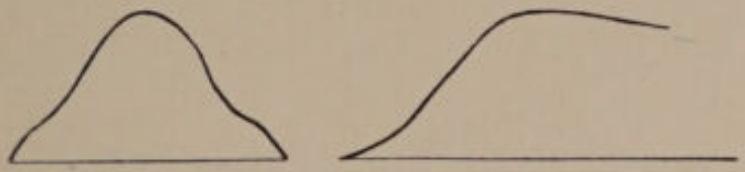
[1] From *Proc. Roy. Soc. Med.*

WITH ADENOIDS.

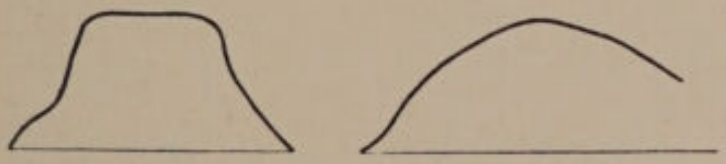


Breast-fed. Adenoids removed at 6 years.

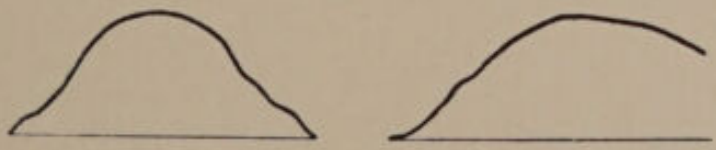


Hand-fed. Adenoids removed at 2, 4, and 6 years.

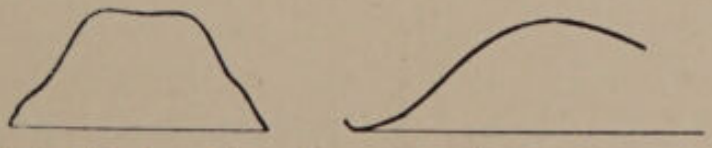
Hand-fed. Adenoids removed at 6 years.



Breast-fed. Adenoids removed at 10 years.



Breast-fed. Adenoids removed at 8 years.



Hand-fed. Adenoids removed at 8 years.

FIG. 15.[1]

[1] From *Proc. Roy. Soc. Med.*

(ii) *The Method of Feeding in Infancy.*—There is reason to think that the method of infant feeding influences the shape of the palate and dental arch.

Breast-fed children have slightly better developed jaws than those fed with the modern-shaped feeding-bottle. This is probably directly attributable to the action of the nipple of the breast upon the palate. Children fed with the tube feeding-bottle often exhibit a

TUBE BOTTLES, WITHOUT ADENOIDS.

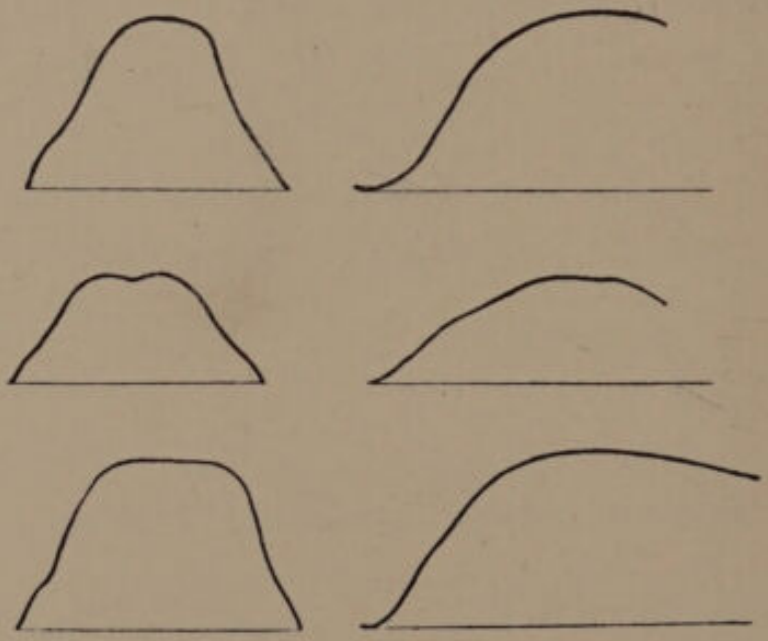


FIG. 16.[1]

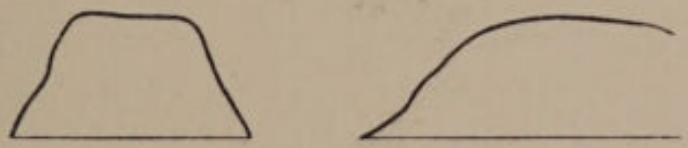
very definite narrowing of the arch. In using this bottle the tongue is closed around the teat and the act of suction is brought into play. The mandible is not used at all, and the whole action of the muscular tissue of the cheeks is towards the median line and not away from it, as is the case with the child at the breast. The action tends to narrow rather than spread the palate.

[1] From *Proc. Roy. Soc. Med.*

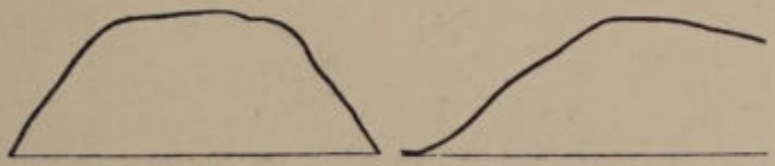
Tracings of the shapes of palates are shown in figs. 14 to 17. The transverse sections were taken across the region of the second premolars, the longitudinal section along the median line.

TUBE BOTTLES, WITH ADENOIDS.

Adenoids removed at 5½ years.



Adenoids removed at 13 years. Came on before the age of 6 years.



Adenoids removed at 15 years. Came on after the age of 6 years.

FIG. 17.[1]

(iii) *Adenoids.*—Nasal obstruction, arising from the presence of adenoids, greatly influences the growth of the maxilla, the effect produced depending on the period of onset and the persistence of the adenoids. The earlier the age at which the adenoids appear, the more marked will be the effect on the teeth and palate, and the longer they persist the greater will be the damage.

If the mouth of a breast-fed child of from 3 to 4 years of age that has not had adenoids be examined, the teeth

[1] From *Proc. Roy. Soc. Med.*

will be found to form a regular arch, with the anterior teeth separated by slight spaces. The vault of the palate in section across the deciduous molars will be oval-shaped, and on longitudinal section will rise with a gentle incline from behind the front teeth to the vault (see fig. 18).

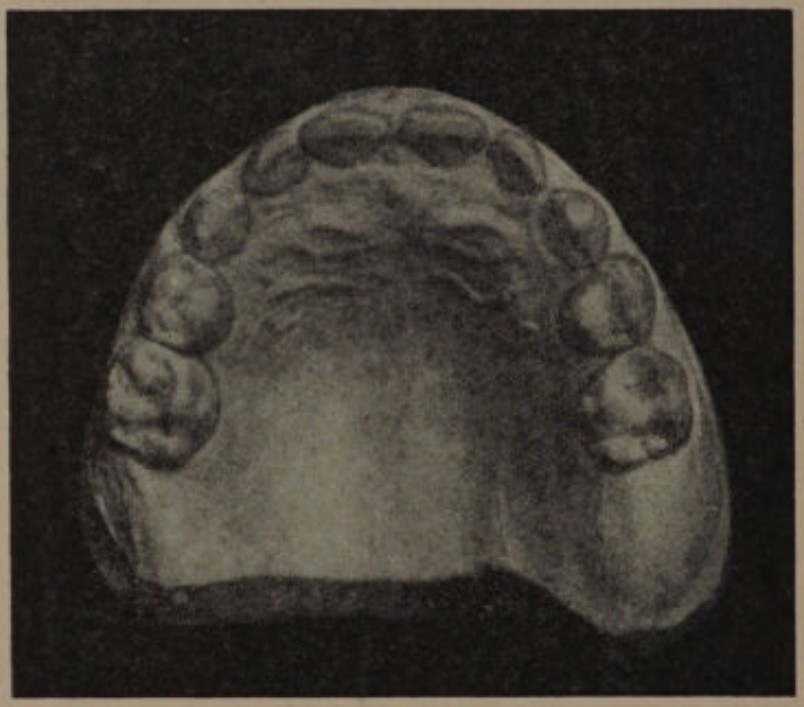


FIG. 18.

In a child that has suffered or is suffering from adenoids, the teeth will be crowded, and the muco-periosteum of the palate will present a somewhat puckered appearance (fig. 19); the vault, on transverse section, will be dome-shaped and, in longitudinal section, will rise somewhat abruptly from the posterior aspect of the incisors. The palate will appear as if it had been pushed up towards the front (fig. 21).

The deciduous molars may occlude too far forward, but this is the exception rather than the rule. If the adenoids

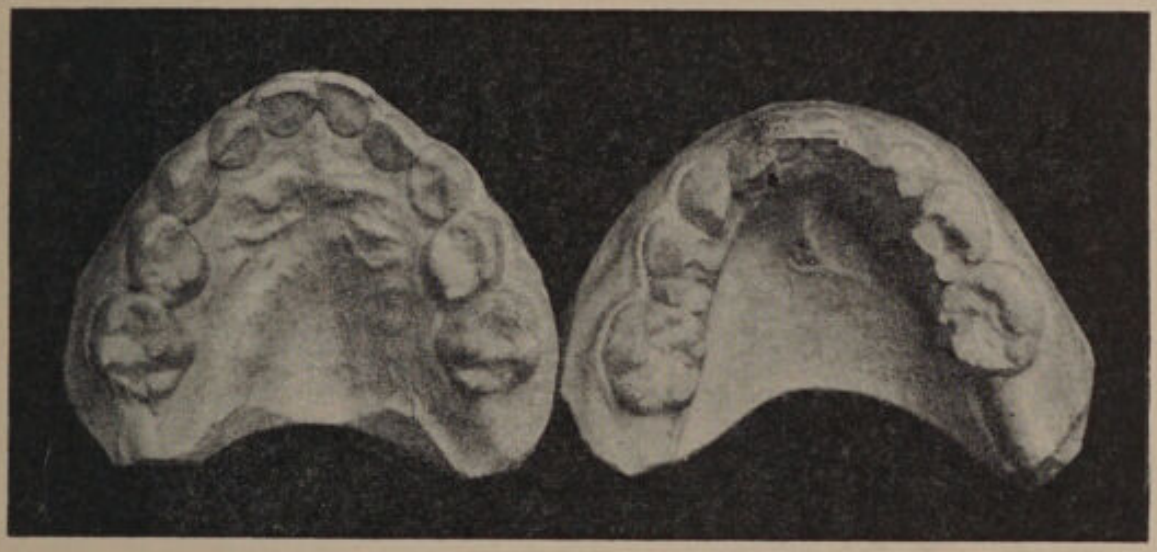


FIG. 19.—Models of a child, aged 5 years 7 months, that had suffered from adenoids from the age of 2 years. The puckered appearance of the muco-periosteum covering the hard palate and the crowding of the teeth are well shown.

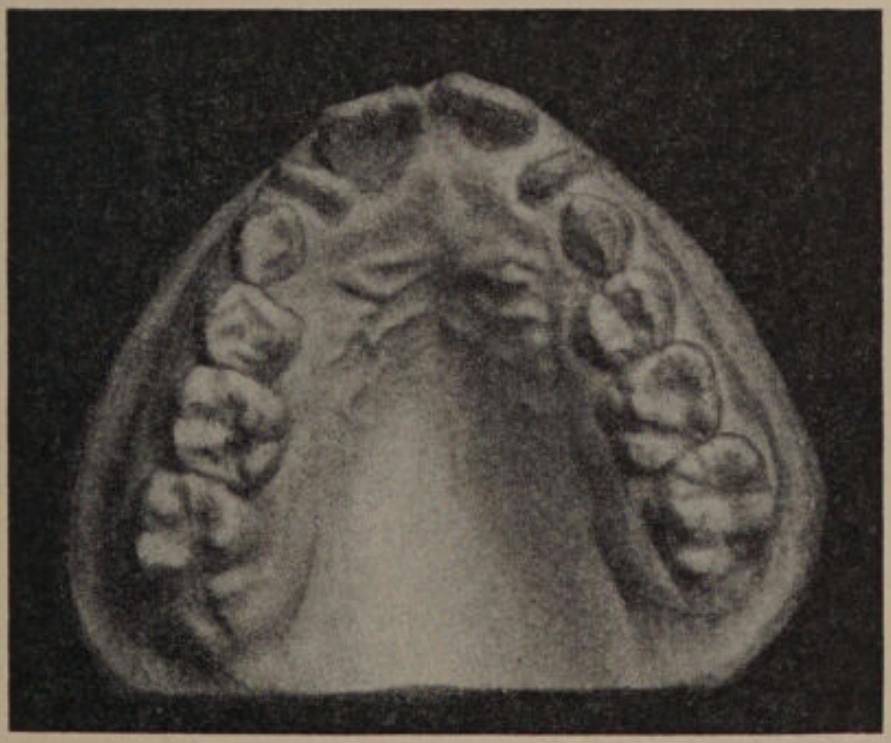


FIG. 20.—Model of the case shown in fig. 19 after the eruption of the incisors. Note the irregular position of the incisors.

have been removed before the age of 6 and have not been severe, the effect on the permanent teeth will be to crowd the upper incisors, and the disturbance will range from a slight tilting of the lateral incisors to an irregularity involving the four anterior teeth (fig. 20).

The first permanent molar erupts in good position, and, provided that the deciduous molars are retained for their normal period, the premolars will erupt in normal

FIG. 21.—Model of a boy, aged 7. He had been operated on for adenoids three times.

occlusion. The canines, when in place, will show a slight slope of the roots towards the median line. The only permanent irregularity will be the crowding of the incisor teeth.

If the adenoids have been severe and of long duration, an additional effect on the arch will be seen in the region of the first molars. The first molar, when erupted, often fails to assume a vertical position, and lies with a general slope backwards. This is well shown in fig. 22, the

models of a child who suffered from adenoids. This tooth is in normal occlusion, but it is not difficult to foresee that, as soon as the second deciduous molar is removed, rapid forward movement of the first molar will result in an encroachment on the space for the premolars and an abnormal occlusion.

In some cases the growth in the molar region is so restricted that the first molar erupts in such a way as to

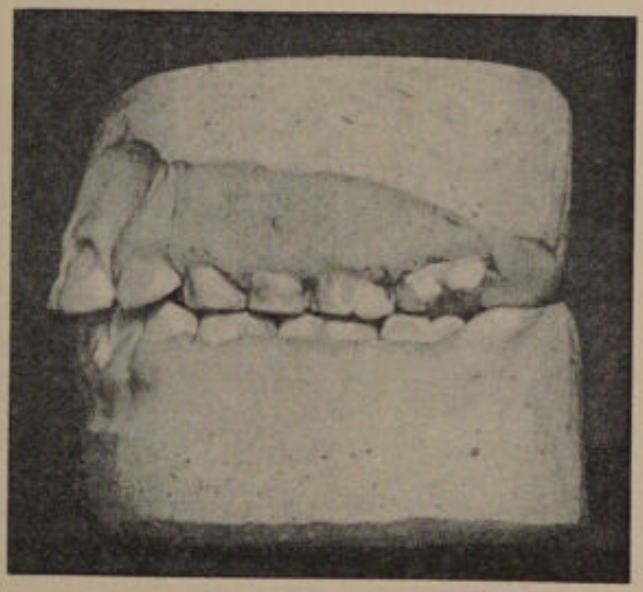


FIG. 22.

lead to absorption of the posterior roots of the second deciduous molar (figs. 23 and 24). Under such conditions, it is obvious that abnormal occlusion must result, and the space for the premolars will be considerably curtailed.

If the permanent molars do not move forward, the premolars and canines erupt in normal position, and the only deformity of the arch that remains is the slight irregularity of the incisors. Where the molar does move forward, a certain amount of general crowding of the premolars and canines results.

There would appear to be a slight decrease in the width of the arch in children with adenoids, as compared with those free from adenoids, the difference being just under 1 mm.

In persons who have suffered from adenoids, the third molars often erupt with the occluding surfaces facing backwards and outwards.

Many of the gross lesions of the jaws so often associated with adenoids are not necessarily due to the

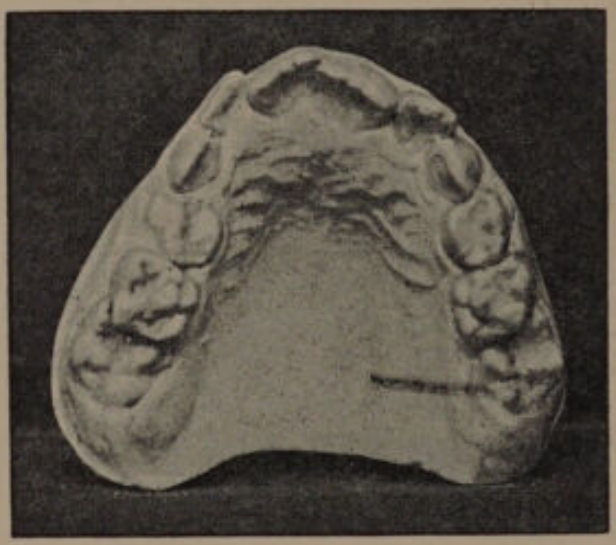


FIG. 23.[1]

presence of adenoids, and the two conditions must not be regarded in the light of cause and effect. These lesions are usually the result of adenoids in conjunction with other contributory causes, such as rickets, lip-sucking, early loss of deciduous teeth, &c.

(iv.) *Rickets.*—There is no evidence to show that rickets by itself can be regarded as the cause of deformed arches, but owing to the fact that the bones in rickety

[1] From *Proc. Roy. Soc. Med.*

persons are imperfectly formed and yield more readily to pressure, the deformities resulting from adenoids and from the use of the "dummy," or from thumb-sucking, will be more marked if rickets is present.

(v) *Habits.*—Certain habits are contracted by children, such as finger-sucking and lip-sucking, which, if persisted in, will produce deformed arches. In the habit of sucking the thumb or finger, the palmar surface of the thumb is placed against the palatal surface of the central incisors, the mandibular teeth being closed on the dorsal aspect. The pressure exerted causes the maxillary

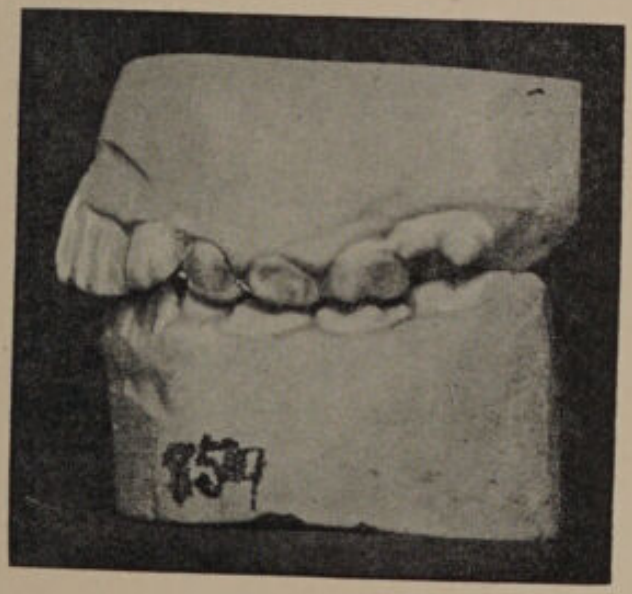


FIG. 24.[1]

central incisors to protrude, so that the lower lip passes behind them, and aggravates the protrusion when the mouth is at rest. At times the thumb or finger is inserted between the teeth in such a manner as to cause the teeth to impinge on the lateral surfaces. Under such

[1] From *Proc. Roy. Soc. Med.*

conditions, the maxillary incisors and canines are forced outwards and frequently upwards, producing a type of irregularity known as "open bite." Sucking of the lower lip or tongue may also produce protrusion of the maxillary teeth.

The prolonged and persistent use of the "baby comforter," or "dummy," is a fruitful source of dental deformity. Fig. 25 shows the models of a child, aged 5, illustrating protrusion of the maxillary arch from this cause. The protrusion was well marked in this child when seen about 18 months old.

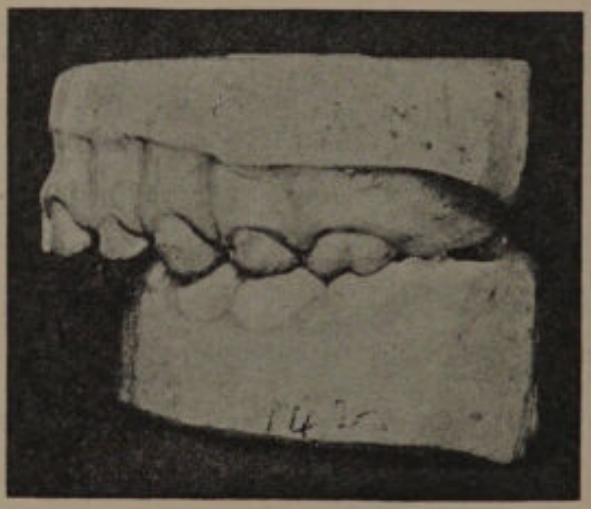


FIG. 25.[1]

Careful enquiry into the use of the "dummy" would seem to show that, if its use is persisted in, deformity of the maxillary dental arch will almost certainly result, the amount of deformity depending on the persistence with which the "dummy" is used, the period over which its use extends, and the presence or absence of rickets.

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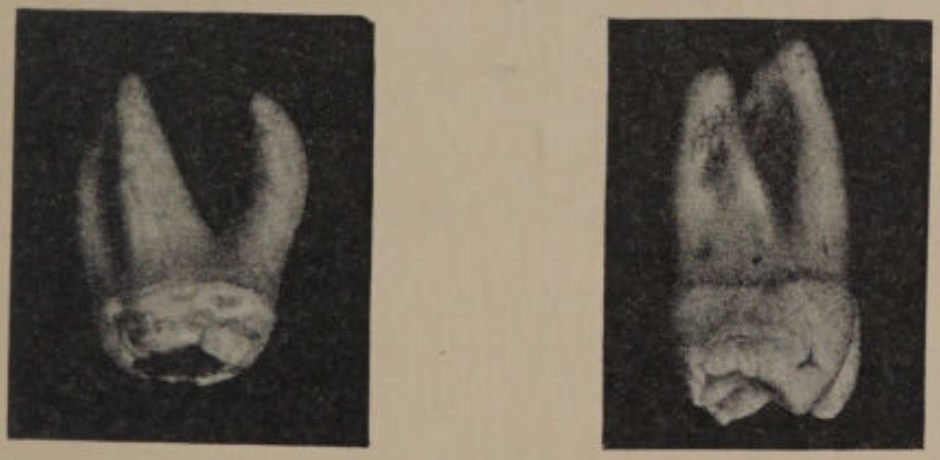
[1] From *Proc. Roy. Soc. Med.*

(C) CONDITIONS WHICH PRODUCE DEFECTIVE FORMATION OF THE TEETH.

Various defects in the structure and form of the teeth are distinctly traceable to certain general pathological conditions.

FIG. 26.[1]—Model showing syphilitic incisors.



(*a*) (*b*)

FIG. 27.[1]—The syphilitic molar (*a*) is shown in contrast with a normal-shaped molar (*b*)—both slightly enlarged.

(i) *Syphilis.*—It is a well-established fact that congenital syphilis may leave, as one of its marks, a characteristic deformity of certain of the teeth.

[1] From a photograph by Mr. G. G. Campion.

The teeth affected are usually the permanent incisors, both upper and lower, at times the canines, and frequently the first molars.

The incisors when affected appear as small, pegtop-shaped teeth. The distal margins of the centrals are generally turned outwards, and it will also be noticed that the alveolar portion of the jaw in the incisor region is imperfectly developed. As a rule the teeth are generally affected symmetrically, but exceptions occur, as, for example, when one incisor is perfectly formed and the other presents the typical syphilitic form.

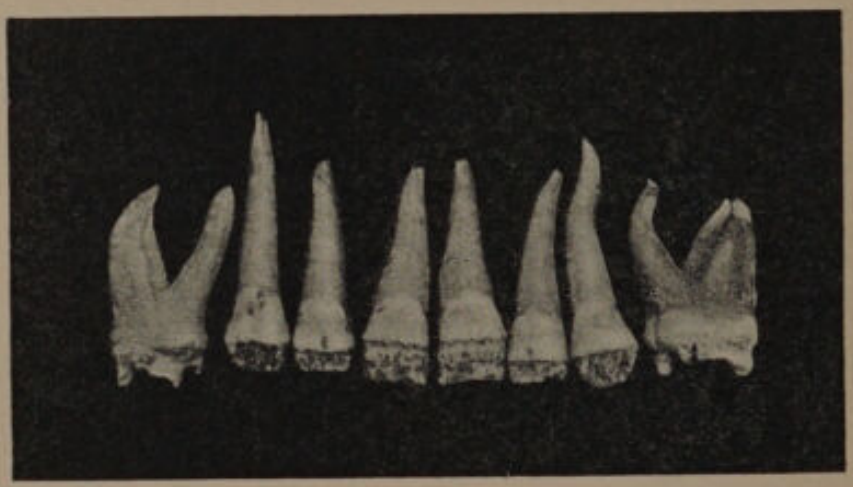


FIG. 28.—Hypoplastic maxillary incisors, canines, and first molars from the mouth of a patient who had suffered from rickets.

When a healthy tooth has just erupted, the cutting edge is seen to be surmounted by three little tubercles separated by two shallow notches, and, the tubercles being rapidly worn down by attrition, the cutting edge soon appears quite straight. In the syphilitic tooth, these tubercles and notches are well marked, and the central tubercle is badly developed: the effect of attrition is to produce one central notch between the two outside tubercles. This central notch is said to

give to syphilitic teeth one of their characteristic appearances, but it must not be solely relied upon in diagnosis, as it appears to be produced in teeth where there is not the slightest taint of congenital syphilis. The laterals are not always deformed in syphilitic patients, the centrals being considered by Mr. Jonathan Hutchison as the " test " teeth.

The deformity of the canines shows itself by a circumferential notch occurring near the cutting edge or point of the crown.

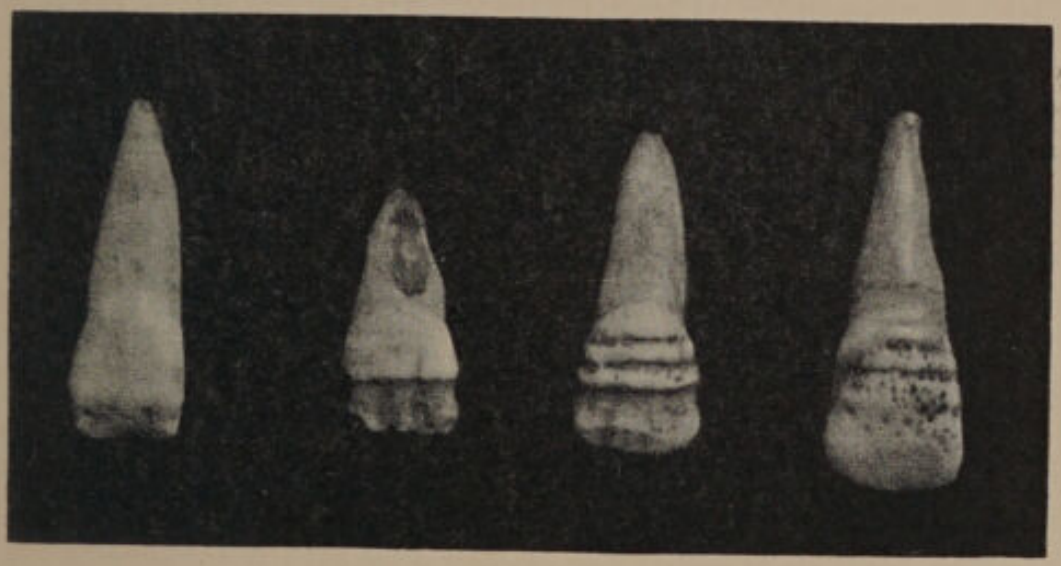


FIG. 29.—Examples of hypoplastic maxillary central incisors.

The affected molars, like the centrals, are smaller than normal. The upper surface appears constricted, the cusps being depressed and are rather flat and irregular (see fig. 27).

The *Treponema pallidum* has been found in the dental follicle in proximity to the vessels and their walls.

(ii) *Hypoplastic Teeth* (honeycombed teeth).—Under this heading it is convenient to describe a condition which affects many teeth and is characterized by a defective formation of the enamel.

The type of hypoplastic teeth most commonly met with is one in which the first molars, together with the incisors and canines, are affected, the defect extending from the cutting edges, and involving, in severe cases, the whole of the crown surfaces. In mild cases, the enamel presents only a slight pitting; in more advanced conditions, the enamel is dark in colour and presents numerous deep pits; while in severe cases the enamel covering is slight and the cutting edge of the tooth presents sharp points, giving the tooth a curious spine-like appearance. In another variety, the enamel is

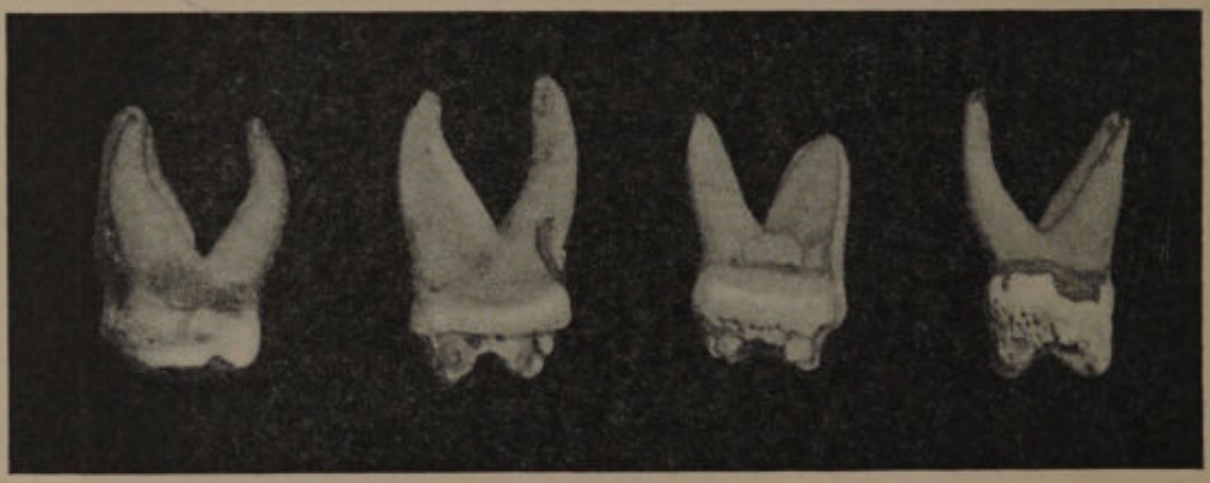


FIG. 30.—Examples of hypoplastic first maxillary molars.

simply deficient in quantity, and shows little or no pitting. In some cases, the rows of pits run transversely across the tooth, separated from one another by well-formed enamel; while other examples are seen in which the surface of the tooth shows transverse grooves, the enamel everywhere presenting a smooth glossy appearance. In rare instances, the defects run in a vertical direction. In some patients, the enamel towards the cutting edge of the tooth may be well formed, the defect starting beyond this point.

In seeking for the cause of the deformity, it must be remembered that the causal agency must have been active when the deformed enamel was being calcified. In most cases, the malformation is on the crowns of the central incisors, the laterals, and the canines towards the cutting edges and on the crown surfaces of the first molars, and this indicates that the agency causing it acted during the first two years of life.

The view that the condition is due to a stomatitis arising from the use of mercury is untenable, well-

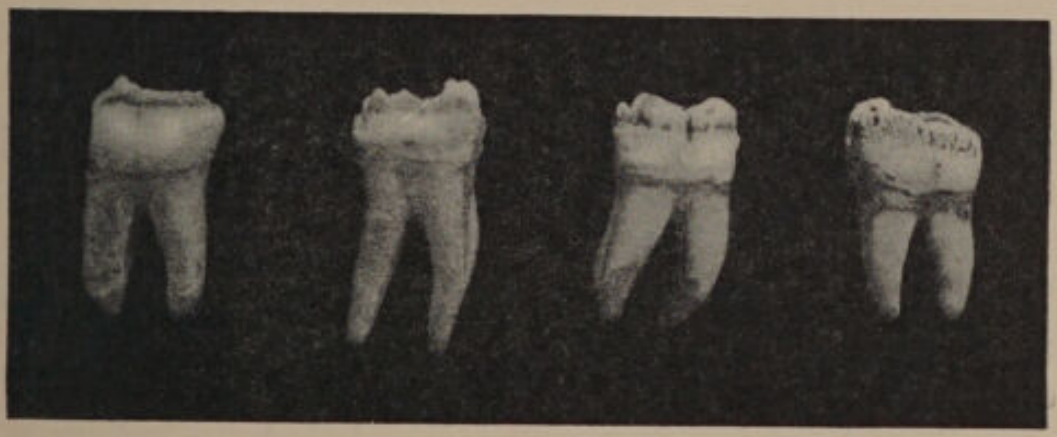


FIG. 31.—Examples of hypoplastic first mandibular molars.

marked hypoplastic teeth being often found in children where no mercury has been given. From clinical investigations it would appear that the defective dental development is due to mismanagement in the feeding of infants, as a large number of children with these teeth have been fed upon artificial foods of a starchy character.

Dr. Kingston Barton, who has given much attention to this question and has kept careful records, states that in 202 children he found 10 cases of hypoplasia in the permanent and 5 cases in the deciduous series. These latter occurred out of 67 hand-fed children. Out of the 202 cases, 54 were fully breast-fed, and in these

no hypoplasia was present. He also adds that in two cases of very bad artificial feeding very early and extensive caries occurred.

There is a type of hypoplastic tooth in which the deformity is limited to a transverse band of tissue in any part of the crown. These defects are traceable to the effect of one of the exanthematous fevers. It must, however, be remembered that only a small percentage of children who have had exanthematous fevers show these defects.

Defects in the structure of teeth may, in rare instances, be produced by local causes. The malformation is usually limited to a single tooth, and may take the form of a pit, ring, or patch, or may extend to the total absence of enamel.

In many of the cases a single premolar is affected, and when this is so, distinct evidence of suppuration in connection with the deciduous predecessor can often be obtained.

CHAPTER III.

Caries of the Teeth.

CARIES of the teeth is the most prevalent of all diseases. It has been found in skulls of the Neolithic period and has since shown a gradual tendency to increase as civilization has advanced. It was common among the Romans in their later and more degenerate days. Among the Egyptians the disease is known to have existed during the early dynasties, and about the year 2,000 B.C. it became prevalent among the aristocratic portion of the race: afterwards gradually increasing and spreading to all classes. Among the British it has been a common disease during the last few hundred years, and would appear to have increased rapidly during the last century, especially during the latter part.

(A) ANATOMY OF THE TEETH.

There are a few points in the anatomy and chemical composition of the teeth to which reference may with advantage be made.

The enamel, which covers the entire crown of the teeth, is composed of prisms hexagonal in form. The prisms start from the dentine surface and take a course outward to the periphery of the tooth. The prisms, although lying on the whole parallel to one another, take a wavy course, the curvature of the prisms being most marked on the masticating surface. The completed enamel

prism is considered by Leon Williams to be a regular beaded, calcified rod, the beadings being embedded in a matrix which fills up the interstices and is itself calcified. In the usual analyses enamel is generally represented as consisting of about 3·5 to 4·0 per cent. of organic matter. Tomes has shown that this supposed organic matter is in reality "water combined with lime salts," the enamel, in his opinion, being devoid of organic matter.

The dentine, which forms the bulk of the tooth, is composed of a matrix permeated by tubes. These tubes commence on the surface of the pulp and pursue a wavy course to the periphery of the dentine. The tubes contain a soft protoplasmic matter which is in continuity with the pulp. Chemically, dentine consists of inorganic and organic material, the proportions being approximately as follows:—

	Inorganic	Organic
Incisors	71·5 ..	28·5
Molars	73·0 ..	27·0

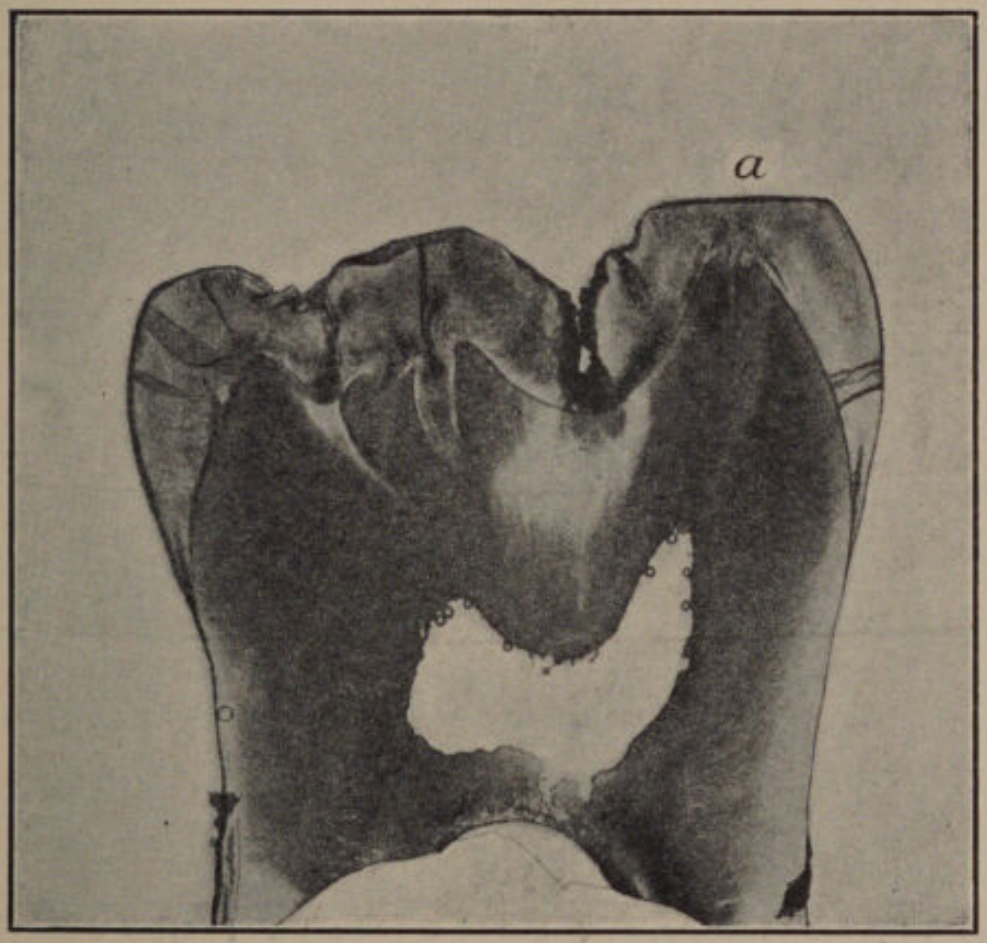
The cementum which covers the root of the tooth is composed of a laminated calcified matrix with a few lacunæ, the latter being met with in greatest numbers towards the apical part of the tooth. Chemically, the cementum contains about 32 per cent. of organic matter and 68 per cent. of inorganic.

(B) THE MORBID ANATOMY OF CARIES.

(1) Macroscopical Appearances.

(*a*) *Enamel.*—Naked eye appearances. In the earliest stages caries, as a rule, appears at certain definite sites on a tooth, namely, (1) in the deep portion of fissures; (2) around, but more especially just below, the point of contact of adjacent teeth; (3) in pits; and (4) on the labial aspects of the teeth near the gum margin.

The first sign of its presence is a slight loss of the normal polish and translucency of the enamel. This gradually increases until a white spot appears which in process of time gradually darkens, the degree of darkening being in inverse proportion to the rapidity of the disease. The matrix of the enamel is dissolved



× 10.

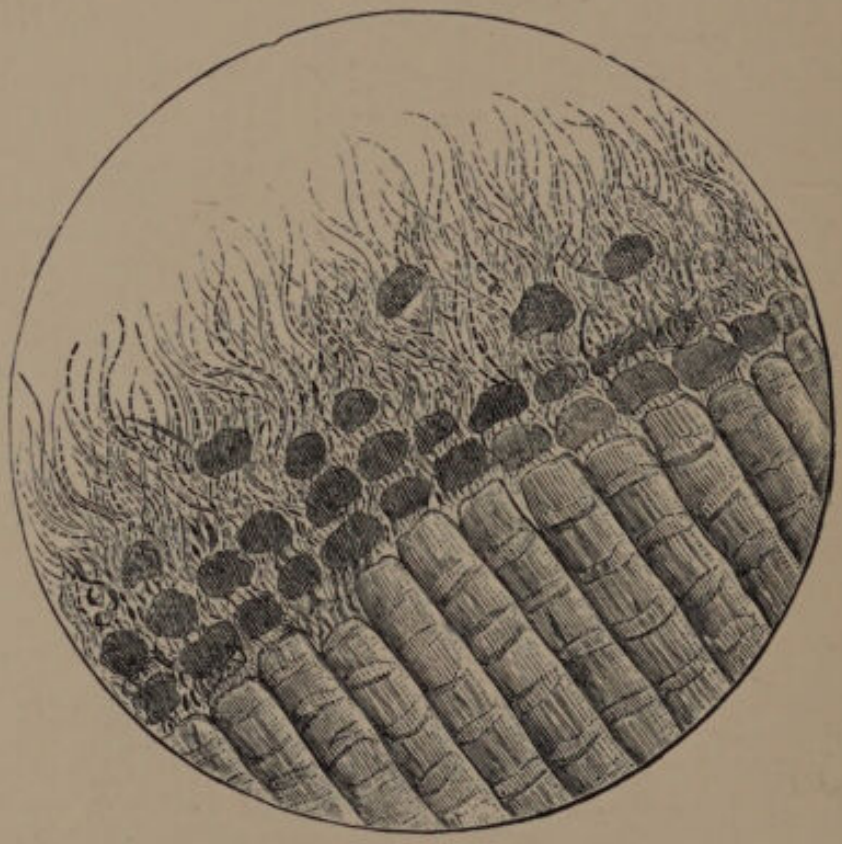
FIG. 32.[1]—Section of molar showing typical transparency. At (*a*) transparency, resulting from abrasion of the cusp (Miller).

before the prisms, which are thus left unsupported and are subsequently partly dissolved and partly mechanically washed away. A cavity is thus created which varies in form, being sometimes broad and shallow with

[1] From *Dental Cosmos*.

indistinguishable margins, at other times deep with sharp and rugged edges. Broken enamel tissue is of a soft and cheesy nature. The destruction of the enamel advances along the course of the prisms until the dentine is reached.

(*b*) *Dentine.*—This tissue becomes of a tough cartilaginous consistency and gradually softens and undergoes disintegration. Pigmentation, due to chromogenic



× 1,500.

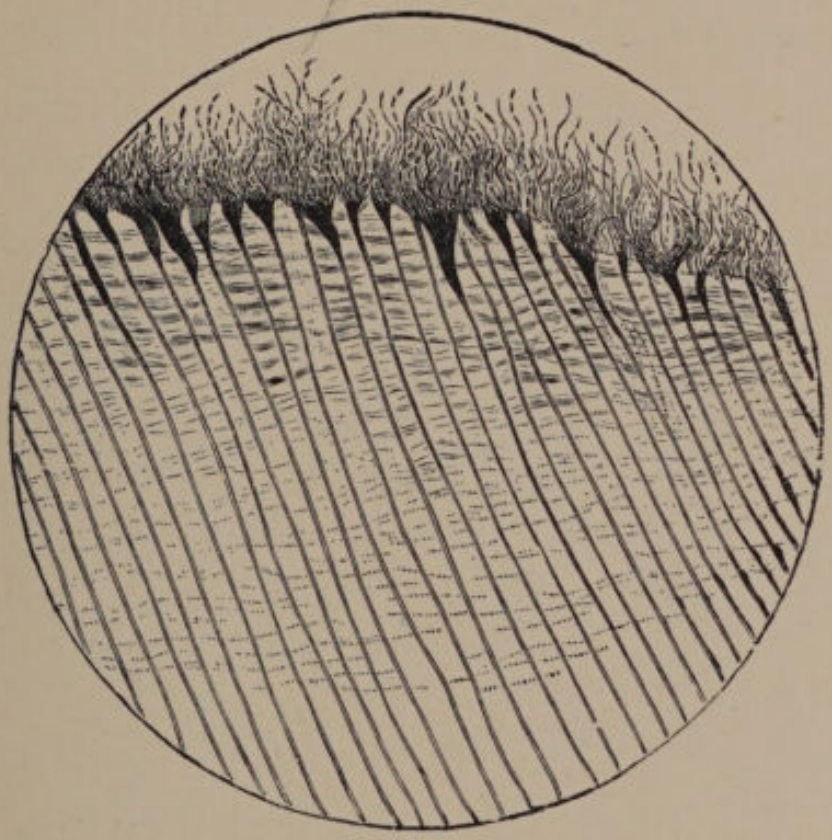
FIG. 33.—The *modus operandi* of the process of caries, according to Dr. Leon Williams, varies somewhat in different specimens. In this specimen, the sectional masses of the enamel rods are being set free by the solution of the cement substance which unites them.

organisms, accompanies the process, and, as in the case of enamel, varies indirectly with the rapidity of the process. The direction in which caries spreads depends upon the anatomy of the tooth; in normal teeth the direction is towards the pulp.

With the approach of caries a change takes place in

the dentine of teeth, the pulps of which are alive. The change consists in the tissue between the pulp and the caries becoming transparent.

This change is probably due to a calcification of the contents of the dentinal tubes and may be regarded as a reaction of the dentine to injury.



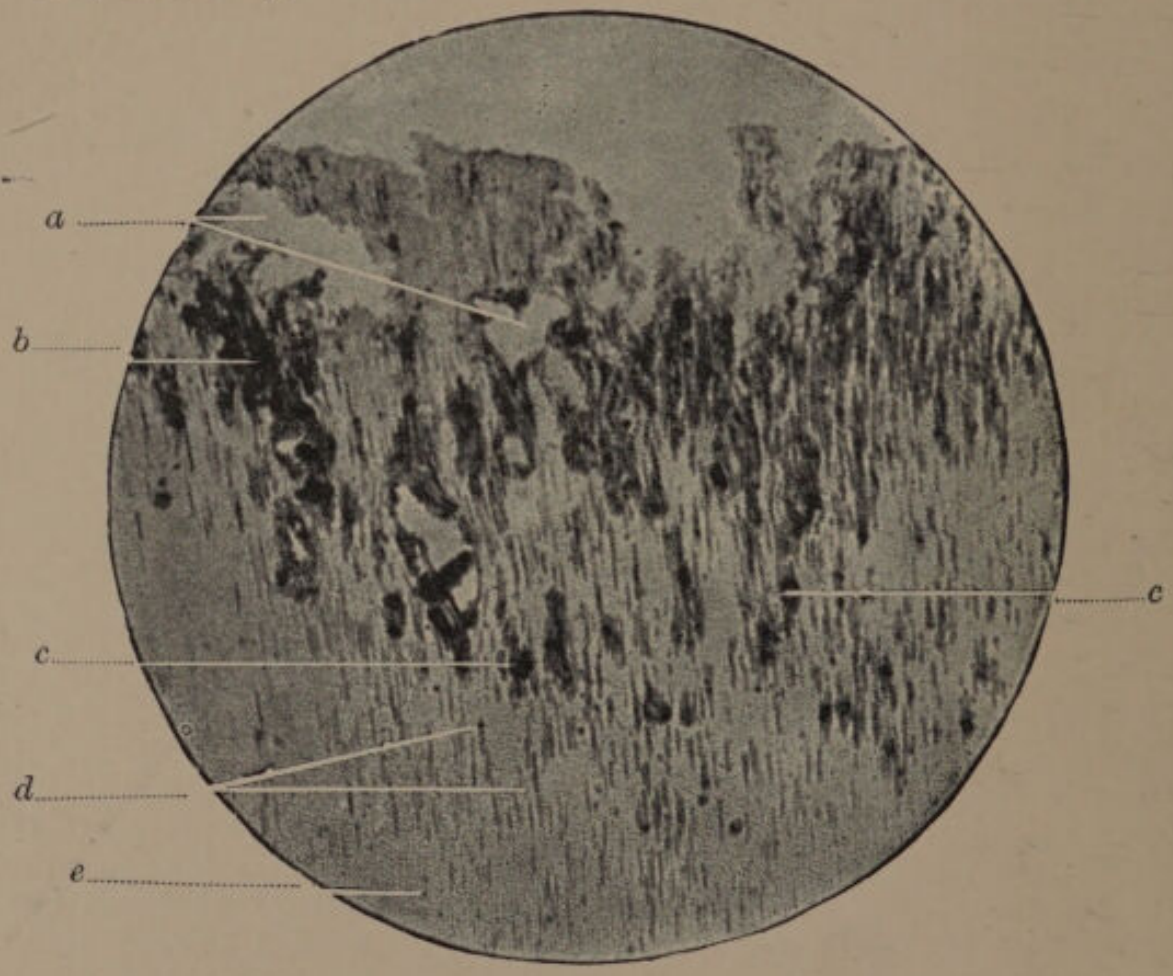
× 800.

FIG. 34.—Showing commencement of the carious process. A felt-like mass of micro-organisms is seen attached to the surface. The cone-shaped cavities between the enamel prisms, produced by solution of the cement substance between the enamel rods, are also shown.

(*c*) *Cementum.*—Caries of the cementum is comparatively uncommon. It starts, as a rule, at the neck of a tooth, but may attack any portion of the root that may be exposed through loss of its periodontal membrane. The cemental tissue becomes softened and subsequently disintegrated. The cavities formed are, as a rule, shallow, though often widely extended.

(2) Microscopical Appearances.

(*a*) *Enamel.*—Sections of carious enamel show that the tissue is decalcified between the rods and the interstitial cement substance; the enamel globules thus unbound being either dissolved or washed away. The enamel may be penetrated to a considerable depth before any breaking down of the tissue occurs.



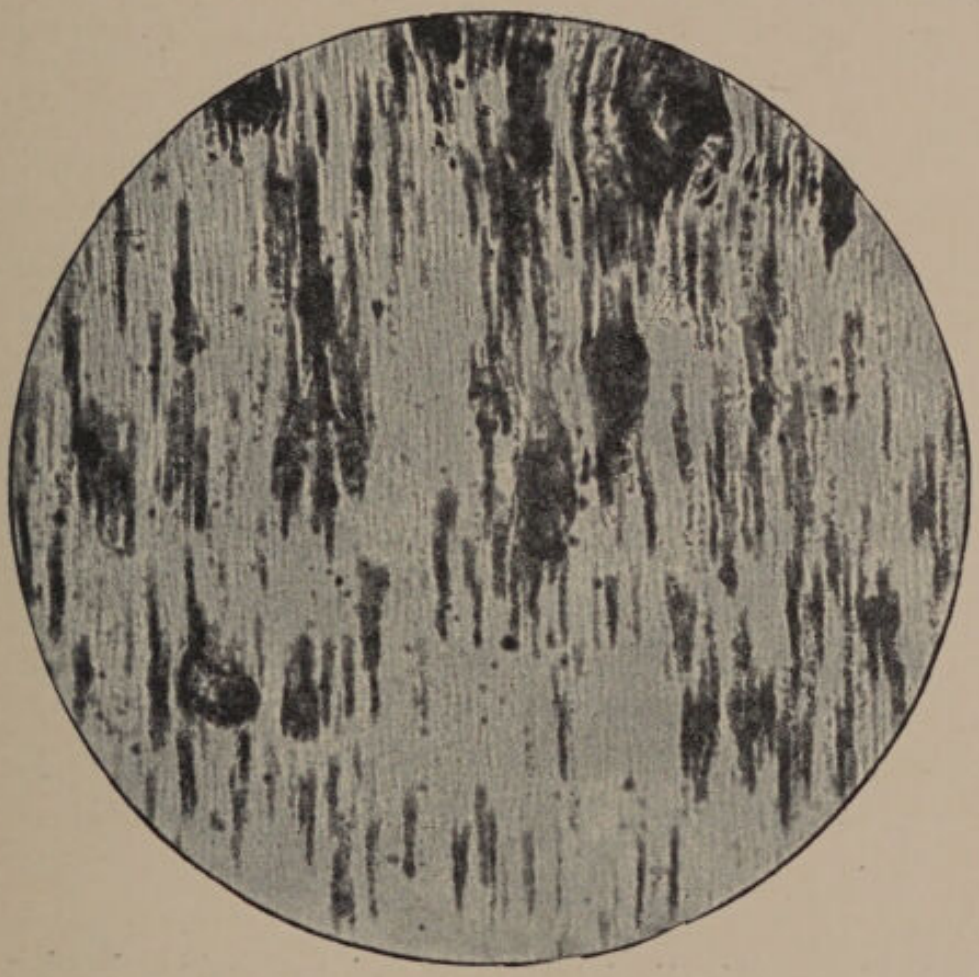
× 80.

Fig. 35.—Longitudinal section of carious dentine. Photomicrograph by Mr. A. Pringle.

(*b*) *Dentine.*—If longitudinal sections of carious dentine which have been stained to demonstrate micro-organisms be examined microscopically, the following points will be observed:—

Under a Low Power (80 magnitude).—Bordering the

surface (fig. 35), the dentine is hollowed out in an irregular manner, the cavities presenting no definite shape (*a*). A little deeper in the substance of the dentine, irregular masses of stain can be detected (*b*), and in places, the stain presents a globular appearance (*c*). Lower down streaks of stain are to be seen (*d*), while still further down the dentine is unstained (*e*).



× 150.

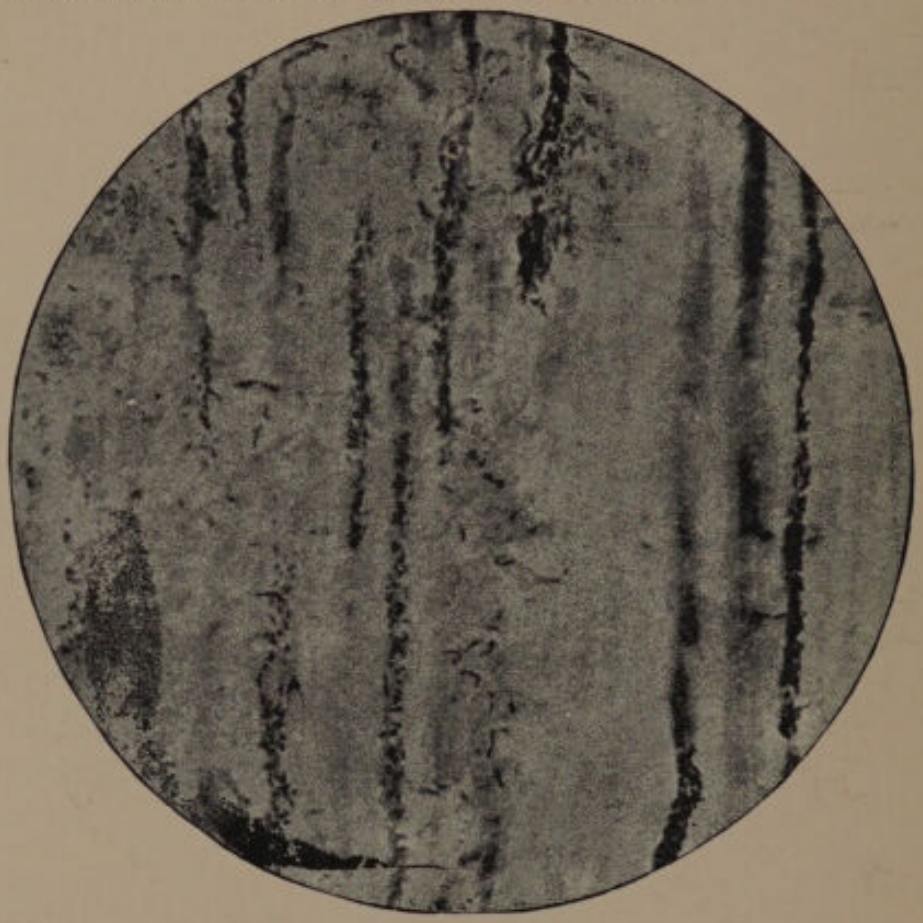
FIG. 36.—Longitudinal section of carious dentine, showing liquefaction foci. Photomicrograph by Mr. A. Pringle.

Under a Higher Power (150 magnitude).—Tracing the process in the reverse order, namely, from within outwards, it will be noticed :—

(1) That the unstained part (*e*) is dentine in a decalcified condition.

(2) The streaks of stain (*d*) are micro-organisms occupying the dentinal tubes.

(3) The globular masses are micro-organisms occupying the tubes and the structure between the tubes. In other words, the dentine matrix has disappeared and micro-organisms have taken its place. These globular masses have been termed *liquefaction foci*.



× 650.

FIG. 37.—Longitudinal section of carious dentine, showing rod-shaped organisms in tubes. Photomicrograph by Mr. J. Howard Mummery.

(4) The irregular masses are formed by the fusion of *liquefaction foci*.

In sections containing interglobular spaces, the spaces are generally seen to be filled with masses of organisms, although occasionally they appear to be quite free from infection. Figs. 35 to 38 are longitudinal sections

through carious dentine stained to demonstrate the micro-organisms.

If sections of carious dentine are stained to demonstrate the micro-organisms only, it can be shown that the softening of the dentine precedes the infection. Examine such a section, and it will be seen that the softened parts towards the cavity are stained, and towards the

x 650.

FIG. 38.—Longitudinal section of carious dentine, showing tubes filled with leptothrix. Photomicrograph by Mr. J. Howard Mummery.

healthy dentine unstained, which clearly shows that the softening precedes the infection. Miller has termed the unstained portion the *non-infected zone*. The micro-organisms show a greater tendency to spread towards the pulp than in a lateral direction, but this is not always the case, especially in irregularly calcified teeth,

such as occur in rickets, in which a large number of interglobular spaces are present. The dividing line between the infected and the non-infected zones is often well marked, and though the majority of tubes near the surface are infected, the infection is not noticeable in the deeper parts. Near the margin leptothrix is mainly found, while the tubes are generally filled with micrococci or simple bacilli.

(C) THE PATHOLOGY.

The primary stage of caries is determined by the presence at a certain portion of a tooth of an acid capable of dissolving the inorganic salts. The matrix of the prisms is the first to be dissolved, and into the spaces thus formed crowd organisms which, given a suitable pabulum, produce more acid and further destruction of the surrounding tissue. There is good reason to believe that, apart from its natural resistance to solution, the enamel offers no vital resistance to attack. After the solution of the enamel the dentine is decalcified in a similar manner, and later the remaining organic portion of the tooth substance is digested by the bacteria, which perform their work by means of secreted enzymes, digesting the tissue in much the same way as the pepsin of the gastric juice acts upon albuminous materials.

(D) BACTERIOLOGY.

Goadby groups the micro-organisms concerned in caries thus :—

(1) Those capable of producing acids by the fermentation of carbohydrates—acid-forming organisms. The weak acids formed during the decomposition of proteids appear to have no action upon the lime salts of the teeth.

(2) Those which, either by their own action or by virtue of a secreted enzyme, lead to digestion of decalcified

dentine (liquefying organisms). Certain organisms which, although not producing acid or enzymes, form gelatinous colonies which adhere firmly to the enamel of the teeth may play some part in the initial stages of caries. These can grow in association with acid-producing organisms, and Goadby believes that a gelatinous plaque on the tooth may act as an osmotic membrane in which, if the necessary pabulum be also present, acid may be formed and diffuse through and attack the tissues beneath.

On the surface of carious cavities the number and variety of bacteria are greater than in the deeper layers of carious dentine. The majority are aerobic liquefiers, the minority anaerobic liquefiers. Many of these organisms liquefy gelatine with great rapidity and in addition blood serum. Those organisms which liquefy coagulated blood serum will, as a rule, digest decalcified dentine. *In the deeper layers* of carious dentine the bacteria are rarely liquefiers, but nearly all are capable of producing an acidic change in carbohydrates, especially glucose, maltose, and lactose. Chromogenic powers are possessed by some of the organisms found on the surface of cavities, but rarely by those in the deeper layers of carious dentine.

The following list of bacteria found in caries is given by Goadby :—

Acid-forming Bacteria.

Streptococcus brevis *B. necrodentalis* (Goadby) *S. albus*	Deep layers of carious dentine.
S. brevis *Sarcina lutea* ,, *aurantiaca* ,, *alba* (Eisenberg) *S. albus* *S. aureus*	Superficial layers of carious dentine.

Bacteria which liquefy Dentine (decalcified).

None isolated as yet. Deep layers of carious dentine.

Bacillus mesentericus ruber (vulgatus), Gisen.
B. mesentericus vulgatus.
B. mesentericus fuscus.
B. septus (B. furvus).
B. liquefaciens fluorescens.
B. subtilis.
Proteus zenkeri.
B. plexiformis (Goadby).
B. maximus buccalis (Goadby).

} Superficial layers of carious dentine.

The above organisms are those capable of existing in the mouth. It is probable, however, that the early stages of caries may often be determined by acid-producing organisms introduced with carbohydrate food, providing that they are capable of existing for a length of time sufficient to bring about the acid change in the medium with which they have been introduced.

(E) THE SOURCE OF THE ACID.

The principal acid formed is lactic, and it derives its origin from the fermentation of amylaceous and saccharine substances retained in contact with the teeth. The carbohydrates fall conveniently into three groups :—

(1) Monosaccharides, or simple sugars, such as glucose, fructose, etc.

(2) Polysaccharides, or complex sugars resembling sugars, such as cane sugar (sucrose), milk sugar (lactose), and malt sugar (maltose).

(3) Polysaccharides, not resembling sugars, such as cellulose, starch, gums.

In the case of sugars, those belonging to the monosaccharides, namely dextrose, levulose (which occur in

fruit and honey and as commercial glucose made by the action of dilute mineral acids on all forms of carbohydrates) are directly fermentable according to the equation :

$$C_6H_{12}O_6 = 2C_3H_6O_3$$

Those belonging to the polysaccharides, resembling sugars, require inversion before the acidic change can take place. Thus :—

$$\underset{\text{Cane sugar}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Dextrose}}{C_6H_{12}O_6} + \underset{\text{Levulose.}}{C_6H_{12}O_6}$$

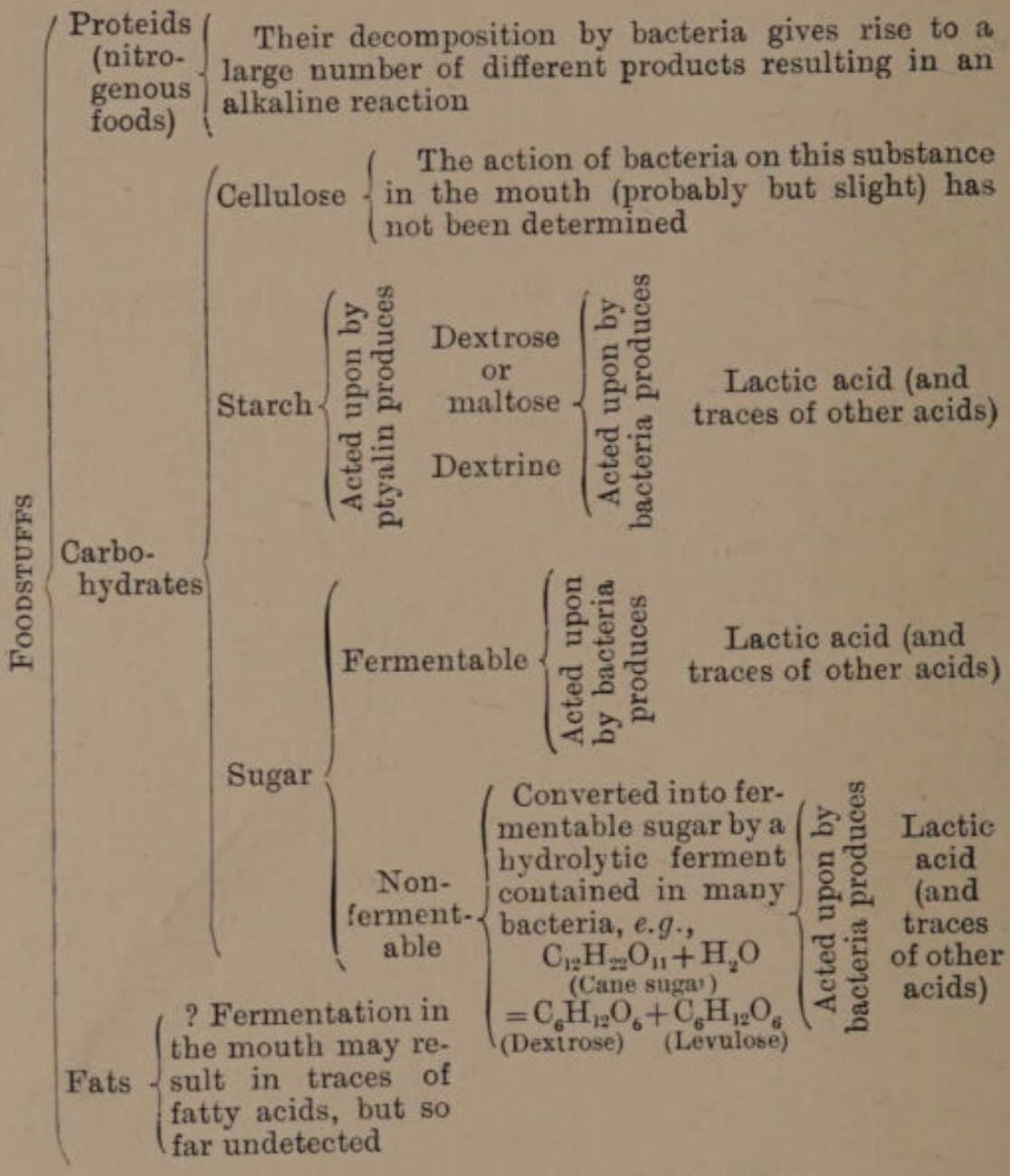
This inversion is brought about in the mouth by the action of invert ferments contained in the bacteria, or secreted by them.

In the case of the polysaccharides, not resembling sugars, probably only those that are capable of being acted on by ptyalin are capable of being fermented in the mouth.

Experiments have proved that when fermentable albuminous materials are combined with saliva little acid is formed, and that this is rapidly neutralized by the various alkaline products which result from the subsequent process of putrefaction.

The appended table, suggested by Mr. Mummery and the late Dr. Miller, shows the part that different foodstuffs play in the direction of dental caries.

Miller believed, and indeed the vast majority of writers still believe, that the acid is formed from the solid food ; but it is probable, for certain reasons which will be dealt with fully later, that carbohydrate food in a state of solution is far more important. Moreover, solid or pulpy food cannot ferment so rapidly as liquid food, for the reasons that the particles cannot move about so freely and the products of the organisms, not being able easily to disperse, remain and inhibit bacterial activity.



(F) THE PROGRESS OF CARIES.

Dental caries is, as a rule, a progressive disease, but under certain conditions its progress becomes arrested. In such cases the teeth generally show an extensive loss of tissue, the enamel of the masticating surfaces, and even of the sides, having disappeared. The exposed dentine is darkly stained, and presents to the eye a polished appearance. An examination with a probe will show that the dentine is as hard as, if not harder than,

in the normal condition. This state, which is known as *spontaneous arrest of caries*, may be found in both the deciduous and permanent teeth; in the latter, the condition is most frequently met with in hypoplastic first permanent molars. It occurs only in teeth with living pulps, and the appearance of the teeth suggests that the disease, when active, was of a general and rapid character.

FIG. 39.—Teeth showing arrested decay.

Microscopical examination shows that the dentine retains the colour of carious dentine, or is perhaps slightly darker. The dentinal tubes, which can be easily seen in the discoloured part, end apparently upon the surface.

(G) SUSCEPTIBILITY AND IMMUNITY TO CARIES.

In connection with susceptibility and immunity of individuals to caries there are certain debatable questions to which brief reference must be made.

The Teeth.—Clinical experience shows that there is a marked difference in the hardness of teeth, and dental surgeons are accustomed to designate them as "hard" or "soft." As a general rule the former are of a yellowish colour, the latter of a bluish-white. Tomes and Black have shown that the dentine of "soft" teeth is not deficient in lime salts. The important portion of the tooth is, however, the enamel, which is the first to be

attacked; and recently S. Mummery has demonstrated that the rapidity with which different enamels are attacked by a solution containing a known strength of lactic acid varies within wide limits. Defects in the form and structure of the teeth influence the carious process by acting as lodging places for food either in a solid or liquid form. In a like manner, abrasions, bruises, and cracks diminish the resistance of enamel.

Putting aside defective and injured teeth, it would seem that the question whether a tooth will become carious depends partly upon its molecular structure, as suggested by Miller, partly upon the degree of concentration of the acid, and partly upon the time that the acid is allowed to act. The inference from this is that although it would not be safe to assume that any form of enamel is absolutely immune to caries, yet it is highly probable that a degree of acidity that would, in a given time, produce caries in one case would fail to do so in another. We can, therefore, only consider the word "immune" as applying to teeth in a comparative and not in an absolute sense. The importance of the above observations will be realized more clearly if it be remembered that the causes which lead to the destruction of enamel *act intermittently and during varying lengths of time.*

The quality of the tooth depends upon two factors, namely:—

(1) Congenital.

(2) Environmental during development of the teeth.

(1) *Congenital.* — The popular belief that certain families are more liable to caries than others is probably true. The greater liability to caries may be dependent upon the structure of the tooth or the nature of the saliva, but more often it depends upon family

habits. Inheritance, however, plays, if any, but a small part in the disease; and it is fortunate that this is so, seeing that we possess no means at present of artificially altering inherent tendencies.

(2) *Environmental.*—These are both pre-natal and post-natal. It is important that the health of the pregnant woman should be good, and that the feeding during infancy should be suitable. Opinions differ as to the effect of infant feeding on the susceptibility to caries.

Dr. Kingston Barton[1] finds, as a result of twenty years of observation, that breast-fed children have the best teeth; those fed on cows', asses', or goats' milk come next; and that when starch or any patent food is added to, or given in place of, cows' milk, the teeth, both deciduous and permanent, almost always suffer seriously. This subject has also been investigated by M. Michael[2], who obtained the following data. Out of 11,762 children examined, 7,763 had been breast-fed, and these showed caries to the extent of 11·46 per cent., or about 8 per cent. less than the average frequency. The average time of nursing was about six and a half months. In 122 cases the children had been nursed for ten months, and in these only 9 per cent. showed caries. In those brought up on cows' milk, the frequency of caries was 22 per cent., and rachitis 8 per cent.; and of 625 children brought up on oatmeal and water, Liebig's or Nestlé's food, the percentage with caries was 27 and with rachitis 16 per cent.

There is, however, no question that an infant, if artificially fed along physiological lines, may develop teeth of excellent structure.

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[1] *Medical Press and Circular*, vol. ii., 1899.

[2] *Brit. Journ. Dent. Science*, May 2, 1904.

The influence of lime salts in water.—Certain authorities consider that individuals living in districts where the soil and drinking water contain a plentiful supply of lime possess better teeth than those in districts where the lime is deficient. It has been contended that the great prevalence of caries in the city of Glasgow is due to the character of the water supply from Lake Katrine. It is, too, a common belief that the defective teeth of many sections of our colonists are due to the absence of lime in the water. In this connection, however, it must be remembered that the natives when still living in these places generally possess good teeth. The difference between the good teeth of the native and the defective teeth of the colonist is probably attributable more to the altered character of the foodstuffs than to the deficient supply of lime in the water.

The Saliva.—It is more than probable that the character of saliva influences the rate of fermentation of the food, but definite work has not been done on the subject. Mechanically, the saliva exerts an influence on the natural cleansing of the teeth. For example, a strong flow of saliva flushes the mouth and tends to remove particles of food from the teeth, while, on the other hand, a viscid saliva may conduce to caries by rendering the self-cleansing action of the mouth more difficult. Rose believes that a definite relationship exists between dental caries and the alkalinity of the saliva. He finds that a high alkaline reaction constitutes the best means of checking caries in its early stages and hindering its progress.

Pregnancy.—There is a current belief that, during the period of pregnancy, there is an increased susceptibility to caries, and it has been assumed that this is due to the absorption of the mineral matter from the teeth to form

fœtal bone. Physiologically there is no foundation for this belief. During pregnancy certain women often seem to be more liable to caries, but an explanation may be found in some change of the oral secretions rendering them a more favourable soil for the development of micro-organisms. It is possible that the vomiting of pregnancy may, to some extent, aid the development of caries. This apparent predisposition of the pregnant to caries is by no means constant, and in practice one meets with many instances of women with large families who have excellent teeth, and who develop no increased susceptibility to caries during the pregnant period.

(H) THE ETIOLOGY.

In the preceding pages caries has been shown to be a disease which is started by the solution of the inorganic matter of the teeth and continued by the peptonizing action of the bacteria on the remaining organic tissue, and that the acids or acid which are responsible for the decalcification are formed from the fermentation of carbohydrate food in contact with the tooth substance. It has been shown also that the earliest stage of caries occurs most frequently in two well-defined regions, namely, at and around the points of contact between adjacent teeth and in fissures or pits, and less frequently in positions where solid food usually lodges, namely, at the necks of teeth. The uniformity and the character of the lesions suggest that they are due to an acid fluid held against them by capillary attraction. It must therefore follow that, if the pathology of the disease be correct, the carbohydrate is in a state of solution. The rôle played by lodged solid food must not, however, be underestimated, for there is no question that with misplaced teeth and in the pits and fissures of teeth, solids may play an im-

portant part. Solid food would appear to act in three ways. Firstly, as a block or obstruction to the free flow of saliva between the teeth and into the fissures and pits. Secondly, as a sponge in which an acid fermentation and a growth of micro-organisms takes place, thus serving as a source of supply of acid, carbohydrate or organisms to the fluid held between the teeth. And thirdly, as a pabulum for increasing the number of acid-producing organisms in the mouth.

The problem of the etiology of this disease is to ascertain what changes have taken place in the food of man which have rendered it possible for the bacteria of his mouth to form acids capable of affecting the teeth. A brief review of the changes that have taken place in man's diet since prehistoric days will show at once how profound and important those changes are. Early man must have lived much as the anthropoids from which he sprang, upon the animals he could easily catch and the products of the uncultivated vegetable world. Thus, fish, birds, fleshy roots, leaves, shoots of young plants, and honey in small quantities formed his diet. It is believed that many thousands of years passed before man discovered the art of shaping weapons for the chase, or the means of fishing, and that after he had made these discoveries he passed through many centuries of increasing carnivorism. The next great discovery, that of *cooking*, brought within range of his diet sources of vegetable food, which hitherto had been too indigestible or unpalatable, or in some cases even poisonous, and thus led to an increased consumption of vegetable food and a proportionate falling off of animal food. Later, he acquired the knowledge of the cultivation of vegetables and the breeding of animals, and, still later, the means of storage against dearth or bad weather.

The introduction of cooking brought into man's diet food of an entirely different nature, namely, soluble, starchy and partly dextrinised carbohydrates, and this in a condition of increased fermentability. It was about this period that caries began to make its appearance. Since the discovery of tropical countries there has been introduced into this and all European countries a food that was hitherto only obtainable in fruits, and in the form of honey, namely, sugar. It may be added here that the Romans in their later days were people of advanced epicurean tastes, and indulged largely in honey and heavily sweetened wines. The Egyptians also were fond of sweet food, and we are told that the confectioner found constant employment in every town. Both these races suffered greatly from this disease.

The phenomenal rapidity with which caries has increased of late years has puzzled those who have studied the question. Considerable light has, however, been thrown on the question by those who contend that a solution is to be found in the altered character of the foodstuffs.

If we examine the foodstuffs as prepared for the table at the present day, we find that every care has been taken to eliminate the fibrous element, and that they are usually presented in such a soft condition as to require very little mastication; indeed, it is extremely difficult, even with the best intentions, to masticate such food. The process of mastication, therefore, remains in abeyance, and the result is that, owing to the pappy character of the food, and the absence of the cleansing operation of mastication, the food tends to cling around the teeth. Dr. Wallace contends that herein lies the true cause of the prevalence of caries. He says: "The cause of the prevalence of dental caries is that the natural foodstuffs are to a large extent ridded of their accompanying fibrous

parts and prepared and consumed in a manner which renders them liable to lodge and undergo acid fermentation in the mouth; while from the same cause and the induced condition the micro-organisms of the mouth lodge, multiply, and augment the rapidity and intensity of the acid fermentation."

In opposition to these views it is argued that there are numerous examples of races living upon soft food and yet not unduly liable to caries. The Kaffirs of South Africa are given as an instance. The porridge of the Kaffirs, which is their staple food, is prepared by bruising and crushing mealies, no attempt being made to eliminate the coarse and fibrous parts, and experience shows that such food *does* require a considerable amount of mastication.[1]

Although there is much to be said in favour of Dr. Sim Wallace's view, it is very doubtful whether it offers a complete solution of the problem. It is more highly probable that there have been other agencies at work tending to accelerate the progress of caries. The use of carbohydrates has probably had an important influence in this direction. *Carbohydrates as used at the present day are more easily soluble and fermentable than heretofore*, and within the last fifty years have undergone an extraordinary amount of variation. Flour is a typical example. In flour the change has been brought about by the altered method of milling. Milling with the old stone mills excluded no part of the grain except the greater portion of the husk, whereas with roller milling the germ is removed and practically all the bran, the flour consisting almost entirely of starch and gluten. With regard to the question of caries, bread made from stone-

[1] References to this question will be found in *Lancet*, September 8, 1900, p. 770; November 3, p. 1307; *Brit. Med. Journ.*, March 18, 1905, p. 629; April 1, p. 749.

milled flour requires more mastication and insalivation than that made from the roller-milled; still further, Mr. T. G. Read[1] is of the opinion that bread made from roller-milled flour becomes acid in the mouth far more quickly than that made from stone-milled. In ten tests it was found that on the average the acidity in bread made from stone-milled flour increased to a very slight extent in the process of mastication; whereas with bread made from roller flour there was an increase of 40 per cent. in the acidity after mastication. The experiments, although instructive, are not sufficiently comprehensive to enable a definite conclusion to be drawn. The subject, however, is deserving of serious attention, seeing that bread and flour form the most important item in the diet of the masses.

Clinical evidence supports the view that there is an intimate relationship between sugar and caries. The use of sugar as a foodstuff has increased enormously during recent years, and this food, it must be remembered, is readily soluble. Dr. Miller[2] would seem to have considered the view that the importance of sugar was overrated. He remarks: "At present sugar is universally regarded by dentists as well as laymen as injurious to the teeth." Again (p. 207), he states that "sugar, being readily soluble, is soon carried away, or so diluted with the saliva as to be rendered harmless," and he held that the chief rôle in the production of decay was performed by bread, potatoes, &c.

Cane and beet sugars are polysaccharides, and before fermentation must undergo the process of inversion;

[1] "Some Chemical Changes occurring in the Mouth during the Mastication of Bread composed of Roller Flour," *Journ. Brit. Dent. Assoc.*, vol. xxii., p. 590.

[2] "The Micro-organisms of the Human Mouth," p. 145.

glucose, on the other hand, is a monosaccharide and is, therefore, directly fermentable. Within the last thirty years glucose has been used extensively as a substitute for sugar, and enters largely into the manufacture of syrups, candies, jams, cheap sweets, &c. In the manufacture of glucose a gummy body, namely dextrine, is formed, and this possibly assists the retention of the sugar in the mouth.[1] The idea suggested, that glucose is rapidly fermentable, requires experimental proof, and the question of the varying rapidity of fermentation of the carbohydrates used as foods opens up a wide field for research. "There can be no doubt that a mistake has been made in the past in regarding all sugars as similar, and that, in the future, in their relation to caries, they must be looked upon as different substances, transformable, to some extent, into each other; just as peptone, being a proteid, is a changed form of other proteids, though possessing many different properties."

In a paper by Dr. L. Ottofy, on "The Teeth of the Igorots,"[2] the relation of sugar to caries is well brought out. In the Igorots of the Philippines, a semi-barbaric race, he found only 2·05 per cent. of carious teeth, while in the Filipinos, the more civilized inhabitants of the islands, the amount of caries was 20·90 per cent. The diet of the Igorots consists of food requiring considerable mastication; bread is unknown and sugar is distasteful to the children. The Filipinos, on the other hand, indulge freely in the practice of chewing sugar, which is sold in small pieces all over the islands, and Dr. Ottofy is convinced that the extensive amount of caries in the Filipinos is due to sugar. There is abundant clinical evidence to

[1] See paper, "The Problem of Dental Caries," S. Colyer, *Dental Record*, vol. xxiv., p. 301.

[2] *Dental Cosmos*, July, 1908.

show that the use of sugar is closely connected with the amount of caries. In practice it is found that children who eat sweets in large quantities will return from school each term with a plentiful supply of fresh and generally rapid caries, but when the eating of sweets is stopped the amount of fresh caries rapidly declines.

(I) THE SYMPTOMS AND DIAGNOSIS OF CARIES.

Caries in its early stage seldom gives rise to pain. If the caries is on the surface of the tooth the patient may be conscious of a cavity, while in cavities between the teeth, lodgment of food is often the first symptom noticed. Pain, when it does occur, is due to irritation of the pulp viâ the dentinal fibrils, and varies according to the situation of the cavity in the mouth and the extent of the cavity.

For the examination of the teeth for caries, a mirror, a pair of tweezers, and a good curved probe are necessary.

The examination should be systematic and the cavities present should be noted on a chart. The pits and fissures on the crown surfaces should be especially examined, the probe being firmly pressed into the various depressions. If caries is present the probe will readily enter the cavity and "stick." Commencing caries on the approximal surfaces usually produces a whitish opaque appearance of the enamel on the crown surface. If caries has progressed so as to form a cavity, the area of opacity will be greater and the probe passed between the teeth will catch in the existing cavity. In the front teeth caries should be looked for especially on the approximal surfaces near the gum margin.

The following method of notation is usually adopted. A horizontal line is drawn, the symbols placed above the line representing the upper teeth and below the line the

lower teeth. A vertical line is drawn to represent the middle line of the mouth. The teeth to the left of this line represent the right-hand teeth, to the right of the line the left-hand teeth. The teeth are represented by the figures 1 to 8 representing the teeth in succesion from the central incisor to the third molar. A carious tooth can be indicated by making an x through the number.

$$\text{R}\ \frac{8\,7\,6\,5\,4\,3\,2\,1\ |\ 1\,2\,3\,4\,5\,6\,7\,8}{8\,7\,6\,5\,4\,3\,2\,1\ |\ 1\,2\,3\,4\,5\,6\,7\,8}\ \text{L}$$

The deciduous teeth are represented by letters a to e, thus

$$\frac{e\,d\,c\,b\,a\ |\ a\,b\,c\,d\,e}{e\,d\,c\,b\,a\ |\ a\,b\,c\,d\,e}$$

(J) THE PREVENTION OF DENTAL CARIES.

Dental caries is a preventible disease. Although it is probable that the knowledge of its etiology is incomplete, it is possible to deduce from the facts known certain methods of prevention both natural and artificial which when carefully observed lead to most satisfactory results. For the purpose of clearness the methods may be divided into :—

(1) Natural.

(2) Artificial.

(1) *Natural Methods.*—These methods aim at leaving the mouth after a meal either free from fermentable carbohydrates or so diluted that any acid formed from them would be of insufficient strength to attack the teeth.

In ordinary life the order of food in meals should be regulated and every effort made to leave the mouth clean and free from fermentable carbohydrates. These results can be attained by ending a meal with a proteid or with substances containing very small quantities of fermentable carbohydrate, such as nuts, apples or other fresh

fruit, not, be it noted, with preserved fruits, such as dates, figs, or plums. If such a practice is regularly observed it is probable that it would be unnecessary in many cases to take any further precautions, more especially if no food is taken for a few hours before going to bed.

(2) *Artificial Methods.*—Whilst it is possible that caries in a race could be greatly diminished by altering the diet of the race, yet to neglect the artificial methods by which it is certain that caries can be prevented would be a short-sighted policy. There are two features of this disease which give the key to artificial prophylaxis. The first is the fact that a clean tooth does not decay; and the second, which is only clinically supported, is that, except in the case of exceedingly susceptible teeth, caries mostly occurs at night, when the functions of the mouth are in abeyance and the organisms are given ample time to bring about the acidic change in food. Those, then, who have the opportunity to clean their teeth after every meal are advised to do so, otherwise it is absolutely essential that the teeth should be cleaned before going to bed. *After this final cleaning for the day no food must be taken.*

The method of cleaning teeth is one of extreme importance, and it is on account of the absence in it of certain details that the toothbrush has come to be looked upon in some cases as useless. A small, hard toothbrush with bristles of different lengths should be used. The brush should be applied to all surfaces of the teeth, and its action may be aided by a mixture of chalk and Castille soap. After the brushing, the mouth should be thoroughly rinsed out with pure water. The brush clears away the solid débris; the water dilutes and carries away the dissolved carbohydrate held by capillary

attraction between the teeth, and in deep fissures on crown surfaces. *The second part of the process is absolutely essential in order to cleanse those portions of the teeth which the brush fails to reach.*

The following is a summary of the means of prevention.

(1) Insistence, when possible, upon breast-feeding.

(2) The early use of foods requiring some effort in mastication.

(3) The arrangement of the order of meal, so that the mouth is left clean and free from carbohydrate residue.

(4) The proper cleansing of the teeth, when possible, after every meal, *always before going to bed.*

(5) No food containing carbohydrate should be taken after the final cleansing for the day.

It may be added that a functional mouth is necessary if caries is to be prevented. Two important factors militate against a functional mouth, mouth-breathing and tender teeth. Any cause of nasal obstruction should, therefore, be removed, and all tender teeth extracted.

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CHAPTER IV.

Diseases of the Pulp Tissue.

THE local adaptive changes resulting from injury to the pulp are generally known as "pulpitis." Two grades of inflammation are recognized, namely, the acute and the chronic; in the former, the development of the inflammatory process is rapid, and is accompanied by marked clinical symptoms, while in the latter the reaction is slow in development and is accompanied by feebly marked clinical signs, indeed, in some instances, no clinical signs are apparent.

(A) ACUTE PULPITIS.

The *commonest cause* of acute inflammatory reaction in the pulp is injury from bacterial toxins. The caries of the dentine progresses until the pulp chamber is invaded and the toxins formed in the carious cavity can thus gain an entrance to the pulp. Other causes are trauma and injury from physical and chemical agents.

Symptoms.—The dominating symptom of acute pulpitis is a sharp shooting pain, often of a throbbing character, which is generally more severe at night when the patient assumes the horizontal position. Thermal changes also lead to severe paroxysms of pain, although in the early stages of acute inflammation cold produces relief by constricting the blood-vessels. Acute pulpitis must be distinguished from acute periodontitis, and the main points of difference are briefly as follows :—

ACUTE PULPITIS.	ACUTE PERIODONTITIS.
Pain sharp, throbbing or lancinating, intermittent and reflected.	Pain dull, heavy, and constant.
Thermal changes to the teeth cause pain.	Thermal changes do not cause pain.
Pressure or tapping on the tooth causes no pain.	Pressure or tapping on the tooth causes pain.
Slight pressure on a piece of cotton-wool in the cavity generally causes acute pain.	Slight pressure on a piece of cotton-wool in cavity does not cause pain, except through pressure transmitted to the periodontal membrane.

Attention to these points will assist in diagnosis, but it must not be forgotten that with acute inflammation of the pulp there is at times a slight inflammation of the periodontal membrane through continuity of the tissues.

Treatment.—The alleviation of the pain of acute pulpitis can usually be brought about as follows: (1) Gently syringe out the cavity with a stream of luke-warm water; (2) dry the cavity with a small pledget of cotton wool and then (3) insert into the cavity a small pledget of cotton wool dipped in pure carbolic acid, and cover this dressing with a further pledget of cotton wool dipped into a solution of gum sandarac, or mastic, &c.

(B) CHRONIC PULPITIS.

Chronic pulpitis follows on injuries similar to those leading to acute pulpitis. The reaction of the tissues in chronic pulpitis is different from that seen in the acute form, and this is due to the fact that in chronic pulpitis the injury is constant, though of lesser intensity.

Where the pulp cavity is exposed, and the pulp tissue is injured by constant doses of bacterial toxins suppuration usually occurs, and the pulp tissue is destroyed by a progressive ulceration. In other cases the effect of the irritant is to cause an overgrowth of tissue (*hyperplasia*), giving rise to a flesh-like mass in the carious cavity. A section through the growth will show that it is mainly composed of granulation tissue, which has a tendency to develop into fibrous tissue. In cases where the pulp is not exposed fresh tissue forms in the pulp cavity and changes, such as fibrosis and fatty degeneration, may occur.

Symptoms.—The symptoms arising in connection with chronic pulpitis vary according to the type of injury and the susceptibility of the patient to pain. Some individuals whose mouths contain several teeth with chronically inflamed pulps experience no objective symptoms; while others suffer severe pain, both local and referred, with the slightest injury to pulp tissue. Suppurative pulpitis in the early stages is generally accompanied by pain to thermal changes. In the later stages, however, cold gives some relief, while heat causes intense paroxysms of pain. This symptom of *increased pain to heat is almost diagnostic of a suppurating pulp.* As the suppuration approaches the apex the inflammation spreads to the periodontal membrane, and symptoms of inflammation of that tissue appear. Pain is more marked during the night than during the day, and is very likely to become wandering in character. It may be referred to another tooth, or to other parts of the head. In exposed cavities the act of mastication will usually cause pain. When the pus is confined, for instance, in suppurative pulpitis under a filling, the pain may be intense. It is usually constant in character,

with acute exacerbation on the application of heat to the tooth. Opening the pulp chamber in these cases gives almost instant relief.

In cases where there is overgrowth of the pulp, symptoms may be altogether absent, the surface of the pulp being insensitive to touch. When the overgrowth of the pulp has encroached on the carious cavity and is fibrous in character, it must be diagnosed from localized hypertrophies of the muco-periosteum. The points of differences are: In the former, absence of pain to pressure, and no great liability to hæmorrhage; in the latter extreme sensitiveness to pressure and liability to hæmorrhage on slight injury. A careful examination will show that the growth in the one springs from the pulp chamber, and in the other from the gum around the neck of the tooth.

When the inflammatory process follows an injury other than bacterial toxins, the only symptom may be slight discomfort to thermal changes, and even this symptom may be absent.

Treatment.—When the chronic pulpitis arises from infection viâ the tooth cavity or periodontal membrane, the pulp must be removed. In cases where suppuration is present, the surface of the pulp should be rendered as aseptic as possible before the removal of the pulp is attempted.

CHAPTER V.

Diseases of the Periodontal Membrane.

THE local adaptive changes resulting from injury to the tooth membrane are generally known as "periodontitis." Two grades of inflammation may be recognized, namely, the acute and the chronic. In the former, the development of the inflammatory processes is rapid, and is accompanied by marked clinical symptoms; while, in the latter, the reaction is slow in development, and the clinical signs are either very feebly marked or absent altogether.

(A) ACUTE LOCAL PERIODONTITIS.

The main cause of acute inflammation of the periodontal membrane is injury from the toxic products of bacteria, which may reach the membrane viâ the pulp canal or the gingival margin—a good example of injury viâ the gingival margin being seen in cases of suppuration in connection with partially erupted mandibular third molars. Other causes are traumatism and the use of strong chemical agents in treating the pulp cavity. When the inflammation arises from bacteria, suppuration frequently supervenes, and gives rise to a dento-alveolar abscess. When once pus has formed it may:—

(1) Become circumscribed, and the condition more or less chronic.

(2) Work towards the surface and point.

(3) Burrow along the fascia and the muscles.

When the pus becomes circumscribed the inflammatory process subsides, and a zone of granulation tissue forms around the pus and encloses it. The abscess may then remain inert, but more commonly the suppuration slowly spreads and gives rise to a type of chronic abscess.

When the pus works towards the surface it follows the direction of least resistance. It may escape at the gingival margin, but in the majority of cases it works its way through the outer alveolar wall and points on the gum over the affected tooth. As long as the pus is confined in the bone, the muco-periosteum is only slightly œdematous, the cellular tissue of the face being unaffected. But when the pus has gained an exit through the alveolar process, a rapid swelling of the cellular tissues takes place owing to a diffuse cellulitis. The extent of the swelling is determined by the virulence of the organisms producing the suppuration and by the resistance of the tissues. The abscess may discharge spontaneously, and, if the tooth or source of infection be removed, healing by granulation usually ensues. If the source of infection remains, the surface is kept constantly infected and a chronic sinus results.

When the pus burrows along the fascia and muscles it may lead to troublesome complications. It may pass to the gingival margin through the outer alveolar plate. In suppuration around the maxillary central, and, occasionally, the lateral incisors, the pus may make its way into the nasal fossa. Pus from the region of the maxillary lateral incisors is prone to burrow under the muco-periosteum of the hard palate, and form a large fluctuating swelling which usually points at the junction of the hard with the soft palate. Although this con-

dition occurs most frequently in connection with the lateral incisor, it may be associated with the second premolar, the first molar, or indeed any tooth in the maxilla. The pus may take a direction towards the antrum, invade this cavity and lead to antral suppuration. When this occurs it is usually in connection with the second premolar or the molars, but may arise in connection with any tooth in the maxilla. Pus around the canine may burrow upwards towards the inner canthus of the eye, and, in one case seen, an abscess in connection with the maxillary first premolar pointed near the angle of the mouth.

In the mandible, the pus, instead of working its way through the outer alveolar plate, may penetrate the inner side of the alveolar process, and open on the floor of the mouth. From the incisors and canines, the pus, especially if penetrating the outer alveolar plate low down, may strip up the periosteum and eventually open under the chin. The insertion of the buccinator influences, in many cases, the direction taken by the pus. If the pus penetrates the bone outside the attachment of the buccinator, it will burrow into the tissues of the cheek, and ultimately, perforating the skin, open on the face. In very rare cases the pus may continue to work among the planes of connective tissue in the neck, producing a diffuse cellulitis (angina Ludovici).

Signs and Symptoms.—In the early stage, the tooth causes the patient discomfort, is slightly raised in its socket, and pressure brings relief. At this period, the blood vessels are in a condition of hyperæmia. The tooth then becomes still further elongated and loose, the gum around it being swollen and painful. Pressure now causes great pain. The vessels at this period are nearing the condition of stasis, and the surrounding tissues are

infiltrated with inflammatory exudation, hence the increased rising of the tooth in its socket. Pressure now increases the pain, because the vessels can no longer be freed of their surplus supply of blood, and each act of mastication only increases the pressure on the already hypersensitive nerves. If suppuration supervenes, the swelling of the gum increases, the tooth becomes more loose, and the pain dull and throbbing. Finally, a distinct fluctuating swelling appears in the sulcus between the gum and the cheek, the face swells quickly, and, the pus being no longer confined in a dense unyielding structure, the tension upon the nerve endings is relieved and the pain considerably lessened. Suppuration is usually accompanied by pyrexia.

Diagnosis.—The diagnosis is usually easy. In all cases where suppuration has taken place, and the surrounding teeth have become implicated in the inflammation, the tooth causing the abscess may be recognized by the fact that it is more movable than its neighbours, and more sensitive to pressure and percussion.

Treatment.—When the periodontitis is due to bacterial toxins the first point to decide is the advisability of retaining the tooth. Teeth that are unsavable by conservative operations should be removed.

The pain in the early stages may be relieved by swabbing the gum with strong tincture of iodine. The pain, however, of acute periodontitis is difficult to relieve, and when persistent the most efficacious remedy is the frequent use of hot fomentations. The administration of a smart saline purge will often be found useful.

If suppuration has occurred, measures must be taken to remove the pus as soon as possible. The removal of the pus may be hastened by the continued application of poultices over the situation of the tooth, and, as soon

as the pus has pointed, the abscess should be opened. The cavity should be thoroughly irrigated with some non-irritating antiseptic solution, and drainage provided. The abscess should be dressed at least twice a day, and this is especially needful when it occurs in the mandible, as the opening is not then in the most dependent part. Acute dento-alveolar abscesses usually heal rapidly, especially if carefully dressed. In the case of extensive abscess, the patient must be anæsthetized, and the suppurating tracts freely opened, irrigated and packed.

(B) CHRONIC LOCAL PERIODONTITIS.

Causes.—The injuries causing chronic periodontitis are similar to those which lead to the acute form, and the difference in the reaction of the tissues in chronic periodontitis is due to the lesser intensity of the injury and its constant presence. The great majority of cases of chronic periodontitis arise from infection viâ the pulp canal. In a few instances, the injury may be caused by the passage of a root filling through the apical foramen.

The character of the reaction in the periodontal membrane varies. We may have (1) a proliferative periodontitis leading to the formation of new tissue on the root of the tooth, a condition often designated exostosis (figs. 40 and 41).

(2) A rarefying periodontitis leading to the absorption of portions of the hard tooth tissue (figs. 42 and 43).

(3) A suppurative periodontitis, leading to the formation of a chronic abscess.

The last condition calls for more than passing notice. The pus formed from the degeneration of the tissues may become more or less confined through being surrounded by a layer of granulation tissue, the outskirts

of which show a tendency to fibrosis, and the abscess thus formed may remain stationary and give rise to no symptoms. More often, however, there is a tendency for the suppurative process to extend slowly, usually in the direction of least resistance, with the result that the alveolar process is destroyed, an opening appears on the gum, and a sinus is formed.

FIG. 40.

FIG. 41.

FIG. 42.

FIG. 43.

In discussing acute suppuration it was explained at what positions the abscess might be expected to point. The same positions may also be regarded as the probable sites of the pointing of chronic abscesses. The destruction of bone resulting from chronic suppuration is at times extensive, especially in the maxilla, which is more susceptible because of the cancellous character of the bone. In fig. 44 is shown a radiograph of a large cavity which has been formed in the bone by suppuration in connection with a maxillary lateral incisor.

At times suppuration from the maxillary incisor

teeth spreads to the floor of the nose, leading to the formation of a sinus. In the premolar and molar region, the suppurative process frequently extends to the antrum and gives rise to antral suppuration. In a few cases, suppuration around the palatine roots of the molars may extend to the nasal fossa. In the mandible, the extension of the abscess is more limited, owing to the denser character of the bone, but at times very extensive excavation of the bone occurs. With the extension of

FIG. 44.—Radiograph showing extensive destruction of bone caused by suppuration in connection with a maxillary lateral incisor.

the suppuration, fresh bone is deposited by the periosteum, and, in this manner, the mandible may become considerably expanded.

Signs and Symptoms.—In teeth where the injury is slight, leading to a *proliferative periodontitis*, symptoms are often absent, and when present, seldom amount to more than a slight tenderness on percussion and occasionally attacks of pain of a dull, gnawing character. *When the inflammatory process is of a rarefying type*, the

principal symptom will be slight pain and tenderness on percussion, and if the inflammation is near the apex and the pulp is alive, there will usually be signs of pulpitis through extension of the disease to the pulp. The gum covering the tooth may be swollen. As the tooth substance disappears, increased looseness will be noticeable, the tooth becoming more tender, and the pulp and the gum more congested. In cases where the diagnosis is not clear a radiograph should be obtained.

A chronic suppurative periodontitis accompanied by a sinus will exhibit but few symptoms. The tooth is frequently free from pain, the patient only complaining of the presence of the "gumboil," which periodically swells and bursts. The sinus is usually situated over the root of the tooth. In cases of sinus on the face, some little difficulty may be experienced in tracing the offending tooth, especially if two or three teeth are pulpless or septic. A digital examination of the sulcus will often disclose a fibrous-like cord running from the base of the offending root to the cheek. The root or tooth causing the trouble may be buried in the gum.

When a sinus is not present (*chronic dento-alveolar abscess*) the tooth is usually slightly tender to percussion, the amount of pain varying according to the activity of the inflammation. The gum over the root of the tooth is at times swollen and congested, but it may be quite normal. A swelling can, as a rule, be felt above the level of the apex of the tooth; this swelling is usually sensitive. In cases in which the maxilla or mandible is much involved, there is often little or no pain or tenderness. A chronic dento-alveolar abscess must be diagnosed from dental cysts and other fluid swellings of the jaws.

Streptothrix Infection.—The question of streptothrix

infection in connection with the lesions of the teeth may be conveniently discussed under "chronic local periodontitis," inasmuch as the infection often takes place viâ the periodontal membrane.

Streptothrix infection in connection with lesions of the teeth may involve the bone itself or the soft parts, the latter condition being more usually met with.

Clinical Features.—A swelling appears on the lower, and less commonly, the upper jaw, and gradually increasing in size, invades the adjacent tissues. At first the mass has a regular and even surface, with the skin slightly hyperæmic. With the progress of the disease, the tissue at places softens and nodular excrescences appear of a yellowish colour; these burst, giving exit to a semi-glutinous fluid in which small granules can usually be observed. The granules vary in colour, being yellowish, yellowish-white or quite clear. The wound generally heals, but is followed by a fresh breaking-out at another spot. The cicatrization following the repetition of this phenomenon gives the surface a curiously characteristic puckered appearance. If left untreated the disease spreads, involving the tissues of the neck and, in some cases, passing upwards to the temporal region. The sinuses formed by the breaking-down of the tissue may persist, and a probe passed down them will often show that they lead to bare bone, but not necrosed bone. On examination, the swelling is found to be of a hard, brawny nature, and to diminish gradually into the unaffected tissue. The disease is not accompanied by a rise in temperature, and there is usually an absence of pain, but should acute suppuration supervene, the general symptoms of an ordinary septic abscess appear.

Diagnosis. — Streptothrix infection can be differentiated:—

(1) From dento-alveolar abscess by the hard, brawny character of the swelling, the long continuance of the disease (as compared to the duration of a dento-alveolar abscess), and by the presence of the multiple sinuses or the puckered cicatrix, the last being almost pathognomonic.

(2) From tubercle streptothrix infection may be differentiated by the fact, that in the former the glands are usually affected, and the swelling is generally well-defined and movable.

(3) From necrosis of the jaw it may be diagnosed by the presence of bare and not necrosed bone. The presence of granules in the discharge and the demonstration of the streptothrix on microscopical examination are positive evidence.

Pathology.—The disease is due to the presence of a form of streptothrix in the tissues, but the ray fungus of Bollinger (*Streptothrix bovis communis*) is an uncommon cause of human infection. The lesion produced by the growth of the streptothrix is of a chronic inflammatory character. A section through the affected tissue shows a number of rounded areas, in the centre of which is the streptothrix surrounded by a zone of granulation tissue. The tissue breaks down, and the streptothrix is expelled in the discharge. A. G. Foulerton[1] has identified six distinct species of streptotricheæ in man.

Method of Infection.—The infection is generally stated to be conveyed by straw, cereals, &c. The parasite probably gains an entrance to the tissue viâ the periodontal membrane, and thence spreads to the neighbouring soft tissue. In specimens of actinomycosis in

[1] *Lancet*, March 5, 1910, p. 626.

oxen the invasion of the tissue viâ the periodontal membrane is often clearly demonstrated.

Treatment.—Locally, the softened spots must be incised and freely scraped, and the sinus packed with iodoform gauze. Internally, iodide of potassium must be administered, in doses increasing up to 50 or 60 gr., three times a day. It is also useful to add 3 to 4 minims of liquor arsenicalis, as this drug is considered by Kellock[1] to have some effect on the disease itself, in addition to assisting in preventing the skin lesions.

The prognosis is good, but the treatment may have to be carried out continuously for a long time.

(C) CHRONIC GENERAL PERIODONTITIS (PERIODONTAL DISEASE, PYORRHŒA ALVEOLARIS).

This disease is peculiarly insidious in its progress, and is far-reaching in its effects on the general health. The disease consists in a progressive destruction of the tooth socket, accompanied sometimes in the earlier, and almost always in the later stages, by a free discharge of pus from the gum margin. The familiar term for the disease, pyorrhœa alveolaris, has led to considerable misconception, and the term "general periodontal disease" is more suitable.

General periodontal disease commences by a slight destruction of the tissues in the inter-approximal spaces, the result of injury from accumulation of food débris, &c. The margin of the alveolar process becomes involved at an early stage, and is slowly but progressively destroyed, the tooth eventually falling out. The gum is involved in the disease, but recedes at a slower rate than the alveolar process.

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[1] "Actinomycosis of the Mouth and Face," *Trans. Odonto. Soc.*, vol. xxxvii, p. 29, 1904.

Maxillæ showing varying stages of periodontal disease are shown in figs. 45 to 49.

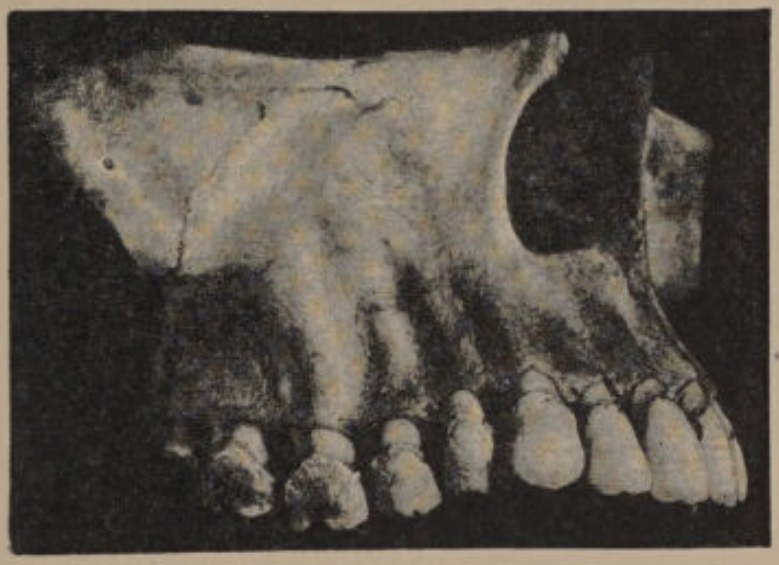


FIG. 45.[1]

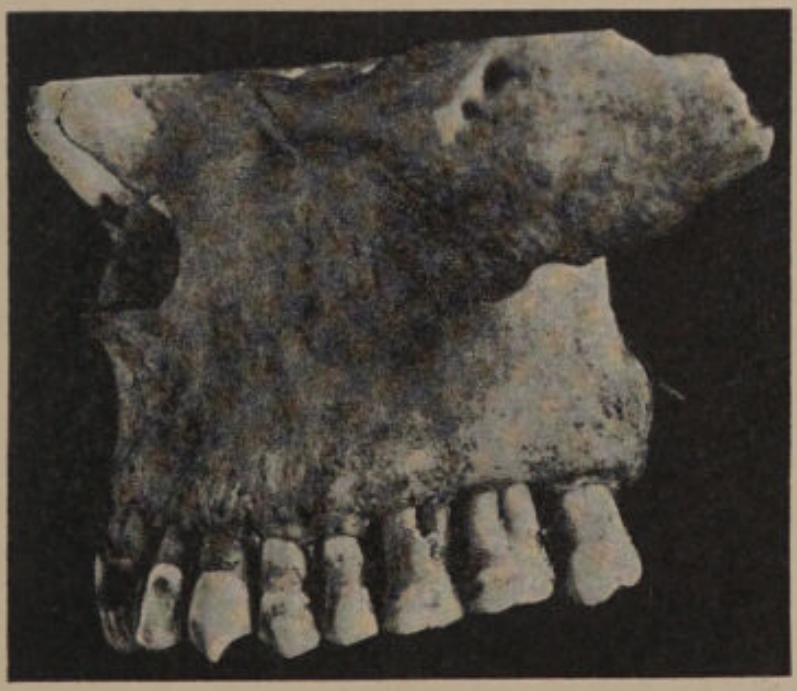


FIG. 46.[1]

The disease is frequently met with amongst the domestic animals and wild animals that have been kept

[1] From *Trans. Odonto. Soc.*

FIG. 47.

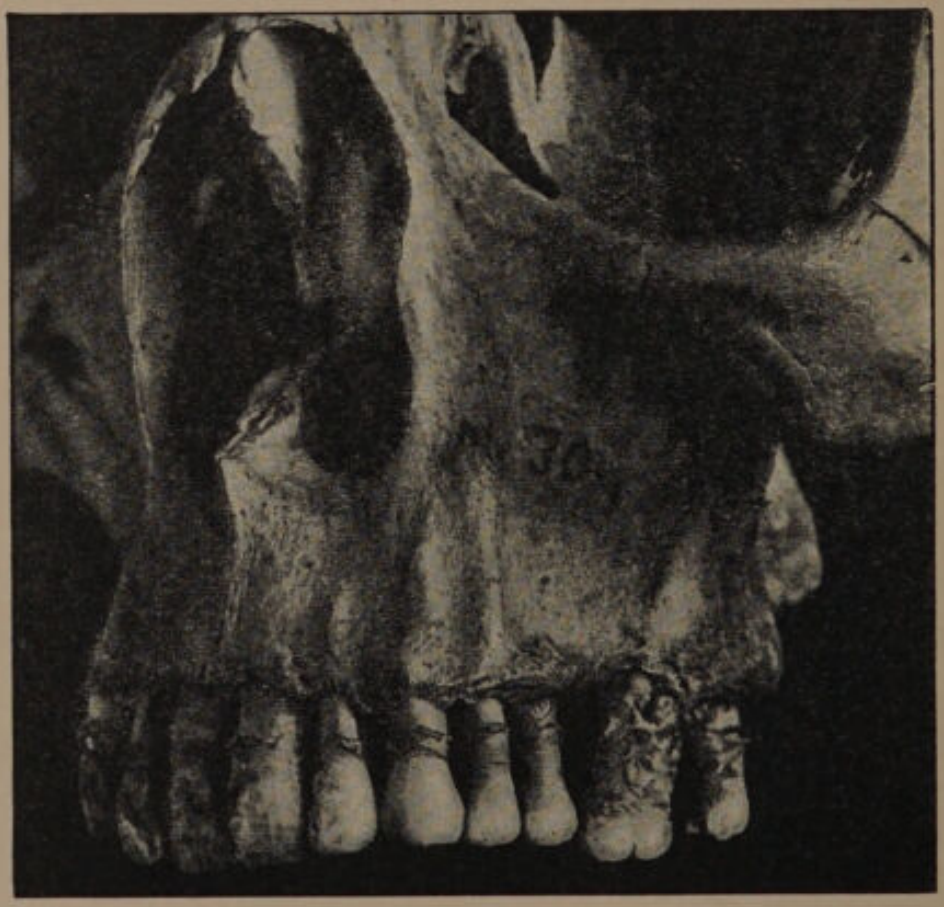


FIG. 48.[1]

[1] From *Trans. Odonto. Soc.*

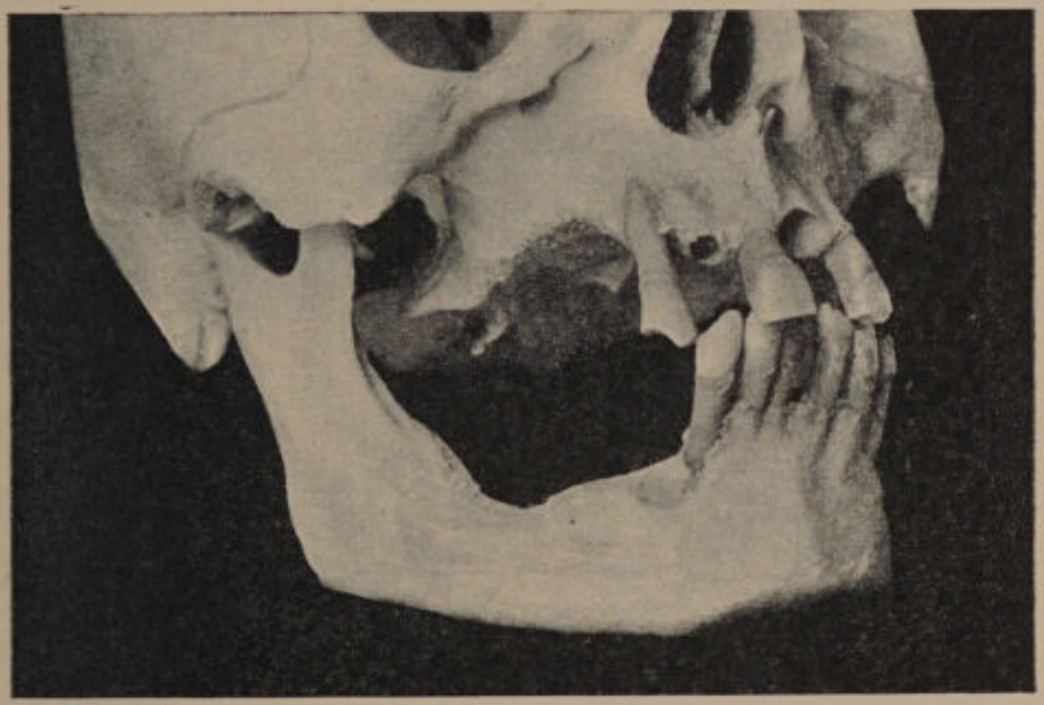


FIG. 49.

FIG. 50.[1]

[1] From *Trans. Odonto. Soc.*

in captivity. Figs. 50 to 52 are samples of the disease in a Panolia deer, a hyæna and the domestic dog, respectively.

FIG. 51.[1]

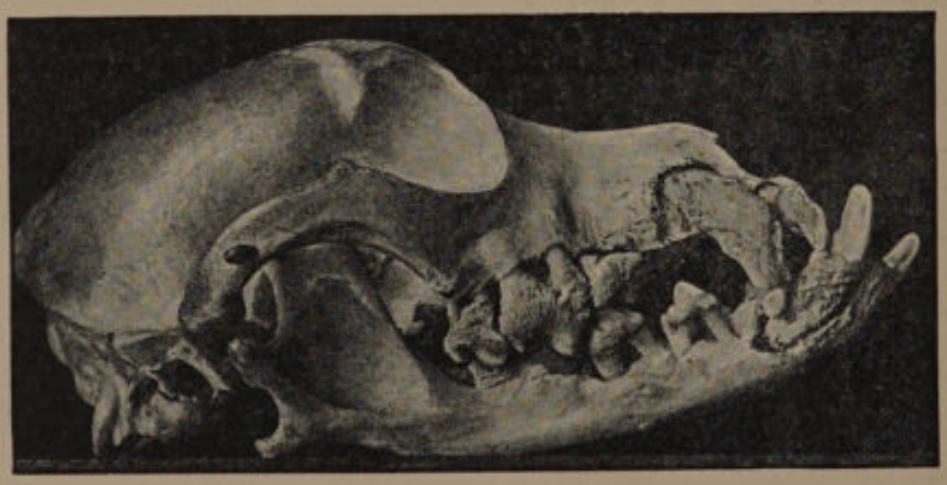


FIG. 52.[1]

Clinical Appearances.—In a young adult whose mouth is in a normal condition it will be noticed that the gums fit closely around the necks of the teeth, the spaces

[1] From *Trans. Odonto. Soc.*

between the teeth being filled with tags of gum. In *the earliest stage of the disease* there is a slight congestion of these tags of gum, and by passing a probe into the interproximal spaces it can be shown that the gum has to some extent been destroyed. The whole of the margins of the gums gradually become congested and bleed readily. The tags of gum between the teeth then gradually disappear and the normal festoons become obliterated. An examination of the teeth at this stage will usually disclose food and other débris in the approximal spaces, while small nodules of calculus are generally present on the teeth. If the gums are compressed, a small quantity of discharge may be caused to escape at the sides of the teeth. Finally, the bone becomes involved in the inflammatory process, and, together with the periodontal membrane, is gradually destroyed. The gums recede, but the recession does not as a rule proceed as quickly as the destruction of the alveolar process. The result of this is that deep pockets are formed around the teeth in which pus and other morbid material accumulate and aggravate the condition.

At this stage the mucous membrane appears deeply congested and the gums are swollen at their free margins and bleed readily on the slightest touch. The roots of the teeth are more or less exposed, and covered with a layer of hard greenish-brown calculus ; there is a fœtid discharge and the breath has a repulsive odour due to indol-forming organisms. A sickly, sour odour of the breath is noticeable when yeasts and bacteria causing carbohydrate fermentation are present. The teeth are freely movable and may become so loose that they can be removed quite easily. If the disease is left untreated, the teeth are lost one by one, and with their loss the inflammatory process completely clears up.

In advanced cases superficial glossitis is often present, and the tonsils and pharyngeal mucous membrane are also inflamed. An unpleasant taste is experienced in the morning. There is constant hæmorrhage from the gums, which is swallowed during the day but occasionally escapes from the mouth at night and stains the pillow. The latter symptom is worthy of special attention, as it might be mistaken for hæmorrhage from the lungs.

The *clinical appearances of the disease*, which *vary considerably*, are determined by the general condition of the patient, the resistance of the tissues, and the hygiene of the mouth. At one period the condition may be acute, with a free flow of pus from the teeth sockets and a rapid destruction of the tissue. This may be evanescent and may be followed by a period during which the inflammation is only slightly marked. In some cases the teeth are comparatively free from calcareous deposits; in other cases they are completely coated. The disease, too, may be more active around the anterior than the posterior teeth or *vice versâ*. Again, the destruction of tissue may be more active in one region of the mouth than another. This is exemplified in cases where the incisors and canines are affected, the destructive process being often more advanced on the palatal than on the labial aspect.

In severe cases the disease may spread, in the maxilla, to the antrum, or in the mandible to the body of the bone.

"The buccal mucous membrane in advanced cases shows a number of small white stellate points, somewhat hard and shotty, lying underneath the mucous membrane, and corresponding in position to the site of the disease of the gum margin. From these glands bacteria

of various species, generally bacilli, may be obtained." (Goadby).

In some cases, where the hygiene of the mouth receives careful attention, the gums appear fairly normal in colour, and abnormal looseness of the teeth is the only indication of the presence of the disease. In these cases the probe discloses deep pockets, and pressure on the gums produces a discharge from the

FIG. 53.[1]

teeth sockets. In a few cases the disease is slow in its progress, and the surrounding bone readily reacts to the injuries caused by the toxic products in the sockets. This is well shown in the case shown in fig. 53.

In certain cases there seems to be a tendency for granulation tissue to form, causing the condition to

[1] From *Trans. Odonto. Soc.*

simulate sarcoma. In one case under observation, associated with myxœdema, the gums were friable and presented a translucent appearance.

The disease may often be well advanced before the patient is conscious that anything is wrong, the only symptom being a tendency for the gums to bleed readily, and this is often unheeded by the patient. With the formation of spaces between the posterior teeth, neuralgia, arising from irritation of the exposed cemental

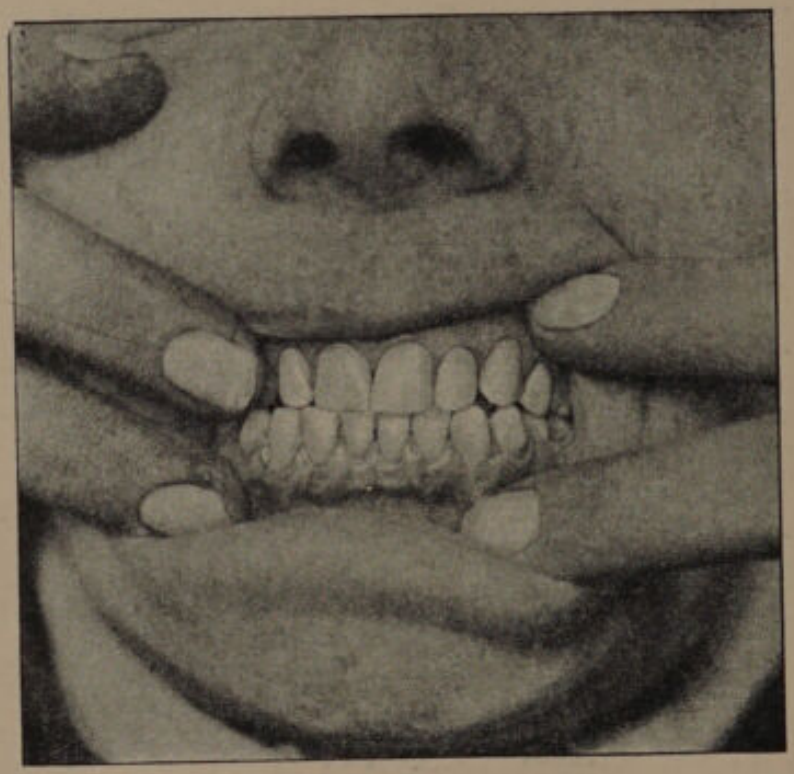


FIG. 54.

tissue, may be present. In the later stages there is often persistent neuralgia owing to the main trunks of the nerves being involved in the diseased process. Where pus formation is active, the patient will often complain of a general itching of the gums.

Whether the disease can be transmitted from one individual to another is an open question, but the balance of evidence is rather against it.

Clinical appearances alone are not altogether satisfactory guides as to the extent of the disease, and it is only by the aid of radiography that the amount of bone destruction can be estimated with any degree of accuracy. The following cases will serve to demonstrate that the

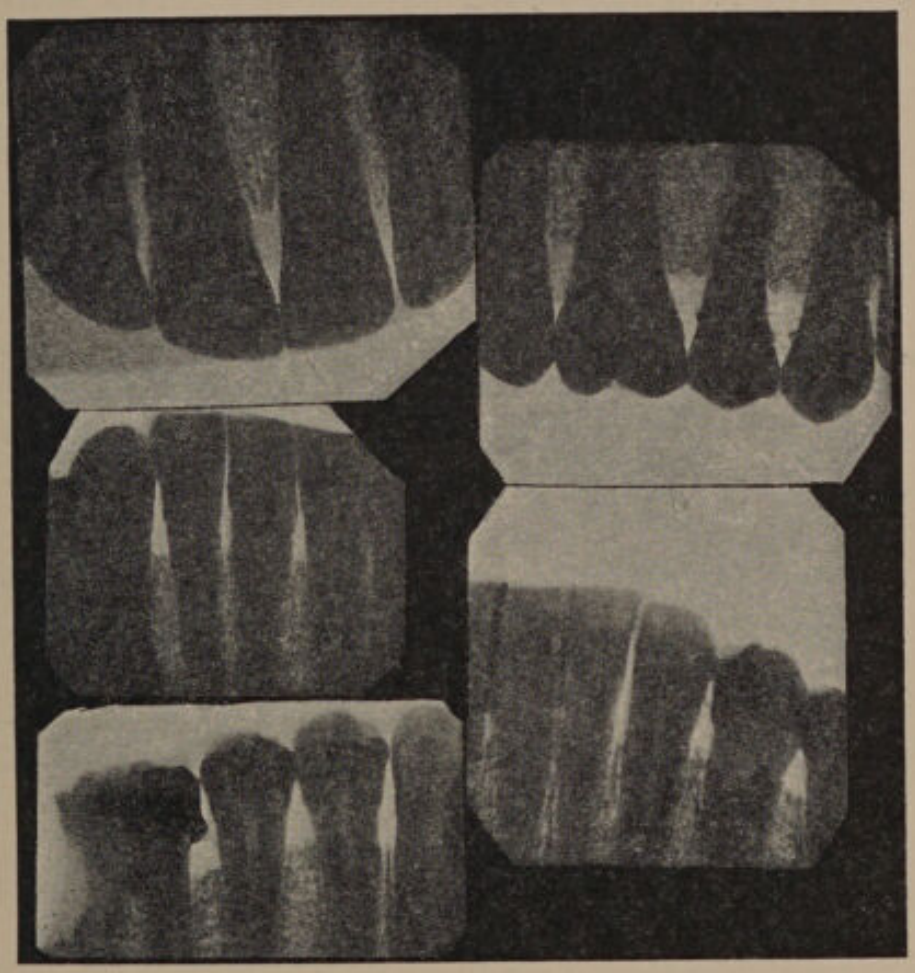


FIG. 55.

amount of bone destruction is greater than the clinical appearances would seem to support.

The case shown in fig. 54 depicts an early stage of the disease. Clinically there is no recession of the gums beyond a partial disappearance of the interdental papillæ between the lower incisors. There was a marked marginal gingivitis, together with a congestion

of the whole of the gums. There was a free discharge from the pockets around the teeth. This patient had a well-developed arch and there was marked attrition. The radiographs (fig. 55) show that the alveolar process is already involved, and that the destruction of bone has advanced to a considerable extent in the lower incisor region.

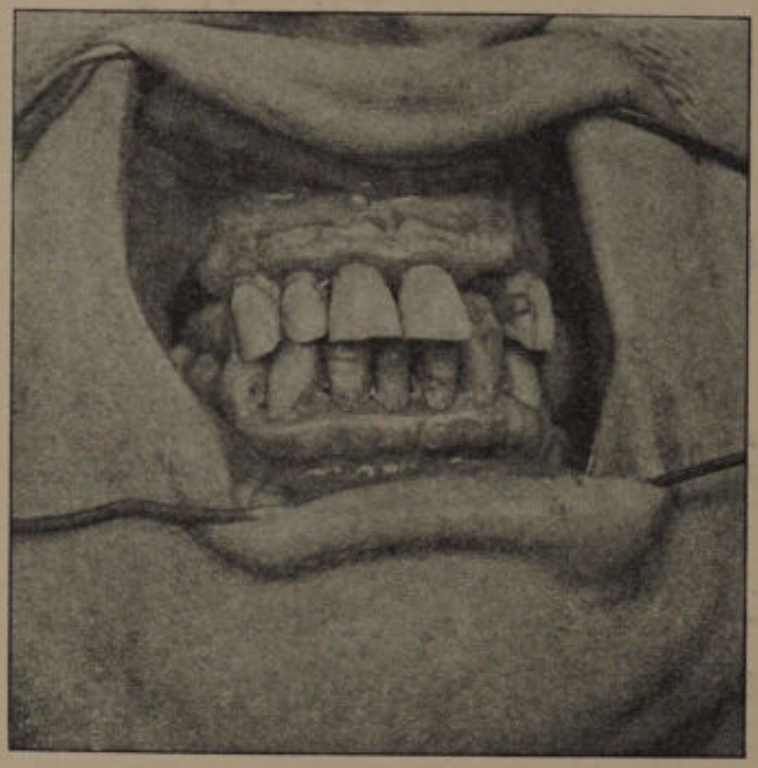


Fig. 56.

In the case shown in fig. 56, the clinical appearances suggest a more advanced condition of the disease than in the preceding case. The muco-periosteum is more swollen and congested. The interdental papillæ have quite disappeared, but the gum margin on the labial aspects of the teeth just fails to cover the necks of the teeth.

The radiographs show that the bone destruction is much more advanced than the gum recession. The level of the gum is well shown in the radiographs (fig. 57).

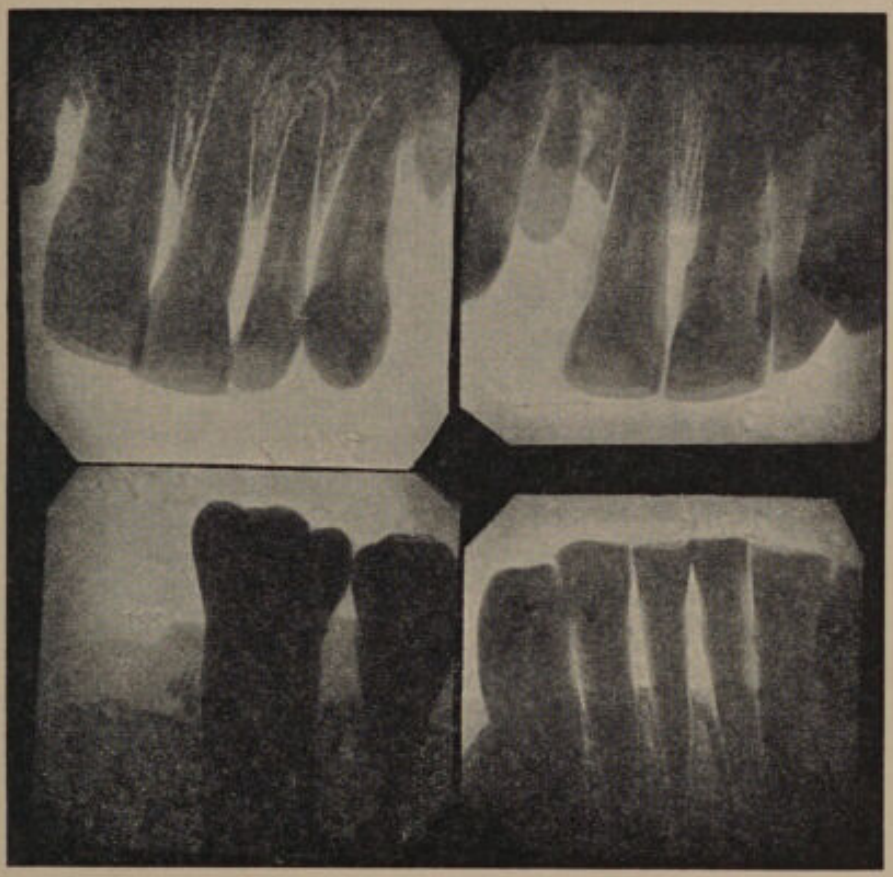


FIG. 57.

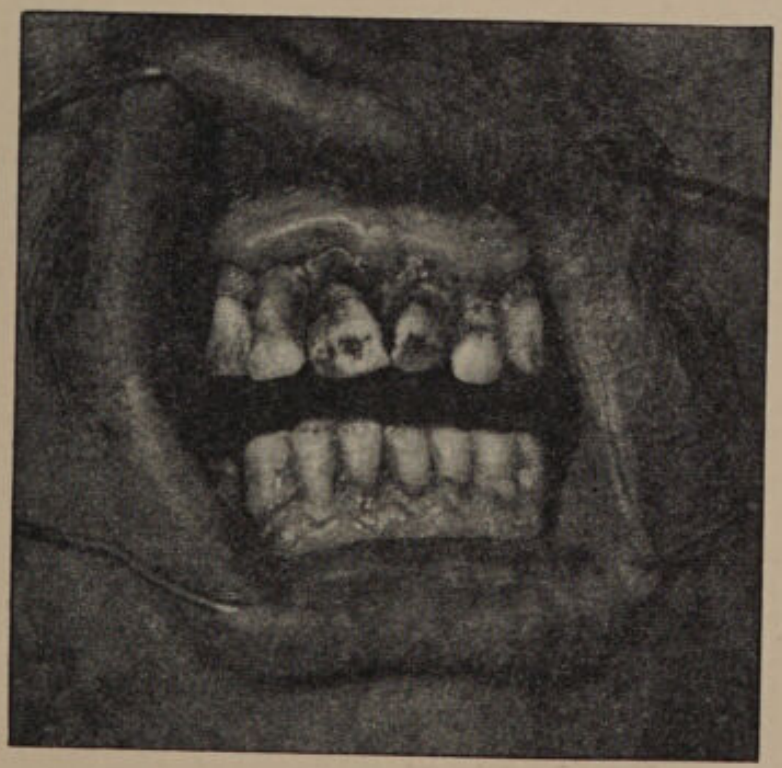
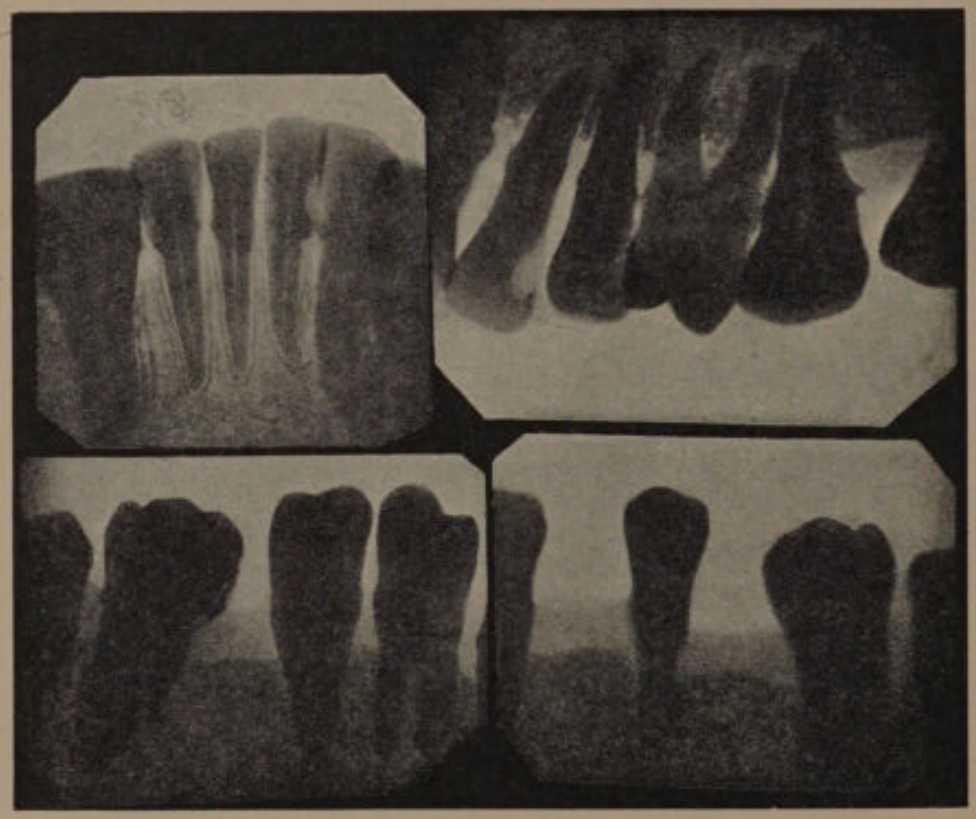


FIG. 58.

The case shown in fig. 58 illustrates an advanced condition. The radiographs (fig. 59) show the extensive destruction of the teeth sockets. Those marked *a* and *b* are also instructive, as they showed a point, namely, that the calculus only extends to just under cover of the gum margin. Rarefying osteitis around the roots of a maxillary molar is shown at *c* (fig. 59).

(*c*)



(*a*) (*b*)

FIG. 59.

Sequelæ.—The various local and general sequelæ that may arise from the presence of pus in the mouth are discussed in Chapter VII.

Pathology.—The study of the disease in the animal kingdom shows clearly that the disease is of local origin, and is due to the lodgment of fermenting food around the teeth. There are one or two important clinical facts

connected with the appearance of the disease in man, which may assist in an attempt to trace its pathology. In non-mouth-breathers the disease commences in the molar region, and gradually spreads to the anterior teeth; while in mouth-breathers the disease starts in the incisor region. The commencement of the disease in the non-mouth-breathers occurs in the region where food is most likely to lodge, and it is probable that the initial lesion is an injury to the gingivæ from food débris. The fact that in mouth-breathers the disease commences in the incisor region is instructive. If the mouth of a child affected with mouth-breathing, but with functional molars, be examined, a gingivitis will be found affecting the incisor teeth, more particularly the mandibular ones. The absence of gingivitis in the molar region is due to the fact that the function of this part of the mouth is being properly performed; the friction of mastication, and possibly the rubbing of the buccal mucous membrane against the gums, removes the superficial layers of epithelium, and stimulates a healthy reaction in the tissues. In the front of the mouth the normal friction of the lips against the gums is absent, the surface epithelium as well as the débris of food and micro-organisms accumulates, and injury results to the gingivæ. As long as mouth-breathing continues the gingivitis persists owing to the constant injury, and eventually the periodontal membrane becomes involved. There is little doubt, therefore, that, with mouth-breathers, the initial stage of the disease is a localized injury to the gingivæ of the anterior teeth.

The consensus of opinion is that, in established cases of alveolar suppuration, one or other of the pathogenic cocci—the streptococcus, pneumococcus, staphylococcus, or *Micrococcus catarrhalis*—is present; and, in addition,

other organisms of the bacillary type predominate in the early stages.

The Facts known regarding Chronic general Periodontitis would seem to indicate:—

(1) That the initial cause, in the majority of instances, is an injury of the tissues of the interdental spaces by fermenting food and other decomposing débris.

(2) That infection with pathogenic organisms rapidly follows, for the pus found even in the early stages contains organisms which are known to be associated with chronic forms of disease in other parts of the body.

(3) That the chronic character of the process is one of slow tissue necrosis, rather than acute inflammation.

(4) That the disappearance of the tooth socket is due indirectly to the action of toxins which are formed in the pockets around the teeth.

(5) That pathogenic organisms and their toxins may gain access to the body via the blood-stream, or gastro-intestinal tract, and, when this occurs, they may cause various diseases often disproportionate in their gravity to the condition of the gums.

(6) That owing to inefficient mastication the tissues around the alveolar process are unduly liable to attack on account of the lack of functional activity.

(7) That the calculus on the teeth is the product of the discharges from the teeth sockets, and that it does not affect the disease beyond interfering with the cleansing of the pockets around the teeth.

Etiology.—A survey of the pathology of the disease inclines one to the opinion that the disease is essentially a local lesion, and that general conditions play only a secondary part. The *immediate cause of the disease* is the accumulation of irritant material in the pocket around the teeth, more especially in the interdental

spaces. This morbid material may be the product of a gingivitis or of food débris. The accumulated material ferments, toxins are formed, and tissue destruction follows. The agencies which are instrumental in producing a marginal gingivitis (see Chap. VI) are, therefore, also to be regarded as the causes of chronic periodontitis. It cannot be too strongly insisted on that mouth-breathing is the principal cause of marginal gingivitis.

The great prevalence of the disease is probably due to the unsuitable character of the diet of the present day. Our foods are now prepared in such a form and manner that they readily accumulate around the teeth, and easily undergo fermentation. *Efficient use of the masticating organs* is almost impossible, and the result is, that the tissues in and around the teeth are deprived of an adequate blood supply, and are rendered less resistant to attack. When once the disease has started, the formation of abnormal pockets assists the accumulation of débris, and accelerates the action of the toxins.

Treatment.—In treating the disease, all that can be hoped for, even in the most favourable cases, is an arrest of the process, and possibly a regeneration in the osseous tissue. The only way of arresting the disease is to promote healthy reaction on the part of the tissues themselves, and to this end the main therapeutic measure to be adopted is the *promotion of asepsis as far as practicable in the pockets by efficient drainage.* Efficient drainage relieves the tissues of the constant injury arising from the toxic matter around the teeth.

In mild cases the following line of treatment often produces good results. The teeth must be thoroughly freed of all calculus, care being taken to avoid as far as possible injury to the periodontal membrane. The

pockets around the teeth should then be irrigated with hydrogen peroxide (15 vols.), and this followed by the application of strong tincture of iodine to the sockets.

The following routine treatment should be carried out by the patient for at least one month :—

The spaces between the teeth must be cleared with floss silk, and the remaining surfaces of the teeth cleaned with a brush if possible after every meal, the mouth then being thoroughly rinsed with an antiseptic mouth-wash. The last thing before going to bed the pockets should be thoroughly irrigated with hydrogen peroxide 15 vols.; for this purpose a small syringe is best. Frequent massage of the gums with the finger will be found useful, and instruction should be given to use the teeth thoroughly during mastication. At the end of a month the irrigation can be reduced to once or twice a week. Tooth powders should be avoided, as the insoluble particles tend to pass under the free margins of the gum and act as irritants.

In more advanced cases where there is much congestion of the gums and active pus formation, in addition to the above routine, the gums should be rubbed every night with powdered tannic acid. An endeavour should also be made to eradicate the pockets by paring away the loose unattached gum and applying to the bare surfaces a mild escharotic, such as trichloracetic acid (25 per cent.). The more thoroughly the pockets are kept free from discharge the less is the damage to the tissues, and naturally the greater the chance of the tissues reacting normally.

Vaccine-therapy has been advocated in treatment, and Mr. K. Goadby states that he has obtained good results. The method he suggests is as follows : "A bacteriological examination is made of the material obtained from the

gum pockets after syringing out with boiled water, the culture being made in ordinary agar and maltose serum agar, and the bacteria planted out. The opsonic indices to the various organisms are then obtained, and a vaccine prepared of the one giving the lowest index. If more than one organism shows a low index, a mixed vaccine of the several species is indicated. If more than one organism is associated, as is commonly the case, subsequent observations of the index of each are recorded, and the vaccine is modified. If improvement is shown, further bacteriological analysis at a later date may be required, as further infection may have occurred." For further details of the treatment the reader is referred to Mr. K. Goadby's paper (*Lancet*, vol. ii., p. 1875, 1909).

Clinical experience teaches us that careful and thorough treatment of the teeth will produce a rapid improvement, not only in the local, but usually in any general symptoms that may be present; and when, therefore, vaccine treatment and local treatment are combined, it is difficult to determine the precise effect of each.

It seems likely that, in advanced cases of pyorrhœa accompanied by definite constitutional symptoms, the employment of vaccines may quickly raise the resistance of the patient to infection, and thereby enable the tissues to respond more rapidly and more readily to local treatment; and the results yielded by vaccine treatment in such cases tend to show that it may act in this way.

In the treatment of periodontal disease it is necessary that the abnormal spaces around the teeth should be eradicated. To obtain this result would seem to be a simple matter, but in actual practice it is extremely

difficult to obtain, and unless patients after treatment by vaccines continue daily to carry out the cleansing of the spaces the disease will progress. Vaccines may assist, and possibly do in some cases assist, the tissues to react, but the most important part of treatment in the disease must not be overlooked—namely, the careful cleansing and treatment of the pockets by local measures, which, as pointed out above, is sufficient in the majority of cases to bring about a rapid improvement.

Dr. E. C. Hort[1] has drawn attention to the fact that vaccine-therapy leaves out of consideration the damage caused to the tissues by the toxic products of autolysis and other cell metabolism. These toxic products are combated by the formation of corresponding antibodies. All conditions which produce hyperæmia in the part, whether by the use of poultices, blisters, or Bier's treatment, act by increasing the fluid exudate in the affected parts, which dilutes the irritant and increases the natural antibodies in the affected area; in other words, the treatment may be regarded as a process of auto-inoculation.

The ideal method of producing hyperæmia would seem to be that suggested by Dr. Bier, but in following this method there are practical difficulties to contend with. In a few cases in which this treatment has been tried radiographic examination has shown that after treatment the destruction of the bone had not progressed, and the clinical appearances suggested a marked improvement. To carry out the treatment a splint is made to grasp the gums as high above the teeth as possible. The spring should be so adjusted as to create pressure

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[1] *Proc. Roy. Soc. Med.* (Med. Sec.), vol. ii, No. 6.

sufficient to congest the gums, but without causing pain. The splint should be applied for about fifteen minutes twice a day.

A useful apparatus for producing hyperæmia of the gums has been suggested by Mr. C. Woodruff. A vulcanite cap is made, with the margins so adapted as

FIG. 60.

to grasp the gums as high above the teeth as possible, the remaining portion of the cap being constructed so that a slight space exists between the gums and the splint. Into the splint a tube is inserted which is connected with an exhaust bulb. The apparatus is shown in fig. 60. The cap is placed over the teeth and the

air gradually exhausted. By this means an effective hyperæmia is obtained.

In dealing with the etiology and pathology of the disease, attention was directed to the effects of mouth-breathing. If this condition is present, treatment must be directed towards its removal, as *local and other remedies will be of little avail while the patient breathes through the mouth.*

If, in cases where the disease is progressing rapidly, the teeth are removed before the whole of the alveolar process has disappeared, and dentures are inserted as soon as the tenderness following the operation has subsided, function will be restored to the bone, and its destruction will, to a great extent, be arrested. By this means an alveolar ridge will remain, rendering the adaptation of dentures an easier task.

The results following the removal of the sepsis, whether by local treatment or extraction, are most satisfactory; there is invariably a rapid improvement in the health, indicated by relief of gastric symptoms, improved appearance of the skin and increase in weight. The improvement which follows will depend to a great extent on the stage at which the disease is recognized. In some cases the injury to the tissues is so severe that, with the removal of the teeth, the utmost that can be hoped for is to prevent further injury. In such cases Mr. Goadby thinks that the infecting organism still remains active in the bone, and their toxins are absorbed into the general circulation.

In cases where there is considerable bone destruction, or where, in spite of local treatment, the formation of pus persists, extraction of the teeth must be resorted to. As a preliminary to extraction, the mouth should be rendered as aseptic as possible by the use of efficient

mouth-washes and irrigation. Opinions differ as to the number of teeth which should be removed at one sitting, and the operator should be guided by the condition of each individual case. Where the removal of the teeth will cause only slight laceration of the tissues, a large number can be removed at one sitting, and the wounds will be found to heal rapidly and with very little subsequent pain. The amount of pain following the removal of the teeth depends, to a great extent, on the care with which the extractions are performed and on the after-treatment of the wounds.

It is in accordance with general experience that wounds following extractions are kept in a septic condition by infection from the remaining septic teeth, and to avoid this it is obviously necessary that all septic teeth should be removed as quickly as possible. In these cases it is very rarely found that the extraction of a large number of teeth causes either local or general disturbance. The after-condition of the mouth depends almost entirely upon the manner in which the operation is carried out.

In cases where the general condition of the patient uggests that the reaction of the tissues will be slight, the removal of the teeth should be delayed, and energetic local and general treatment persevered with in the hope of a general improvement, and therefore more healthy reaction of the tissues.

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CHAPTER VI.

Diseases of the Gums and Adjacent Mucous Membrane.

Hypertrophy of the Gums—Gingivitis—Stomatitis—Leucoplakia—Pemphigus—Syphilitic Lesions.

(A) HYPERTROPHY OF THE GUMS.

HYPERTROPHY of the gums consists of a general overgrowth of the tissues covering the alveolar process. The disease has not been shown to be hereditary, although it may occur in members of the same family. It may be local, but, as a rule, is general in distribution. In its origin the disease would seem to be in some way related to the eruption of the teeth, and has shown itself as early as the age of seven months at the appearance of the incisor teeth. An interesting peculiarity of the disease is that it is not infrequently associated with abnormalities of other tissues, such as molluscum fibrosum, or an excessive development of hair on the body.

The hypertrophied tissue is of normal gum colour unless inflammatory changes have taken place in it, in which case it becomes darker than normal, and bleeds on very slight injury. If the inflammatory reaction continues, the hypertrophied tissue may increase enormously and the condition become very distressing.

Microscopical examination of such cases generally disclose numerous cells between the delicate fibrous tissue and many blood-vessels scattered throughout the growth.

Treatment.—This consists in the free removal of the hypertrophied gum, including the margins of the affected alveolar process.

(B) REACTION OF THE GUMS AND ADJACENT MUCOUS MEMBRANE TO INJURY—INFLAMMATION.

In inflammation of the gums two distinct conditions are recognized, namely, "gingivitis," in which the inflammation is confined to the gums; and "stomatitis," in which the gums, buccal mucous membrane and other mucous surfaces of the mouth may be involved.

(1) GINGIVITIS.

The condition may be general, or limited to the gum margin—"marginal gingivitis."

(*a*) *Marginal gingivitis* is a common affection, and, if left untreated, may lead to chronic periodontitis, or may spread and involve adjacent surfaces.

Etiology. — (1) *Local causes:* Mechanical irritants, such as badly-adjusted crowns, bands of tooth plates, and accumulation of tartar; decomposing food *débris*, &c.

(2) *General causes:* The prolonged administration of such drugs as mercury, lead, or iodine; certain diseases such as gout, diabetes, and nephritis.

(3) *Direct infection:* Gonococcus, and possibly other organisms.

Pathology.—In a mouth that is functional, the friction of mastication, and of the cheeks and lips against the

gums, and the constant flow of saliva is sufficient to prevent the collection of food and epithelial *débris* at the gingival margin. Anything which interferes with the normal mouth condition will favour such collections, which, undergoing decomposition, will lead to an inflammatory condition of the adjacent tissues. The relation of marginal gingivitis to mouth-breathing has already been pointed out. In mouth-breathers, not only are the gums deprived of the natural friction of the lips and cheeks, but there is a constant change of air over the anterior part of the mouth which serves to dry the parts and aid the retention of the *débris*.

In acute illnesses the marginal gingivitis would appear to be caused by mouth-breathing and the "slop" diet.

Progress.—The disease, if left untreated, leads to chronic periodontitis. On the other hand, if the conditions giving rise to the disease be removed, a rapid improvement takes places.

Varieties.—The disease may be either catarrhal or purulent. In certain patients the tissues readily undergo hypertrophic changes resulting in the formation of tags of tissue between the teeth. The so-called polypus of the gum is formed in this way. This variety is usually designated "chronic hypertrophic marginal gingivitis." Marginal gingivitis must be clearly distinguished from general chronic periodontitis.

Signs and Symptoms.—The free margins of the gums are red and painful, and bleed freely on slight injury. Pressure with the finger against the gum margin will lead to the exudation of a thick creamy discharge. In acute attacks the infection spreads rapidly to the periodontal membrane, the teeth become loose and bathed in pus; there is marked salivation and a rise of temperature.

Treatment.—Attention should in the first place be directed to removing the predisposing or more general causes. Thus, adenoids should be removed and care taken that nasal breathing is established, or any offending drug such as mercury or iodine stopped. In the second place, the local condition must be treated. The tissues around the teeth should be syringed with a solution of hydrogen peroxide and an astringent mouth-wash prescribed. If there be much pain, a mouth-wash consisting of equal parts of tincture of iodine and spirits of camphor will be found useful. In marked cases, and when resolulution does not rapidly occur, powdered tannin may be applied twice or thrice a day and the parts carefully massaged with the finger. Redundant tissue should be cut away.

In acute attacks, the parts should be syringed with hydrogen peroxide and afterwards swabbed with a solution of silver nitrate (10 gr. to the ounce) twice a day.

(*b*) *Chronic gingivitis* is usually the result of local irritation and is seen frequently in patients wearing artificial dentures. The gums are slightly swollen, red, congested and painful. A temporary removal of the denture and the use of an astringent mouth-wash is sufficient to effect a cure.

Lead Poisoning in Relation to the Gum Margins.—In some cases of lead poisoning a blue line *in* the gum margins may be seen. It occurs more frequently in the lower than in the upper gums, and in the incisor more often than in the molar region. It is more common in dirty than in clean mouths. It is probably due to the precipitation of the sulphide of lead from the lead circulating in the blood, the sulphur being supplied from the decomposition of *débris* around the necks of the teeth. It does not occur if the teeth are absent. It is an

important sign to look for in all cases of abortion in which the cause is not clear.

It should be diagnosed from (1) a delicate line of blue at but not in the margin of the gum, which occurs in persons exposed to the dust of white lead for a few hours.

(2) A deposit under the gums in people who clean their teeth with charcoal, and in miners and others exposed to coal dust.

(3) The line caused by copper and bismuth poisoning.

(2) Stomatitis.

(*a*) *Catarrhal Stomatitis.*—This form is generally associated with inflammation of the throat or nose, and probably arises from the same cause as catarrh of those parts. It is frequently seen in the course of the exanthematous fevers and gastro-intestinal disturbances. The excessive use of tobacco, or too hot or too highly-seasoned foods, may be cited as causes.

Signs and Symptoms.—The serum from the congested mucous membrane filters into the sub-epithelial space. There is an increased production of surface epithelium and also of mucus. This mucus continues and, with the serum, forms the peculiar sticky discharge seen in these cases. In more severe cases the epithelium may be detached in large masses, the sub-mucous tissue becomes greatly infiltrated with leucocytes and the whole mucosa swollen, giving rise to a purulent or muco-purulent condition. The margins of the gums become acutely inflamed and painful to the slightest touch. In the early stages the gums are dry, but this is soon followed by an excessive secretion. The portion of gum attached to the margin of the alveolar process is pale, while the reflection of the gum from this point on to the cheek will show the vessels to be congested. The gums appear whitish and

mottled, and pus is generally seen welling up from the sulci around the teeth.

The mottled appearance of the gums is due to the fact that in inflammation the epithelium proliferates, and appears whitish, but through the friction which occurs between the gums and the cheeks, the epithelium covering the papilæ of the gum is rubbed off and leaves reddish patches here and there. The breath is fœtid, the patient has a sensation of heat and pain, and the taste is impaired. The tongue is furred, there is a loss of appetite, derangement of the bowels, and a feeling of malaise.

Treatment.—The cause should, if possible, be removed, antiseptic and astringent mouth-washes should be used, and the general condition of the patient treated by appropriate remedies.

(*b*) *Mercurial Stomatitis.*—Prolonged use of mercury is likely to lead to gingivitis, which rapidly spreads to contiguous parts. The ptyalism, so often seen in cases of mercurial stomatitis, is due to the spread of inflammation to the salivary glands. The early symptoms are soreness and discomfort in the mouth, accompanied by a metallic taste and fœtid breath. The gums become inflamed, and present a deeply congested appearance at the free edge; the portion attached to the margin of the alveolar process remains whitish, and the portion beneath presents the whitish mottled appearance referred above. The teeth become loose, sloughing and ulceration occur near the margin of the gum; the slough separates, and the teeth fall out. The inflammation spreads rapidly to the cheeks, tongue, floor of the mouth, and to the salivary glands, leading to a profuse flow of saliva. There is much pain in swallowing, speaking, and on moving the jaws. In severe cases, if not quickly treated, extensive sloughing and necrosis may supervene.

With regard to the pathogenesis of mercurial stomatitis, Alonkvist,[1] who examined specimens from subjects who had died in the course of mercurial treatment, concludes: "That the deposition of mercurial granules takes place in the vessel walls, and in preference in the capillary loops nearest to the epithelium. During the evolution of the disease the following phenomena occur in the gum and mucous membrane. Deposition of granules of mercuric sulphide, vascular dilatation, diapedesis of leucocytes laden with the mercury granules, degeneration and death of the histological elements of the tissue. The mercury is deposited through the action of the hydrogen sulphide, formed during the decomposition of proteid matter in the mouth or intestinal tract. The latter compound precipitates the mercury which circulates through the blood-vessels and that which is eliminated through the oral mucous membrane in the form of mercuric sulphide. Hence the necessity of keeping the mouth and teeth in a perfect state of cleanliness during mercurial treatment, and of paying as much attention as possible to the digestive functions, in order to avoid the formation of excessive amounts of hydrogen sulphide. Inflamed gums, caused by carious teeth, do not hug the teeth tightly at the neck, and in the interstices thus produced food *débris* accumulates, and in undergoing decomposition hydrogen sulphide is formed, which causes the precipitation of the mercury eliminated through the mucous membrane.

"In animals, mercurial intoxication is not, as a rule, accompanied by stomatitis, which, however, may be induced by detaching the gums around the teeth, and

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[1] See "Mercurial Stomatitis: Present Status of the Question." Raymond Lulle, *Le Progrès Dentaire*, September, 1907.

irrigating the pockets for some time with solutions of hydrogen sulphide."

Treatment.—Local: In mild cases, the use of an antiseptic mouth-wash is sufficient. In severe cases, local depletion will be found advantageous.

General: The administration of the drug must be stopped, and the bowels made to act freely by means of saline purgatives. A plentiful use of alkaline mineral water should be enjoined. The diet should be in a liquid form, and, if there is much pain, opium in the form of pulv. opii. co. may be given at night. The general anæmic and debilitated condition which follows severe "ptyalism" requires a supporting form of treatment.

(*c*) *Ulcerative Stomatitis.*—This condition is most frequently met with in children. It may prevail in an epidemic form in institutions where insanitary conditions exist. Want of cleanliness, faulty hygienic surroundings, and general ill-health favour the development of the disease.

The inflammation commences at the free margin of gums, and is said to be more frequent in the maxilla. In the early stages the gums are swollen and congested, the congested veins leading to the part being distinctly visible. The ulceration, commencing at the free margin, gradually spreads, denuding in its course the alveolar process, and leading to necrosis of the teeth. The adjacent mucous surface of the cheek usually becomes attacked from contact, and if the case is left alone, the ulceration may extend and lead to extensive necrosis, sloughing, and ultimately death of the patient. In well-developed cases an ulcer with sharp irregular edges is seen, the margin displaying a bluish ring, the ulcer, being covered with a greyish or yellowish slough, the neighbouring lymphatic glands being enlarged.

Bacteriology.—In this disease, the *Bacillus fusiformis* and *Spirochæta dentium* are always present in large numbers; and observers who have examined such cases have generally come to the conclusion that the fusiform bacillus and the spirochæte are the causative organisms.

No satisfactory evidence of the pathogenicity of either of these two organisms is, however, forthcoming. Ellermann and Muhlens and Hartmann, who isolated respectively the fusiform bacillus and the *S. dentium*, were unable to obtain pathological lesions by infections of pure cultures. By epidemiologists the disease is often thought to be related to the foot-and-mouth disease of cattle, and in the latter disease no definite micro-organism has been isolated; but whatever the organism may be, it is apparently smaller than any organism yet known, as the living virus will readily pass through the pores of a Pasteur and Chamberland filter.

Guezette[1] considers that ulcerative stomatitis is closely related to noma. The most constant feature in three cases he examined was the presence of a spirillum and a bacillus. He injected upon his own lip the discharge from a case, and found that the spirillum speedily disappeared whilst the associated streptococcus flourished freely.

In addition to the spirochæte and *B. fusiformis*, in three cases of ulcerative stomatitis which Mr. Goadby has examined he found the *Saccharomyces albicans* present in large numbers, both on the films direct and upon cultivation. The organism takes some little time to develop, and may easily be overlooked. It is quite possible that this organism may be only a concomitant to the inflammatory condition, but it is not common in ulcera-

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[1] *Arch. de la Sci. Med.*, vol. xxiii, No. 1.

tive lesions of the mouth, whereas the *B. fusiformis* and the spirillum occur whenever any diseased condition of an inflammatory nature is present in the mouth.

Signs and Symptoms.—In the early stages there is little pain, and the disease may be far advanced before it is discovered by the parents, and then the odour of the breath is the first symptom noticed. Constitutional symptoms are marked in severe cases, and death sometimes results.

Treatment.—Treatment consists in administering internally chlorate of potash, a drug which seems to be a specific for this disease. Children should be given 5 to 10 gr. according to age, adults 15 to 20 gr. It is well to give at the same time a tonic.

An endeavour should be made to improve the surroundings of the patient, should these be at fault. Plenty of exercise in the fresh air should be recommended.

Locally, all unhealthy teeth should be removed, and the ulcer should be painted with a strong solution of nitrate of silver, the mouth being kept clean by frequent irrigation with hydrogen peroxide. Should the cheek be involved as well as the gums, a strip of lint moistened with carbolized oil should be placed between the two surfaces. A gargle of chlorate of potash should be prescribed, and its frequent use recommended. Under this treatment most cases speedily improve.

There is *a type of stomatitis which seems to possess distinctive clinical features.* The condition usually starts around a septic tooth, and at first differs but little from a simple ulcerative stomatitis. Within from twenty-four to forty-eight hours, the infection spreads rapidly along the gingival margin, involving both jaws, and often extends backwards to the pillars of the fauces, to the hard palate, and the floor of the mouth. The gums bleed

readily, and present a ragged margin of necrotic tissue; the periodontal membrane is rapidly involved, and the margin of the bone becomes exposed.

With the gum condition are usually associated lesions of the mucous surfaces of the fauces, tonsils, soft palate and pharynx, which become covered with patches of exudation forming a yellowish to greenish-yellow membrane, which may remain superficial or tend to spread into the deep layers of the tissues. The breath has a most offensive odour, and pus and blood wells up around the teeth. If left untreated, the teeth rapidly loosen and

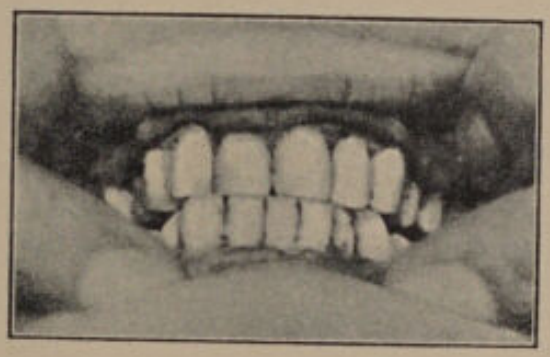

FIG. 61.—From photograph of gums of patient suffering from the type of stomatitis described in the text.

are lost, and the adjacent tissues are deeply ulcerated and the lymphatic glands enlarged. The local condition is attended by a rise in temperature varying from 100° to 103° F., which usually falls to normal about the fourth or fifth day.

In one case under observation, the disease started around a loosely fitting crown, and in three other cases the focus of infection was around a misplaced third mandibular molar, whence it spread rapidly forwards along the gingival margin, and backwards on the pillars of the fauces and soft palate. In a series of seven cases

recorded by Messrs. Harwood-Yarred and P. N. Panton[1] carious teeth were present, and it was apparently around these teeth that the disease arose. In five of the seven cases, a greyish-white necrotic patch appeared in the mucous membrane of the cheek, and opposite the carious teeth. In every case the authors were able to demonstrate one or both of the organisms described by Vincent.

The symptoms and general course of the disease are similar to those characterizing the condition usually known as Vincent's angina; "indeed, it is probable that they are one and the same affection, the main focus of the disease lying in the gingivæ." Two varieties of Vincent's angina are recognized. A mild or diphtheroid type, in which the process is limited in extent of surface and does not penetrate to any depth; and a severe or ulcero-membranous type in which the process extends rapidly over the surface and involves the mucous membrane deeply.

Bacteriological examination of cases shows that in addition to pyogenic cocci, a characteristic bacillus is present in the exudation frequently associated with a spirillum. In the mild type, the bacillus predominates; in the severe type, the spirillum is "much more frequent and may even be present in greater numbers than the bacillus." The bacillus described by Vincent is called the "fusiform bacillus."

Treatment consists in thorough irrigation of the pockets around the teeth, and also of the affected mucous membrane, with hydrogen peroxide, followed by the application of strong tincture of iodine. The acute symptoms rapidly improve under this course of treatment, but a chronic condition often remains which requires careful hygienic

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[1] *Lancet*, February 17, 1906.

treatment on the part of the patient. Teeth which are obviously the source of chronic sepsis should be removed, and in cases where the stomatitis spreads from misplaced third molars these teeth should be removed.[1]

(*d*) *Gangrenous Stomatitis ; Noma ; Cancrum Oris.*— This very serious disease is a rapidly spreading gangrenous inflammation, which usually attacks the cheeks, and occurs in children from two to six years old. The disease may, however, occur in adults.[2] It is more common in girls than in boys. It is frequently seen in those just recovering from one of the exanthematous fevers. Unhygienic surroundings and weakening of the system by long-continued administration of mercury also act as predisposing causes. The disease may start either in the substance of the cheek, or in the mucous membrane, the latter being the more usual common situation. The cheek becomes hard, brawny, and very swollen, a dark red colour showing in the centre, the surrounding parts being œdematous. At this stage, if the mouth be examined, an ulcer will be seen on the mucous surface of the cheek, corresponding to the dark spot on the skin. The ulceration, or rather sloughing, leads to perforation of the cheek, and, if the disease still pursues its course, the soft parts rapidly become gangrenous. The child becomes exhausted, delirious, and eventually dies of blood-poisoning, or some septic affection of the lungs.

The fact that the whole cheek becomes gangrenous, and that the disease is not amenable to the action of

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[1] In connection with this question, the following papers will be found worthy of perusal: On Vincent's Angina, H. W. Bruce, *Lancet*, July 16, 1904, p. 135. Cases of stomatitis and tonsilitis in which Vincent's Spirochæte and Bacillus were present, W. H. Harwood-Yarred and P. N. Panton, *Lancet*, Feb., 1906, p. 438.

[2] See *Lancet*, December 21, 1901, p. 1730.

chlorate of potash, help to distinguish it from ulcerative stomatitis; but most authors think that the difference between cancrum oris and ulcerative stomatitis is only one of degree. The disease is said to be caused by thrombosis of the capillaries induced by the presence of a specific micro-organism, and is similar to the gangrenous inflammation known as noma which occurs upon the female genitals.

The diseases in rare instances may commence at the gingival margin. A case illustrating this point is recorded by C. Lockyer.[1] The patient, a youth aged 18, removed three roots of teeth by means of a wooden pen-holder sharpened and used as an elevator. Four days subsequently the face began to swell; gangrene ensued and spread rapidly over the palate and the alveolar process, the patient eventually succumbing to general septic infection.

Bacteriology.—Walsk[2] found in eight cases of noma the diphtheria bacillus. In one case the culture was pure; in the remaining seven it was associated with other bacteria. Four of the cases started as ulcerative stomatitis, but in fifteen cases of the latter condition the diphtheria bacillus could not be detected. Dr. Hellsen[3] states that he has isolated from a case of cancrum oris a diplococcus which he was able to cultivate, and which, when injected into an animal, caused the identical disease. From the latter source it was obtained in pure culture. Four generations of bacteria were obtained from the original growth, and each caused a well-defined

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[1] "A Case of Gangrenous Stomatitis," *Brit. Dent. Journ.*, vol. xxix, p. 605.

[2] *Proc. Path. Soc.*, Philadelphia, June, 1901.

[3] *Münchener med. Wochenschrift*, 1907, No. 5.

case of necrosis in the specimens experimented upon. It is worthy of note in this connection that the *Diplococcus pneumoniæ*, an organism frequently present in suppurative conditions and infections of the mouth, when injected into guinea-pigs causes a fibrinous exudate, later breaking down at times into necrotic pus.

Professor von Babes, in Kolle and Wassermann's "Handbuch der pathogenen Micro-organismen," gives a number of references to spindle-forming and thread-forming organisms which have been described from time to time as associated with gangrenous stomatitis. The *Bacillus fusiformis* of Vincent and the *Spirochæta dentium* are supposed by some to be associated with this disease, and some time since Bernheim considered that the spirochæte was the cause of the disease, as it was usually present in large numbers. Other observers have thought that the *Bacillus necrosis*, or a streptobacillus of Zohr, was an organism which played a part in the production of gangrenous stomatitis. In one or two instances the bacillus of malignant œdema has been described, but as yet there is no settled bacteriology with regard to the disease. The disease is fortunately uncommon, at any rate in England; and there is little opportunity for making bacteriological examination.

The *prognosis* of cancrum oris is bad. The *treatment* consists in carefully drying the soft parts, removing all gangrenous portions, and cauterizing the remaining surface with nitric acid, or the actual cautery. The general treatment should consist in supporting the patient's strength with a plentiful supply of beef-tea and other nutritious remedies. It has been suggested that free excision of the gangrenous surfaces would be an effective treatment, and in cases recorded it has proved

beneficial. Corrosive sublimate locally applied has been used; it has proved successful in three cases recorded by Kingsford in the *Lancet* of May 4, 1889. Disinfectant mouth-washes must be prescribed, and the raw surfaces carefully dressed with antiseptics. The disease may spread until the whole side of the face disappears, the cavity extending from the nose to the ear, and from the lips to the upper eyelid.

(*e*) *Follicular Stomatitis.* — This inflammation is similar to an ordinary herpetic eruption. A cluster of vesicles first appears, which, on breaking down, coalesce and form a small circular and well-defined ulcer. This ulcer is surrounded by a zone of redness, and is extremely painful. The ulcers are said to occur more frequently near the frænum of the lip, on the under surface of the tongue, and in the sulcus between the gums and the lip. The little round punched-out ulcers met with in the cheeks are probably a variety of this form. Follicular stomatitis occurs in adults less frequently than in children. It is often associated with some gastro-intestinal disturbance.

The *treatment* consists in the application of an astringent solution, and, in intractable cases, the ulcers may be touched with a crystal of sulphate of copper or nitrate of silver. The general condition of the patient must also be treated.

(*f*) *Parasitic Stomatitis.*

(1) *Due to Saccharomyces albicans (Thrush).*—Thrush is a parasitic inflammation of the mucous membrane of the mouth, dependent upon a fungus, the *Saccharomyces albicans.* It is common in infants, but may occur in adults, and, in the latter, is generally associated either with some of the acute specific fevers, or with chronic wasting diseases, such as phthisis. When associated with

phthisis it generally proves fatal. The fungus develops in the upper layers of the mucosa, the filaments forming a dense network among the epithelial cells. In this manner the mucous membrane becomes covered with numerous white spots, which are firmly adherent, and, when removed, a deep red colour is revealed. These spots appear mostly near the angles of the mouth and on the tongue, but may occur elsewhere in the oral cavity, the affection spreading at times to the pharynx and œsophagus. The spots are about the size of a pin's head, are circular in form, and they gradually coalesce and form larger patches, giving rise to the appearance of a false membrane with a slightly yellowish aspect. These patches come away, leaving a reddish surface beneath. The patches are found to consist of epithelium and fat, together with sporules of the *Oidium albicans*, the vegetable parasite which causes the disease.

When thrush occurs in children they are generally found to be out of health, the bowels relaxed, the evacuations green and sour. The motions are generally acrid, and irritate the margins of the anus, giving rise to an erythematous blush over the buttock, the appearance of the edges of the anus being similar to that seen in the mouth. Sucking and deglutition are impaired by the condition of the mouth, and the child will usually be in a state of drowsiness and torpor. Many cases of thrush in infants are distinctly traceable to the use of dirty feeding-bottles.

The *treatment* of thrush may be divided into local and general. The *local* treatment consists in carefully wiping the mouth with soft lint after each meal, care being taken to burn the lint after use. The exposed surfaces thus left are touched with a solution of 3 drachms of borax to 1 oz. of water. In severer cases, it will be

advisable to use nitrate of silver, 5 gr. to the ounce, or dilute carbolic acid in glycerine. It is needless to say that attention should be given to the condition of the feeding-bottle. The *general* treatment should consist in careful attention to the diet, with the administration of a mild aperient.

(2) *Due to the Aspergillus nigrescens.*—An inflammatory condition due to the presence of the *Aspergillus nigrescens* has been reported.[1]

The following is a brief account of the case: "A small ulcer first appeared on the middle line of the roof of the mouth, about half-way between the incisors and the soft palate. The patch increased slowly in size, and others formed in the neighbourhood, the condition appearing like a lumpy patch extending from just behind the incisors to within one-fourth of an inch of the soft palate. Cup-shaped elevations on the soft palate appeared on either side of the middle line. A firmly attached membrane, giving rise to hæmorrhage when forcibly removed, covered the areas. The colour of the recent deposit suggested the sulphur-coloured scutula of favus; where it had remained undisturbed, it was darker. With low power the growth was recognized under the microscope as a fungus differing from the achorion. The mycelium network was composed of delicate fibres, bearing perpendicular fructifying hyphæ. Scattered over the field were a number of fruit receptacles and a few spores. The manner of fructifying showed that the fungus did not belong to the oidium, but to the ascomycetous genus. Cultures showed it to be *Aspergillus nigrescens* which had caused the inflammation. Upon applying 25 per cent. ethereal solution of pyrozone,

[1] *Medical Record*, October, 1896 (The Dental Digest, p. 641, 1896).

improvement was immediately noticed. The pseudo-membrane disappeared and new patches ceased forming. After seven weeks' treatment, the patient was well.

"The spores were supposed to have been implanted in the mouth through the medium of cheese, strong and mouldy varieties of which the patient was very fond of eating."

(*g*) *Aphthous Stomatitis.*—This form rarely occurs in adults. It is characterized by the formation of fibrous deposits on and under the epithelium, and is considered by some authors to be contagious. It is frequently seen in rachitic and weakly children, and is most commonly met with during the periods of dentition. In adults it occurs in those debilitated by illness, or may be associated with general inflammatory conditions. In women, it occurs during menstruation, pregnancy, and during the puerperal period.

Bacteriology.—Out of ten cases of aphthous stomatitis examined by Mr. Goadby, a streptococcus was present in six, having the characters of the *Streptococcus fæcalis* and not of the ordinary *Streptococcus brevis* of the mouth.

Signs and Symptoms.—Small yellowish-white patches, slightly elevated and extensively sensitive, are present on the mucous membrane. The patches are surrounded by a zone of inflammation. They have a tendency to spread and coalesce, forming large patches. When not associated with any active general disease, constitutional symptoms may be present, namely, slight elevation of temperature, thirst and loss of appetite.

Treatment.—Locally, a mouth wash of chlorate of potash should be used, and the mouth thoroughly cleansed after each meal. If the aphthæ persist, they may be treated with nitrate of silver. The constitutional condition also requires attention.

In cattle, *aphthous stomatitis appears in an epidemic form*, and is commonly known as "*foot-and-mouth disease.*" It is communicable to man. In man it is an eruptive fever, running a fixed and definite course, usually in eight to ten days, and is characterized by a vesicular eruption in the mouth, on the lips, gums, and tongue as well as on the hands and feet. In the early stages of the disease there is a headache and loss of appetite, with a quickly rising fever. Pustules appear in the mucous membranes of the mouth, more frequently on the lips and the tongue than on the hard palate and the throat. After a few days the pustules burst, leaving eroded spaces with greyish-yellow surface, which, during the second week, commence to heal. In the acute stage, the patient suffers from a sensation of heat and burning in the mouth, the cervical and salivary glands swell, producing severe dysphagia.

(*h*) *Chronic Neurotic Stomatitis.* — This condition appears usually in patients suffering from mental worry. It is an uncommon condition. Knowsley Sibley[1] considers that it is not of the nature of pemphigus, as sometimes described, but is a distinct affection.

"It generally commences as a crack or streak, or from the beginning as a small superficial bright red ulcer. Occasionally in the tongue it begins in an inflammatory localized thickening just beneath the mucous membrane, which rapidly breaks down and forms an ulcer, usually with a slough in the centre and considerable inflammatory redness around. It sometimes happens that the ulceration is preceded for a day or two by a heaping up of the epithelium, often forming a pale, gelatinous-looking ridge fitting in the spaces between the teeth.

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[1] *Brit. Med. Journ.*, April 1, 1899, p. 900.

At other times, the ulcers are preceded by small gelatinous-looking bodies about the size of millet seeds, and occasionally by small vesicles; accompanying the ulcers is usually a considerable desquamating catarrh of the surface of the tongue. There is usually a good deal of burning sensation and great distress, accompanied by profuse salivation, and if the ulcer is very indolent, with œdema of the parts around. If the lesion is situated in the mucous membrane of the lips, these may become so swollen as hardly to permit of the mouth being opened and the tongue protruded. The ulcers are produced by a distinct tropho-neurosis, and they are quite different from the common catarrhal or dyspeptic ulcer."

In one case recorded by Sibley the ulcers had appeared intermittently for twenty-three years.

Treatment.—Complete rest from worries is needful. Locally, the healing of the ulcers may be expedited by the application of tincture of iodine. The pain and distress may be mitigated by the use of cocaine, or, in severe cases, opium.

(C) LEUCOPLAKIA.

Leukoplakia of the cheeks and the palate is of frequent occurrence in syphilitics and those addicted to heavy smoking. It is more usually seen in the muco-periosteum covering the back of the hard palate, and in marked cases presents the appearance of "ichthyosis." At times the condition may be limited and give rise to a definite swelling of a white, cauliflower-like growth. H. S. Pickerill[1] has recorded a case of this character.

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[1] "A Note on a Case of Ichthyosis Gingivæ," *Brit. Dent. Journ.*, vol. xxx, p. 733.

On the tissue being removed and examined microscopically, the growth was found to consist of epithelial cells, which, instead of becoming squamous and then desquamating, had become swollen and hardened and remained *in situ*. The sub-mucous tissue was normal. This author considers the condition similar to the pre-cancerous conditions of the tongue described by H. T. Bullin.

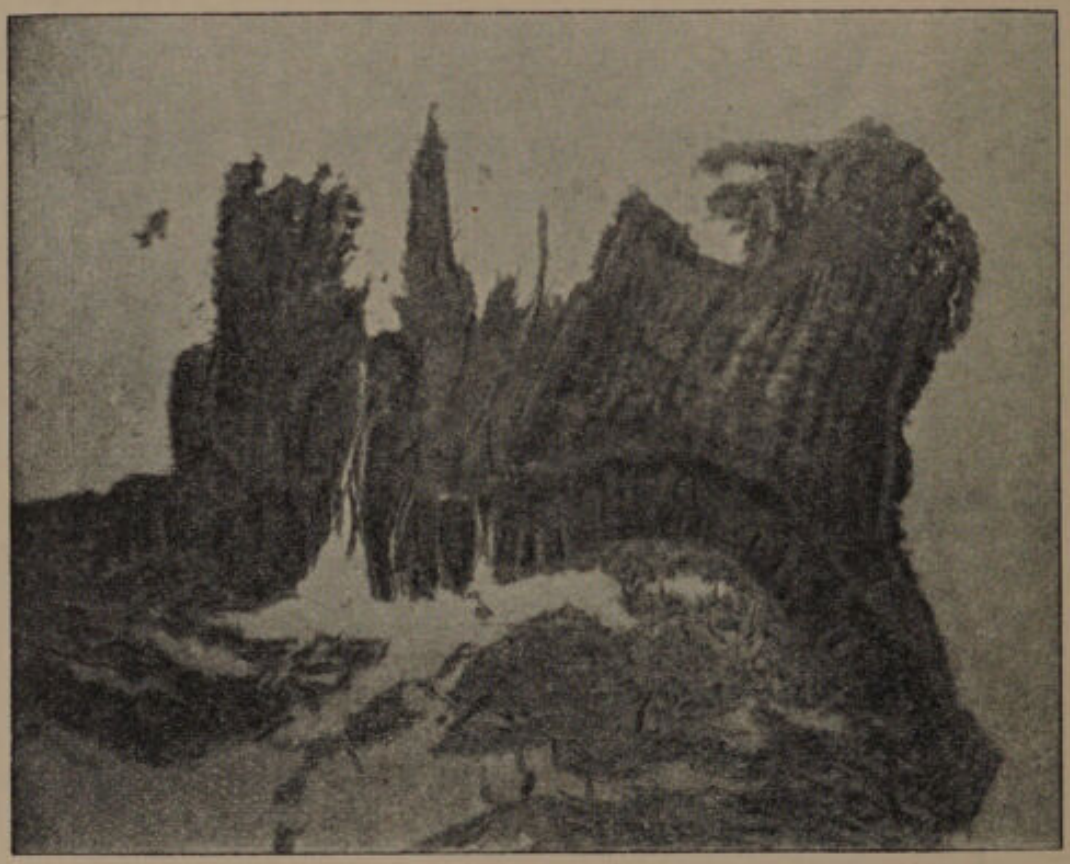

FIG. 62.[1].

(D) PEMPHIGUS.

The eruption of pemphigus may occur in the mouth. and one rare form, *pemphigus vegetans*, may remain localized to the mouth and adjacent cavities. The gums are also sometimes affected in "purpura hæmorrhagica."

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[1] From *Brit. Dent. Journ.*

(E) SYPHILITIC INFLAMMATION AND ULCERATION.

Syphilis may affect the gums and oral mucous membrane in all stages.

The primary lesion may occur on the gums, tongue, or other parts, and often assumes an unusual appearance. Ulcers of peculiar appearance and uncertain origin should always be regarded with suspicion, and the possibility of their being of syphilitic origin should be carefully inquired into.

Secondary lesions may appear in the form of mucous tubercles and in almost any part of the mouth; the favourite situations are the inner surfaces of the cheeks, the edges of the tongue and the lips. In weak and debilitated patients, the tubercle may break down, leaving an ulcer with a sinuous outline. The ulceration may extend and lead to extensive destruction of the tissue, followed, on healing, by adhesion of contiguous parts and extensive contraction.

In the tertiary stages the ulcerations are generally of a deep, excavated character, and are preceded by gummata, which, undergoing degeneration, produce the ulceration.

CHAPTER VII.

Oral Sepsis and its Influence on the Body.

THE term "oral sepsis" is used, not to denote a specific disease, but, collectively, to include all chronic inflammatory diseases about the mouth.

With the onset and progress of oral sepsis, there occurs an increase in the number and variety of the organisms commonly found in the mouth, especially those of the pyogenic class. Under the altered environment, many of these organisms may undergo changes in virulence, either in the direction of exaltation or attenuation, and thus some which were previously non-pathogenic may become pathogenic. The catarrhal and suppurative products which result from the bacterial activity undergo partial putrefactive changes in the mouth, and in this condition are constantly passing into the gastro-intestinal tract, together with enormous numbers of bacteria.

The pollution of the alimentary canal by this septic material leads to an alteration in the usual intestinal flora, and to the presence of abnormal putrefactive products, and these, together, may induce certain changes in the economy of the body. Thus (1) the invasion of the tissue by organisms will be largely increased, and these organisms, which are said, normally, to pass through the intestinal wall during digestion, will be of an altered character; (2) the new flora may act locally

upon the walls, and induce a catarrhal condition, and variations in the normal secretions, and so lead to a disordered digestion; (3) the products of digestion, and the usual putrefactive changes that take place in the intestine, may be directly affected by the organisms and unnatural products result.

Against this formidable attack, the body possesses certain defences which may be divided into "local" and "constitutional."

The local defences consist of:—

(1) Phagocytosis in the mouth, the process being assisted by the positive chemio-tactile properties of the saliva.

(2) The antiseptic action of the gastric juice. This property depends upon the presence of hydrochloric acid, which is only present during digestion, and hence its action is intermittent.

(3) The problematical struggle for existence among organisms in the small and large intestine. But little is known in connection with this question; it must therefore, for the present, be disregarded.

The constitutional defences are those brought about by the reaction of the tissues to the action of bacteria and their products. Collectively, they constitute what is known as the resistance of the body, and they vary in degree of resistance in each individual. As the effect of oral sepsis is to throw a continuous and increasing strain upon these natural defences, it is not surprising that at times the defences break down, and that constitutional changes of a marked character follow.

CONDITIONS ASSOCIATED WITH SEPTIC PROCESS ABOUT THE MOUTH.

(*a*) Local infections.

(*b*) Affections due to the spread of infective material

along natural channels, such as the œsophagus, Eustachian tube, parotid duct, trachea, &c.

(*c*) Affections due to the continuous passage of bacteria into the tissues, or absorption of toxins or other abnormal oro-gastro-intestinal products (sapræmic or toxæmic conditions).

(*d*) Conditions due to bacterial infection of the tissues at some point or points in the alimentary tract (septicæmic conditions).

(*e*) Diseases influenced by oral sepsis.

(A) LOCAL INFECTIONS.

(1) Of the upper respiratory tract.
(2) Lymphadenitis.

(1) Upper Respiratory Tract.

The infection may spread backward and infect the tissues with which it comes into contact. Thus it may lodge in the crypts of the tonsils, infect them, and lead to their enlargement. Stewart examined 231 cases of unilateral enlargement of the tonsil in school children. He found that 135 had dental caries, generally of the lower molars, and oral sepsis on the side of the enlarged tonsil; that sixty-seven had dental caries and oral sepsis on both sides; that sixteen had no oral sepsis; and that fifteen had caries and oral sepsis on the opposite side. He states that unilateral enlargement of the tonsil is not found in infants. In the same paper, he records cases of laryngitis that cleared up on the treatment of the associated oral sepsis.

In *chronic pharyngitis* it is often impossible to trace any source of infection other than from the mouth. Hunter states that this disease is always associated with septic gastritis due to oral infection.

Bellei records a case in which a woman who had suffered from chronic general periodontitis for many years developed suddenly symptoms of mastoid suppuration. She was operated on for this condition, but subsequently developed septicæmia and died. He was of the opinion that the mastoid suppuration was due to infection from the mouth.

(2) Lymphadenitis.

Chronically enlarged glands in the neck are due, with few exceptions, to the influence of bacteria. Such enlargements in children occur with very slight stimuli and their resolution, even after the removal of the cause, may take many years and is seldom complete.

Enlarged cervical glands will thus be found to be due either to *pre-existing* causes such as acute fevers, stomatitis, catarrhal or suppurative conditions of the oral or nasal mucous membranes, &c.; or to *existing* causes, namely, septic conditions of associated parts. There is often great difficulty in diagnosing with any certainty which tissue or organ is the source of infection. It may be impossible to do so, and a combination of causes, such as chronic stomatitis, inflamed or dead tooth pulps, tonsilitis, and adenoids, must be held responsible.

The numerous points from which a cervical gland can be infected and the want of agreement upon what is to be called "enlarged" has led to a marked difference in the statistics which have been published upon the subject. It is questionable whether a gland should be described as enlarged simply because it is palpable. From a clinical point of view a very satisfactory standard may be established by the following method: turn the head to the side opposite the glands to be examined and

let the light fall athwart the neck. If the glands cause irregularities of the surface they may be considered as enlarged. Clinical experience shows that enlarged cervical glands are more frequently due to infection through the tonsil than infection through the tooth pulp. The teeth which give rise to most trouble are the first and second lower molars when inflammation and necrosis of the pulp occur before the closure of the apices of the roots.

Enlarged cervical glands are in a condition of lessened resistance and often become tuberculous. For this reason they are often spoken of as being in a *pretubercular condition*. There are probably three important paths by which the cervical glands may become infected with tubercle bacilli, namely, the intestine, the adenoid tissue about the throat, and the teeth. The intestine at the present moment is considered to be their most frequent point of entrance into the body. Tubercular organisms have occasionally been demonstrated in enlarged tonsils and adenoids, which is indirect evidence that these may at times be the gate of entrance. Although infection through the tooth pulp is possible, the evidence at present rests upon somewhat insufficient ground. Starck records two cases of some importance. In one man, aged 18, he found tubercle bacilli in coverslip preparations made from his decayed teeth; in the other, a female, aged 14, the bed of the first left lower molar consisted of characteristic tubercular tissue. Both cases had tubercular cervical glands on the same side as the oral lesions.

It is not always possible to be sure whether cervical glands are tuberculous or not. Under such circumstances, if an obvious source of septic infection be present, it should be treated and the patient kept under observa-

tion for some time before operative measures on the glands are undertaken. If this rule be followed, it will be found that many glands, some possibly slightly tuberculous, will resolve in a remarkable manner, and the patient be thus saved the hazard and disfigurement of an operation. If, on the other hand, the glands are obviously tuberculous and it is considered abvisable to operate upon them at once, all sources of possible infection should at the same operation be dealt with, so that a recurrence of the trouble may be rendered less probable.

(B) SECONDARY INFECTIONS DUE TO THE PASSAGE OF INFECTIVE MATERIAL ALONG NATURAL DUCTS.

(1) Gastro-intestinal affections.
(2) Secondary parotitis.
(3) Secondary pancreatitis.
(4) Septic diseases of the bile duct.

(1) Gastro-intestinal Affections.

(*a*) *Septic Gastritis, Enteritis, and Colitis.*—There is ample evidence to show that a close relationship often exists between oral sepsis and gastric affections. In many instances the gastric affection is the direct result of the constant swallowing of septic saliva, and cases are constantly occurring in practice in which, with the removal of the oral sepsis, the gastric condition completely clears up. It is no exaggeration to say that the majority of cases of dyspepsia in middle life owe their persistence to oral sepsis. Many patients, however, especially those under forty years of age, have a remarkable resistance to oral infection, and it is by no means uncommon to meet with cases in which a pyorrhœa of

many years' duration is unaccompanied by any apparent or constitutional trouble; but after this age such cases become less common.

It is possible that the organisms themselves, acting upon the normal mucosa of the stomach, may be able to give rise to a septic gastritis; but it is probable that the chain of events is usually as follows: The food, imperfectly masticated and incorporated with infected saliva, undergoes excessive fermentation, with the result that sooner or later a catarrhal inflammation is started. The sticky exudation from the inflammation forms an excellent nidus, upon which the organisms can grow and directly affect the gastric walls. "A septic catarrh is set up, never to be got rid of, but continuously sustained by an influx of septic organisms into the stomach; if continued long enough, the chronic catarrh leads to the usual effects of a glandular catarrh — viz., glandular atrophy, with increase of interstitial tissue around" (Hunter).

A case of an infective character is quoted by Hunter, who believes that the gastritis was directly traceable to the mouth condition. The following is the history: "This was a case of subacute gastritis in a woman, aged 62. The patient suffered from severe intermittent sickness and gastric pain, necessitating the use of morphia, lasting eight months, with loss of weight and increasing weakness. Cancer was suspected, but on examination no sign of malignant disease was visible in the stomach, the abdomen, the rectum, or the uterus. Constant complaint was made of a bitter taste in the mouth, nausea, and a loathing and distaste for food. The vomit in this case was loaded with streptococci, staphylococci, and a few bacilli. The only teeth present were three roots, around which there was a free dis-

charge of pus. With removal of these teeth the gastric condition rapidly improved."

The sepsis from the stomach escapes through the pyloric opening and directly infects the bowel. Thus we often find associated with septic gastritis symptoms of intestinal trouble, such as constipation, diarrhœa, and possibly some instances of mucous colitis. *Post-mortem* evidence offers ample proof of the frequently concurrent sepsis of the whole intestinal tract. Peter Daniel, indeed, maintains that in cases of oral sepsis the lower part of the ileum is always in a congested state.

The *prognosis* in these cases depends chiefly upon the duration and nature of the symptoms, and upon the age of the patient. It may, as a rule, be safely concluded that the younger the patient the better the prognosis; and that old-standing cases in middle-aged and old patients will only improve in so far as the damaged stomach is capable of resolution in the absence of further infection. Experience teaches that improvement of more or less degree occurs in all cases. It is clear, however, that if there be an increase of interstitial tissue the glandular elements of the stomach must become progressively disabled.

(*b*) *Gastric Ulcer.* — Many eminent surgeons have expressed the opinion that oral sepsis is responsible for many gastric ulcers. But such an opinion cannot at present be unreservedly accepted. There is no doubt that the so-called gastric ulcers of young girls may occur in patients whose mouths are perfectly healthy; and it is equally certain that if these patients are fed for long on "slops," or by the rectal method, oral sepsis does result, unless scrupulous care be taken of the mouth. Patients suffering from chronic duodenal or gastric ulcers

generally have unclean, often foul, mouths; but here, again, it is difficult to decide whether the oral condition preceded or followed the duodenal and gastric. If oral sepsis is not a direct cause in these cases, it is certain that it exercises, when present, a marked influence over the course of the ulcers; and very little improvement takes place until it is treated.

(2) Secondary Parotitis.

In cases of fistulæ in man and in the normal ducts of animals Bond showed that particles of pigment deposited at their orifice or in their lumen were carried up the ducts in a direction contrary to the flow of the normal secretion. He concluded that the movement of these particles was due to an upward current in the mucous which normally covers the epithelium of the ducts. Micro-organisms may thus be carried up to the glands and washed down again during active secretions, and no infection of the gland will appear unless certain abnormal predisposing causes be present. Bucknall, in a paper dealing with secondary parotitis, states that one or other of the following conditions must be present:—

(1) The number or virulence of the organisms at the mouth of the duct must be increased.

(2) The general vitality of the patient must be lowered.

(3) The quantity of secretion passing down the duct must be diminished.

(4) The quality of the saliva must be changed.

Bucknall adds to the last condition, lowered bactericidal quality of the saliva; as recent research has proved that this property does not exist, it must be omitted. In cases of oral sepsis the first condition is always present and the second generally.

Microscopical evidence furnishes strong proof that secondary parotitis is an ascending infection. According to Bucknall, the appearances are as follows: "(*a*) the ducts become blocked with débris containing micro-organisms; (*b*) inflammation first begins in the centre of each lobule around the ducts, and at a point furthest away from the vessels; (*c*) many lobules are simultaneously affected. Each becomes centrally necrosed, and then finally, by extension, they fuse to form a multilocular abscess cavity." Bacteriologically it has been frequently shown that the organisms present in the duct and the oral cavity are identical.

Secondary infection of the parotid (and of the submaxillary and sublingual glands) is uncommon. It occurs in cases of gastric ulcer, of typhoid fever and after surgical operations, or, speaking more generally, in those cases in which the functions of the mouth are not properly exercised, and in which artificial oral cleanliness has not been carefully observed. A case is reported by Maggs which was directly traceable to an abscessed molar.

(3) Secondary Pancreatitis.

Certain cases of pancreatitis would appear to be due to an ascending infection, the microscopical examinations showing that the inflammation begins and is most marked around the ducts in the centre of the lobules, just as in the case of secondary parotitis. It is highly probable that some of these cases may depend indirectly upon oral infection. The same may be said of the rare condition known as hypertrophic biliary cirrhosis (Hanôt's disease) which is probably secondary to an ascending infection. Both these diseases would probably repay investigation from this point of view.

(4) Septic Disease of the Bile-ducts.

In the fatal disease known as suppurative cholangitis, probably nearly always an ascending infection, the common bile-duct must be infected from the duodenum. In one case of this nature, the patient, a young adult male, had a most foul mouth; his gall-bladder was full of stones and suppurating, and his liver was riddled by numerous abscesses.

(C) CONDITIONS DUE TO THE CONTINUOUS PASSAGE OF BACTERIA INTO THE TISSUES, OR TO THE ABSORPTION OF TOXINS, OR OTHER ABNORMAL ORO-GASTRO-INTESTINAL PRODUCTS.

(1) Certain forms of anæmia and debility.
(2) Cardiac irregularity.
(3) Diseases of the nervous system.
(4) Rheumatism and infective arthritis.
(5) Deformities of weakness.

(1) Certain Forms of Anæmia and Debility.

Anæmias.—In a series of papers communicated by Hunter during recent years to various journals, he has drawn attention to the relation of oral sepsis to the severe anæmias.

Septic Anæmia.—Under this name Hunter describes a condition in which the "septic factor" is predominant, and owes its origin to sepsis in connection with the oral, gastric, and intestinal tract. This anæmia in its severe form is characterized by (1) "an oligocythæmia comparable to that found in Addison's anæmia, including the existence of poikilocytes, normoblasts, and at times megaloblasts"; (2) hæmorrhage; (3) dirty yellow and anæmic complexion; (4) the frequent existence of oral, gastric and intestinal sepsis and symptoms; (5) fever;

(6) the severe and often fatal course the disease takes; (7) nervous effects and symptoms in many cases; (8) chronic nephritis; (9) favourable prognosis if the cause is removed in time; (10) absence of hæmolytic and bone marrow changes found in progressive pernicious anæmia. The relationship of this form of anæmia and oral sepsis is shown by the rapid improvement which follows the removal of the sepsis.

Pernicious Anæmia.—Hunter gives to oral sepsis the important position of being the chief predisposing cause of this anæmia. He considers that it acts by producing an unhealthy condition of the stomach and intestines, "which favours the contraction of the specific (hæmolytic) infection responsible for the disease, and which favours its continuance after its contraction," and he holds that the essential feature of pernicious anæmia is an excessive destruction of blood corpuscles which takes place in the portal system. The usual argument brought against this view of the etiology is that cases of pernicious anæmia are comparatively rare, whereas cases of oral sepsis are extremely common. Nevertheless, the association of pernicious anæmia with oral sepsis, either past or present, is usually found, and until clear evidence is brought forward that the disease does occur independently of oral sepsis, the opinion of Hunter cannot be disregarded.

Chlorosis.—This form, so common in young females, does not appear to be due to oral sepsis. Should oral sepsis, however, be present, it is usually found that the cure of the case will either be retarded or even impossible until the mouth is attended to.

General Debility.—In cases of so-called "general debility," which is often apparently due to the absorption of abnormal intestinal products, most convincing results

are obtained by the treatment of oral sepsis if it be present. The relation of oral sepsis to general debility in children is extremely important, and will be found dealt with in a later chapter.

(2) Cardiac Irregularity.

Cardiac irregularity may be due to oral sepsis in two ways: firstly, by the absorption of bacteria or toxins from the mouth or alimentary tract which have a specific affinity for cardiac nerves; secondly, by leading to the form of dyspepsia known as flatulent dyspepsia. Douglas Powell records an interesting case of a lady who suffered from cardiac irregularity and frequent attacks of spasmodic heart pain, unassociated with valvular disease. She refused to have her mouth, which was in a very unhealthy condition, attended to, and went through a course of treatment with only slight relief. She subsequently consented to have her oral condition treated, and from that time her cardiac symptoms ceased.

(3) Diseases of the Nervous System.

Under the term of "toxic neuritis," Hunter and Goadby have published cases which appear to owe their origin to oral sepsis. In one of Hunter's cases, a male, aged 33, the illness began with diarrhœa, pains in the stomach and vomiting, and these symptoms lasted three weeks. A month later he had weakness of the hands accompanied by a sensation of "pins and needles." During this time he suffered from acute pains in his stomach, and was very depressed and anæmic. Two and a half months after the onset of his illness, he exhibited marked weakness and atrophy of all the muscles of both arms, including his deltoids. His mouth presented the most intense condition of oral sepsis, which

the patient admitted had been present for twelve years. He improved rapidly on the treatment of the oral sepsis.

The disease known as *subacute combined degeneration of the spinal cord*, commonly associated with more or less profound anæmia, is held at the present day to be due to a toxin. It is thought by Batten and others that the toxin, in many cases, has its origin in chronic infections of the mouth and gastro-intestinal system.

The late T. D. Savill held oral sepsis responsible for many cases of neurasthenia. One patient, a woman aged 32, who consulted him, showed all the symptoms of aggravated hysteria. The usual remedies had no effect upon her until some stumps concealed beneath her artificial plate were removed, when she rapidly recovered. He believed that in a large proportion of his out-patients exhibiting nervous symptoms the cause of their trouble could be traced to pyorrhœa alveolaris.

(4) Rheumatism and Infective Arthritis. (5) Deformities of Weakness.

Under the term "Arthritis Deformans" three distinct diseases may be classed, viz., osteo-arthritis (hypertrophic arthritis), rheumatoid arthritis, and infective arthritis. The first appears to be distinct from the other two in its etiology, course, morbid anatomy and prognosis; and infective conditions do not play an important part in its genesis. Of the other two it is probable that rheumatoid arthritis, though often indistinguishable clinically from infective arthritis, is a definite disease; but as wide differences of opinion exist as to its etiology it will not be dealt with further in this chapter.

In *infective arthritis* (non-suppurative) the affected joints are generally the larger; sometimes the small

joints of the hand are affected, but not as a rule the terminal ones. The swelling around the joints is fusiform in shape, the skin is reddened, and the glands in the neighbourhood are enlarged. In endeavouring to locate the source of the infection it is necessary, first of all, to exclude gout, syphilis and acute gonorrhœal rheumatism. The commonest points of infection are the mouth, the female generative organs and the male urethra; less common are aural sepsis, or any other form of sepsis, such as bronchiectasis. It is most important to remember that *more than one source of infection may be present.* When the infection arises from two or more sources, it will be found that the treatment of one source of infection will lead to some improvement in the patient's condition, but that the condition will remain stationary or recede unless the other source or sources be dealt with also. That the amount and degree of arthritis depends upon the amount of septic absorption taking place is brought out clearly in a case reported by Dr. Percy Kidd. The patient, a female, aged 26, was affected with bronchiectasis. Six months after the onset of the illness, the sputum became offensive and more copious, the ankle joints were affected, and later the wrist and other joints. The joint affection improved with the lessening in quantity and the decreasing in foulness of the sputum, and vice versa.

Wirgman and Turner give short notes of thirteen cases of rheumatoid arthritis (? infective arthritis), in all of which, with two exceptions, pyorrhœa alveolaris was present. In most cases distinct improvement followed the treatment of the mouth.

The prognosis depends upon the changes that have taken place in and around the joints, the capacity of the

patient for recovering, and the possibility of removing all forms of sepsis. Although a complete cure, as a rule, cannot be looked for, it generally happens that the disease becomes stationary for a time, and later a very gradual improvement takes place. So important are these cases, and so essential is it to deal with them in their early stages, that septic foci in the mouth, if they do not at once respond to treatment by conservative methods, should be dealt with radically by the removal of the teeth. Moreover, it must never be forgotten that more than one focus of infection may be present.

Chronic Rheumatism. — The condition known as chronic rheumatism implies a class of symptoms of a somewhat similar nature, but due often to dissimilar causes. The belief that many cases are traceable to intestinal disturbances such as oral sepsis, flatulent dyspepsia, and constipation is certainly supported by clinical experience. In this connection the reader is referred to an interesting paper on "'Nodular' Fibro-myositis," by Maxwell Telling. This author clearly demonstrates that the pain in many of the cases of so-called muscular rheumatism is due to areas of "focal inflammatory hyperplasia of the connective tissues," which result in nodular thickenings of varying degree. Such inflammatory foci "may be limited in number, even to a single area, or they may be scattered widely through the fibro-muscular structures of the body." He believes that disturbances of the alimentary tract play a somewhat important part in the origin of this state.

Fairbank holds oral sepsis responsible for many cases of deformities of weakness. He maintains that no examination of these cases which does not include the mouth is complete, and no treatment thorough that does not deal with it when found. The most striking effects

he finds in the cases of flat feet occurring in young men and lads who are pursuing an ordinary healthy life. This aspect of the subject is one of great interest and well worthy of investigation.

(D) CONDITIONS DUE TO INFECTION BY BACTERIA OF THE TISSUES AT SOME POINT OR POINTS IN THE ALIMENTARY CANAL. (SEPTICÆMIC MALADIES).

(1) Infective endocarditis.
(2) Septicæmia and Pyæmia.
(3) Sub-acute Septicæmia (?)

(1) Infective Endocarditis. (2) Septicæmia and Pyæmia.

The etiologies of these conditions are closely allied and may be conveniently considered together in their relation to oral sepsis. The fact that the mouth is the most septic part of the body and the spot where slight injuries of the surface are of frequent occurrence renders it probable that it is often the source from which the infection in these cases gains entrance to the body. Such a supposition is rendered more probable when it is remembered that of the various organisms that have at one time or another been considered the causative agents in these conditions, many are commonly found in the mouth.

Cases of septicæmia are generally associated with an acute oral condition, but may at times be indirectly related to chronic sepsis. Thus Bellei records a case which illustrates the sequence of events that preceded the general infection : namely, (*a*) Suppurative periodontitis, (*b*) Septic tonsilitis, (*c*) General infection.

The patient was a man, aged 48. He had been in

good health until the age of 40 years, when he began to suffer from suppurative periodontitis. One day he was attacked with a severe sore throat which he disregarded, and went to business as usual. The same evening he had an attack of shivering followed by high temperature. In the course of the next few days an abscess formed in the right tonsil. Four days after the beginning of the pharyngeal inflammation the abscess was opened. The pus on cultivation showed the presence of a very small streptococcus and of a saccharomyces. The patient improved after the operation, but the next morning he was suddenly seized with a shivering fit, followed by a rise of temperature to 104°. This attack proved to be the commencement of a general infection to which the patient succumbed. A micro-organism was isolated from blood drawn from a vein during life, and was found to possess the same microscopical character as the micro-organisms found in the abscess.

Many cases of infective endocarditis attributed to oral infection have been reported. The following case, recorded by Ewart, is well worthy of attention.

J. C., aged 26, an in-patient in St. George's Hospital. Whilst at work he had had a sudden seizure, which left him aphasic and paralysed on the right side. Nothing abnormal was found on physical examination of the chest and abdomen with the exception of a slight systolic murmur. During the week following his admission, his temperature vacillated from 100° or 101° in the evening to normal in the morning; the pulse and respirations gradually rose to a maximum of 120 and 134 respectively. The diagnosis remained doubtful until the discovery of a changing and increasing murmur defined the case as one of a malignant endocarditis. No improvement took place under treatment. Death was rather

sudden and unexpected. "The state of the mouth was unusually bad. Besides stomatitis there was an exceedingly foul condition of numerous stumps. The odour of the breath was intense, and reminded one of the worst smell of decaying and macerating bones. Some of the hollow teeth contained plugs of offensive decomposing material." A very thorough *post-mortem* examination was made. Dr. Ewart's remarks are of importance. "The choice lies between classing it in the group of primary or 'idiopathic' cases, because, had all examination of the mouth been omitted no disease recent or old would have been found throughout the body; or, on the other hand, giving full recognition to the fact that the mouth was profoundly diseased, that it was infected with organisms, and that it presented large ulcerated surfaces through which access to the circulation must have been given to the infection."

(3) Subacute Septicæmia (?).

Apart from the acute form of infection there is a large class of cases, characterized by irregular temperature of long duration, to which no cause can, as a rule, be assigned. Many of these, undoubtedly, owe their origin to oral sepsis and depend upon a *continuous* subacute infection of the tissues. For convenience they are here classed as subacute septicæmia.

Smith and Barnes give a very accurate account of a case suffering from peculiar general symptoms and oral sepsis, of which the following is an abstract:—

"The patient, a male, aged 35, had travelled about in the British Isles, but never abroad; he had enjoyed good health except for several attacks of influenza and a nervous breakdown at the age of 16. There was no

history of rheumatism, gout, syphilis, gonorrhœa, or alcoholism. On March 27, 1909, he had aching in the head, back and limbs. He was better the next morning, but was taken ill on April 2, with violent headache. In two days he recovered, only to have further attacks on the 9th, 12th, and 15th of the month. On the 16th he consulted his doctor, and gave a clear account of his attacks, which, he said, were all alike. The first thing he noticed was the appearance of a red spot on one limb (near a joint), which enlarged until it was about ¼ in. in diameter. At first painless it soon began to burn, and then the neighbouring joint became painful and swollen. In the course of a few hours he felt chilly and his temperature rose to 103° F.; with the temperature the headache and pain increased, so that the joint could not be moved. About an hour later, perspiration commenced, which soon became so profuse that the bedclothes were saturated with moisture. The pain lessened with the onset of the perspiration and disappeared in about an hour, whilst the perspiration itself continued for many hours. After such an attack he passed a good night and felt comparatively well next morning.

"On examining the patient (April 16) the tongue was found clean, but of rather brighter red than usual. The bowels were regular; urine neutral and clear. There was a slight macular rash over the abdomen. With the exception of some labial herpes there were no sores of any description. No disease in the ears, nose or throat was found. He wore an artificial plate, but had some teeth of his own, which were carious in places, and around which the gums were retracted and spongy. He was considered to be suffering from rheumatism and was treated accordingly.

"Two days later (April 18) he had a similar attack, and

again on April 21 and 24. On the last date there was definite swelling on either side of the ligamentum patellæ, and patches of erythema were present on various parts of the body; there was a soft, systolic murmur at the apex; the spleen was enlarged, but not tender. Blood smears were examined: leucocytosis and a slight eosinophilia (6 per cent.) were present. The diagnosis was changed to septicæmia, with infective endocarditis, and it was suggested that the teeth were at the root of the matter. He was treated with 5 gr. doses of sulphocarbolate of soda, ordered to leave out his denture, and given a mouth-wash of permanganate of potash.

"The next attack, on April 27, was slighter; the pain and swelling were in the left heel. After this the attacks were more frequent but of less severity. From May 3 onwards the teeth were removed at intervals. After the removal of the last tooth he had no further attacks, and gained ground rapidly. After a fortnight's rest at the seaside, he returned to work in good health, which has since been maintained."

The quartan periodicity of the attacks was difficult to explain, and made it necessary to exclude malaria by examination of the blood. The authors consider that the result of the treatment affords strong evidence that the oral condition was the cause of the obscure symptoms.

This case, which was very carefully reported, has been abstracted rather fully because it brings out certain important points. Although the attempt to cultivate an organism from the blood failed, the symptoms most certainly point to septicæmia. The result of treatment showed that the *condition of the patient improved pari passu with the gradual removal of the sepsis.* This latter point not only serves to differentiate this case from

acute septicæmia, in which the bodily defences are supposed to be suddenly overwhelmed by a single infection, but it suggests the importance of the treatment of the mouth in septic diseases *even though the disease appears to be definitely located elsewhere.*

Sidney Spokes records the case of a woman who was supposed to be suffering from typhoid fever. Streptococci were found in the blood. She was found also to be suffering from general suppurative periodontitis, and immediately began to get better on the removal of her teeth.

Such cases of persistent temperature of unknown origin are familiar to most practitioners.

(E) DISEASES INFLUENCED BY ORAL SEPSIS.

It has been pointed out several times in the course of this chapter that oral sepsis influences the recovery from many diseases, especially in regard to those connected with the intestine and anæmia. Speaking generally, it may be said that the course of all diseases may be influenced by oral conditions, for recovery depends upon the reactive qualities of the tissues, which themselves depend very largely upon the nutritive substances absorbed from the intestinal tract. In acute illnesses, such as the exanthematous fevers, septicæmia, and pneumonia, the prognosis must certainly be influenced by the presence or absence of oral sepsis. It has been shown by Hunter that the severity of the mouth lesions in scarlet fever, and the duration of the disease, are markedly influenced by oral sepsis. Again, in diphtheria the symptoms vary in intensity. This intensity may depend upon the relation of the individual's resistance and the toxicity of the organisms, and it is reasonable to believe that

superadded sepsis may also be of marked importance. Further, it is probable that oral sepsis, by damaging the mucous membrane of the fauces, may form a favourable medium for the growth of the Klebs-Loeffler bacillus.

The importance of oral sepsis in connection with acute pneumonia can scarcely be doubted. The fevered mucous membranes, the rapid breathing, the open mouth, the frequent expectoration, are in themselves sufficient to lead to unhealthy oral conditions; but add a pre-existing oral sepsis to this picture and the condition and the prognosis must be infinitely less hopeful.

An attempt has been made in the foregoing to give a general rather than an exhaustive account of the important part that oral sepsis plays in the domain of medicine. Its influence is more extensive than that of sepsis in other parts of the body, for the reason that the mouth stands at the entrance to the alimentary tract, and consequently the sepsis pollutes that important canal. The principal effects that are associated with it do not, as a rule, arise directly from oral infection, but indirectly by organisms, toxins, and disordered products of digestion and putrefaction absorbed from the intestinal canal, the defects of which are due in the first place to oral infection. When these essential facts are fully understood, the position of oral sepsis in the realm of pathology will be readily conceded.

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CHAPTER VIII.

Diseases arising from Reflex Irritation from the Teeth.

Affections of the Nervous System—Affections of the Ear—Affections of the Eye.

In past years various lesions of the eye, ear and other organs were attributed to reflex irritation from the teeth. In reading the recorded accounts of many of these cases, one is driven to the conclusion that the diagnoses were often fallacious, and that diseases which were merely coincident were frequently wrongly associated in a causal relationship. The histories of the cases, when read by the light of modern pathology, would seem to suggest that, in the majority of them, the lesions attributed to the teeth were the result of septic absorption rather than reflex irritation.

(A) AFFECTIONS OF THE NERVOUS SYSTEM.

Epileptic and Convulsive Seizures.—It is quite possible that epilepsy may be induced by dental as by any other peripheral irritation. Dr. Brown Sequard found that, after section of one of the lateral columns of the cord anywhere between the medulla and tenth dorsal vertebra, a seizure could be determined by very slight irritation of the fifth nerve. The following case is recorded by Dr. Bakewski [1]:—

[1] *Brit. Dent. Journ.*, vol. xii, p. 280.

"A young female had suffered from epileptic seizures for nine months, the fits having increased in frequency, there being several every day. The usual drugs were tried without effect. Finally, the teeth were examined, and the right maxillary first molar and the left mandibular first molar were found to be carious. These were removed, and the fits ceased entirely and did not return, the patient being kept under observation for six months subsequently. Upon being closely questioned the girl remembered that before the fits commenced she had had some unpleasant sensations in the affected teeth, but nothing that could be described as pains."

In persons subject to epilepsy it is not uncommon for a fit to be induced by the irritation attending dental operations.

Hysteria.—Cases of hysteria supposed to arise from dental irritation have been recorded. Hysteria is such an obscure condition that it is quite conceivable that continued pain from dental trouble might act as an exciting cause in a patient with the peculiar predisposition.

Chorea.—Some medical writers are of opinion that chorea may, in some instances, be connected with dental irritation. If chorea is a functional condition it is quite possible that the dental lesion may induce it, either directly by reflex irritation or indirectly by lowering the vitality. The following case, quoted in the "American System of Dentistry," would seem to indicate that the dental trouble was intimately connected with the choreic condition. The case is related by Dr. C. N. Pierce[1] :—

"A boy, aged 9, had always been in good health until two years ago, when he was attacked with choreic movements, chiefly in the muscles of the face, though present also in the muscles of the neck and shoulders. Owing to their local character, and the absence of the causes usually assigned for the appearance of chorea, it was thought possible that the condition of the teeth might offer some explanation of the trouble. Careful examination of the mouth

[1] "American System of Dentistry," vol. iii, p. 598.

revealed, in addition to considerable overcrowding of the teeth, persistence of the deciduous incisors. Upon their removal the choreic movements at once subsided. After an interval of a year there was a return of the symptoms. Examination of the teeth again showed a persistence of the deciduous molars delaying the eruption of the premolars. Removal of the offending teeth was followed by complete recovery."

Spasmodic Closure of the Jaws.—The following case was reported by Dr. Ewart [1]:—

"The patient, a man, aged 47, moderately addicted to alcohol, was suffering from an extensive chronic ulcer of the right leg. His illness began with a 'severe cold' three weeks before his admission into St. George's Hospital on September 20, 1899. The trismus set in quite suddenly during the night a week later, when he awoke in a fit of suffocation due to the closure of his lips unsupported by teeth. The same nocturnal attacks continued to occur in the hospital for five weeks. The rigidity of the jaws, of the floor of the mouth, of the platysma of the neck, and of the abdominal muscles was intensified by excitement, but there was neither opisthotonos nor any spasm of the limbs. Speech, respiration, and alimentation were much impeded, leading to loss of flesh and weakness. There was a tender and slightly swollen spot on the gum, and the case was diagnosed from the first as one of reflex spasm due to periosteal irritation and tenderness at the left posterior extremity of the upper jaw, and local treatment was recommended. This was finally resorted to after various remedies had proved ineffectual, at the end of October, and the symptoms rapidly disappeared after the tender gum had been freely incised."

This case was probably purely reflex, the absolutely edentulous and healthy condition of the gums excluding the theory that septic infection from the mouth was the cause of the spasmodic closure of the jaws.

Many cases of so-called trismus arising from the teeth are not due to spasm of the masseter muscles as is usually supposed. The closure of the jaw in most of the cases arises from infiltration of the muscles and

[1] *Brit. Journ. of Dent. Sci.*, January 11, 1900.

tissues around; the interference with the movement of the jaw being due partly to mechanical impediment from the inflammatory products and partly to involuntary closing of the jaw on the part of the patient.

Paralysis of the Arm.—It is interesting to note that cases of supposed paralysis of the arm have been recorded as due to dental lesions.

Before arriving at a diagnosis of paralysis, it would always be well to exclude hysteria, and carefully to ascertain that the case is not one where the part is involuntarily kept inactive in order to avoid pain.

Spasm of the Sterno-mastoid (Wry-neck).—A case of spasm of the sterno-mastoid has been recorded by Hancock.[1] The patient, a woman, had suffered considerable pain in the left shoulder, apparently owing to the presence of a carious tooth on the left side of the mandible. The head was drawn down nearly to the left shoulder. On removal of the tooth, the condition disappeared. In this case, as in others recorded, there was probably no spasm of the sterno-mastoid, the head being placed involuntarily in the bent position as a result of the pain.

Tonic Spasm of the Upper Extremities. In the following case, the spasm of the muscles would seem to have been related to the pulp irritation :—[2]

"A robust, strong man, aged 29, who was not in the least nervous, suffered from toothache, caused by an exposed pulp in the upper left third molar. During repeated attempts at luxation of the tooth, which was fixed very firmly, the forceps slid off and struck directly into the pulp. There was at once a tonic cramp of the flexor muscles of all fingers on both hands. Especially the left

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[1] *Lancet*, 1859, p. 80.

[2] This case was recorded by G. Randorf in "Items of Interest."

hand was closed so tightly that the patient could not open it, whilst he had some difficulty in opening the right. The flexibility of the arms was also affected. The patient, describing his condition, said he felt as though a very strong electric current passed through his head and arms, or as if the latter had pins and needles in them; a painful condition which caused the patient, usually showing great powers of endurance, to groan pitifully. Concentrated carbolic acid was applied to the pulp, whereupon the cramp gradually ceased. On the tooth being extracted the patient became normal, and could follow his business undisturbed all day long."

(B) AFFECTIONS OF THE EAR.

Affections of the ear, due to reflex irritation from the teeth are uncommon, and in a large number of cases formerly attributed to reflex irritation from the teeth it is probable that septic absorption from the teeth only acted as an aggravation of a condition started by other causes.

Otalgia.—Otalgia is frequently traceable to teeth. Politzer[1] states that carious teeth are the most frequent causes of otalgia in children. In cases of otalgia, where there are no inflammatory or other abnormal phenomena in the ear itself to account for the trouble, the teeth should always be examined, as under such conditions the otalgia is invariably connected with the teeth. Even when there is evidence in the ear to account for the otalgia, the possibility of the teeth aggravating the pain should not be forgotten.

Deafness without any definite lesions in the ear is at times apparently traceable to reflex trouble from an unerupted third molar. In a case under my care, a patient developed what she termed a "worry" in the ear, which interfered with hearing. The ear was free from organic disease, and the only possible cause in the

[1] Edition, 1909, p. 686, translated by Messrs. Baillie and Heller.

mouth was an unerupted, misplaced third molar. With the eruption and removal of this tooth, the symptoms passed away. Mr. J. Howard Mummery[1] records a case of considerable deafness on the left side, which had existed for months in association with the delayed eruption of the third maxillary molar. The hearing was much relieved immediately on the extraction of the tooth, and was fully restored the same day.

(C) AFFECTIONS OF THE EYE.

Lachrymation. Apart from an emotional flow of tears from the distressing pain of odontalgia, the eyes will occasionally become suffused with tears through sudden pain caused by biting on a sensitive tooth. This is similar to the reflex overflow of tears produced by pungent vapours, *e.g.*, strong ammonia.

Spasm of the Orbicularis Palpebrarum: *Blepharospasm.* Excessive blinking is a mild variety of chronic spasms of the orbicular muscle of the eyelids. Knies has seen a case of this kind disappear immediately after the removal of a painful tooth.

Blepharospasm is occasionally traceable to reflex dental irritation, and in such instances can be cured by attention to the teeth. Blepharospasm may, however, occur in elderly people with edentulous jaws. In these cases it is supposed that the osseous sclerosis present causes the spasms reflexly by the imprisonment of filaments of the dental nerve. Such cases are difficult, in fact almost impossible, to cure. The spasm involves not only the orbicularis palpebrarum, but many of the other muscles supplied by the facial nerve. It varies from slight twitching of the orbicular muscle, to a violent, spasmodic

[1] *Brit. Med. Journ.*, September 9, 1905, p. 553.

contortion of the whole side of the face. It is usually asymmetrical, and is seen more often in elderly females. It has no association with tic convulsif.

Spasmodic Contraction of the Internal Rectus Muscle is manifested by an internal or convergent strabismus. A squint not infrequently develops during the period of the eruption of the deciduous molars, and many mothers assert that the squint is due to the dental irritation. This is true only so far as the disturbance created by the eruption of the teeth acts as the determining cause of the strabismus. The fundamental cause, hypermetropia, being present, any condition which impairs the health of the child, and interferes with the disestablishment of convergence (a dissociation which must inevitably take place to avoid a squint) must lead to convergent strabismus. Convulsions, measles, whooping-cough, adenoids, &c., are all in turn credited as the cause, and early dentition is perhaps as common a determining cause as any.

Spasm of Accommodation.—Spasmodic contraction of the ciliary muscle, similar to spasm of the internal rectus, seems occasionally to have a reflex dental origin. Attention to the teeth will sometimes cause a chronic ciliary spasm to relax, and a troublesome case of apparent myopia to be cured.

Paresis of the Levator Palpebræ Superioris.—Hancock and Nicol[2] have each recorded a case of ptosis which disappeared after the treatment of carious teeth. In all probability, however, the ptosis in these cases was purely functional.

Paralytic Strabismus.—It is impossible for a paralytic

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[1] *Lancet*, 1859, p. 80.

[2] *Trans. Odonto. Soc.*, November, 1895.

squint to be produced reflexly from any source of irritation, though some observers think that they have seen a paralysis of an ocular muscle produced by dental irritation. Such cases are probably due to direct continuity of disease.

Muscular Insufficiency with Diplopia has been attributed to painful carious teeth.

Paresis of Accommodation.—Accommodative failure, attended by asthenopia, is recognized by many surgeons as due to diseases of the teeth. Schmidt[1] found it present seventy-two times in ninety-two cases of dental caries. It probably results, as Knies[2] suggests, from the lack of vigorous innervation on account of distressing pain. On the other hand, Priestly Smith found unimpaired accommodation in fifteen out of sixteen cases of odontalgia.

Amaurosis and Amblyopia.—Amaurosis, or complete functional loss of sight, and amblyopia or impaired visual acuity, are two functional conditions which have over and over again been attributed to reflex dental irritation. There are a great number of such cases on record. In some of these there was probably an acute retrobulbar optic neuritis, the result of inflammation by continuity. In others the elements of hysteria were probably very predominant. Galezowski records a case by a lady who suffered from impaired vision upon a tooth being stopped. When the filling was removed, her vision improved, but relapsed again when the tooth was re-stopped. The removal of the tooth wrought a permanent cure.

The following case of amaurosis consecutive to the extraction of a tooth is recorded by Dr. Santamaria (*La Stomatologia*, Milan, July, 1906).

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[1] *Arch. f. Ophthal.*, xiv, p. 107.

[2] "Relations of Diseases of the Eye to General Diseases," xiv, p. 267

"The patient was a soldier who, prior to enlisting, had followed the occupation of portrait painter. Six months after his arrival at the Florence barracks he suffered from a violent attack of pulpitis in the upper right first molar, which tooth, being badly decayed, was extracted at once. A few hours after the extraction the patient again suffered from a neuralgic attack, the pain radiating from the right cheek to the eye of the same side, and from a rapid and progressive diminution in the visual power of the right eye, which very soon became amaurotic. This condition lasted five days, and, ceasing abruptly, was followed by intense amblyopia. An examination showed that the visual field had decreased both on the right and left sides, but to a greater extent on the former than on the latter, and that there was, in addition, an intense dichromatism for the blue and red primary colours. An external examination revealed nothing worthy of note; in the mouth no pathologic lesion could be seen."

The author's own conclusions regarding the peculiarities of this case are that the amblyopia was not of reflex nature; that the dichromatism was an interesting feature of the case by virtue of the previous occupation of the patient; that the amaurosis, and the dichromatism confirm the fact observed by Trombetta, viz., that the symptoms exhibited by those affected with traumatic neurosis become in preference localized in those organs which for each patient are of pre-eminent importance in his daily occupation; that the nature of the traumatism was entirely out of proportion to the severity of the consecutive neurotic manifestations; that even the most insignificant stimuli may be followed by serious and unexpected consequences in the case of neuropathic patients.

Glaucoma.—In referring to convergent strabismus it was pointed out that the irritation from eruption might in some cases be the determining cause; in like manner it is possible that odontalgia may be the determining cause of a glaucoma. If an eye is anatomically in a

condition favourable for the onset of an attack of glaucoma, severe pain arising from a carious tooth may so lower the vitality of the patient, that the disease may be started. Von Hippel and Grunhagen[1] consider that irritation of the fifth nerve raises the intra-ocular pressure. Priestly Smith's[2] experiments, however, seem to show that the tension is not increased in odontalgia.

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[1] *Arch. f. Ophthal.*, xiv, 1, p. 107.

[2] Priestley Smith, "Glaucoma," London, 1879.

CHAPTER IX.

The Treatment of Dental Disease in Children.

A HEALTHY mouth is essential for the well-being of the child. Unfortunately this is not sufficiently appreciated by the parents and those responsible for the up-bringing of children.

In treating dental disease in children *the importance of rendering the mouth functional* must be clearly kept in mind. If the mouth is rendered functional it will be kept clean by natural means. The chief causes of a functionless mouth in children are :—

(*a*) Mouth-breathing.

(*b*) Caries of the teeth.

(*a*) *Mouth-breathing* arising from nasal obstruction produces a persistent gingivitis of the gum in the front of the mouth, and so predisposes the teeth to caries, especially the incisor teeth. Mouth-breathing is often overlooked, more particularly in children who suffer from intermittent nasal obstruction. A sign of nasal obstruction which may be regarded as diagnostic consists in a marginal gingivitis limited to the incisor teeth, the gums at the back of the mouth being healthy.

Too much importance cannot be attached to the proper performance of nasal breathing, and, if there be sufficient nasopharyngeal trouble present to cause even intermittent mouth-breathing, the trouble must be removed. *Unless a child regularly breathes through the nose it cannot have a healthy mouth.*

(*b*) *Caries of the Teeth.*—It is a matter of common experience that children with tender teeth "bolt their food," and so place in abeyance the function of mastication. If mastication is inefficiently performed the mouth cannot be kept healthy. It is therefore essential that children's teeth should be rendered capable of performing their function without the least discomfort.

Small cavities where the pulp is not involved should be treated by means of filling. If the pulp of the tooth is exposed, the better line of treatment, in the majority of cases, is to remove the tooth. This practice may not coincide with that of the majority of practitioners, but the impossibility of thoroughly treating pulp chambers in children, and the fact that suppuration frequently occurs after treatment, inclines one to the opinion that by extraction the mouth is more likely to be rendered functional. The only instances where conservative treatment is indicated are cases of second deciduous molars in children under the age of 6 years, that is, in cases where the first permanent molars have not erupted; but even in these cases the teeth should be carefully watched, and, if periodontitis appears, they should be removed.

A tooth with a septic pulp chamber in a child often causes no objective symptoms, but yet is the seat of a slight chronic periodontitis; moreover, the constant absorption of the septic matter from the pulp chamber is liable to cause chronic lymphadenitis.

It follows therefore that in the treatment of children our efforts should be directed to establishing the two conditions stated, namely: (*a*) Proper nasal breathing and (*b*) functional teeth. If we fail to obtain these two conditions, a condition of oral sepsis is very likely to be present, even though the greatest care may be taken in adopting artificial methods of cleaning the teeth.

The cases, however, which demand the most earnest attention are those in which the majority of the deciduous teeth are hopelessly decayed. Children presenting this condition are usually ill-nourished, under weight and frequently show symptoms of intoxication from intestinal sepsis. The goal to be aimed at in the treatment of these cases is to give the child a "functional and therefore a clean mouth," and with this object it has been the practice, for several years, of one of the writers to treat this type of case by the extraction of most of the deciduous molars. In detail the treatment would be as follows. If the first permanent molars are in position, the best course is to remove all the deciduous molars. This treatment has the advantage of removing all sources of sepsis and of isolating the first molars, a point of the greatest importance in view of the value of this tooth in mastication. One does not hesitate to remove sound molars if the opposing teeth have been removed. Perhaps this point can be made more clear by giving an example. Suppose that the right maxillary deciduous molars and the left mandibular molars are unsavable, and their removal is called for, then the remaining teeth, namely, the right mandibular and the left maxillary molars, are rendered functionless, and can serve no good purpose; they will form a lodgment for food, and so prevent the mouth being kept naturally clean. The removal, then, of all *non-functional* deciduous teeth is called for, if we hope to render the mouths of children clean. Again, in cases where at, say, the age of 8 years the first permanent molars are carious and have exposed pulps or septic pulps, these teeth are removed in addition to the deciduous molars, the child being left with only the permanent incisors and the deciduous canines.

It has been urged that this treatment deprives the child of masticating power, but such teeth are useless as far as the function of mastication goes; indeed, the tender teeth render the first permanent molars functionless, because a child with tender teeth cannot chew and therefore "bolts" its food.

The improved condition of the child following this rather drastic treatment is most marked. This can best be shown by detailed reference to a few cases.

M. L., a girl aged 4 years 6 months, was suffering from gastro-intestinal trouble when first seen.

Teeth removed: $\frac{\text{d c b a}}{\text{e d}} \Big| \frac{\text{a b c d}}{\text{d e}}$

The $\frac{\text{e}}{\text{e}}$ were not carious and are being retained until the $\frac{6}{6}$ erupt, when they will be removed.

This patient lost her gastro-intestinal trouble and, between June 8 and October 2, increased in weight from 2 st. 4 lb. to 2 st. 8 lb. The normal weight of a girl aged 4 years 6 months is about 2 st. $8\frac{3}{4}$ lb., and the normal increase in weight about 5 to 6 lb. in a year. This child, however, increased 4 lb. in four months.[1]

W. G., a boy, aged 7 years 4 months, suffering from indigestion.

Teeth removed: $\frac{6\ \text{e}\ \text{d}\ \text{c}}{6\ \text{e}\ \text{d}\ \text{c}} \Big| \frac{\text{c}\ \text{d}\ \text{e}\ 6}{\text{c}\ \text{d}\ \text{e}\ 6}$

The teeth were removed on December 6, the patient weighing 3 st. $2\frac{1}{2}$ lb. On February 7, the weight had increased to 3 st. 5 lb., and all symptoms of gastro-intestinal disturbance had ceased. This boy should have weighed about 3 st. $8\frac{1}{2}$ lb.; the normal increase in

[1] The normal weights are taken from "The Diseases of Infancy and Childhood," by L. Emmett Holt.

weight between 6 and 7 years of age being about 5 lb. The boy increased 2½ lb. in two months.

A. R., a boy, aged 6 years 7 months, complained of constant feverish attacks.

Teeth removed: $\frac{e\ d\ b\ a}{e\ d} \mid \frac{a\ b\ d\ e}{d\ e}$

Between December 1909, and October 23, 1910, the weight had increased from 3 st. 5 lb. to 3 st. 10 lb. The feverish attacks have ceased and the child now sleeps and eats well. This boy was of about normal weight. The increase in weight following the treatment was slightly above the normal.

The rapid increase in weight following the extraction is at times very marked. For example, in a boy aged 10 years 2 months, who was suffering from "glands," it was found advisable to remove $\frac{e\ d\ c}{e\ \ c} \mid \frac{c\ d\ e}{c\ d\ e}$

Between June and October this boy increased in weight from 4 st. 6 lb. to 4 st. 11 lb.

In another case, a boy aged 6 years 6 months was seen on November 25, 1910, suffering from marked gastro-intestinal disturbances and glands in the neck. He weighed 2 st. 12 lb. The following teeth were removed: $\frac{d\ b\ a}{e\ d\ b\ a} \mid \frac{a\ b\ d}{a\ b\ d\ e}$

When next seen January 18, 1911, he showed marked improvement in health, and then weighed 3 st. 3½ lb., a gain of 5 lb. in about seven weeks.

The increase in weight is, however, not always shown; in a few cases there is a slight decline, which is frequently traceable to the presence of sepsis in some other parts—for example, the tonsils or the naso-pharynx.

L. G., a girl aged 7 years 8 months. For this child the following teeth were removed : $\frac{e\ d\ c}{e\ d\ c} \Big| \frac{d\ e}{d\ e}$

With the result that in four months the girl gained 4 lb. in weight.

G. R., a boy aged 10 years 2 months. This boy was suffering from glands and general mal-nutrition.

The following teeth were removed : $\frac{e\ d\ c}{e\ \ c} \Big| \frac{c\ d\ e}{c\ d\ e}$

Weights : June, 4 st. 6 lb. ; August, 4 st. 8 lb. ; October, 4 st. 11 lb. The normal weight of this boy should have been about 4 st. 12 lb., and the average increase in weight in four months about 2 lb. The weight increase following the extractions was in the four months 5 lb.

To show how the presence of tender teeth will affect the child the following case is of interest, because the mother has kept a regular record of the child's weights.

The girl from birth had increased progressively in weight until September, 1909, the weight then being about 24 lb., and the child aged 3 years. The mouth then began to get tender and in February, 1910, the child had lost $1\frac{1}{2}$ lb. in weight. The deciduous molars were removed and attempts made by local treatment to preserve the incisors. Within one month the child had increased in weight to $24\frac{3}{4}$ lb. By September 3, the weight was $27\frac{1}{2}$ lb. The incisors now began to give trouble and the child was seen on October 6, when it weighed $25\frac{3}{4}$ lb. These teeth were removed, and the increase in weight was as follows :—

October	6	..	..	..	..	$25\frac{3}{4}$ lb.
,,	15	..	..	..	..	$26\frac{1}{4}$,,
,,	29	..	..	..	..	$26\frac{3}{8}$,,

The next case is instructive, as it shows that the improvement in health does not necessarily follow the removal of teeth, if other sources of sepsis are present.

A. Y., a girl, aged 7 years 6 months, was suffering from septic teeth, tonsils and adenoids.

On February 23, the following teeth were removed :—

$$\frac{6\ e\ d\ c}{6\ e\ d} \Big| \frac{c\ d\ e\ 6}{d\ e\ 6}$$

The child decreased in weight and did not show any improvement till after the removal of the tonsils and adenoids, when a speedy improvement followed :—

		st.	lb.	oz.
February	23	.. 3	.. 10	.. 8
March	24	.. 3	.. 9	.. 12
May	3	Tonsils and adenoids removed.		
,,	25	.. 3	.. 9	.. —
July	26	.. 3	.. 10	.. 12
October	15	.. 4	.. 0	.. 6

An argument advanced against this treatment is, that the removal of the deciduous molars allows the first permanent molars to come forward, and so cause, in the future, crowding of the anterior teeth. With regard to this argument, it may be pointed out that such travelling forward of the permanent teeth occurs mainly in mouths where the growth of the jaws is interfered with by want of function, due either to insufficient mastication or to lack of nasal breathing. From observation—but one expresses oneself guardedly in this respect—one is inclined to think that if by removing the deciduous molars the first molars can be rendered functional, the growth of the jaw will be stimulated, and room will be made for the development of the second and third molars. With no forward pressure from the second and third molars, and with the first molars occluding correctly, there will be little, if any, forward movement.

But, granting that the suggested treatment by extraction does cause a forward movement and subsequent crowding, the removal of four teeth will easily alleviate the condition. One has to decide between the disadvantage arising from the possible loss of four teeth on the one hand, and the constant presence of oral sepsis and its sequelæ on the other. The risk of sepsis is certainly by far the greater evil. Still further, it must always be remembered that, even if the deciduous molars are retained with the object of preventing a forward movement of the molars, it is quite possible that the removal of teeth to prevent crowding would be necessary, because, in mouths such as these in question, some interference with the development of the jaws would probably have taken place.

In cases where the deciduous teeth are decaying on all surfaces, a condition not infrequently arising from the sucking of sugar bags, or the constant presence of an easily fermentable carbohydrate on the surfaces of the teeth, a considerable improvement can be effected by careful regulation of the diet, and regular cleaning of the teeth, combined with local treatment of the teeth. For local treatment no drug acts better than nitrate of silver, which should be applied to all the carious surfaces of the teeth. To apply the drug, a small piece should be melted on to the end of a steel instrument so as to form a small bead; the nitrate of silver can then be applied at any point with accuracy and safety. About four applications should be made at intervals of about one a week, and after this regularly at intervals of three months. Once a day spirits of wine should be applied to the teeth as follows: the surfaces of the teeth should be thoroughly dried, the spirit applied on cotton-wool, and the saliva kept away,

if possible, for one or two minutes. The spirit in evaporating dehydrates the dentine and apparently hardens the surface. Twice a day, morning and evening, an alkaline mouth-wash should be used. If the directions above given are faithfully carried out, the teeth can often be retained for the normal period. Nitrate of silver must be applied with caution. On no account should it be held between the blades of conveying forceps, as it may slip from them and pass either into the larynx (as is known to have happened in one case), or into the stomach. In the first case, inversion might be tried, but skilled surgical aid should immediately be sought; in the second, a plentiful supply of common salt should be given in order to create a chemical reaction, leading to the formation of the insoluble and inert chloride of silver.

The practitioner should impress on parents the necessity of keeping the first permanent molars scrupulously clean during eruption, explaining to them that these teeth form the vanguard of the second dentition, and that during the period of eruption the destructive agents are most active, the loose flap of gum overlying the crown acting as a food-trap.

As soon as the first permanent molar shows signs of caries in the fissures on the occluding surface, the fissures should be cut out as thoroughly as the patient will permit and filled with amalgam.

CHAPTER X.

Dental Disease in relation to Life Assurance.

THE importance of dental disease in its relation to life assurance has not hitherto received adequate recognition. Although it is now an established fact that dental disease is productive of harm and often serious and lasting injury to the individual at all periods of life, yet, in the medical examination of applicants for life assurance, very little attention is paid to the condition of the mouth. Unfortunately there are available no statistics to assist us in forming a definite opinion as to the extent to which length of life may be affected through the instrumentality of dental disease.

In the case of assurance of infants, it is of course impossible to forecast the condition of the teeth in later life, as dental disease must necessarily be dependent in a very great measure on the use made of the teeth, their environment and other conditions.

Formerly, hereditary transmission was regarded as a very important factor in dental disease. Many authorities appear to have accepted this view on the evidence afforded by the history of carious teeth or of crowded conditions of the teeth in many individuals of the same stock. The advance made in recent years in the pathology of dental diseases and the abandonment of the theory that acquired characteristics are transmitted have greatly modified, if not entirely

changed, the opinions formerly held on the question of heredity, and few dental pathologists now believe that dental disease is inherited. A family history of dental disease is now regarded as of little value in forecasting the future dental condition of any child.

If examination is made of a series of individuals who were insured during their infancy it will be found that, while a few have clean healthy mouths, the great majority are suffering from some form of dental disease, and in many cases the teeth are in such a deplorable condition that very little hope of saving them can be entertained. It is obvious that, in point of general health, the individuals with oral sepsis are at a great disadvantage compared with those who possess healthy mouths ; and it is not unreasonable to assume that their lease of life will be shorter. The insurance companies, in the protection of their own interests, naturally fix the premiums on infant insurance at an amount sufficient to cover the maximum risk, and consequently in cases where the insured possess healthy mouths and the risk attendant on dental disease is thus removed, too high a premium is paid.

The insurance companies cannot be expected to alter their practice as regards the amount of the premiums payable on infant insurance, but a way out of the difficulty might be found by insuring children up to a certain age and then re-insuring them after a proper medical examination which should include the condition of the mouth.

In the case of children between, say, 4 and 10 years of age, the condition of the mouth has a most important bearing on general disorders. When the teeth are carious and have become painful the children bolt their food and gastro-intestinal fermentation is started.

Although the gastric condition thus started is harmful, it is difficult to estimate, even approximately, how far it will affect the future life of the individual, and whether the expectation of life is materially lessened. The longer the condition persists, the greater is the damage to the tissues; on the other hand, as soon as the teeth are rendered efficient a rapid improvement takes place in the general condition of the children (see Chap. IX).

Mouth-breathing as an agency in the production of a septic condition of the mouth and as the forerunner of general periodontal disease has already been referred to. The effects of mouth-breathing on the general health of the individual are, we venture to think, much more serious than is usually supposed, and it is questionable whether at any period of life a mouth-breather should be accepted as a first-class life.

In considering the bearing of dental disease on the insurance of adults it is important to bear in mind that, although the mouth may be in a healthy condition at the time of insurance, immunity from dental disease in the future cannot be relied on. In this connection, however, clinical experience may be of some assistance. If an individual at the age of from 23 to 25 years is practically free from caries, and there are no signs of periodontal disease, there is a strong presumption in favour of a healthy mouth being retained through life, provided that reasonable care is taken of the teeth. In cases where extensive caries is present, but no periodontal disease, the teeth may be rendered efficient by careful treatment, and if proper hygienic measures are taken there is no reason why the mouth should not continue healthy. Where periodontal disease is present, even though it be only in an early stage, there is great probability that the

mouth will become the focus of septic infection later on. Scrupulous care of the mouth may, in some cases, keep the disease in check, but it is found from experience that in the majority of instances the disease makes headway, and the difficulty of preventing a septic condition of the mouth is increased, more particularly in the case of mouth-breathers.

The question naturally arises as to whether, in the case of individuals who are entirely free from periodontal disease between 20 and 30 years of age, it is possible by careful attention to the mouth, to ensure immunity from periodontal trouble in the future. In considering this point, it should be remembered that, when periodontal disease does appear, it is invariably present, at least in the early stages, before the age of 30 years, and usually before the age of 20.

In considering periodontal disease as a focus of infection, it must be borne in mind that the focus of infection is capable of being removed. Also, it must be remembered that, in general periodontal disease, the teeth gradually loosen and are lost one by one, and in this way the focus of infection is removed; but during the process, which often lasts over ten, twenty, or thirty years, the absorption of the sepsis from the mouth may cause irreparable damage to the tissues.

General periodontal disease in the early stages frequently eludes notice, partly no doubt because it is not easily detected, and partly because the condition is not fully understood. It is, however, very important that the disease should be recognized in its earliest stages and that, when present, it should influence the report of the medical examiner.

The following case is a typical example of the serious importance of the disease and of its insidious nature:–

A man, aged 36, at the beginning of last year developed a swelling on the left maxilla, and examination of the mouth showed slight necrosis in this region, with general suppurative periodontitis, necessitating the removal of all the teeth. The previous history of the patient was as follows: Until two years ago his normal weight was 13 st.; he was an exceptionally fine type of man from a physical culture point of view, and had succeeded in carrying off many prizes in important athletic military contests. About two years ago he discovered that he was losing weight, and in February last year he weighed about 9 st. During this period his mouth, he said, had caused him very little trouble, except that he had neuralgia occasionally, and his teeth had from time to time become loose. On questioning the patient, one was able to obtain a very definite history of periodontal disease spreading over a dozen years. Unknown to himself, he had been the subject of periodontal disease for years. For a long period he was able to combat the absorption of the toxic matter, no doubt by the formation of antibodies and so remain immune, but after a time this immunity had broken down, and the effects of the toxic material then became rapidly destructive to the tissue. While suffering from the disease he had been accepted more than once as a first-class life. The gum condition which leads to the general toxic condition was present at the time of medical examinations, and one cannot help feeling that if it had been recognized it would have influenced the opinion of the medical examiner.

In another case which came under my notice recently, a man, aged 40, had been accepted as a first-class life five years ago. His appearance was suggestive of septic poisoning, but he stated that he felt quite well, with the exception of slight flatulence after meals. This patient's

teeth were almost free from caries, but the gums were congested and there was marked periodontal disease. There was a plentiful supply of pus around the teeth, the condition having probably been present for ten or fifteen years. This individual was constantly swallowing toxic matter, and so indirectly damaging his tissues.

As regards life insurance, it seems obvious that in point of general health there is a wide difference between lives such as those quoted above, and individuals possessing clean, healthy mouths ; yet, as a matter of fact, both are treated alike.

In conclusion, we are constrained to say that in our opinion the condition of the teeth has not hitherto received the attention it deserves in medical examinations for life insurance, and we consider that individuals with general periodontal disease should not be accepted, at least not as a first-class life, until the condition has been relieved by proper treatment.

INDEX.