Notes on dental surgery and pathology : with over 150 illustrations ;it is intended as a ready reference for the busy practitioner and a note-book for students; interleaved with blank pages for the reader's own notes and drawings. / by T.W. Widdowson.

Notes on Dental Surgery and Pathology

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IT IS INTENDED AS A READY WORK OF REFERENCE FOR THE BUSY PRACTITIONER AND A NOTE-BOOK FOR STUDENTS.

INTERLEAVED WITH BLANK PAGES FOR THE READER'S OWN NOTES AND DRAWINGS.

BY

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"The Care and Regulation of Children's Teeth,"
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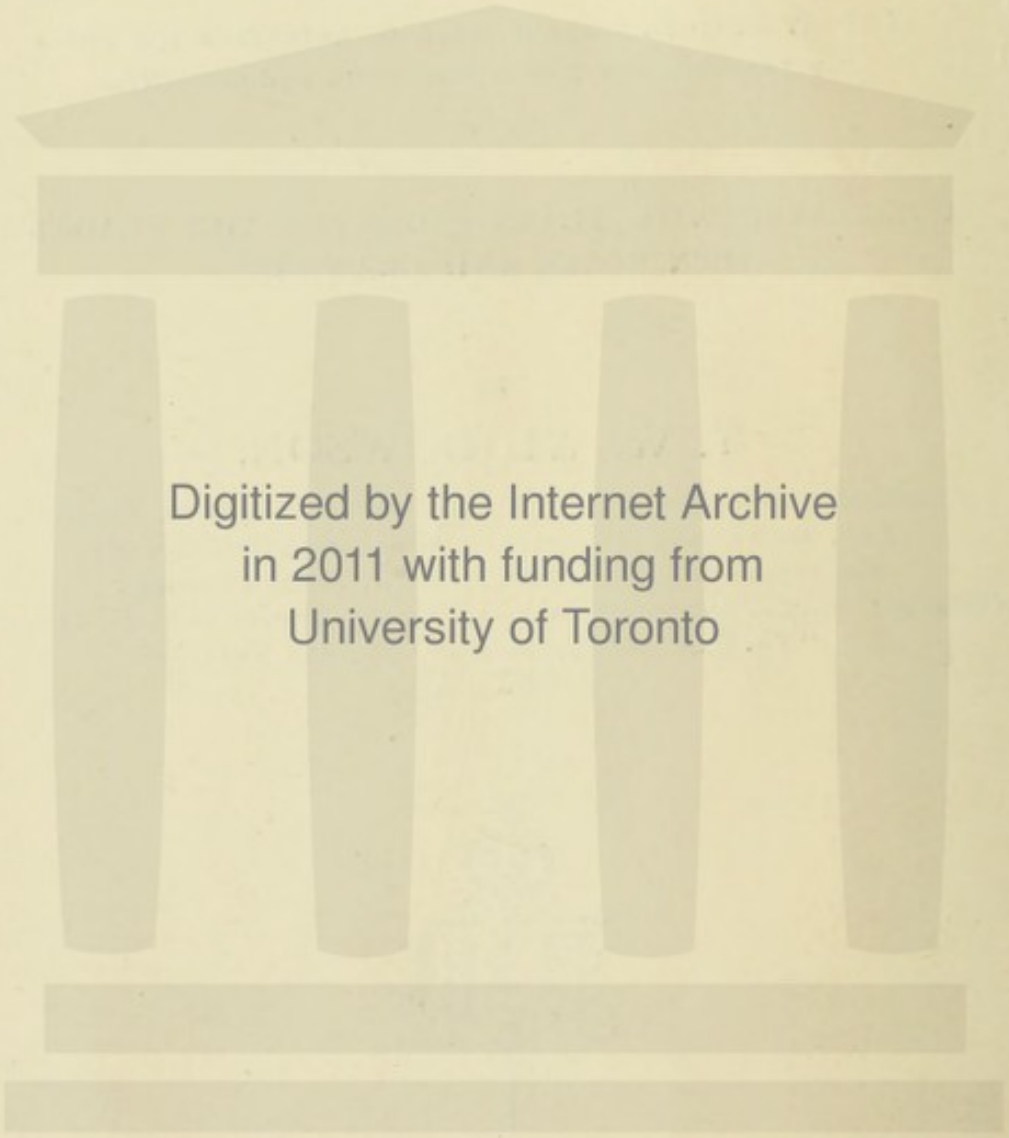

To my Friend,

A. B. CHARLICK, Esq., L.D.S., R.C.S.Eng.,

of Birkdale, Southport,

this Book is Dedicated.

PREFACE.

THIS work is intended as a companion to the author's "Notes on Dental Anatomy and Dental Histology." Originally compiled by the writer for the use of the members of his tutorial class, it has since been enlarged and brought up to date and is now published in book form in the hope that it will be of use to a much larger circle, both to students as a notebook in simplifying their work, and to busy practitioners as a ready work of reference.

The notes are a combination of the author's observation and practical experience with a compilation from the standard works on dental surgery.

Acknowledgment must be made of valuable help obtained from the following:—

"Dental Surgery," by *Tomes and Nowell.*

"Dental Surgery and Pathology," by *Colyer.*

"Histology and Patho-Histology of the Teeth," by *Hopewell-Smith.*

"Aids to Dental Surgery," by *Underwood.*

"Operative Dentistry," by *Marshall.*

"Practice of Dentistry," by *Greenbaum.*

"Dental Materia Medica," by *Glassington.*

"Surgery for Dental Students," by *Mills and Humphreys.*

"Practice of Surgery," by *Spencer and Gask.*

Most of the illustrations are diagrammatic, which, in the author's opinion, are often better for teaching

purposes than are sketches, in which perspective and shading of colour are considered essential, or even photographs.

Thanks are due to Messrs. J. and A. Churchill, and Messrs. Longmans, Green and Co., for their permission to use and for their preparation of blocks of the illustrations taken from several of their publications, and to Messrs. Bale, Sons and Danielsson, the publishers, for their valuable aid in the production of the book.

T. W. Widdowson.

55, *St. James's Street,*
London, S.W.

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CHAPTER I.

PRIMARY OR DECIDUOUS DENTITION.

FORMULA.

$$i\ \frac{2}{2}\ c\ \frac{1}{1}\ m\ \frac{2}{2}$$

There are therefore twenty deciduous teeth.

Fig. 1, adapted from *Colyer's* "Dental Surgery and Pathology," gives the approximate amount of calcification of tooth substance of the several deciduous teeth at various ages.

The development of the deciduous teeth is complete about 2½ years after their eruption begins.

DATES OF ERUPTION.

Most authorities differ with respect to the dates of eruption. The following are approximate:—

	After birth.
Lower central incisors	7 months.
Upper ,, ,, ...	9 ,,
All lateral incisors	12 ,,
First molars	14 ,,
Canines	18 ,,
Second molars	26 ,,

The canines are the only deciduous teeth to erupt between already erupted teeth.

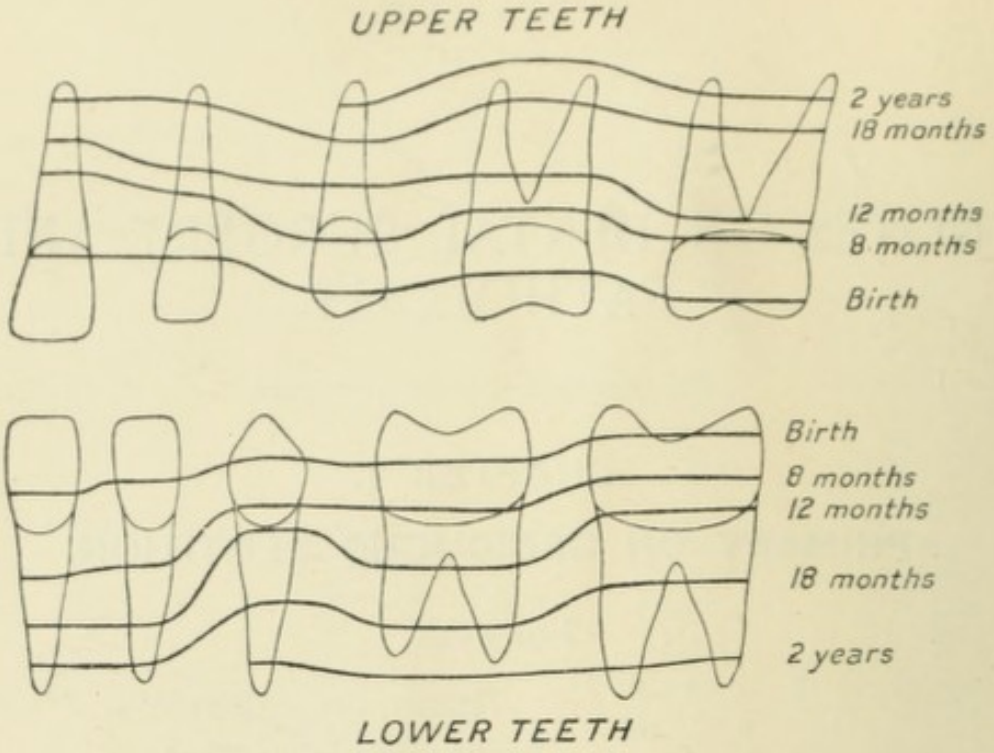

FIG. 1.

Eruption is not a continuous process, and the periods of rest which occur allow of recuperation, preventing prostration or death from exhaustion.

THEORIES FOR CAUSE OF ERUPTION.

The true cause is unknown. The chief theories are:—

(i) Blood-pressure is the active mechanical factor (*Constant*). This is the most plausible, as during eruption there exists an excessive quantity of blood in the part, and pressure from this may force the tooth in the direction of least resistance.

(ii) Rotary movement of the mucous membrane carrying the teeth with it (*Underwood*).

(iii) Pressure from elongation of the root.

(iv) Pressure from deposition of bone in the crypt.

(v) Enamel is an epithelial structure and tends to return to the surface.

(vi) According to *Delabarre*, it is due to a contractile power of the sac wall which forces out the tooth in the same way as the fœtus is expelled from the uterus.

(vii) It may be due to the building of the alveolus around the roots.

Objections to theory (i):—

Why does eruption cease?

Why do some teeth move sideways?

How is the eruption of the lower incisor of a rodent explained? Here the tooth grows from a persistent pulp, and the base develops backwards to some extent, impinging upon and bending the nerve, although the apex moves forwards.

Objections to theory (ii):—

This idea does not explain everything satisfactorily, although, as *Tomes* states, "This would seem tantamount to attributing the tooth's movements to changes in the alveolo-dental periosteum, and the direction of the fibres in an erupting crocodile tooth would lend some support to it."

Objections to theories (iii) and (iv):—

The crown of a tooth often travels a further distance than would have been effected by either of these causes.

Teeth with stunted roots often erupt.

Fully formed teeth sometimes never erupt, and in erupted deciduous teeth at birth the roots are not formed.

Objections to theory (v):—

Glands and nerves which are epithelial structures do not return.

Teeth devoid of enamel (*edentates*) erupt.

Objection to theory (vi):—

No elastic fibres occur in the walls of the tooth sac.

Objection to theory (vii):—

The alveolus is not formed around the roots until the crown has been erupted.

Process of Eruption.

The alveoli of the teeth are absorbed. This absorption goes on chiefly over the labial wall which is removed, the bone behind remaining to help in the formation of the crypts for the permanent teeth. When the crown of the tooth appears through the gum a deposition of bone commences and this embraces the neck, loosely at first but later on firmly. The roots and jaws deepen first in the incisor region. The eruption of the first dentition is usually complete at the end of the second year. When eruption of the permanents is due (sixth year) there is spacing of the temporaries, and their position is more anterior.

General Relation of Unerupted Permanent to one another and to the Erupted Deciduous Teeth.

The crowns of the permanent front teeth lie behind the roots of the deciduous teeth. The crowns of the upper central incisors lie either just behind or just in front of the lateral incisors, being overlapped slightly by the laterals or overlapping them slightly as the case may be. The lower permanent front teeth are very closely packed.

The upper cuspid is far above and out of the arch. The lower cuspid is near the lower border of the mandible, is more anterior than the lateral incisor, and leans towards the median line. The crowns of the pre-molars lie directly under the roots of the temporary molars and are sometimes enclosed by them, so that it is often difficult

to remove the deciduous tooth without removing, or at any rate injuring, the permanent one beneath. The first permanent molar lies immediately behind and in close contact with the second temporary molar. The remaining permanent molars lie behind and on a lower level than the anterior one. If the child is healthy and vigorous the arch gradually spreads at from 4 to 6 years to make room for the larger permanent teeth.

After absorption, the permanent teeth erupt in a similar manner to the temporaries. The permanents stand obliquely when erupted, the temporaries vertically.

Signs and Symptoms of Eruption.

Although in a normal case of eruption of the deciduous teeth there should be few symptoms, such a case is so comparatively rare, and so few infants are free from trouble during the process, due to civilization, &c., that the following may be included in signs and symptoms:—

(*a*) Local.
(*b*) General.

(*a*) *Local:*—
- (i) Heat, redness, swelling, pain.
- (ii) Tendency to bite at anything, such as toes, fingers, dummy teat, &c.
- (iii) Increased flow of saliva.
- (iv) Tenseness and shininess of the gums.
- (v) Gradual disappearance of the swelling as the tooth erupts.

(*b*) *General:*—
- (i) Irritability.
- (ii) Restlessness.
- (iii) Rise of temperature.

NOTE.—This is higher in the morning than at night, the condition being the reverse of what occurs during ordinary fevers, when the temperature is higher at night. A temperature of 104° F. is often reached. This in an adult would occasion alarm, but in a child it would not be nearly so serious.

Disturbances accompanying Eruption.

Although in the majority of cases the teeth should be cut without much trouble it is a process which, when completed, produces relief in the minds of parents because of the popular but mistaken belief that it is the primary cause of most of the disturbances from which children suffer during these two years. Whether it be the cause of or merely coincident with these troubles it is often the case that the following disturbances accompany teething, and although a brief *résumé* of the causes, symptoms, and treatment of these affections is given, it is nearly always advisable to call in the aid of a medical practitioner when such cases arise. They are:—

(*a*) Local.

(*b*) General or reflex.

(*a*) *Local:*—

(i) Simple stomatitis.

(ii) Ulcerative stomatitis.

(iii) Phagedænic stomatitis.

(i) *Simple Stomatitis:*—

Causes:

Local irritation from the erupting teeth.

Putrefaction of food in the mouth.

Rough handling of tooth-brush, &c., in cleansing the mouth, and rough feeding of a refractory child, &c.

Symptoms:

Heat, redness, swelling, pain.

Pyrexia (temperature of 104° to 105° F.).

Treatment:

Mild purge (rhubarb and magnesia).

Scarification or incision of the gums.

Note.—In incising it is not sufficient to make one incision. Make two semi-lunar incisions and remove the portion of gum between (fig. 2).

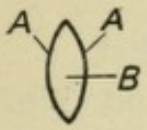

FIG. 2.

AA, the two incisions; B, the portion of gum to be removed.

Some dentists make a crucial incision, but the method just described will be found more satisfactory.

(ii) *Ulcerative Stomatitis:—*

This affection does not occur in healthy and robust children, and is very rarely seen in children of the better classes. The average age at which it is seen is 6 years, and it usually attacks one side only.

Causes:

Malnutrition.
Bad hygiene.
Infected milk.
Scurvy.
Administration of lead, mercury, or arsenic.
Digestive disturbances, &c.

Symptoms:

Heat, redness, swelling, pain.
Bleeding from the mouth during sleep.
Pyrexia.
The swellings over the erupting teeth ulcerate.
The ulcers spread.
The teeth become loose and may fall out.
Excessive flow of saliva.
Disturbance of digestive organs.
Fœtid breath.
Necrosis may occur.

Treatment:

Remove any tooth or teeth causing irritation, or which are septic.

Purge.

Nourishing diet.

Give potassium chlorate internally in doses of from 3 to 10 gr.; 1 gr. for each year of the child's age.

Apply locally silver nitrate, 10 gr. to the ounce; or potassium chlorate with honey, in doses of from 3 to 5 gr.

Frequent overdoses of potassium chlorate may produce *hæmoglobinuria*, a disease, rare in *England*, in which the urine is red or brown.

(iii) *Phagedænic Stomatitis*:—

Causes:

Bad hygiene.

Debilitated condition after exanthemata.

Symptoms:

Ulcerations followed by sloughing.

If untreated this spreads and blood poisoning and death result.

Treatment:

Touch the sloughs with phenol.

NOTE.—Sometimes a blue or purple-coloured cyst occurs over the erupting tooth. This yields to pressure, and if opened up exudes a transparent fluid. It is due to a secretion of fluid between the enamel and the tissue above.

(*b*) *General or Reflex*:—

(i) Disturbances of the cerebrospinal system. (Convulsions, irritability, &c.)

(ii) Disturbances of the respiratory tract. (Cough, bronchitis, &c.)

(iii) Disturbances of the alimentary tract. (Diarrhœa, vomiting, &c.)

Pulse small and quick.
Coma, sometimes death.

Treatment:—

To temporarily relieve the child immediate immersion up to the abdomen in a hot bath should be resorted to, and if the fever be high a cold sponge should be applied to the head.

If teething be the cause, semilunar incisions, as before described, should be made over the erupting tooth or teeth.

Should the condition be due to any cause other than teething a medical man should be consulted immediately, as delay might lead to serious results.

Diatheses affecting Eruption.

Rickets, idiocy, and cretinism (a disease, due to the atrophy or absence of the thyroid gland, producing a stunted, ill-developed, and mentally weak being) retard eruption. Exanthemata, struma, and probably syphilis accelerate eruption, children suffering from congenital syphilis often being born with one or several teeth already erupted. *Colyer* states that syphilis more often than not retards eruption.

The administration of thyroid extract accelerates the eruption of teeth as it accelerates growth generally.

In healthy children there is often a slight irregularity in the dates of eruption, the process being slightly earlier or later than normal. Such an event need cause no anxiety.

Abnormalities of the Temporary Teeth.

(*a*) In size.
(*b*) ,, number.

(*c*) In position.

(*d*) ,, structure.

(*a*) *In size:*—

(i) Large. Upper canines. Second lower molars.

(ii) Small. None specialized.

(*b*) *In number:*—

(i) Excess. Supplemental supernumerary incisors.

(ii) Deficiency. This is rare. If it occurs it is usually symmetrical and in connection with the upper lateral incisors.

NOTE.—*Supernumerary teeth* may be divided into *supplemental*, *conical*, and *tuberculated*. *Supplementals* are those which simulate normal teeth in the arch, and are usually laterals or pre-molars. The *conical* are generally found in the median line between the central incisors of the upper jaw. Sometimes they are found geminated to molars. The *tuberculated* are found on either side of the median line, behind the upper central incisors. These may also be geminated to molars. Supernumerary teeth are nearly always single-rooted. They may be due to *atavism*, as there are six incisors in the typical mammalian dentition, to a bifurcation of the normal tooth germ, or to an accidental inclusion of epithelium along the lines of developmental folds. *Temporary* supernumeraries are often followed by permanent ones.

(*c*) *In position:*—

The individual teeth are very rarely abnormal in position, but in connection with the whole jaw superior or inferior protrusion or open bite may occur.

Superior protrusion:—

Causes:

(i) Excessive development of the upper jaw.

(ii) Arrested development of the lower jaw.

(iii) Thumb, toe, lip, and tongue sucking.

(iv) Mouth-breathing, due to adenoid growths and enlarged tonsils.

Inferior protrusion:—

Causes:

(i) Excessive development of the lower jaw.
(ii) Arrested development of the upper jaw.
(iii) Finger sucking.

Open bite:—

Causes:

(i) Arrested development of the pre-maxilla.
(ii) Arrested development of the ascending ramus.
(iii) Mouth-breathing, due to adenoids.
(iv) Thumb, toe, lip, and tongue sucking.

The habit of sucking the thumb, &c., may be arrested by covering the part with thick bandages, or applying something unpalatable to the organ. Adenoids and tonsils should be removed, and the child taught to breathe correctly.

(*d*) *Structure:—*

The deciduous teeth are rarely *hypoplastic* or honeycombed (see figs. 15, 16, 17, and 18), a condition in which the enamel is pitted and marked, due to the exanthematous fevers or mercury in teething powders. They, however, often show marked signs of *attrition* (see fig. 46), which is a wearing away of tooth substance due to mastication. The pulp becomes protected by the formation of *secondary or adventitious dentine* and under the microscope the tissue shows the *translucent zone*, an appearance which is dealt with in the chapter on *Caries*, well marked. Accessory cusps and roots may occur. With cretinism and congenital syphilis the teeth are not affected structurally, but with rickets there may be a secretion between the follicle wall and the enamel organ, a thickening of the follicle wall, and the formation of a cyst.

Gemination of two or more teeth may exist, usually between a lateral and a canine or a lateral and a central.

NOTE.—*Gemination*, which is more common in the deciduous than in the permanent dentition, is the fusion together of two or more teeth. There are two forms, the *physiological* or *true* and the *pathological* or *false*. The former takes place during development, and fusion occurs between all the tissues of the several teeth, whilst there is only one pulp chamber for all the teeth united. This is the form which is usually seen in connection with the *temporary* teeth. The latter is due to disease and the cementum only of the tissues is fused. It occurs chiefly in connection with the *permanent* teeth.

FIG. 3.

FIG. 4.

Figs. 3 and 4 show cross-sections just below the cervical margins through geminated central and lateral incisors. Fig. 3 is the *pathological* variety, where the cementum only is joined and there is a pulp chamber for each tooth. Fig. 4 shows the *physiological* variety, where all the tissues are joined and there is one pulp chamber only.

EXTRACTION OF TEMPORARY TEETH.

Temporary teeth should not be extracted until the second permanent molar has erupted, except under the following conditions:—

(i) If the permanents are erupting abnormally.
(ii) If the temporary is abscessed.
(iii) If the roots of the temporaries are ulcerating through the gum.
(iv) In most cases where deciduous teeth are retained beyond the normal period. Should these *retained teeth*, however, be firm, healthy, useful in mastication, and not causing any disturbance, they may be left alone.
(v) Teeth causing enlarged glands.

NOTE.—The early removal of the deciduous incisors does not usually lead to abnormality, but the early removal of the second deciduous molar or the canine allows, in the first case, the first permanent molar, and in the second case, the first premolar to erupt into a more anterior position than normal, thus causing crowding.

It is rather a common occurrence for members of the deciduous dentition, especially the upper canines, to be retained until long after the normal time for their natural removal, and this may be due to:—

(i) The absence of permanent successors.
(ii) The tardy development of the permanent successors.
(iii) The development of the permanent successors into abnormal positions.
(iv) The non-eruption of the permanent successors, due to obstruction.
(v) The death of the pulps of the deciduous teeth resulting in septic inflammation and the probable destruction of the absorbent organ. The resulting absorption is slow, irregular, and *pathological*.
(vi) The impaction of deciduous teeth between others.

ABSORPTION OF THE TEMPORARY TEETH.

Some assert that absorption of the temporary teeth is due to pressure from the permanents, others that it is due to the action of an absorbent organ acting in one of three ways, *viz.*:—

(i) Acid secretion.
(ii) Phagocytosis.
(iii) Amœbiform process.

Absorption, however, is probably not dependent upon pressure. "In the frog and the crocodile the successional tooth sac passes bodily into the excavation made before it in the base of the preceding tooth, and therefore if

the eruption had been due to pressure the cells of the enamel organ, &c., would have been crushed and destroyed. Again, absorption not dependent upon pressure attacks the roots of the permanent teeth." (*Tomes.*) Absorption also often begins on a portion of the tooth which has not been in contact with the permanent one. *Peirce* and others state that absorption has been completely accomplished in the absence of a successor.

Process.—Upon any part of the roots of the temporary teeth, about twelve months after their completion, cup-shaped depressions occur. These coalesce, enlarge, and destroy the tissue. The cementum is attacked first, and then the dentine, except a very resistant portion in close proximity to the pulp. Very closely applied to the tooth or teeth undergoing absorption there exists a soft vascular tissue, *the absorbent organ*, upon the surface of which are *giant cells, osteoclasts, or absorption cells.* These are contained in the cup-shaped depressions (*Howship's lacunæ*). The absorption of the temporary teeth is *physiological*, whilst that of the permanents is *pathological*, although the process is similar. In the latter case irritation has produced soreness, a roughening of the surface, and a formation of pus; whilst in the former there are no inflammatory symptoms, and the surface is smooth. The absorption of a temporary tooth is completed about four years after the process sets in. Absorption is sometimes retarded, due to:—

(i) Death of the pulp of a deciduous tooth preventing a quick natural, or *physiological* absorption.

(ii) Late development of the replacing permanent tooth.

(iii) Eruption of what should have been the replacing permanent tooth into an abnormal position.

(iv) Non-development of what should have been the replacing permanent tooth.

(v) Non-eruption, due to obstruction, of what should have been the replacing permanent tooth.

Origin of Absorbent Organ.—The outer layer of the dental follicle of the permanent teeth. (*Hopewell-Smith and others.*)

Points to Distinguish a Temporary Tooth from a Permanent.

Temporary teeth are smaller, the roots are more divergent, the necks are more constricted, the enamel ends abruptly at the necks, and the implantation is perpendicular. They show signs of attrition, and are on a lower level in the jaw. The first upper molar has three cusps, the second four, whilst all the permanent uppers have four; the first lower molar has four cusps, and the second five, whilst the permanent usually have five on the first, four on the second, and five on the third.

CHAPTER II.

THE CARE AND TREATMENT OF THE DECIDUOUS TEETH.

Our duty as dentists should not begin and end with the treatment of disease. The prevention of a disease or a deformity is a more satisfactory achievement than its cure, and this is particularly applicable to disease of the deciduous teeth. It is, therefore, our duty not only to treat disease, but to educate that portion of the public with which we come into contact professionally, in its prevention. It should be impressed upon the minds of our patients that the teeth are developing before birth and that some of them are even partially calcified prior to that event, so that no amount of care and attention, after this has occurred, can change weak and unhealthy tissue into tissue which is the reverse. These facts being recognized, the importance of indirect attention to the primary dentition before birth, by strict attention of the mother to hygiene, diet, and general health, will be thoroughly appreciated.

The period of gestation is often coincident with caries of the teeth of the mother, which during this period should undergo frequent and careful examination, and, if necessary, be rendered sound and aseptic.

Such conditions are absolutely essential to the proper mastication and digestion of the mother's food, and the resulting nutrition of the developing deciduous teeth.

In much the same way as the developing permanents are affected in structure, after birth, by the exanthematous

fevers and other disorders, so also are they and the deciduous teeth affected, before birth, by a disturbance in the general health of the parent, thereby depriving them of their proper nourishment.

Parents and guardians realize that a child's bodily health is just as important as its mental development, but all do not appreciate the fact that the former can often be seriously affected by inattention to the mouth and teeth, which from birth should receive constant attention. The adoption of such a course may and does necessarily involve an increased amount of care, but surely the result, namely, a child with a healthy body and a sound mind, is worth all the added labour and anxiety.

Present civilization, the desire to be relieved of responsibility and, therefore, of motherhood, love of pleasure, &c., are all tending to race deterioration. Many women dispense with the natural feeding of their offspring and its attendant cares and ties. The mother who is healthy and capable of suckling her child and yet refuses to do so, is not only unmotherly, and therefore unnatural, but may be the means of creating an unhealthy and weakly being unpossessed of the power of resisting to any extent the attacks of disease germs. Even in healthy bodies and mouths, pathogenic or disease-producing germs are existent in great numbers; but they fail to produce their specific affections except under certain conditions, when the vitality of the organ which they attack is lowered.

It behoves every mother, therefore, to do all that she possibly can to ensure a healthy child sufficiently strong to resist these attacks, and in most cases this can best be thoroughly effected by natural feeding.

Of course cases occur when either through weakness of the mother or the child, or both, or from some unforeseen cause, artificial feeding has to be resorted to; but

whatever means are adopted, it is wise to keep the child's mouth clean and healthy by sponging daily with boracic lotion or borax mixed with honey or glycerine. This is a preventive of thrush (*parasitic stomatitis*), which is so prevalent among artificially fed babies through the agency of feeding-bottles, the tubes, &c., of which have not been rendered thoroughly aseptic. It is probably

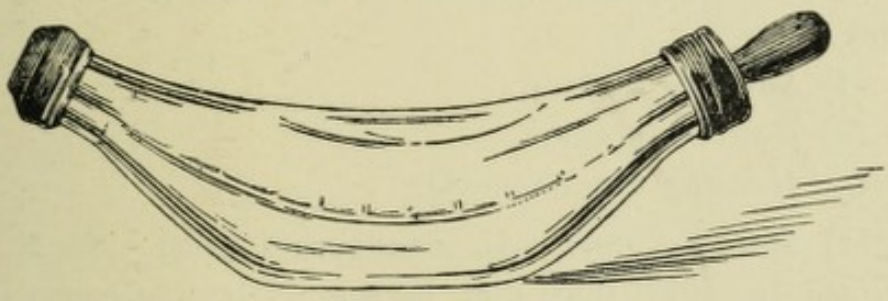

FIG. 5.
Allen and Hanbury's Feeding Bottle.

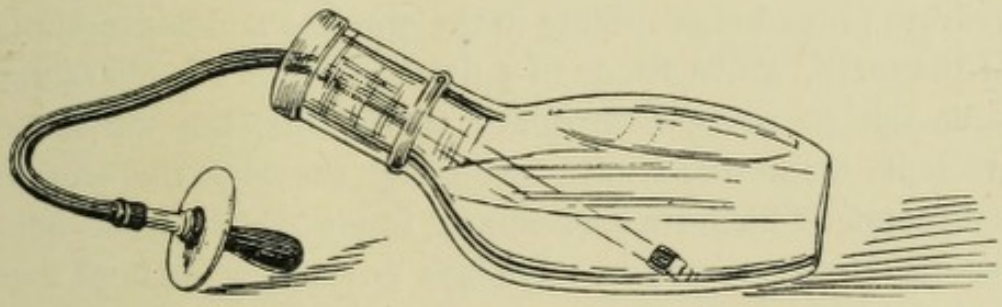

FIG. 6.
The Long Tube Bottle.

unnecessary for the author to suggest that should it be found impossible to feed a child naturally, *Allen and Hanbury's* bottles (fig. 5), which are so readily cleansed and sterilized, should be used for the administration of artificial foods, and not the long-tube bottles (fig. 6), which are cleansed with difficulty.

It should be impressed upon mothers that in the first few years of life the greatest and most active development of the various tissues and organs is proceeding.

Improper feeding, therefore, can readily produce disastrous results to the teeth.

Neither dummy-teats, nor teething powders, such as *Steedman's* or *Stedman's*, which contain mercury, should be allowed, as the former produce abnormality of the jaws, and the latter affect adversely the structure of the teeth.

Three important conditions for the prevention of caries are:—

(1) *Suitable diet.*

(2) *Good hygiene.*

(3) *Healthy environment.*

(1) *Suitable Diet.*—In connection with this *J. Sim Wallace* believes that diet is the most important consideration in the prevention of caries. He states that the diet to be suitable should leave the mouth physiologically clean and healthy. He finds that highly refined carbohydrate foods lodge unduly in the crevices and depressions of the teeth, whilst foods of a fibrillar or fibrous character have not so great a tendency to do this, but stimulate efficient mastication and probably prevent the undue lodgment of bacteria and mixed foods which cause acid fermentation and caries. He lays down four rules of diet to be observed, *viz.*:—

(i) During the first 2½ years of life all starchy or sugary food (except milk) should be given in a firm or fibrous form, so as to stimulate mastication and insalivation, and promote the healthy growth of the jaws and the regular arrangement of the teeth.

(ii) After the age of 2½ years children should always have a considerable amount of the farinaceous food in a form which will stimulate a pleasurable amount of efficient mastication.

(iii) Three meals daily is to be preferred to any greater number.

(iv) Water should be taken *after* the meal.

A diet which, he states, does not produce caries is as follows: —

Breakfast.—Fish, bacon, toast and butter, coffee, and tea.

Luncheon.—Meat or poultry, potatoes, salad, baked bread, pudding, fresh fruit, water.

Supper.—Rusks, toast, or bread-rolls and butter, chicken or fish, an apple, tea, or coffee.

(2) *Good Hygiene.*—Although the axiom "*Clean teeth cannot decay*" is not strictly correct, still, caries can, to a great extent, be prevented by the mechanical cleansing of the mouth and teeth. This prevents the collection of food *débris* upon and between the latter,

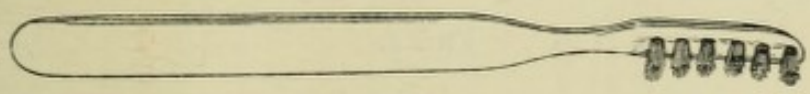

FIG. 7.
Baby's Tooth-brush.

the formation of acids, and the production of an ideal soil for the development and activity of bacteria.

In a very young child this can be accomplished by means of a soft cloth, or a tooth-brush of badger-hair (fig. 7).

When the child is 3 years old a fine horse-hair tooth-brush should be used, whilst after the age of 12, brushes of a harder nature will be necessary. The bristles should be arranged as in figs. 8 and 9.

The brush should not be too hard, as it will then not be sufficiently elastic; nor too soft, since it will then be wanting in strength to fulfil its purpose. The smaller and more numerous the bristles, the better. Horse-hair tooth-brushes are the best.

The cleansing should take place after each meal, if possible, but certainly twice a day at the least, *viz.*,

before going to bed at night, *after which no more food should be taken until the morning*, and on rising in the morning.

Children should be taught *how* to use the brush; brushing not only from side to side, but also with a rotary upward and downward movement. Brushing merely from side to side will cleanse the prominent portions only, of the teeth, leaving the interstices uncleansed. With the upper teeth the brush should be held with the bristles directed obliquely downwards, and with the lower with the bristles directed obliquely upwards towards the crowns, in order to cleanse as far as possible the inter-

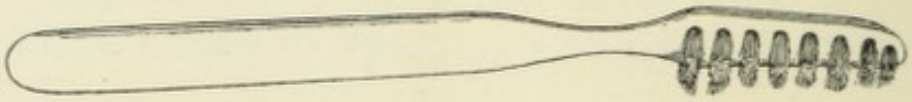

FIG. 8.

FIG. 9.

spaces. The posterior surfaces should be rendered clean by means of a tooth-brush, the handle of which is bent at a convenient angle for the purpose. Brushing, however, is not all that is desirable, and this alone will not free the approximal surfaces from the *débris* of food, &c. This must be removed either with the aid of floss silk or, better still, a quill tooth-pick. Many children, and even adults, brush the teeth simply with the idea of rendering the visible surfaces clean, and do not think of the particles of food remaining between, which eventually putrify and create disease and decay.

After the age of 3 some form of tooth-soap or powder can be used and is necessary for thorough cleanliness, and in the opinion of the author a saponaceous

tooth-powder is preferable, as powders alone are often gritty and tend to lodge between the gums and the teeth, in this way producing irritation and recession of the former. It is often the case that, although the teeth and mouth are conscientiously brushed and cleansed, the method of brushing and the tooth-powder have been such that the roots of the teeth have become exposed by recession of the gums due to inflammatory trouble. Care should therefore be taken never to injure the gums in the process. It is popularly supposed that tobacco-ash or charcoal make good tooth-powders. They should not be used, as not only do they wear away the enamel, &c., but they also collect between the teeth and gums, giving rise to irritation. Camphorated chalk is an excellent and harmless medium for brushing the teeth, as is also the following saponaceous tooth-powder: —

℞	Precipitated chalk	ʒii.
	Light magnesia	ʒss.
	Powdered white soap	ʒii.
	Orris-root powder	ʒii.
	Oil of cloves	♏iv.

A powder consisting of equal parts of calcined magnesium and prepared chalk, a little sugar, and a few drops of oil of wintergreen is also good.

With a tooth-powder or soap there should be *no chemical action*, such action being liable to affect the teeth adversely. The *action* should be merely *mechanical*. It should be borne in mind that friction is the most important item in brushing, for no matter what soap or powder is used it will be of little avail unless the teeth are scrubbed thoroughly and efficiently.

As a general mouth-wash benzoic acid ʒii to ʒii of water, used with the tooth-brush, is to be preferred.

The foregoing treatment for the prevention of decay

is not the only treatment necessary. Parents should be impressed with the desirability of periodical visits to the dentist being made every three months for a thorough examination—scaling, cleaning, polishing, and, if necessary, the filling of any cavities present, or, if judicious, the extraction of abscessed teeth, teeth ulcerating through the gum, or teeth which are producing crowding or other abnormality.

The periodical scaling of the teeth is *most* important. The deposits of tartar start at and under the gum margins, and give rise to slight but imperceptible inflammation. The deposits continue, and should they be allowed to remain, the irritation and inflammation increase, and absorption and suppuration may result.

(3) *Healthy Environment.*—Healthy exercise and fresh air in combination with the systematic attention to the mouth and teeth are also important to health, upon which the perfect structure of the teeth depends. It is also of importance that children should be taught to breathe correctly—*viz.*, through the nose—and any obstruction which prevents this, such as adenoids, tonsils, &c., should be removed, as breathing through the mouth produces crowded arches, and thus caries.

Colyer and others state that a diagnostic sign of mouth-breathing is marginal gingivitis limited to the incisor teeth.

THE DECIDUOUS TEETH IN THE ORDER IN WHICH THEY ARE SUSCEPTIBLE TO CARIES ARE:—

Approximal surfaces of upper incisors and upper and lower molars.

Crown fissures of molars and upper canines.

Lower incisors.

The *etiology*, *symptoms*, and *pathology* of caries of the deciduous teeth are the same as in the permanents

and will be discussed in the chapter on "*Caries*." The disease is an important cause of ill-health in children, and may produce digestive disorders, loss of appetite, loss of flesh, enlargement of the abdomen, pains in the abdomen, anæmia (probably due to chronic poisoning), tuberculous glands, headaches, &c.

Treatment of Caries in the Deciduous Teeth.

When this becomes necessary it is advisable to remember two important rules:—

(1) Gain the confidence of the child by talking about, and taking an interest in, the things which interest and please him or her, and do very little work on the first visit.

(2) Never deceive. Do not tell the patient you are not going to hurt when you know it is impossible not to. A child once deceived in this way will have no faith in you, and will become very difficult, if not impossible, to treat.

Excavate the carious tissue very carefully, and if the cavity is wide, shallow, and sensitive, touch with silver nitrate and remove some decay at each visit. Sometimes a little bicarbonate of soda dropped into a sensitive tooth and rubbed on the gum will relieve tooth-ache. Creosote is a good obtundent for sensitive deciduous teeth before excavating. Before filling, sterilize with a 10 per cent. solution of formaldehyde in alcohol, or a 1 in 500 solution of mercuric chloride in hydrogen dioxide, and then dry thoroughly.

For fillings, cements, the oxyphosphates of zinc and copper are preferable.

If desired, these, when set, may be cut into and layers of amalgam placed externally. *Hill's* permanent gutta-percha is also a good filling material for the deciduous teeth, and on occasions in difficult approximal cavities

which are too sensitive to prepare properly, two fillings may be joined together for support without harm.

Should an exposure of the pulp have occurred pure carbolic acid, silver nitrate, or arsenic trioxide, may be used to devitalize it.

NOTE.—In applying arsenic to a temporary tooth it is wise to use *Ash's* or *S. S. White's* fibre preparation with a little oil of cloves and allow the dressing to remain not longer than from twelve to twenty-four hours, on account of the open nature of the end of the root. The dose of arsenic trioxide for local application in deciduous teeth is 1/30 gr.

After death of the pulp remove the coronal portion only, and do not attempt root-filling. Fill with a suitable filling material.

Colyer advocates the extraction of all septic deciduous teeth, and all deciduous teeth in which the pulps are exposed, except the second molars, in children under 6. The author of these notes is quite in agreement with the radical treatment of hopelessly septic deciduous teeth, but prefers, if possible, to save those suffering only from exposed pulps. The different teeth have different functions, and the loss of any one of them destroys to some extent their organization and the proper preparation of food prior to digestion. The extraction of the deciduous molars, also, allows the six-year molars to move forward, and eventually produces crowding. *Colyer* believes that there is little, if any, forward movement due to the extraction of the temporary molars.

Whether this is so or not, it seems preferable to save the temporary teeth as long as possible, up to the time when they should be naturally removed, if it can be done without causing the child any great distress or inconvenience.

CHAPTER III.

THE PERMANENT DENTITION.

FORMULA.

2	1	2	3	There are there-
i	c	pm	m	fore thirty-two
2	1	2	3	permanent teeth.

THE APPROXIMATE DATES OF ERUPTION OF THE PERMANENT TEETH ARE:—

First molars	6 years
Lower central incisors...	7 ,,
Upper ,, ,, ...	8 ,,
All the lateral ,, ...	9 ,,
First pre-molars	10 ,,
Second ,, ...	11 ,,
Canines	11-14 ,,
Second molars	12 ,,
Third ,, ...	17-26 ,,

The lower of each series usually erupt before the upper. The process is similar to that of the temporary teeth after absorption of the alveoli. The following figure shows the amount of calcification of the various

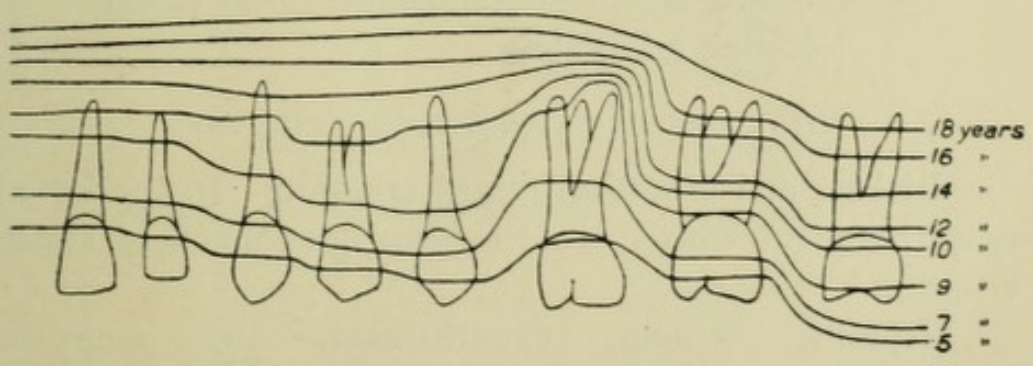

FIG. 10.

permanent teeth at various ages, the lower teeth being a little further in advance of the upper.

Signs, Symptoms, and Disturbances of the Erupting Permanent Teeth:—

These are usually slight and mild, and may be divided up into (a) Local.
(b) General.

(a) Local.

Usually slight salivation and inflammation. There may be profuse salivation, severe swelling and tenderness of the gums, or ulcerative stomatitis. *Trismus* may occur, due usually to the impaction and difficult eruption of the third lower molar. The symptoms, treatment, &c., of this affection will be dealt with in a later chapter.

(b) General.

The following may be present:—

Hemicrania (pain on one side of the head).
Otalgia (earache).
Neuralgia.
Paralysis.
Chorea (St. Vitus's dance).
Epilepsy.
Angina Ludovici (suppuration between the two layers of the deep cervical fascia).
Reflex odontalgia.

Diatheses, &c., affecting Eruption.

Rickets and cretinism retard eruption. In the latter case treatment with thyroid extract does much to assist eruption.

Syphilis, struma, exanthemata, and neurosis accelerate eruption.

Dilaceration of a tooth retards its eruption.

NOTE.—A *dilacerated* tooth (fig. 11) is one in which the root is bent at an abrupt angle with the crown, due to it having been obstructed or otherwise injured during development, or to it having been injured during the extraction of a deciduous tooth. It is more common in connection with the incisor teeth, and retards their eruption.

FIG. 11.

In children of the poorer classes the teeth are erupted later than in children of the well-to-do.

The canines and second molars are cut earlier in girls than in boys.

IRREGULARITIES OF THE PERMANENT TEETH.

(*a*) *In size.*
(*b*) *In number.*
(*c*) *In structure.*
(*d*) *In position.*

(*a*) *In size:*—

Large.—Usually upper central incisors, second lower pre-molars, and second lower molars.

Small.—Usually upper lateral incisors and upper third molars.

(*b*) *In number:*—

Excess.—Supernumeraries.

Deficiency.—Any tooth may be missing from the series. This most commonly occurs in connection with the lateral incisors and the third molars. In the author's practice a case came under notice where the patient retained her lower deciduous incisors until she was 20,

and although she is now between 45 and 50, she has never erupted the permanent successors. She has never had a left upper permanent canine, and still retains the deciduous tooth.

The causes which may produce deficiency are:—

(i) Alveolar abscess of a deciduous tooth destroying the permanent germ beneath.

(ii) Traumatism, involving the removal or destruction of a permanent tooth or germ, usually occurring in extracting a deciduous tooth.

(iii) Exanthematous fevers causing necrosis, involving the destruction of the alveolus and tooth germs.

(iv) Obstruction preventing eruption.

(v) Odontome.

Cases have been recorded of a total absence of the permanent teeth. *Mr. W. Rushton* records such a case in a boy of 16, and *Dr. Thorvald Kjer*, of Copenhagen, a case in a man of 25 who still retained five of his temporary teeth, which on the whole had been very defective. Neither of his parents had suffered from any serious complaint, nor had the patient, and in no other member of the family had a similar condition existed.

(*c*) *In structure:*—

Causes: (i) Local. (ii) General.

(i) *Local:*—

Traumatism, producing chipping of the enamel, &c.

Traumatism, producing dilaceration.

Alveolar abscess of a deciduous tooth, affecting the permanent germ beneath.

Disease of the cementum producing gemination (*pathological*). *Physiological* gemination may occur.

Causes producing accessory cusps, roots, or enamel nodules.

Enamel nodules are found upon the roots of teeth, usually in a multiple rooted tooth at the junction of the roots. They consist of dentine and enamel. Vascular canals sometimes occur in dentine, and these are usually associated with enamel nodules.

The author in his own practice has a case of a girl aged 11, where all the erupted permanent teeth are, or seem to be, devoid of enamel. There is no history of disease or malnutrition, and it is the only case in a family of six. This condition is described by *Hopewell-Smith* as *nanoid* or *dwarfed* enamel. Prolongations from the pulp into abnormal positions in the crown may, but rarely, occur.

(ii) *General*:—

Syphilis.—The central incisors are peg-shaped and notched. In the first instance, as in the lateral incisor (A, fig. 12), there are three tubercles and two notches on the cutting edges. The central tubercle then becomes worn away, leaving two tubercles and a semi-lunar notch (B, fig. 12).

The lateral incisors are, however, rarely affected, and the canines have circum-

ferential notches at their tips (fig. 13). This abnormality of the canine is not really typical of syphilis. A similar appearance may be seen in connection with canines affected by exanthemata (see fig. 17), and is due to intermittent calcification.

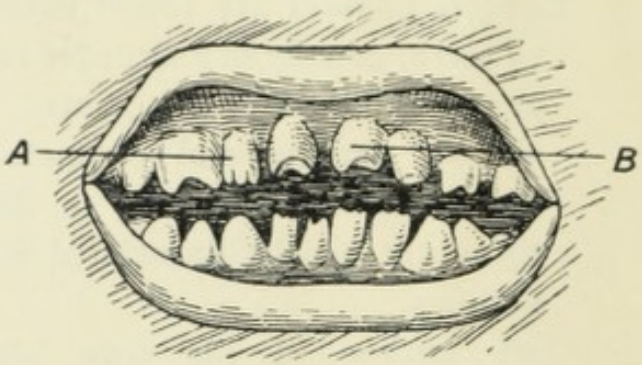

FIG. 12.

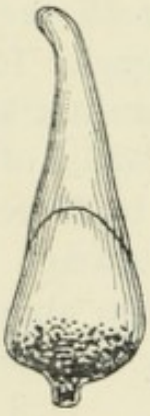

FIG. 13.

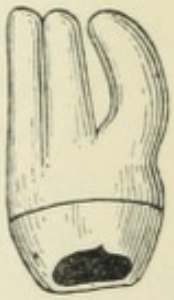

FIG. 14.

The pre-molars are rarely affected, and the molars are dome-shaped (fig. 14).

The teeth generally are small, spaced, dusky and opaque. *Jonathan Hutchinson*, who first called attention to the peculiar effects of inherited syphilis upon the permanent teeth, and to which affected teeth, therefore, the term *Hutchinsonian teeth* has been applied,

regarded the appearance of the central incisors as diagnostic of the disease.

The *Spirochæta pallida*, the specific organism of syphilis, will be found in the follicle walls.

Rickets.—The teeth are bluish, sparse, tapering, and notched.

Gout.—Causes recession of the gums and attrition of the teeth.

Malnutrition, *Teething powders* (containing mercury), *Exanthematous fevers* (scarlet fever, measles, chicken-pox, &c.) } produce *hypoplastic* or *honeycombed* teeth.

Hypoplasia occurs more frequently in the lower classes and hand-fed children than in the upper classes and naturally fed children. It is due to some alteration in the activity of the enamel-forming cells (*ameloblasts*) during illness, malnutrition, or the administration of mercury, and occurs during the first two years of life. In teeth thus affected the enamel is yellowish or brownish, dull, opaque, pitted, and marked by lines, and the dentine presents numerous interglobular spaces. The first molar, the incisors, and the canines are the teeth most affected (figs. 15, 16, 17, and 18).

Hutchinson states that all hypoplastic teeth are caused through mercury in teething powders. *Mr. J. G. Turner* records a case of *hereditary hypoplasia* traced through five generations. This condition, however, is very rare.

(*d*) *In position:*—

Causes: (i) Local. (ii) General.

(i) *Local:*—

Undue retention of the deciduous teeth, especially the upper incisors, causing the

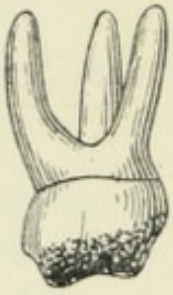

FIG. 15.—Hypoplastic molar.

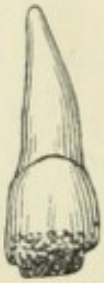

FIG. 16.—Hypoplastic incisor.

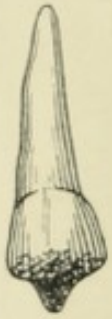

FIG. 17.—Hypoplastic canine.

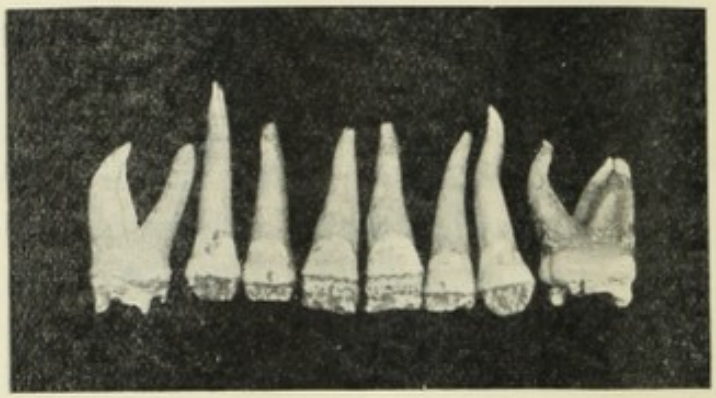

FIG. 18.—Hypoplastic maxillary incisors, canines, and first molars.

(From *Colyer's* "Dental Surgery and Pathology," by permission of *Messrs. Longmans, Green and Co., the publishers.*)

permanent incisors to erupt in front of them.

Presence of supernumerary teeth.

Too early extraction of the deciduous teeth, especially the second molar (allowing the first permanent molar to move forward), and the canine (allowing the first pre-molar to do likewise).

Injudicious extraction of the permanent teeth, especially canines and lateral incisors.

Delayed eruption of the permanent teeth.

Rhinitis.

Mouth-breathing due to adenoids or enlarged tonsils.

Cicatrices.

Tumours (hypertrophy of the gums, &c.).

Frænum of the upper lip passing between the upper incisor teeth and spacing them.

The habit of pushing the lower jaw forward.

Undue development of either jaw.

Arrested development of either jaw.

Ill-fitting regulation plates, dentures, and crowns, causing open bite, &c.

Thumb, toe, lip, finger, or tongue sucking.

Dummy-teats causing undue and abnormal pressure.

Approximal caries of teeth allowing teeth to close together.

(ii) *General:*—

Heredity.—Dr. Sim Wallace denies that heredity has any influence, but many cases exist of the transmission of peculiarities through many generations, *e.g.*, the underhung jaws of the *House of Hapsburg*.

Civilization, including love of luxury, and lack of use due to living upon soft food, &c.—*Mr. Coleman*, who has investigated the matter very closely, states that 69 per cent. of the well-bred children he examined had contracted arches, whilst only

7 to 8 per cent. of the more lowly bred suffered similarly, thus tending to prove that the higher the intellectuality and civilization, the greater the tendency to deformity.

Race-crossing } The inter-marrying of
Cross-breeding } two individuals, one dolichocephalic with large teeth, and the other brachycephalic with a small mouth, will tend to irregularity, &c.

Sexual selection.—Man's selection of the oval type of face with a small mouth is not conducive to broad and strong jaws.

Malnutrition.

Syphilis.

Neurotic tendencies.

Infantile scorbutis.

Rachitis.

CLASSIFICATION OF IRREGULARITIES IN POSITION.

(i) In connection with the individual teeth.
(ii) In connection with the whole jaw.
(iii) Contracted arches.

(i) In connection with the individual teeth.

Individual teeth may be:

(*a*) Extruded.
(*b*) Rotated (completely turned round, the labial becoming the lingual surface and *vice versâ*).
(*c*) Transposed (occupying a position usually occupied by another tooth, which takes the place of the displaced tooth).
(*d*) Within the arch.
(*e*) Without the arch.

(*f*) In other abnormal positions (under the chin, &c.).

(ii) In connection with the whole jaw:—

(*a*) Superior protrusion.

(*b*) Inferior protrusion.

(*c*) Open bite.

Superior protrusion:—

Causes of:

Heredity.

Excessive development of the upper jaw.

Arrested development of the lower jaw.

Undue retention of the deciduous teeth.

Presence of supernumerary teeth.

Frænum of the lip causing separation of the upper central incisors.

The biting of the lower incisors on the cingula of the upper, a condition, according to *Colyer*, invariably associated with adenoids.

Thumb, toe, lip, or tongue sucking.

The use of comforters or rubber teats.

Inferior protrusion:—

Causes of:

Heredity.

Excessive development of the lower jaw.

Arrested development of the upper jaw.

Loss of back teeth.

Habit (pushing the lower jaw forward).

Finger and tongue sucking.

Cicatrices.

Open bite:—

Causes of:

Arrested development of the pre-maxilla.

Arrested development of the ascending ramus.

Ill-fitting regulation plates and dentures.
Adenoid growths and enlarged tonsils causing mouth-breathing.

(iii) Contracted arches:—

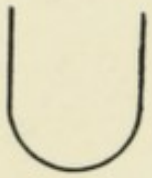

FIG. 19.

FIG. 20.

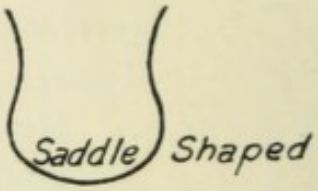

FIG. 21.

Of the three the *U-shaped* is the least abnormal.

Causes of:

The *V-shaped* arch usually occurs in the mentally and physically weak. It may be due to pressure from the masseter, combined with the actions of the buccinator and temporal muscles, upon bones lacking in density due to rachitis. The *saddle-shaped* arch may also be produced in this way, and it may often be due to the too early extraction of the deciduous molar teeth, allowing the six-year molars to move forward, so partially closing the space intended for the pre-molars, and crowding them within the arch. It may also be due to mouth-breathing (caused through the presence of adenoids) and resulting pressure from the buccinator muscles upon the soft parts in the region of the pre-molars. It is often found in the mentally and physically weak.

CHAPTER IV.

INFLAMMATION.

BEFORE dealing with the diseases of the dental tissues, it will be advisable to give a brief summary of the causes, symptoms, pathology, &c., of *inflammation* generally.

Definition:—

Inflammation is the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality. (From *Spencer* and *Gask's* "Surgery; its Theory and Practice.")

Varieties:—

(*a*) Acute.

(i) Simple.
(ii) Septic.
(iii) Infective.

(*b*) Chronic.

(*a*) ACUTE INFLAMMATION.

(i) Simple.—It is limited, and subsides quickly; as a rule, there is no suppuration. (This is not so, however, in connection with the pulp, as this tissue is of a delicate structure, has no lymphatics, and is enclosed in a non-elastic bony cavity; suppuration, therefore, usually results.) It is nearly always due to mechanical injury (in connection

with the pulp it is more often due to caries).

(ii) Septic.—This is due to the presence in the wound of the products of suppuration (blood poisoning).

(iii) Infective.—Due to the presence of pathogenic micro-organisms.

Inflammation is due to irritation, and is for the purpose of removing it.

The *causation* of inflammation may be:—

(*a*) *Ectogenous* (irritation of the tissues occurring from without, *e.g.*, chemical irritants, &c., &c.).

(*b*) *Endogenous* (irritation arising within the body, *e.g.*, harmful excretions from tumours, &c., &c.).

(*c*) *Metastatic* (irritation arising in one part through the passage to that part of excitants, along the blood or lymph stream, from another affected area).

Causes:—

(*a*) *Predisposing:*

(i) Old age.

(ii) Poisons in the blood (phosphorus, lead, &c.).

(iii) Feeble circulation.

(iv) Disease of nerve centre.

(v) Excessive use of alcohol and mercury.

(vi) Poor supply of healthy blood.

(vii) Diseases (syphilis, gout, diabetes, &c.).

(viii) Strumous diathesis.

(*b*) *Exciting:*

(i) Physical irritation (cold, acids, alkalies, electricity, friction, heat, direct violence, foreign body such as a filling, &c.).

(ii) Chemical irritation (putrefaction or fermentation).

(iii) Micro-organisms.

Micro-organisms.

The following are a few brief notes on micro-organisms:—

Micro-organisms may be divided up into

(i) *Schizomycetes* (fission fungi, bacteria).

(ii) *Hyphomycetes* (mould or thread multicellular fungi, *e.g.*, *Oïdium albicans*, which causes thrush).

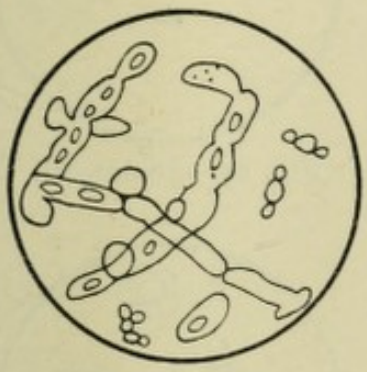

Fig. 22.—Yeast fungi.

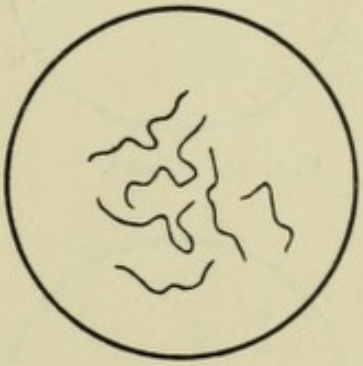

Fig. 23.—Spirilla.

(iii) *Blastomycetes* (multicellular bud or yeast fungi—fig. 22).

(iv) *Protozoa* (unicellular animals, such as the *parasite* of malaria and the *spirochæta pallida* of syphilis—fig. 33).

The first group is the most important from the standpoint of disease.

Micro-organisms may also be divided up according to their behaviour in the presence of oxygen into

(*a*) Obligatory aerobes.

(*b*) Obligatory anaerobes.

(*c*) Facultative aerobes and anaerobes.

Most are *obligatory aerobes*, and require free oxygen in order to live, *e.g.*, *yeast fungi* (fig. 22).

The *obligatory anaerobes* live in the absence of oxygen, or in a medium where oxygen is very scarce, *e.g.*, *Bacillus tetani* (fig. 31).

The *facultative* can live either with or without oxygen.

(i) *Bacteria or Schizomycetes*.—These are the simplest and lowest forms of vegetable life, and may be divided up, according to their form, into

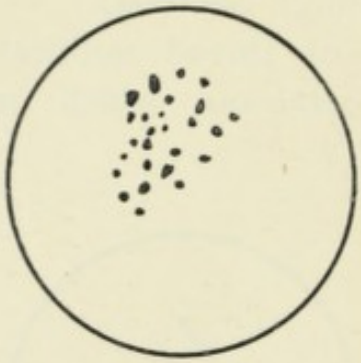

FIG. 24.—Cocci.

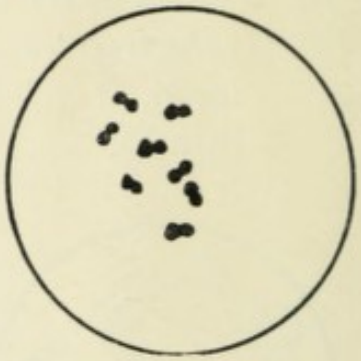

FIG. 25.—Diplococci.

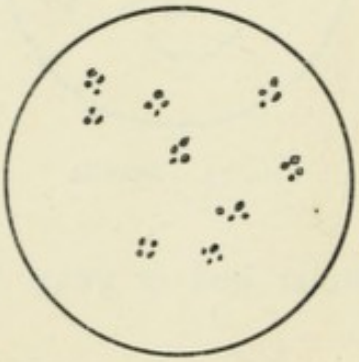

FIG. 26.—Tetracocci.

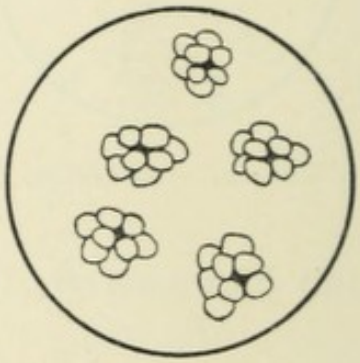

FIG. 27.—Sarcinæ.

(*a*) Micrococci.
(*b*) Bacilli.
(*c*) Spirilla (fig. 23). (Of no surgical interest.)

(*a*) *Micrococci*.—These are round or oval, multiply by fission or division, occur singly (fig. 24), in pairs (*diplococci*—fig. 25), *e.g.*, gonococcus,

in fours (*tetracocci*—fig. 26),
in packets (*sarcinæ*—fig. 27),
in chains (*streptococci*—fig. 28), *e.g.*, *Streptococcus pyogenes*,
or in grape-like masses (*staphylococci*—fig. 29), *e.g.*, *Staphylococcus pyogenes aureus*.

(*b*) *Bacilli* (fig. 30).—These are rod-shaped, and

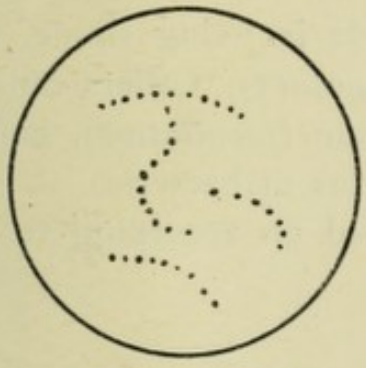

FIG. 28.—Streptococci chains.

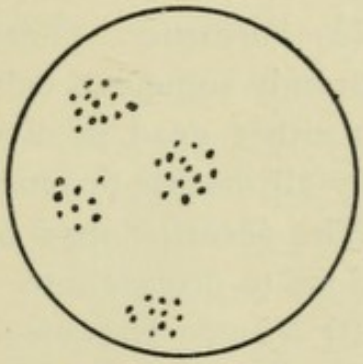

FIG. 29.—Staphylococci.

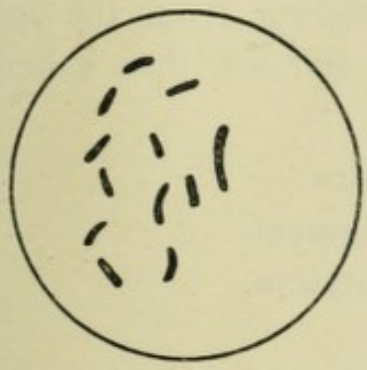

FIG. 30.—Bacilli.

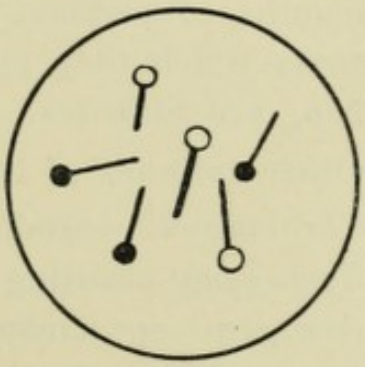

FIG. 31.—*Bacillus tetani*, showing spores.

multiply by *spores* or division. The spores are developed in the bacilli, and when the latter are destroyed the former are liberated. The spores (fig. 31), *on account of their possessing dense enveloping membranes*, are more resistant to the action of external agents, such as antiseptics, boiling, &c., &c., than the bacilli.

Micrococci and bacilli may be divided up into

(*a*) *Saprophytic*. (These live on dead, and cannot

exist on living tissue. They act as indirect causes of disease by entering the system through a wound, &c., and producing poisonous substances which are absorbed and give rise to *ptomaine fever, septic intoxication*, or *septicæmia*. They themselves, however, cannot penetrate the tissues nor enter the circulation. If by any chance they get into the blood they are quickly killed).

(*b*) *Parasitic*. (These reside in living tissue, upon which only some can exist (*obligatory*). Others can exist upon either dead or living tissue (*facultative*), and are nearly all disease-producing forms of bacteria).

The *parasitic* may be divided up according to their relations to disease into

(i) *Non-pathogenic*.

(ii) *Pathogenic*. These can enter the tissues and the blood, and produce disease directly. They may occur in the mouths of persons not suffering from the specific affections which they produce, and are then called, by *Hueppe, œco-parasites*.

Other varieties of bacteria are:—

Chromogenic—producing pigment.

Zymogenic—causing fermentation.

Aerogenic—producing gas.

Saprogenic—endowed with intense putrefactive properties.

The thermal death point of most bacteria is between 65° C. and 70° C. for half an hour. Extreme cold does not seem to destroy them. The temperature most favourable to their growth is about 37° C., and is known as the *optimum temperature*.

Most bacteria dislike sunshine, but the following are essential to their growth:—

(i) A certain amount of heat.

(ii) Moisture.

(iii) The presence of organic matter.
(iv) Rest.
(v) The removal of the products of the organisms.

Infection.

Definition:

The access of living, virulent, pathogenic organisms to a region from whence their toxins may act on the tissues of the body.

Varieties:

(*a*) *Local* (occurring at the point where the micro-organisms enter).

(*b*) *General* (the entrance of micro-organisms into the blood-stream and their development and multiplication there).

Immunity.

Tissues are protected from micro-organisms by

(*a*) *Natural immunity.*
(*b*) *Acquired immunity.*

(*a*) *Natural immunity:*

This condition is due to the possession, by the human being, of the power of resisting the action of micro-organisms, and is dependent upon the ability of the leucocytes to destroy bacteria, or to produce bactericidal substances, which effect the same thing.

This power may be weakened through malnutrition, unhealthy environment, excessive use of alcohol, &c., &c.

(*b*) *Acquired immunity:*

This may be *active* or *passive*. The former may be due to

(i) Exhaustion of the soil through a previous attack or attacks.

(ii) The poisonous products of bacteria staying the development of other organisms.

(iii) The destruction of bacteria by leucocytes.

(iv) The destruction of bacteria by the chemical constituents of the blood serum. In the blood bactericidal substances (*antibodies*) are formed, and these render inert the toxins produced by bacteria.

Passive acquired immunity is obtained artificially by injecting into one animal the serum obtained from another animal possessed of *active acquired immunity*.

For the lists of mouth bacteria which follow the author is indebted, to a great extent, to *Mr. Kenneth Goadby's* works on bacteriology.

The following *pathogenic* bacteria may be found in the mouth:—

Streptococcus brevis.—This is the least pathogenic of all the cocci, and is found in all mouths, healthy or unhealthy.

Streptococcus pyogenes (longus).

Streptococcus fæcalis, *Streptococcus angiosus*} These are the pathogenic streptococci most frequently found in the mouth.

Staphylococcus aureus.—This is usually the most pathogenic of the staphylococci mentioned here, and is not often found in the mouth.

Staphylococcus albus.—This is more frequently found in the mouth than is the *S. aureus*.

Staphylococcus citreus.—Is very common in the air, but is not often found in the mouth.

Staphylococcus granulatis citreus, of Freund.—This is very often present in the pus of the mouth.

Pneumococcus or Streptococcus pneumoniæ.—This is sometimes found in healthy mouths.

Micrococcus tetragenus.—This is not often found in the mouth.

Meningococcus or Diplococcus intracellularis.—It causes cerebrospinal meningitis, and is not common in the mouth.

Micrococcus catarrhalis.—This is often present, associated with periodontal trouble or coryza (common cold).

Gonococcus.—The organism of gonorrhœa.

Bacillus diphtheriæ (Klebs-Löffler bacillus).—It is often found in healthy mouths, and is sometimes

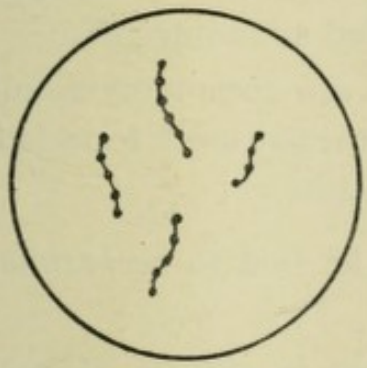

FIG. 32.—*Bacillus tuberculosis.*

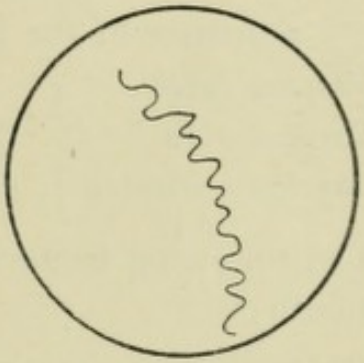

FIG. 33.—*Spirochæta pallida.*

associated with the ulcers found on the tongues of children suffering from measles.

Bacillus tuberculosis (fig. 32).—This is more common in the mouth than the *Bacillus diphtheriæ.*

Bacillus lepra.—This is the leprosy bacillus, and is not common in the mouth.

Bacillus pyocyaneus.—This is not common, but when present it is associated with dento-alveolar abscesses.

Bacillus friedlander.—It is associated with bronchitis.

Bacillus influenzæ.—It is often present in the mouth, and is associated with epidemic influenza.

Spirochæta pallida (fig. 33).—This is associated with

syphilitic lesions, and is the direct cause of syphilis.

Saccharomyces neoformans.—This is associated with acute pyorrhœa alveolaris.

Saccharomyces albicans (*Oïdium albicans*).—It is associated with parasitic stomatitis or thrush, and is found in the mouths of children who have been fed from bottles which have not been thoroughly cleansed and sterilized. It is also associated in adults with wasting diseases.

Actinomyces (*streptothrix*) *bovis.*—This has also been termed the *ray fungus*, and is the cause of a disease of the jaw called actinomycosis.

Sarcina lutea, *Sarcina alba*, *Sarcina aurantea*: These are commonly found in the mouth, but more especially in dirty mouths.

The following bacteria may be said to be *peculiar* to the mouth:—

Leptothrix racemosa.—Found in nearly all mouths where there are deposits of food or tartar, or artificial plates.

Bacillus maximus buccalis.—This organism is the largest bacillus occurring in the mouth, and is very common in that position, especially if dirty.

Bacillus fusiformis.—It occurs in healthy mouths, but is found in large numbers associated with pyorrhœa alveolaris and ulcerative stomatitis.

Spirochæta dentium, *Spirillum sputigenum*: These organisms are often found in the mouth.

Streptothrix buccalis.—Not uncommon in the mouth, and is occasionally found on the tongue.

The following is a list of some of the *non-pathogenic* bacteria of the mouth:—

The *Bacillus mesentericus* group.—These are associated with caries, and include the *Bacillus mesentericus ruber*, the *Bacillus mesentericus fuscus*, and the *Bacillus mesentericus vulgatus*.

The *Proteus* group.

The *Bacillus lactis* group.—Often found in milk, and occur naturally in soil, water, and air.

Bacillus necrodentalis.—This organism is found in the deeper layers of carious dentine, and closely resembles the *Streptococcus brevis*.

Micro-organisms of Caries.

Mr. Goadby divides these into two groups, viz.:—

(i) Acid-forming bacteria, or bacteria capable of producing acids by the fermentation of carbohydrates. The weak acids formed from the decomposition of proteids are incapable of attacking the lime salts of the tooth.

(ii) Liquefying bacteria which, either by their own action or the production of proteolytic enzymes, cause digestion of the decalcified dentine.

Goadby's Classification of Bacteria of Caries.

Acid-forming Bacteria.

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

BACTERIA WHICH LIQUEFY DENTINE (DECALCIFIED).

None isolated as yet. Deep layers of carious dentine.

Bacillus mesentericus ruber (vulgatus), of Gizen
Bacillus mesentericus vulgatus
Bacillus mesentericus fuscus
Bacillus septus (Bacillus furvus)
Bacillus liquefaciens flourescens
Bacillus subtilis
Bacillus maximus buccalis (Goadby)
Bacillus plexiformis (Goadby)
Proteus zenkeri

} Superficial layers of carious dentine.

STERILIZATION.

In surgical operations on the mouth and teeth, it is of the utmost importance that absolute cleanliness should be ensured, not only of the *instruments* and the *hands* of the *operator*, but also of the *mouth* and *teeth* of the *patient*. The dentist should thoroughly wash and scrub his hands with hot water and an antiseptic soap, taking care that the nails are thoroughly cleansed, and this should be carried out *before the patient*.

The mouth and teeth of the patient should then be thoroughly cleansed, scaled, and washed with a good antiseptic mouth-wash before any other operation is begun. All instruments, such as lances, forceps, excavators, chisels, scalers, clamps, &c., &c., should be sterilized by boiling in water containing a few grains of bicarbonate of soda (1 dr. to a quart of H_2O), the latter preventing rusting. Burs and drills can be dipped in alcohol, and then passed quickly through a flame, but a better method, and one which does not dull the edges, is to brush them with soap and water, dip them in boiling water, and then place them in a vessel containing alcohol.

Finer instruments may be sterilized by heating them up in oil or glycerine.

Rubber-dam may be used several times for the same patient, and may be sterilized by placing it in a glass containing a solution of 1 in 3,000 bichloride of mercury, and washing it in distilled water before again using.

A good disinfectant is a 1 in 200 solution of *Lysol*.

Signs and Symptoms of Acute Inflammation:—

Macroscopically—(*a*) *Local:—*

Heat—due to increased quantity of blood and increased chemical action, but the heat of the part is always less than that of the blood.

Redness—due to increased vascularity.

Swelling—due to distension from increased quantity of blood, and the exudation into the surrounding tissues from the blood-vessels.

Pain—due to pressure upon nerve fibrillæ. This is more intense in connection with inflammation of the periodontal membrane, on account of the great degree of tension present.

Loss of Function—due to impaired nutrition.

(*b*) *General:—*

If the inflammation is slight there may be no fever, but if it is intense there are:—

Feverish Symptoms, such as rise of temperature, quick pulse, dry skin, headache, loss of appetite, constipation, furred tongue, scanty urine, sometimes delirium. The fever may be:—

(i) *Sthenic:* Temperature 103° to 105° F., strong pulse.

(ii) *Asthenic:* Temperature falls, weak pulse, typhoid in character, mouth in offensive state.

(iii) *Irritative:* Nervousness and delirium added to either of the above set of symptoms.

Microscopically (fig. 34).—Changes occur in order as follows:—

(i) Momentary *constriction* of the blood-vessels.

(ii) Prolonged *dilatation* of the blood-vessels. This is first noticed in the arteries and is due to an alteration in the tone of the vessel walls.

(iii) *Acceleration* of the blood flow and an increased supply of blood (*active hyperæmia* or *determination of blood*).

(iv) The blood flow *slackens*, due to the altered condition of the vessel walls; first in the veins, then in the capillaries, and then in the arteries. Two streams of blood exist—the *axial* and the *circumferential*—the latter flowing along more slowly than the former, due to the friction with the vessel walls. The leucocytes or white blood corpuscles pass from the axial to the circumferential zone, collect, and adhere to one another and then to the vessel walls (*margination*), thus narrowing them and impeding the blood flow.

(v) *Diapedesis or emigration* now occurs. This is the passage of leucocytes through the vessel walls from the circumferential zone, the red blood corpuscles still passing along the axial stream. It is caused through the *amœba-like movements* of the leucocytes,

according to some authorities, and according to others, through *positive chemiotaxis* (the attractive power of an irritant or poison for leucocytes). It occurs normally, but is very much more excessive during inflammation. As the inflammation increases, the number of leucocytes exuded is increased. Not only are the leucocytes exuded, but they also multiply considerably by direct division after *margination*. In the early stages, there is merely a *serous exudation*, but later, in addition to the passage of leucocytes, there is a *plastic exudation* into the surrounding tissues causing *œdema*. The *plastic exudation* or *inflammatory lymph* differs from the *serous* in containing not only leucocytes, but also the fibrin-forming elements of the blood, and is coagulable. The purpose of the exudation is to dilute and wash away the irritant. Sometimes the red corpuscles, which do not normally do so, pass through, due to pressure, and then the part, macroscopically, is mottled. In some acute inflammations the capillary walls may give way, resulting in an extravasation of blood (*rhexis*) into the surrounding tissues.

(vi) *Oscillation* of the blood-stream now results. It passes onwards during the systole, and backwards during the diastole of the heart.

(vii) *Stasis* follows. The blood flow stops. If the irritation ceases, *oscillation* recurs, the leucocytes cease dropping out of the blood-stream, and *resolution* results, the exudation and cells being taken up by the lymphatics.

(viii) Should the irritation continue, *thrombosis* or coagulation of the serum follows. Resolution cannot occur at this stage, but death of the part takes place through the action of the leucocytes, liquid exudation, and the plugging of the vessels.

In addition to the above phenomena, changes occur in the *fixed cells and tissues.*

The first changes are *degenerative*, the fluid soaking into the surrounding tissues and breaking down the connective tissue fibres. The cells break up into granules, and they and the fibres are swallowed up in the fluid.

Regeneration may then take place. Proliferation of the cells occurs by what is called *karyokinesis*, or indirect division of nuclei, and these mix with the leucocytes. They may act in either of two ways:—

(*a*) As *fibroblasts* in the process of reparation.

(*b*) As *phagocytes* in arresting the activity of irritants and promoting the process of absorption.

Sequelæ of Inflammation.

(*a*) Resolution.

(*b*) Suppuration and abscess. (This is the usual termination of inflammation of the pulp, as it is of such a delicate structure, is contained in a dense body cavity, thus preventing expansion, and has no lymphatics.)

(*c*) Gangrene, necrosis, sloughing.

(*d*) Scarring and fibrous thickening.

(*e*) Ulceration.

(*b*) Chronic Inflammation.

Pathology.—Similar to acute. The disease is less severe, but of longer duration. The exudation contains less albumin and fibrin-forming materials.

Causes.—Similar to acute.

Symptoms.—All the symptoms are less marked than in the acute, except swelling, which is a marked sign. Fever may or may not be present.

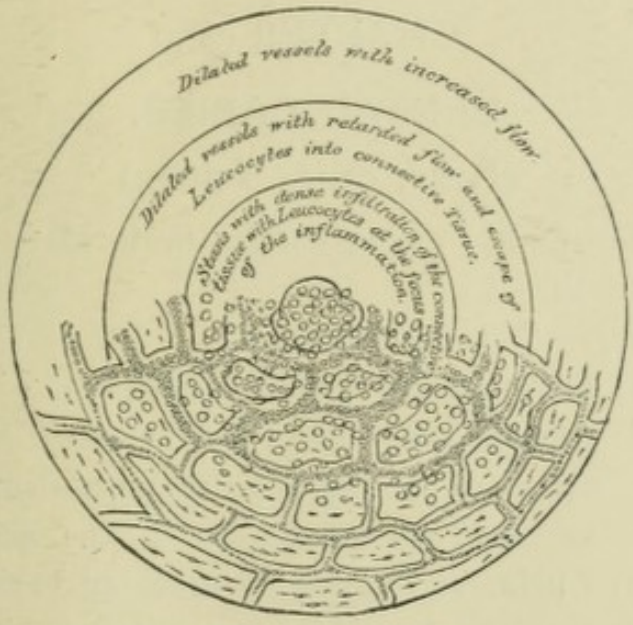

FIG. 34.—Diagram of the minute changes in inflammation. From *Walsham's* "Theory and Practice of Surgery," sixth edition.

Treatment.—The treatment of dental inflammation will depend upon the cause, variety, &c., and will be dealt with under the various dental affections.

CHAPTER V.

DENTAL CARIES.

(Latin, *caries*, rotten.)

Definition.—The progressive molecular disintegration of a tooth.

Varieties.—Old classification.

(i) *Caries acuta* (rapid caries).

(ii) *Caries chronica* (slow caries). Here the action of the peptonizing organisms is almost as rapid as the action of the acids.

(iii) *Caries sicca* (dry or arrested caries).

(iv) *Caries humida* (saturated with fluids). *Colyer* describes *Caries humida* or *Caries rapida* as a condition where the action of acids is more rapid than the action of the peptonizing organisms and in which the dentine can be removed in large leathery masses.

Modern Classification (*Marshall* and others).

1st Stage (superficial or incipient).

Involving only enamel or cementum. Fig. 35 (A).

2nd Stage (progressive).

Enamel or cementum has been penetrated and the dentine is being attacked. Fig. 35 (B).

3rd Stage (deep seated).

Dentine attacked almost to pulp chamber. Fig. 35 (C).

modern races is due to the gradual adoption of a soft diet and the increased use of carbohydrates.

Good hygiene is quite as important as food. The most highly civilized races are the most prone to caries due to their luxurious habits.

Sex.—Females are more prone than males. Married women are probably more prone than the unmarried, caries occurring especially during pregnancy and lactation.

Age.—Caries is most frequent before 25 and after 50.

Occupation.—Millers, bakers, alkali workers, &c., &c., due to the constant presence in the mouth of fumes, flour, dust, sugar, &c., &c.

Diseases.—Exanthematous fevers, syphilis, dyspepsia, cancer of the stomach, rachitis, scrofula, diabetes, phthisis, aphthæ, anæmia, and any inherited disease.

Vaccination.—Herbert Spencer suggested that this might increase the susceptibility of teeth to caries.

Climatic Influences.—These, acting upon the general system adversely, tend to produce caries.

Miscegenation (the crossing of distinct races).—*Thompson* states: "Human hybrids are notably deficient in physical completeness and vital power, nutrition is more or less impaired, and the physiologic processes are weakened, so that the entire economy is depressed and abnormal. . . . The depression that has ensued to the physique of the people of the *United States* as a natural result of these inter-marriages may have

contributed somewhat to the existing extra defectiveness of the teeth of Americans over those of nations who have been less subject to the effects of miscegenation."

Excessive Mental Strain in Growing Children.—This lowers the general tone of the system. *Dr. John Sayre Marshall* states : "I have often found it necessary to advise parents and guardians to remove a child from school to save the health from being weakened, and in many cases the rapid decay of the teeth was the prominent symptom which attracted my attention and led to an investigation of the general health and surroundings of the child."

(ii) Local.—

Mucoid or acid *saliva*.

Structural deficiency of teeth (pits, depressions, inter-globular spaces, &c.). As *Burchard* states: "The forces of attack being equal, a poorly organized and badly formed tooth will succumb sooner than one perfectly formed and of completely organized tissues; this law is constant in all biology."

Traumatism (chipping of enamel, &c.).

Irregularity and *crowding* of the teeth, rendering it difficult to thoroughly cleanse them.

Food and water.—With respect to the former, the more liable it is to ferment, the more likely will caries result. *Dr. Rose*, of Freiburg, states that where water and soil have good supplies of lime, caries is less prevalent than in places where this is not the case.

Living teeth resist the attacks of caries better than *devitalized* ones. The production in the

former of the *translucent zone*, and in many cases, of *secondary* or *adventitious dentine*, during an attack, as protective measures, prove this.

Cider drinking induces caries.

(*b*) Exciting.—The theories which have been advanced are as follows:—

(i) *Inflammatory.*—Inflammation of dentine (*Galen*).

(ii) *Electrical.*—The root of the tooth invested with gum and alveolus is the positive pole, whilst the crown is the negative, the saliva being the exciting fluid (*Bridgeman*).

(iii) *Chemical.*—Due solely to acids (*Paul Ægina*).

(iv) *Humoral.*—According to *Hippocrates*, the body contained blood, phlegm, black and yellow bile. If the proportions necessary to health of these four fluid humours, as they were called, were disturbed then disease resulted, therefore caries was due to such a disturbance.

(v) *Putrefaction.*—Putrified food clinging to teeth causes caries (*Pfaff*).

(vi) *Parasitic.*—Due solely to micro-organisms, the organisms secreting acids (*Erdl*, *Milles*, and *Underwood*).

(vii) *Chemico-parasitic Theory.* — Carbohydrates are taken into the mouth and changed into grape sugar by means of the ptyalin in the saliva. This undergoes fermentation and *lactic* acid results. The lactic acid breaks down the enamel prisms, and the bacteria enter and peptonize the dentine. This is the accepted theory and is advanced by *Miller*.

MILLER'S CONCLUSIONS WITH RESPECT TO THE CAUSE OF CARIES ARE:—

(i) Micro-organisms are always present.

(ii) Softening or decalcification takes place before infection occurs.

(iii) Artificial caries may be produced in the presence of acids and bacteria but not in the presence of either separately.

Teeth with putrid pulps do not necessarily undergo caries. This is on account of the absence of carbohydrates and the alkalinity of the pulps, under which conditions the bacteria are inert.

COMPARATIVE TABLE OF THE OCCURRENCE OF CARIES IN INDIVIDUAL TEETH (TOMES).

From 2,638 cases of extraction on account of caries.

Central incisors	25
Canines	36
Lateral incisors	62
First premolars	227
Third molars	230
Second premolars	393
Second molars	575
First molars	1,090
	2,638

Generally speaking the upper teeth are far more prone to caries than the lower, with perhaps the exception of the second and third molars. The lower incisors of all the teeth are the least prone and the six-year molars the most prone.

MACROSCOPICAL APPEARANCES OF CARIOUS TOOTH TISSUE.

Enamel.—The tissue becomes opaque.

A white spot occurs which gradually darkens and a cavity appears.

The carious tissue is soft and cheesy.

Dentine.—The cavity is usually triangular with the apex directed towards the pulp (*penetrating caries*).

The disease spreads laterally if the tissue is ill developed and there are a large number of interglobular spaces.

The carious tissue is tough, cartilaginous, and pigmented.

Cementum.—The cavity is expanded, saucer shaped, shallow, and very sensitive to the touch.

Nasmyth's Membrane.—This tissue is pigmented (green) and is the appearance often seen in connection with the teeth of the young.

NOTE.—Pigmentation in caries is probably due to *chromogenic* bacteria, and progresses in inverse ratio to the rate, that is to say, the darker the pigmentation the slower the rate of progression and *vice versa*. Pigmentation is probably not essential to caries.

MICROSCOPICAL APPEARANCES OF CARIOUS TOOTH TISSUE.

Enamel.—In the early stages there is a furze-like layer of micro-organisms upon the surface. According to *Black*, gelatinous plaques exist upon the free surface, and securely attach the groups of organisms to the tissue. Later the organisms penetrate between the prisms.

The striæ on the prisms are well marked.

The prisms eventually break down, and according to *Tomes* do so at their axes before at their peripheries. As *Hopewell-Smith* states, "This, no doubt, is the case where the tissue is at all ill-formed."

Hopewell-Smith mentions three zones in carious enamel, *viz.*:—

(i) The zone of partial decalcification, where

the rods are pronounced, the striæ marked, and the cement substance partially disintegrated.

(ii) The zone of complete decalcification, in which the prisms are structureless, but possess a marked degree of granularity.

(iii) The zone of "*secondary enamel decay*" (*Miller*) in which masses of micro-organisms only can be demonstrated.

Dentine.—On the surface of the cavity is a furze like layer of micro-organisms. Below this is seen a large black mass of stain. This is a mass of micro-organisms which has disintegrated the dentine. Just below this are seen streaks of stain marked in numerous places by small masses of stain. The streaks are the dentinal tubes filled with micro-organisms, the small masses being micro-organisms which have broken through the walls of the tubes and partially disintegrated the matrix (*liquefaction foci*). Below this are streaks of stain, which represent the tubes filled with micro-organisms that have not yet broken through the walls. Below this is an unstained portion. This is called the "*non-infected zone of Miller,*" and is that area which has been softened by acids but not yet infected by bacteria. The existence of this area proves that acids act before bacteria or that *softening precedes infection*. If a transverse section across the tubes in this area be taken the appearance known as the "*tobacco pipe appearance of Tomes*" (fig. 36) is seen. The appearance has been better described by *Underwood*,

who likens it to that of the ends of a bundle of carbon pencils, the carbons representing the dentinal fibrils, whilst the surrounding portions of wood represent the dentinal sheaths. The appearance is also seen in caries in human teeth worn on artificial plates, and in teeth in which caries has been produced artificially.

It is due to an enlargement of the diameters and a thickening of *Neumann's* sheaths through, according to *Hopewell-Smith* and others.

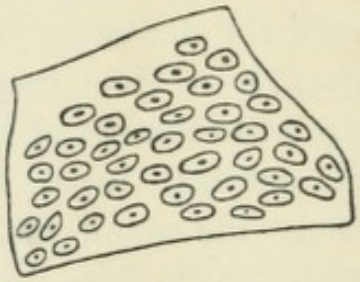

FIG. 36.—The tobacco pipe appearance of *Tomes*.

(i) Expansion from pressure of micro-organisms in an outward direction on the decalcified dentine matrix.

(ii) Slight thickening of the sheaths due to the peptonizing action of the micro-organisms within.

Leptothrix occur upon the surface of carious dentine whilst micrococci and bacilli exist in the tubes, usually in separate ones (figs. 37 and 38). The infection, however, may be mixed (fig. 39).

In the tubes may also be seen *rod-shaped fragments* and *rows of shining granules*. The former are probably pieces of consolidated fibrils, pieces of the sheaths of

Neumann, or casts of enlarged fibrils. *Wedl* is not in agreement with the view that they are pieces of consolidated fibrils, whilst *Miller* believes that they are calcified fibrils.

The *rows of shining granules* are probably due to the breaking down of the *rod-shaped fragments*. *Miller* leans towards this view, and thinks that as they are found in caries in pulpless as well as living teeth, they are not

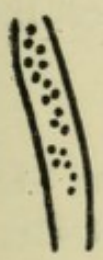

FIG. 37. Micrococci in a tube in carious dentine.

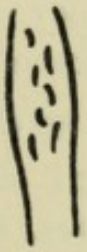

FIG. 38. Bacilli in a tube in carious dentine.

FIG. 39. Mixed infection of a tube in carious dentine.

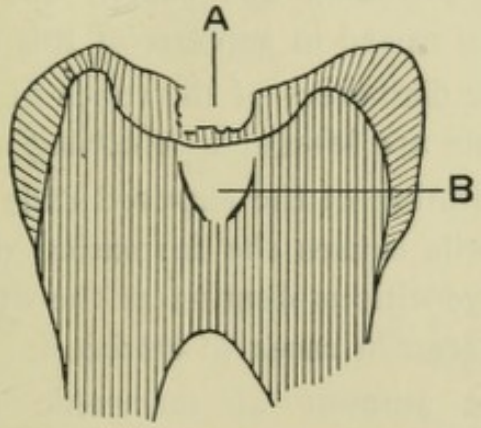

FIG. 40.—A, Carious enamel ; B, translucent zone in dentine.

lime salts thrown out from the pulp. *Magitot*, however, thought them lime formations excreted from the odontoblasts to resist advancing disease. *Wedl* and *Black* have described them as fat globules, whilst *Baume* disproved this view by showing that they do not disappear when treated with sulphuric ether.

Another *microscopical* appearance in the dentine of a tooth attacked by caries is the *translucent zone* (fig. 40). This is seen in the dentine of a carious tooth, between the pulp and the caries, and is best marked when the enamel only is affected. The zone is conical in shape, with the apex directed towards the pulp, and on either side is bounded by an opaque line.

In normal dentine the opacity is due to the different refractive indices of the matrix and the contents of the tubes, and this translucency is produced through the equalization of these refractive indices.

This equalization may be due to:—

(i) Calcification of the contents of the tubes (*Sir John Tomes, Magitot, Miller* of Berlin), or,

(ii) Decalcification of the matrix (*C. S. Tomes, Wedl, Black, Abbott, F. J. Bennett*).

It is more probably due to (i), and the following points have been raised in support of this contention:—

(*a*) The diameters of the tubes in the translucent zone are lessened (*Walkhoff*).

(*b*) Irritation of the exposed ends of the dentinal fibrils causes the formation of secondary or adventitious dentine at the other end. This is seen in cases of attrition.

(*c*) The amount of inorganic matter in the translucent zone is not less than in healthy dentine matrix, in fact, it is 69.5 per cent. against 68 per cent. (*Jeserich*).

(*d*) Decalcified dentine stains readily, whereas the dentine in the region of the translucent zone stains with difficulty.

(*e*) In the dentine of teeth in which caries has been produced artificially the zone is absent,

tending to prove that its production is a vital process.

(*f*) In the dentine of pulpless teeth which have undergone caries the zone is absent, also tending to prove that its production is a vital process.

In some cases it has been shown to be present, but it is probable that it was present before the pulp was removed.

(*g*) *Mr. Charters White* found that in decalcified specimens coloured collodion readily permeated the dentinal tubes, whereas in the dentine in the region of the translucent zone the tubes were only slightly permeable.

(*h*) The translucent zone is present only in the dentine where the fibrils have been affected and irritated by the carious process, but if the zone is due to decalcification the appearance should be seen on all sides of the carious cavity.

The chief argument in favour of the theory that the translucent zone is due to decalcification of the dentine matrix is that it is seen in some human teeth which have been mounted upon artificial plates and undergone caries. It is probable, however, that the zone was present whilst the pulps of the teeth were alive.

Another argument in favour of this theory is that in this zone the tubules are enlarged, presenting the tobacco pipe appearance of *Tomes*.

Cementum.—Like dentine, but the bacteria are in, and follow the canals containing *Sharpey's* fibres.

Arrested Decay (fig. 41).

This condition is occasionally seen, and occurs only in teeth with living pulps. There is a great loss of tissue and the dentine exposed is darkly stained and exceedingly hard. The condition may be found in both deciduous and permanent teeth. The first permanent molar is the tooth most often affected, and hypoplasia is usually an

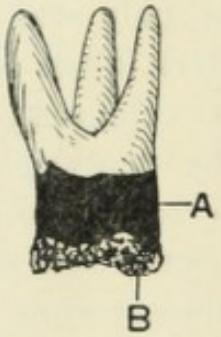

Fig. 41.—Molar showing—A, arrested decay; B, hypoplasia.

accompanying feature. Microscopically the dentinal tubes are seen to end abruptly on the surface and no micro-organisms are present. It has been stated by some to be due to dehydration, but as the teeth are constantly bathed in saliva this is probably not the cause. It is more probably due to some vital process, as it never takes place in pulpless teeth.

Artificial Production of Caries.

Miller produced caries artificially by cutting up a tooth into portions and placing them in a mixture of saliva, meat, and bread. The mixture was then kept at a temperature of 98° F., and renewed frequently to prevent putrefaction.

Symptoms of Caries.

(i) *Pain.*—There may be none, or it may be slight or severe, depending upon the amount of tissue involved and the health and temperament of the person affected. This symptom

is due to the exposure of the dentinal fibrils and nerves, and is increased by the action of thermal changes, sweets, acids, and instrumentation. The pain may often be very severe in the early stages, due to irritation of the fibrils and nerve endings, and gradually disappear as the disease progresses and the cavity becomes deeper. *Mummery* has proved that nerves exist in dentine (fig. 42),

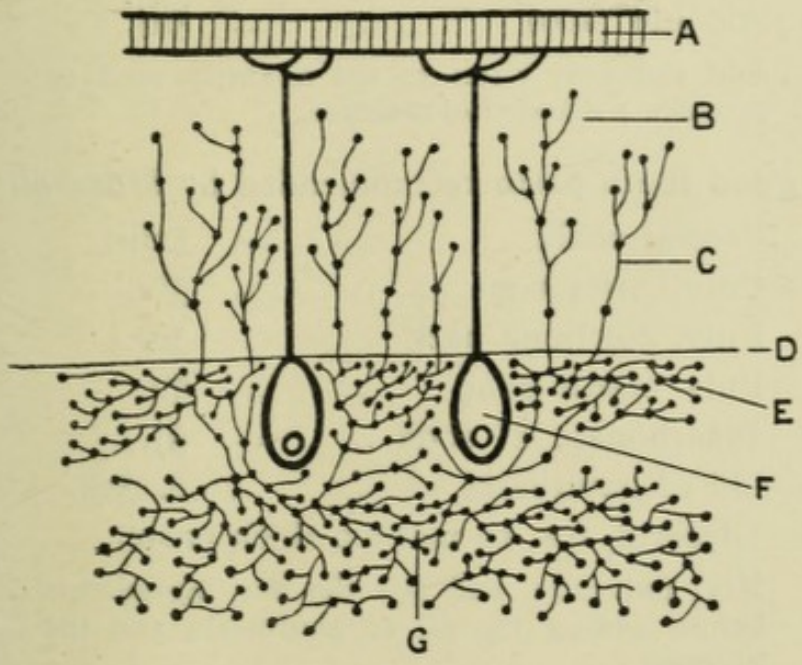

FIG. 42.—A, enamel; B, dentine; C, nerves in dentine; D, a line, dividing off the pulp from the dentine, for descriptive purposes; E, a narrow plexus of nerves on the surface of the pulp next to the dentine; F, odontoblast; G, the plexus of nerves in the pulp known as "*the plexus of Raschkow*," which is continuous with E.

and this fact accounts for the hypersensitiveness of the tissue, superficially, in many instances.

(ii) Sensation of a cavity.

(iii) Lodgment of food.

Treatment.—The treatment for caries may be either

(i) *Prophylactic.*

(ii) *Therapeutic* or *remedial.*

The writer has already dealt with prophylactic treatment in Chapter II, and most of the advice given there

for the care of the deciduous teeth will apply equally well to the care of the permanents. The following prescription for an antiseptic mouth-wash, suggested by *Miller*, will be found useful:—

℞ Thymol ... ... gr. iij.
Benzoic acid ... ... gr. xlv.
Tincture of eucalyptus ... ʒss.
Oil of wintergreen or
Oil of peppermint ... ... gr. xxv.
Alcohol (100) ... ... ʒiij.

Add sufficient water to the quantity used to produce a decided cloudiness.

A good tooth paste recommended by *Marshall* is:—

℞ Precip. chalk ... ... ʒviii.
Pulv. orris root ... ... ʒiv.'
Pulv. cinchona bark ... ... ʒiv.
Pulv. castile soap ... ... ʒij.
Bicarbonate of soda ... ... ʒj.
Oil of gaultheria ... ... fl. ʒss.
Glycerol, q.s. to make a thick paste.

Mix, pulverize, and sift the dry ingredients before adding the oil of gaultheria and the glycerol.

For those who prefer a powder, however, the following prescription of *Marshall's* is to be recommended:—

℞ Precip. chalk ... ... ʒviii.
Pulv. orris root ... ... ʒiv.
Pulv. cinchona bark ... ... ʒiv.
Pulv. cinnamon ... ... ʒv.
Pulv. castile soap ... ... ʒij.
Pulv. white sugar ... ... ʒiv.
Oil of lemon ... ... gtt. xx.
Oil of rose ... ... gtt. ij.

Mix, pulverize, and sift through a fine hair sieve.

Colyer advises the following alkaline mouth-wash for swabbing the mouth the last thing before going to bed in cases where caries is progressing rapidly:—

℞ Mag. carb. levis ... ... ʒiv.
Aq. rosæ ... ... ʒvi.
Aquam ad. ... ... ʒxij.
Misce. Shake before using.

After cleansing the teeth a tablespoonful of the above should be taken into the mouth and sluiced between the teeth.

It is not the intention of the author to deal with the remedial measures of treating caries, such as filling, crowning, &c. For a knowledge of these the reader must refer to the usual text-books.

CHAPTER VI.

INJURIES TO TEETH DUE TO CAUSES OTHER THAN CARIES.

Erosion (figs. 43 and 44).

Definition.—A gradual molecular destruction of tooth substance due to some cause not yet recognized and universally accepted.

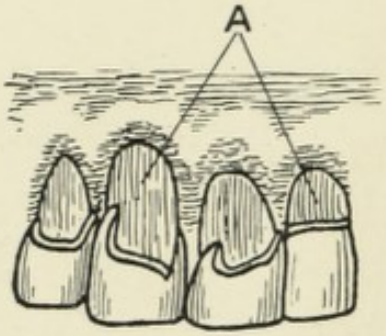

Fig. 43.

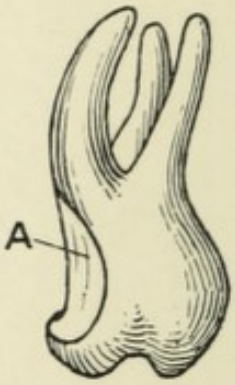

Fig. 44.

Fig. 43.—A, eroded labial surfaces of incisors.
Fig. 44.—A, eroded lingual surface of an upper molar.

Theories for cause:—

(i) *Electrolytic action* (*Bridgman, Garretson*). *Black* has produced, artificially, in sound human teeth, an appearance like erosion by the agency of electrolytic action.

(ii) It is due to the *imperfect structure of the enamel*, so lowering the power of its resistance to the action of external agents (*Bell*).

Black, however, states that erosion does not follow the developmental lines which would be the case if portions of the teeth could be worn away on account of any softness from faulty development.

(iii) The *solvent property of saliva* produces erosion (*Fox*).

If this is so one would reasonably expect to find teeth generally affected, and not certain teeth in certain positions. For instance, the lower teeth should be attacked to a greater extent than the upper, as they are being constantly bathed in saliva. The exact opposite is, however, the case, and the upper teeth are the more often affected.

(iv) There is some connection between erosion and *defective development* (*Bland Sutton*).

Sutton draws attention to a skull of the *Otaria jubata*, a specimen in which an appearance, similar to that seen in skulls of rachitic animals at about the period of puberty, is to be observed, *viz.*, a peculiar, porous, soft hyperostosis. The teeth also suffer from the effects of erosion.

He also found in a racoon-like dog, besides eroded teeth, characteristics of a peculiar bone disease known as mollities ossium.

(v) According to *Magitot and others* erosion is the manifestation of a *spontaneous cure of incipient caries* due to the filling up of the dentinal tubes.

Against this theory is the fact that the cavities are nearly always hypersensitive, whereas, if *Magitot's* theory is correct, they should be insensitive.

Again, the writer has had under observation many erosion cavities which have been filled with gold, and which have eventually extended from the edges of the fillings without the slightest sign of caries being present.

(vi) *Baume* believes that the surface layers of dentine not covered by gum, enamel, or cementum, *atrophy* and fall off, and that the resulting spaces are polished by friction from the lips, &c. With relation to this denuda-

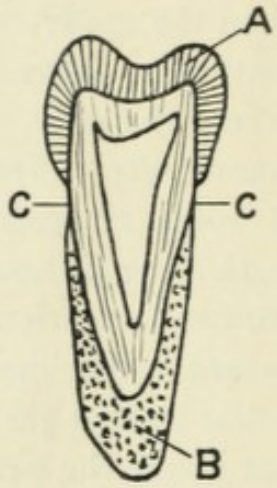

FIG. 45.—A, enamel. B, cementum. C, uncovered dentine.

tion of enamel and cementum, *Choquet* has shown that in some teeth neither the enamel overlaps the cementum nor the cementum the enamel, but that a portion of dentine remains uncovered (fig. 45).

Hopewell-Smith, in writing of erosion, states: "It is exceedingly probable that the condition is predisposed to by the denudation of dentine at the necks of teeth by the thin edges of enamel and cementum which, as a rule, overlap it, when—in other

words—these two tissues do not meet *bout à bout.*"

(vii) It is due to the combined action of *acids and friction* (*Bastyr, Walkhoff, and others*).

According to this theory a very thin layer of dentine is decalcified and then removed by friction from a tooth-brush or other external agent.

(viii) Erosion is due to the action of *secretions from the labial glands* in individuals suffering from gout and rheumatism. The active chemical agent is *potassium sulphocyanate* (*Michaels*).

(ix) It is due to the *solution of the organic matrix by ferments and the action of friction.*

The two theories which receive the most support are:—

(x) It is mainly due to *friction*, such as the action of tooth-brushes, tooth-powders, gritty food, the lips, the cheek, the tongue, &c. (*John Tomes, Salter, Von Melnitz, Zsigmondy, jun., Miller, and others*).

This theory is not altogether satisfactory, although the cavities may eventually be enlarged by these means. It has been pointed out by *Dr. Murie* that in certain sea lions erosion occurs. This, according to *Coleman*, may be due to the fact that these animals take stones, sand, and hard fish scales into the mouth, but against this suggested cause is the fact that the cavities are often in positions which could not have been affected by friction.

The writer has seen many cases of erosion in human teeth which, on account of position, have probably not been produced by

friction. He has also filled erosion cavities and stopped the use of tooth-brushes and powders, and yet the cavities have extended, after a lapse of time, and this without any appearance of caries or history of friction.

And (xi) Erosion is due to *exposure to the action of acids*, which may be derived from the mucous glands of the cheeks, gums, and lips.

Kirk believes that the affection is associated with gout and rheumatism and that *lactic acid* is the active agent when the disease is general, but that when it is local the agent is *acid sodium phosphate* or *acid calcium phosphate*.

Cook states that *mucic acid* plays an important part.

Truman, *Black*, and *Harris* believe that erosion is due to some acid solvent.

C. Tomes records cases where the appearance was due to sucking lemons and grapes in such a way as to bring them in contact with the teeth.

Some of the many arguments raised against the theory that erosion is due to the action of acids are: —

(*a*) The surface of an erosion cavity is not soft, but hard and polished.

(*b*) The portion of dentine affected does not stain more deeply than the surrounding dentine, whereas decalcified dentine stains more deeply.

(*c*) *Bastyr* and *Miller* state that acids, or in general, agents which dissolve the calcium salts of the teeth, can never produce these wedge-shaped defects. In all such cases only decay at the neck of the tooth is the result.

Position of Erosion Cavities.

They occur more often in the upper than in the lower teeth, usually on the labial or buccal surfaces, and at the cervical margins. They sometimes occur on the approximal, and less often on the lingual surfaces. *Marshall* states that when they occur in the lower jaw, they are seen more often in connection with the premolars. In the upper jaw the incisors are the teeth first attacked.

Description of Cavities.

They are horizontal, often wide, shallow, and saucer-shaped, sometimes wedge-shaped with the apices directed towards the pulp, polished, and have white, smooth, and hard bases. They are usually very sensitive, especially to thermal changes, sweets, acids, and instrumentation, and very rarely show signs of discoloration. Secondary or adventitious dentine is frequently present, formed as a protective measure from possible exposure of the pulp.

Microscopical Appearances.

According to *Underwood* the structure of the enamel at the seat of erosion has an exaggerated granular appearance, and presents interglobular spaces and calco-spherites. The dentine with its tubules and fibrils gives the appearance of having been cut with a sharp instrument. The *translucent zone* is well marked. According to *Black*, " Neither the dentine nor the enamel immediately adjacent to the portions being removed, even up to the immediate surface, shows any changes whatever, except it be a slight discoloration, which is present in only a portion of the cases."

Treatment.

Should the cavity be shallow and it is decided to dispense with filling, the *hypersensitiveness* may be treated by any of the following methods: —

(*a*) Apply the rubber dam, moisten the cavity with alcohol, and dry gradually with hot air from a

chip or electric hot air syringe. This treatment should be continued until the desired result is obtained.

(*b*) Apply the rubber dam, dry the cavity with alcohol and hot air, swab it with tincture of aconite to relieve the excessive pain which would be caused by the application of zinc chloride, a small piece of which is next placed in close contact with the cavity, and allowed to remain for from three to six minutes. Zinc chloride is a severe irritant, and should only be used in shallow cavities, otherwise death of the pulp might result.

Another method is to apply to the cavity a pledget of cotton wool saturated with the following solution and evaporate to dryness:—

℞ Zinc chloride ... ... gr. xx.
Alcohol ... ... fl. ʒiv.
Chloroform q.s. ad. ... ... fl. ʒj.
Mix.

Colyer suggests the following paint to be used by the patient, who should dry the cavity, paint it with the solution, and then keep it dry to allow the material to harden:—

℞ Gum mastic (powder) ... ... ʒj.
Chloroform ... ... ʒss.
Zinc chloride ... ... gr. v.
Mix.

The therapeutic action of zinc chloride is due to its powers of coagulating albumin, dehydration, and penetration of the dentinal tubes.

(*c*) *Robinson's* remedy is a combination of equal parts of caustic potash and carbolic acid.

Apply the rubber dam to the tooth and introduce the mixture into the cavity on a pledget of

cotton wool. Then direct a stream of hot air upon the part. There is at first sharp pain, which soon subsides.

(*d*) Apply the rubber dam and rub the surface of the cavity with a stick of silver nitrate, or better still, to prevent accidents, with a bead of silver nitrate fused upon a piece of platinum wire. Its action as an obtundent is due to its power of coagulating albumin, and to its penetration of the dentinal tubes. It does not penetrate very deeply. The objection to it is that it stains the tissue with which it comes into contact, and its use must therefore be restricted to situations which do not show. One point in its favour is that it stays decay. Its action is, however, slow, and several applications at intervals of several days are usually necessary.

A solution of sodium chloride should always be at hand dring its use, where it is impossible to use the rubber dam, in case of accidents, such as the swallowing of a piece, when the solution should be administered internally, or the accidental touching of the mucous membrane with the drug, when the rinsing of the mouth with the solution will counteract its injurious effects by the formation of silver chloride.

(*e*) Use the rubber dam as before and apply a pledget of cotton wool soaked in a 15 per cent. to 20 per cent. solution of trichloracetic acid.

Should the operator decide to fill the cavity any of the afore-mentioned methods may be employed, or, if preferred, one of the following:—

(*a*) Fill the cavity with a temporary filling, and cover the tooth with absorbent cotton. Direct a spray of ethyl chloride upon the tooth, gradually

removing the cotton and filling until the cavity is exposed, when the spray may be directed upon the cavity almost, if not quite, painlessly.

(*b*) Fill the cavity temporarily with oxychloride of zinc, or with oxyphosphate of zinc to which has been added a little oil of cloves or carbolic acid, and leave the filling in for six weeks, when the extreme sensitiveness will probably have disappeared, and it will be possible to prepare for and insert a permanent filling.

(*c*) Seal powdered copper sulphate in the cavity with gutta percha and leave for some days.

(*d*) Administer a general anæsthetic, such as nitrous oxide gas, and prepare the cavity during anæsthesia.

(*e*) High pressure anæsthesia may be adopted. In this case a hole is drilled into the dentine. This is then countersunk for the purpose of ensuring a perfect joint. Then with a high pressure syringe having a tapered point, such as *Myer's*, a 2 per cent. solution of cocaine is forced into the dentine, the pressure being applied for from one to one and a half minutes. The cavity may then be prepared, or should the dentine be still too sensitive for this to be carried out properly, the hole may be deepened and a further use of the syringe made, when the work may probably be finished without discomfort.

(*f*) Cataphoresis has been used in this connection, but very often the process occasions more pain than would the ordinary preparation of the cavity.

It consists of conveying chemical substances through the dentine by means of electrolytic action.

To the tooth under treatment the rubber dam is applied and securely ligatured to prevent a leakage of the current or the saliva. It is also a wise precaution to varnish the neck of the tooth and any metallic fillings in close proximity.

A pledget of cotton-wool soaked in cocaine solution, which may vary in strength from 10 per cent. to 40 per cent., is then applied to the cavity.

The cathodal electrode is next placed in contact with the cheek or neck, or held in the patient's hand, or fastened to the wrist, and the anode applied to the cotton in the cavity.

The current is now turned on, starting with the lowest voltage and the weakest amperage, and the strength gradually increased. The sensations of the patient are not at all pleasant, but when these pass away the current may, by degrees, be turned off and the preparation of the cavity proceeded with. The application usually lasts from 10 to 20 minutes.

(*g*) Apply the rubber dam, dehydrate with alcohol and hot air, apply a pledget of cotton-wool saturated in a 50 per cent. solution of aromatic sulphuric acid, and then dry with hot air, repeating the treatment until the cavity is insensitive.

(*h*) The writer has had good results from injecting into the adjacent gums, as for the extraction of the affected tooth, a local anæsthetic, such as a 4 per cent. solution of novocain, or a solution of cocaine hydrochloride, ½ gr. in 10 minims of water.

In all cases sharp, small, or medium-sized burs used with a touch-and-go movement will greatly lessen the discomfort of the patient.

Large and blunt burs are often the cause of much pain and annoyance due to pressure and the evolution of heat from friction, and this is also the case where the bur or drill is used for too long a spell without short intervals. Fillings of porcelain or oxyphosphate of zinc are valuable, and, according to *Miller* and *Marshall*, these materials seem to arrest the disintegration better than gold. Gutta-percha can also be used in many cases with advantage.

The patient should be advised to dispense with tooth-brushes and powders, and to use a soft cloth and an alkaline mouth-wash, such as sodium bicarbonate or milk of magnesia.

In most cases, however, treatment does not seem to arrest the fresh removal of tooth substance. *Mr. Royston* removes the labial glands by cauterizing them with silver nitrate, as he believes that some abnormality of them produces erosion, and he states that in those cases where he has adopted this treatment the disease has been retarded.

Attrition (fig. 46).

Definition.—The wearing away of tooth substance due to the friction of mastication or habit.

The condition occurs chiefly in deciduous and senile teeth and may be local or general. If local it is due to some abnormality of the bite, and if general to such conditions as:—

(i) *Density of Tooth Structure.*—According to *Black*, however, the differences of density of teeth are so slight as to be of very little account in relation to their resistance to external influences.

(ii) *Character of Food.*—Pre-historic teeth and the

teeth of the South American negro, &c., &c., show marked signs of attrition due to their coarse and gritty diet.

(iii) *Form of Occlusion.*—The loss of posterior teeth causing an excess of work to fall upon the anterior ones.

(iv) *Habits*, such as grinding the teeth together.

(v) *Diatheses*, such as gout and rheumatism. *Colyer* states that it occurs frequently in the teeth of adults having gouty or rheumatic tendencies.

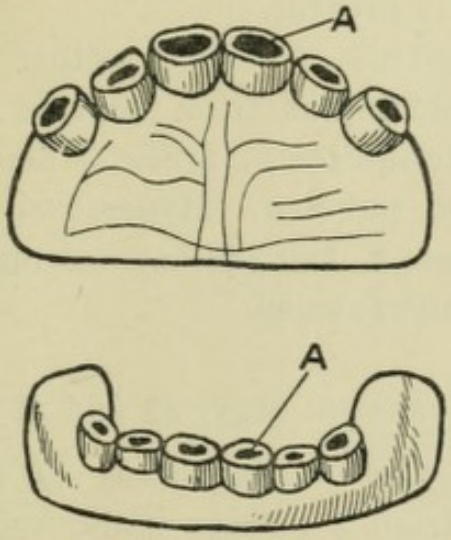

FIG. 46.—Diagram showing the worn occluding surfaces of teeth due to attrition. The dark centres A represent secondary dentine which has been formed to protect the pulps from exposure.

Description of Worn Surfaces.—There may be no cavities, but simply worn surfaces, which are polished and bright. The enamel on the masticating surfaces of the cheek teeth is worn away and the dentine exposed. This softer tissue wears away more rapidly than the surrounding walls of enamel, so that eventually saucer-shaped cavities are formed. The enamel edges may be rough and jagged, and the dentine hypersensitive. Grooves may be worn in the upper and lower incisors.

Teeth affected by attrition are often very brittle and

so strongly embedded in the jaw as to be very difficult of removal. Secondary or adventitious dentine often occurs as a protective measure from impending exposure of the pulp, and *Underwood* states that inflammation of the pulp and periodontal membrane occasionally co-exists.

Microscopical Appearances.—The enamel is pigmented. The enamel prisms are cleanly cut. The dentine presents a similar appearance to dentine in erosion, where the tubules and fibrils appear to have been cut with a sharp instrument. An increased number of inter-globular spaces is present.

Treatment.—This is often neither necessary nor advisable. Should the anterior teeth be affected, due to the loss of posterior teeth, artificial dentures, relieving the bite, may be supplied. Hypersensitiveness may be treated as in erosion, and the cavities may be filled with gold, or the teeth crowned.

Abrasion (figs. 47 and 48).

Definition.—The wearing away of tooth substance due to friction from a foreign body, such as wires and bands on dentures and regulating plates, tooth-brushes and powders, pipes, tobacco, and betel nut.

Description of Cavities.—They resemble erosion cavities, are V-shaped, with the apices directed towards the pulps, superficial, dull, rough, horizontal, and often very hypersensitive. Secondary or adventitious dentine may be produced as a protective measure from impending exposure of the pulp, or caries may result.

Position of Cavities.—They occur more often on the left than on the right side, due to the greater pressure and friction exerted in brushing the teeth with the right hand. The left upper canine and the left lower canine and pre-molars appear to be the teeth most affected.

Microscopical Appearances.—Pigmentation.

The dentine, with its tubules and fibrils, appears, as in erosion and attrition, as if it had been cut with a sharp instrument.

Treatment.—Remove cause and treat as in erosion and attrition.

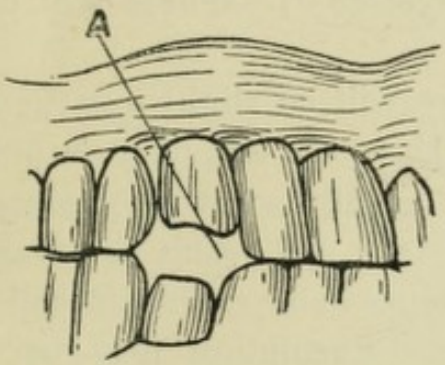

FIG. 47.—A, Abrasion due to friction from a pipe-stem.

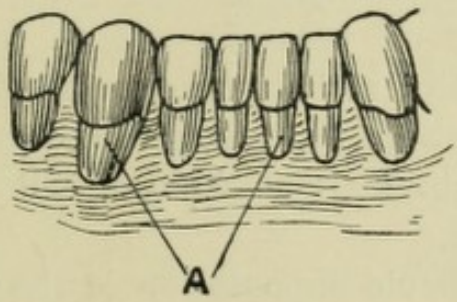

FIG. 48.—A, Abrasion cavities due to excessive use of the tooth-brush and powder.

CONCUSSION.

Definition.—The state of being shaken by sudden contact.

Cause.—Traumatism, such as a direct blow on the tooth or teeth. An indirect blow on the tooth or teeth from a blow on the jaw, &c.

Result.—This, according to the degree, may be

(*a*) Slight inflammation of the alveolar dental periosteum.

(*b*) Severe inflammation of the alveolar dental periosteum.

(*c*) Inflammation of the pulp associated with inflammation of the alveolar dental periosteum.

(*d*) Rupture and death of the pulp associated with inflammation of the alveolar dental periosteum.

Signs and Symptoms.—These will depend upon the *result.*

If (*a*), there will be slight looseness and extrusion of the tooth, and pain on pressure. Resolution usually soon takes place.

If (*b*), pressure will result in severe pain, and the looseness and extrusion will be more noticeable. Resolution may occur, but the inflammation may involve the pulp.

If (*c*), the looseness of the tooth, extrusion, and pain on pressure will be accompanied by pain of a sharp lancinating character, which may sometimes be referred to another tooth. Slight thermal changes will produce severe pain.

Should suppuration of the pulp spreading to the alveolar dental periosteum take place, thermal changes will not cause pain, but pressure will produce severe pain of a throbbing character.

If (*d*), there may be simply looseness of the tooth, extrusion, and slight pain on pressure, due to the condition of the alveolar dental periosteum, but, later on, marked discoloration of the tooth, due, according to some authorities, to the passage into the dentinal tubes of the hæmoglobin of the blood, results.

Suppuration of the pulp may occur and then the symptoms will be as in (*c*).

In addition to these, absorption of the root of an affected tooth, or spontaneous fracture, may take place.

Treatment.—If possible treat immediately. The mouth should be cleansed and rendered as aseptic as possible and the tooth or teeth then treated according to the *result*.

If (*a*) or (*b*), dry the gum over the affected tooth or teeth and paint it, using a camel hair brush, with equal parts of tincture of aconite (*Fleming's*) and tincture of iodine. During the application keep the cheeks away from the part and dry with warm air to prevent washing of the drug away by the saliva. Capsicum applied to the dried gum in the form of a plaster is also often efficacious as a counter-irritant.

If (*c*), in conjunction with the above treatment, if the inflammation of the pulp does not subside in a day or two, open up the tooth into the pulp chamber, extirpate the pulp, fill the root canal or canals and eventually the tooth.

If (*d*), apply counter-irritants as before, open up the tooth immediately, extirpate the pulp, and then proceed as in (*c*).

Dislocation.

Definition.—The displacement of a tooth from its normal situation.

Varieties.—*Partial*, where the tooth has been partially forced out of place, such as outwards, inwards, into the floor of the antrum of Highmore, or into the alveolus, &c., &c.

Complete, where the tooth has been forced

completely out, or right into the antrum, &c., &c.

Cause.—Severe traumatism, such as:—

(*a*) A blow.

(*b*) Inadvertent removal or displacement in extracting another tooth.

(*c*) Forcing out a tooth with a gag under an anæsthetic.

Signs and Symptoms.—In most cases these are obvious. Where the tooth has not been forced out of sight it is usually loose, painful to pressure, and displaced. History of traumatism. Usually rupture of the pulp. Often fracture of a portion of the alveolus.

Treatment.—The *general* treatment in all cases is to:—

(i) Irrigate the mouth with an antiseptic wash.

(ii) Thoroughly cleanse the part from blood coagula and portions of fractured alveolus.

Further treatment will depend upon:—

(*a*) The variety of the dislocation.

(*b*) The age of the patient.

(*c*) The health of the patient.

(*d*) The period of time that has elapsed between the accident and the proposed operation.

If the dislocation be *partial* and *only slight*:—

(i) Force the tooth back into its normal position.

(ii) Mould into their normal positions, as far as possible, any saveable portions of fractured alveolus.

(iii) Ligate.

(iv) Carefully take an impression in composition of the dislocated and adjacent teeth.

(v) Apply a temporary composition or gutta-percha splint.

See next page

(vi) Cast a metal splint, preferably aluminium, or make a vulcanite one.

(vii) Remove the temporary composition or gutta-percha splint and apply the permanent one. Ligatures may be used in place of a splint, but the latter will give more satisfactory results.

(viii) Apply a counter-irritant occasionally.

(ix) Advise the patient to bathe the mouth frequently with an antiseptic solution.

Should the dislocation be a *partial* one into the *floor of the antrum* or into the *alveolus*, anæsthetize the patient before attempting to restore the tooth to its normal position, as this operation often requires considerable force with a suitable pair of forceps.

After the above treatment the tooth will probably become firm in from seven to ten days, but should the alveolus be considerably involved a much longer period of time will elapse before perfect union takes place.

When dislocation is *complete, occurs in healthy youth,* and *is seen directly after the accident, e.g.*, forcing out a tooth with a gag under an anæsthetic, or accidentally removing one tooth in extracting another, the following treatment should be adopted:—

(i) Cleanse the tooth by placing it in a weak antiseptic solution, such as a 3 per cent. solution of carbolic acid, a solution of bichloride of mercury 1—1,000, or a weak borax solution.

(ii) Syringe the socket with a weak antiseptic wash until hæmorrhage ceases.

(iii) Replace the tooth firmly.

(iv) Ligate, take an impression, and proceed as with a slight partial dislocation.

Cases have been recorded where immediate replacement of dislocated teeth, even though the vessels, &c., have been completely ruptured, has resulted in the latter's reunion. In most cases, however, this does not take place, and it is therefore advisable, when the replaced tooth is perfectly firm, if it does not respond to thermal changes, to open up into the pulp-chamber, extirpate the pulp, and fill the root or roots.

In *complete* dislocation in *adults seen some time after the accident* it is advisable to extirpate the pulp and fill the root-canal or canals. This may be done:—

(*a*) Before the replacement or

(*b*) After the replacement.

If it is carried out before the replacement proceed as follows:—

(i) Place the tooth in a weak antiseptic solution for some time.

(ii) Protect the cementum and the alveolar dental periosteum (leaving the apex or apices of the roots exposed) with cotton-wool soaked in a weak antiseptic solution.

(iii) Hold the tooth by the crown, and if single rooted, enlarge the apex, withdraw the pulp, and cleanse the root thoroughly under the strictest antiseptic conditions. In a double or multiple rooted tooth open up through the crown to withdraw the pulp, &c.

(iv) Seal the apex or apices of the root or roots and fill the canals and cavity.

(v) Syringe the socket and irrigate the mouth with an antiseptic solution.

(vi) Replant.

(vii) Apply splint, &c., &c.

Should it be decided to carry out the operation after the replacement it will be wise to drill a hole through

the crown into the pulp-chamber as a vent for the escape of any gases that may form, replant, and proceed with the treatment on the tooth becoming firm.

Union takes place through the medium of the alveolar dental periosteum.

Sequelæ:—

(i) Looseness of teeth.

(ii) Absorption of roots.

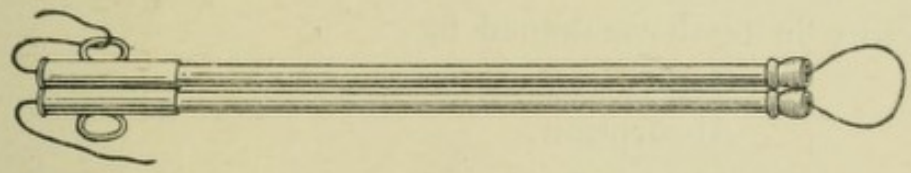

FIG. 49.

From *Marshall's* "Operative Dentistry."

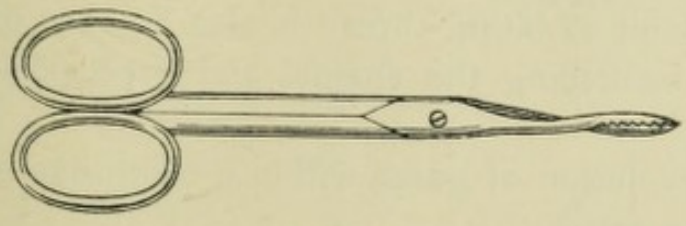

FIG. 50.

From *Marshall's* "Operative Dentistry."

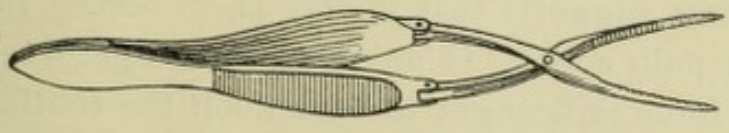

FIG. 51.

From *Marshall's* "Operative Dentistry."

Should the tooth have been *forced into the antrum of Highmore* it may be discovered and removed by means of an adaptable loop of wire (fig. 49) or either of the pairs of forceps shown in figs. 50 and 51.

FRACTURE.

Definition.—The sudden solution of continuity of a tooth.

Causes.—Traumatism, such as:—

(*a*) A direct blow.

(*b*) An indirect blow, such as a blow on the chin bringing the teeth into sudden contact.

(*c*) Attempted extraction.

(*d*) Excising a tooth without grooving it sufficiently may produce fracture of the root, often longitudinal.

Slight pressure in mastication, &c., may produce fracture in teeth weakened by

Caries.
Absorption.
Abrasion.
Erosion.
Attrition.

The habit of biting thread is also a cause of cutting into and weakening the enamel and predisposing it to fracture.

The evolution of gases within a tooth has been suggested as a cause.

The expansion of metal fillings through thermal change has also been suggested as a cause.

Tomes suggests that it may be produced through a calcified pulp acting as an internal wedge when a distorting force is brought to bear upon the tooth.

Varieties of Fracture:—

(*a*) *Simple*, when the hard tissues only of the tooth are involved.

(*b*) *Compound*, when both the hard tissues and the pulp are involved.

(*c*) *Comminuted*, when the tooth is broken into numerous fragments.

Degrees of Fracture:—

(i) Slight cracks and fissures in the enamel.

(ii) Chipping of the enamel.

(iii) Chipping of the enamel and dentine.

(iv) Transverse fracture of the crown exposing the pulp.

(v) Transverse fracture of the root.

(vi) Oblique fracture.

(vii) Longitudinal fracture.

Symptoms.—These will depend upon the degree.

If (i), there is no pain, but discoloration may occur at the seat of the lesion.

If (ii), there is loss of enamel.

Usually no pain.

The fractured surface may be slightly sensitive.

If (iii), there is:—

(*a*) Impairment or loss of function.

(*b*) Hypersensitiveness.

(*c*) Hyperæmia of the pulp.

(*d*) Pain to thermal changes, sweets, acids, and instrumentation.

(*e*) Pain on pressure through passage of the inflammation to the alveolar dental periosteum.

(*f*) Sometimes swelling of the gums.

If (iv), as in (iii) with exposure of the pulp.

If (v), there is:—

(*a*) Impairment or loss of function.

(*b*) Hyperæmia of the pulp.

(*c*) Pain on pressure through passage of the inflammation to the alveolar dental periosteum.

(*d*) Swelling of the gums.

(*e*) *Crepitus* (a harsh grating feeling produced by rubbing one portion of the fractured tooth over the other). This is not always noticeable. The manipulation should be as gentle as possible.

(*f*) Neuralgic pain if the pulp is involved.

If (vi), as in (iii), with probable exposure of the pulp.

If (vii), as in (v).

Longitudinal fracture is more common in the posterior teeth.

Treatment.—This will depend upon the degree.

If (i), leave alone.

If (ii), smooth and polish with stones, discs, &c.

If (iii), apply counter-irritants, such as aconite and iodine or a capsicum plaster, to the gum, or make incisions to deplete the part. Apply an obtundent, such as zinc chloride, alcohol and hot air, &c., &c., to the sensitive dentine. Trim and smooth.

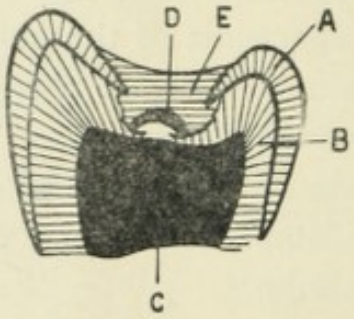

FIG. 52.—A, enamel; B, dentine; C, the pulp; D, cap; E, filling.

If a large portion of dentine is involved wait for the inflammation to subside and then fill. Should suppuration of the pulp ensue, open up, extirpate, and fill.

If (iv), the following points must be taken into consideration:—

(*a*) *Age.*—If the root of the tooth is not completed, save the pulp, if possible, until this has occurred. This may be effected by *capping* (fig. 52).

The cap should be circular, have a concave surface, and be made from tin, platinum, or other suitable material. In

a very shallow cavity a small flat piece of cardboard may be utilized as a cap.

The process is as follows:—

(i) Stop any hæmorrhage, and this may be done with spirits of camphor.

(ii) Syringe out the cavity with tepid water.

(iii) Apply the rubber dam.

(iv) Swab with a non-irritant antiseptic, such as a warm weak borax solution.

(v) Dry thoroughly with warm air.

(vi) Flow a very small quantity of a melted mixture of equal parts of salol and thymol thickened with a little very fine zinc oxide, or a mixture of oxysulphate of zinc and oil of cloves, or of chloroform and gutta-percha over the exposure.

(vii) Fill the concave surface of the cap with the same mixture used for flooring and apply it accurately over the pulp, the edge resting on the dentine all round.

(viii) Fill the cavity with *Hill's* gutta-percha or cement.

The following conditions are necessary to successful capping:—

A perfect fitting cap.
A non-irritant cap.
A non-conducting cap.
Absence of pressure.
Asepsis.

Should it be deemed inadvisable to cap, the root may be extracted and an artificial denture made, to prevent the movement of the approximate teeth into the space and the alteration of the centre line with consequent disfigurement of the arch.

Should the root be completed the pulp may be devitalized and extirpated, and the root treated and crowned.

(*b*) *Sex.*—From an æsthetic standpoint it is much more important to save the tooth in a girl than in a boy.

(*c*) *Crowding.*—It might be advisable to extract in any case in order to rectify or prevent this irregularity.

If (v) or (vi), extract if the fracture is high up and there is no chance of union. If near to the cervical margin the root may be treated and crowned. Union can and has been known to take place. It is more likely to occur in young developing than in adult teeth and when the fracture is near to the apex of the root. It occurs through the formation of secondary or adventitious dentine by an irritated pulp, and the fresh activity of the alveolar dental periosteum resulting in the formation of cementum.

Two important conditions are absolutely essential to union, *viz.*:—

(*a*) Perfect immobility of the parts.

(*b*) An uninjured pulp.

If (vii), extract, although banding and crowning may be resorted to.

Perforation of Root.

This may accidentally occur during enlarging, &c., resulting in injury to the alveolar dental periosteum.

In most cases extraction of the tooth is the wisest course to pursue.

If it is decided to save the tooth, however:—

(i) Syringe out the canal with a warm non-irritant antiseptic solution.
(ii) Stop the hæmorrhage.
(iii) Cleanse canal.
(iv) Locate the perforation.
(v) Cap perforation as in capping pulp (see pages 94 and 95.)
(vi) Fill the root canal, and seal up temporarily for a few days.
(vii) Fill permanently if everything satisfactory.

CHAPTER VII.

DISEASES OF THE PULP.

Hyperæmia.

Definition.—An inflammatory condition in which there is an excessive quantity of blood in the part and an increased flow.

Varieties:—

(i) Intermittent.
(ii) Continuous or acute.

Causes:—

(i) Traumatism.
(ii) Thermal changes.
(iii) Sweets and acids.
(iv) Caries.
(v) Injudicious use of drugs, such as arsenious acid or zinc chloride.
(vi) Erosion, attrition, abrasion.
(vii) Irritation from a filling.
(viii) Instrumentation.
(ix) Galvanic action between two metal fillings, such as gold and amalgam.
(x) Pulp stones.
(xi) Septic infection through a carious cavity.
(xii) Recession of the gum.
(xiii) Neurasthenia, pregnancy, internal use of mercury, rheumatism of the jaw, dyspepsia, gout, &c., &c.

Irritation, through any of the above causes, produces inhibition of the vaso-motor nerves.

Symptoms.—If it occurs in a tooth in which the hard tissues are sound thermal changes produce pain.

There is, however, usually a shallow cavity, a filling, or some other obvious cause present. The patient may complain of slight discomfort only.

This discomfort may eventually become pain of a sharp, lancinating, and severely paroxysmal character.

The pain may be referred.

There may be pain on pressure due to the spread of the inflammation to the alveolar dental periosteum.

Pathology.—The blood-vessels are irregularly dilated, varicosed, tortuous, and have thin walls.

There is a proliferation of pulp cells and an increase in the number of odontoblasts.

Red blood corpuscles, which have migrated through the vessel walls, are sometimes noticed.

Patches of secondary dentine may be present.

The translucent zone in the adjacent dentine is present.

Treatment.—Should the hard tissues of the tooth be sound, apply counter-irritants, at intervals, to the gum. *Black* suggests covering the tooth with a closely fitting gutta-percha cap to protect it from external influences. This rests the part and resolution may take place. If the pain still continues after a short interval, open up the tooth, extirpate the pulp, treat the root canal or canals, and fill.

In other cases *remove the cause*, such as a filling, &c., and *keep the tooth at rest* for a few days by inserting in the cavity a sedative, antiseptic dressing, such as a mixture of *Fletcher's* artificial dentine, carbolic acid, and cotton-wool. As soon as the tooth is comfortable, floor the cavity with a non-irritating, non-conducting material,

such as a mixture of oxysulphate of zinc and oil of cloves, and fill.

Should the cause be galvanic action, remove one of the fillings and replace with a similar filling to that contained in the adjacent tooth, or with a non-conductor such as oxyphosphate cement.

If due to pulp stones, or septic infection through caries, devitalize, extirpate the pulp, treat the canals, and fill.

Where there is recession of the gum the necks of the teeth should be dried thoroughly and touched occasionally with silver nitrate or zinc chloride, as indicated.

The general troubles, such as neurasthenia, must be treated constitutionally.

With pregnancy advise an aperient, a warm foot-bath, and anodynes at night.

Pulpitis.

Definition.—Inflammation of the pulp.

Varieties:—

(i) Acute.

(*a*) Simple (unassociated with micro-organisms).

(*b*) Infective (complicated by the presence of micro-organisms). More common than the simple variety.

(ii) Chronic.

(i) Acute.—

Causes.—As in hyperæmia.

Symptoms.—Impairment or loss of function. Pain of a sharp, lancinating, throbbing character, which is less paroxysmal and inclined to be more continuous than in hyperæmia.

The pain is worse after exertion, after lying down, and in the young, where the pulps of the teeth are large

and the amount of adventitious dentine small. Thermal changes, sweets, and acids cause severe pain, although, as *Colyer* points out, cold, in the early stages, gives relief, on account of a constriction of the blood-vessels.

Pressure of food, &c., in the cavity causes pain.

When the pulp is not exposed the symptoms are much more severe, but do not last as long as when exposure has taken place.

Diagnosis from Acute Periodontitis.—Adapted from *Colyer's* "Dental Surgery and Pathology":—

	Acute Pulpitis.	Acute Periodontitis.
Pain	severe, sharp, lancinating, throbbing, intermittent, reflected.	dull, heavy, constant.
Thermal changes	cause pain	do not cause pain.
Sweets and acids	cause pain	do not cause pain.
Pressure on tooth	does not cause pain unless the inflammation has spread to the alveolar dental periosteum.	causes pain.
Pressure in cavity	causes pain	does not cause pain unless the inflammation has spread to the alveolar dental periosteum.

Pathology.—The same as in inflammation in any vascular tissue. (See pages 52, 53, and 54.) There is a multiplication in the number and layers of odontoblasts.

In certain situations these cells are shrunken, and in others absent.

The definite structure of the nerve bundles is altered.

On the border of the dentine, between it and the pulp, are seen portions of calcified material (*adventitious dentine*). These are due to a fresh activity of the pulp in an effort to protect itself from the oncoming irritation.

Where infection has occurred micro-organisms are present.

Sometimes, where a portion of the pulp has degenerated into pus, fibroblasts are formed, enclosing,

or dividing off the pus from the rest of the tissue, and so protecting it.

Sequelæ:—

(i) Suppuration (usually). (See page 54.)

(ii) Gangrene, due to the strangulation and consequent cutting off of the blood supply at the apex.

(iii) Chronic inflammation.

(iv) Resolution (uncommon).

Treatment:—

(i) *Remove the cause*, such as a filling, &c. If due to caries, remove as much carious tissue as possible.

(ii) Wash out the cavity with an alkaline solution, such as—

℞ Bicarbonate of soda ... ... ʒj.
Tepid water ... ... fl. ʒj.

to neutralize the acidity.

(iii) Apply the rubber dam.

(iv) Dry the cavity with warm air.

(v) If there is no exposure and the tooth is too sensitive to allow of a complete removal of the carious tissue, *keep the part at rest* for a day or two by protecting it from external influences with a sedative, antiseptic dressing, such as a mixture of *Fletcher's* artificial dentine, carbolic acid, and cotton-wool.

(vi) Apply a counter-irritant to the gum.

(vii) After a day or two floor the cavity with a mixture of oxysulphate of zinc and oil of cloves, and fill permanently.

Should the inflammation still continue, or an exposure of the pulp have occurred, the latter may be—

(*a*) Extirpated after devitalization with arsenious acid (arsenic trioxide) or cobalt.

(*b*) Extirpated under pressure anæsthesia (the induc-

tion of local anæsthesia by the forcible application of such agents as cocaine).

(*c*) Extirpated under anæsthesia after injection into the gum.

(*d*) Extirpated during general anæsthesia.

(*e*) Capped.

(*f*) Mummified.

Or the tooth may be extracted.

(*a*) *Extirpation after devitalization with arsenious acid or cobalt.*

Process.—

(i) If there is much inflammation seal a small quantity of the following, on cotton-wool, in the cavity with a temporary cement, such as calxine, for twenty-four hours.

℞ Acetate of morphine ... gr. xx.
Tannic acid ... ... gr. xxx.
Creosote ... ... ℥ij.

As an anodyne it prevents or lessens the severe pain which would result from the immediate application of arsenic. A dressing of oil of cloves or carbolized resin may be used for the same purpose.

(ii) Remove as much decay and make as free an exposure as possible. It is important to remove all the carious tissue and to wedge out or remove any flap of gum at the cervical margin to prevent any escape of arsenic. Puncturing of the pulp, after obtunding the surface with cocaine, is advantageous, as it lessens the congestion and so prevents or lessens any pain that might otherwise have resulted from the application of arsenic. It also prevents discoloration, which otherwise might result from a distribution of hæmoglobin through the dentinal tubes.

(iii) Syringe with warm water and bicarbonate of soda to neutralize the acidity.

(iv) Apply the rubber dam.

(v) Disinfect the cavity and the pulp with a warm 2 per cent. solution of carbolic acid, or with peroxide of hydrogen.

(vi) Dry the cavity with warm air.

(vii) Apply one-sixteenth of a grain of arsenious acid (arsenic trioxide) on a small piece of cotton-wool (about the size of a pin's head) which has been dipped into oil of cloves, in close contact with the pulp.

NOTE.—It is better to apply arsenic in the morning, so that any pain that may occur will be over before night. The larger the dose of arsenic the greater will be the resulting pain. Large doses of arsenic often prevent successful devitalization.

The following paste will be found useful:—

℞ Arsenious acid ... gr. x.
Acetate of morphia ... gr. x.
Creosote ... ... q.s. to make a paste,

or a fibre preparation, such as *Ash's* or *S. S. White's*, may be used.

(viii) Cap, to prevent pressure from the temporary filling and from the expansion of the pulp, fill with a temporary cement, such as calxine, and leave for from twenty-four to forty-eight hours if the paste has been used, or from five to seven days should the fibre have been applied.

The following illustration (fig. 53), borrowed from *Colyer's* "Dental Surgery and Pathology," by permission of *Messrs. Longmans, Green and Co.*, the publishers, is useful in explaining a safe method of using arsenic in approximal cavities near the gum.

Action of arsenic on the pulp:—

There is at first arterial hyperæmia, then pain (if any), then acute congestion of the blood-vessels, then

exudation, and finally strangulation of the pulp at the apex, causing stasis and death.

The connective tissue fibres do not change. The odontoblasts do not change. The connective tissue cells are increased in size. Some axis cylinders of the nerve elements disappear.

Powdered dry cobalt (*arsenical ore*) applied, for from four to six days, on a small piece of cotton-wool dipped in oil of cloves, is often used for devitalization. Its advantages over arsenic are:—

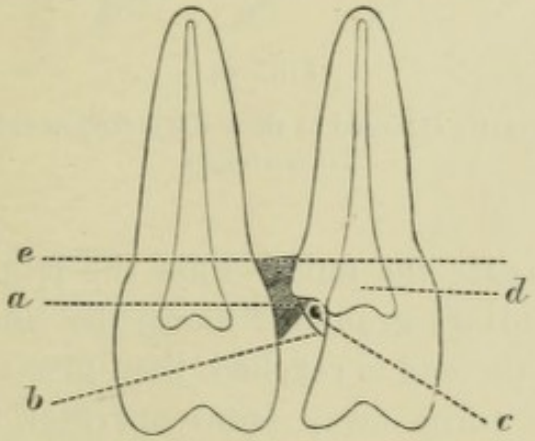

FIG. 53.

(*a*) Gutta-percha.
(*b*) Metal cap covering dressing.
(*c*) Dressing of arsenious acid.
(*d*) Pulp chamber.
(*e*) Line indicating margin of gum.

There is less pain and inconvenience.

The pulp becomes harder and drier.

There is less hæmorrhage in removing the pulp.

Its disadvantages are:—

It often stains the tooth pink.

Its action is longer than that of arsenious acid.

(ix) If time allows, insert a dressing of tannin for several days to harden the pulp and so facilitate its removal.

(x) Apply the rubber dam.

(xi) Open up the pulp chamber, remove all trace of arsenious acid, if used, and wipe out the cavity with a solution of dialysed iron to neutralize any that may have been left.

(xii) Remove all carious tissue, prepare the cavity for filling, and disinfect by swabbing with a 2 per cent. solution of carbolic acid or with peroxide of hydrogen.

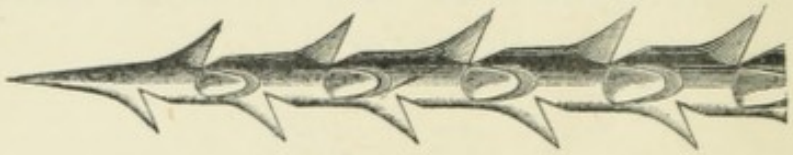

FIG. 54.

Pulp broach greatly enlarged to show the arrangement of the minute sharp hooks.

(xiii) Extirpate the pulp. Pass the pulp broach (figs. 54 and 55) gradually along the side of the canal until the end is reached, then give it two or three quick turns away from operator and withdraw it sharply, tearing away the pulp in the process. Should a small portion of the pulp be broken in the canal and sensitive, do not use arsenious acid

FIG. 55.

Pulp broach. The lengths of the broach and the handle vary according to the tooth to be treated and its position.

again, but introduce on cotton-wool a 95 per cent. solution of carbolic acid and leave for a day or two, when the broken portion can probably be painlessly removed.

(xiv) Stop any hæmorrhage. This may be effected by syringing with warm water and then introducing

into the canal on cotton-wool a 95 per cent. solution of carbolic acid and leaving it there for some little time.

(xv) Thoroughly dry the canal with alcohol and hot air.

(xvi) Fill the root or roots and finally the tooth.

(*b*) *Extirpation of the pulp during pressure anæsthesia.*

Process.—If there is an exposure:—

(i) Remove as much carious tissue and prepare the cavity as well as possible.

(ii) Syringe out with warm water and bicarbonate of soda to neutralize the acidity.

(iii) Apply the rubber dam.

(iv) Swab the cavity with a disinfectant and analgesic, such as a 2 per cent. solution of carbolic acid, or with peroxide of hydrogen.

(v) Place in the cavity over the pulp a pellet consisting of $\frac{1}{6}$ gr. cocaine hydrochlorate and $\frac{1}{300}$ gr. adrenalin.

(vi) Press on this, gently at first, to avoid pain, with unvulcanized rubber, gradually increasing the pressure until the pulp is anæsthetized.

(vii) Open up the pulp chamber and prepare the cavity.

(viii) Extirpate the pulp.

(ix) Stop the hæmorrhage.

(x) Dry the canal or canals.

(xi) Fill the roots and cavity.

If there is no exposure, and a fair thickness of dentine remains between the cavity and the pulp, *high pressure anæsthesia* (page 80) may be resorted to. Some experimenters state, in this connection, that, in cases of

pulp degeneration, where the use of cocaine is not effective, three or four drops of

1 part of alcohol
3 parts of water

is a valuable substitute.

(*c*) *Extirpation of the pulp may be carried out during local anæsthesia*, which may be produced by the intra-alveolar or sub-mucous injection of a local anæsthetic, such as a 4 per cent. solution of novocain or a solution of cocaine hydrochloride, ½ gr. in 10 minims of water.

(*d*) *Extirpation of the pulp during general anæsthesia.* Nitrous oxide gas is preferable.

Process:—

(i) Have the anæsthetic administered.

(ii) Extirpate the pulp with a pulp broach.

(iii) Insert into the canal with a smooth broach a wisp of cotton-wool soaked in a 95 per cent. solution of carbolic acid. This keeps the canal as aseptic as possible and stops the hæmorrhage.

(iv) Shortly after the recovery of the patient from the anæsthetic, apply the rubber dam, and proceed to treat and fill the canals and the tooth, or the cavity may be temporarily filled and the work completed on a subsequent visit.

Some operators fill the root canals of teeth from which healthy pulps have been removed at the same sitting.

The writer prefers to do this on a subsequent visit when the hæmorrhage has quite subsided.

In this connection also, where the pulp has been extirpated during *pressure* or *local anæsthesia*, the soft tissues about the apical foramina are also anæsthetized,

and it is difficult to tell whether the whole of the pulp has been extirpated or whether the root fillings have reached or passed through the apices.

(*e*) *Capping the pulp.*

This is a very difficult operation to perform satisfactorily and is not advised by the author unless the pulp is quite healthy and uninjured, or it is desired to preserve it for the completion of an undeveloped tooth. (See pages 94 and 95.)

(*f*) *Mummification of the pulp.*

This is another operation not recommended by the author. It tends to lazy and careless operating and is imperfect and hygienically unsound.

The only cases in which its adoption can possibly be justified are:—

(*a*) In teeth with tortuous and inaccessible roots, or where the roots have become partially filled in and constricted by the deposition of secondary dentine.

(*b*) In the teeth of patients who are physically, or on account of age, unfit to undergo the comparatively tedious processes of pulp devitalization and root treatment.

(*c*) In the teeth of patients who find it quite impossible to give the necessary time for the more conservative treatment.

Process:—

(i) Carry out the process of devitalization with either arsenious acid or cobalt. Arsenious acid is a preservative and therefore aids in mummification.

(ii) Apply the rubber dam.

(iii) Remove the coronal portion only of the pulp with a sterilized rose head bur.

(iv) Syringe out the cavity with warm water, dry

with alcohol, and swab out with cotton-wool soaked in formalin.

(v) Place a portion of the following paste (recommended by *Soderberg*), mixed with a small quantity of formalin, in the pulp chamber, and, with a suitable broach, carry it into the canals. Then fill the pulp chamber with it, floor with cement, and fill permanently. The paste is

℞ Alum ex. sic, Thymol, Glycerol ... āā ... 3j.
Zinc oxide q.s. to make a stiff paste.

The alum mummifies or tans.
The thymol acts as an antiseptic.
The glycerol acts as a binding and penetrating agent.
The zinc oxide is the conveying medium.

NOTE.—Never apply the paste unless the pulp is completely devitalized, otherwise severe pain, for several days, may result. Should pain arise after mummification apply a counter-irritant to the gum and prescribe ammonal internally in 10 gr. doses.

Those operators who favour mummification advance the following points in its favour:—

(i) It is easy for the operator and the patient.
(ii) There need be no extensive destruction of tooth substance.
(iii) It is difficult to anticipate the presence of curved and tortuous roots.
(iv) In those cases in which mummification fails, the extirpation of the pulp and root treatment can then be resorted to.

NOTE.—Mummification should never be attempted in connection with septic pulps or inflamed pulps associated with periodontitis.

(ii) Chronic.—

Definition.—An inflammation of the pulp due to similar causes to the acute, acting less intensely and over a longer period.

Causes.—Similar to acute.

More often the predisposing general. Is often the termination of acute pulpitis.

Symptoms.—Not so active as in acute.

The *pain* is irregular and usually not continuous, is sometimes dull and continuous, often referred and neuralgic, and often intense if the affected pulp is under a filling.

Thermal changes and external agents, such as sugar and salt, may cause paroxysms of pain.

There may be pain on mastication due to the inflammation having spread to the alveolar dental periosteum.

There may be no pain. The pulp is deep red, extremely sensitive, and bleeds readily.

Pathology.—There is a continuous dilatation of the vessels, which lose their tone.

The blood flow is slow.

The exudation is less than in acute pulpitis.

The proliferation of tissues is more excessive than in acute pulpitis.

Terminations:—

(*a*) RESOLUTION never occurs.

(*b*) FORMATION *of cells*, which enlarge the pulp cavity by an absorption of the dentine (*destructive pulpitis*).

(*c*) HYPERPLASIA (*polypus of the pulp or productive pulpitis*).

(*d*) SUPPURATION.

(*e*) DEGENERATIONS.

Fibroid (senile atrophy).

Fatty.

Calcareous.

(*c*) Hyperplasia (*polypus of the pulp or productive pulpitis*).

Definition.—An increase of the elements which compose the tissue of the pulp, resulting in an increase in its bulk.

Cause.—Irritation from the jagged edges of a carious tooth.

Pathology.—A formation of granulation tissue which, when old, becomes fibrous at the neck.

Masses of endothelial cells may be found at the surface.

Symptoms.—It is not painful on pressure.

It does not bleed readily on pressure.

It can be traced into continuity with the pulp.

It frequently ulcerates at the surface.

The first three differentiate it from *hypertrophy* or *polypus of the gum*, which is painful on pressure, bleeds readily on pressure, and can be traced into continuity with the gum.

Squamous epithelium is often found on the surface of a polypus of the pulp. It has been stated that this is due to a process of skin grafting from the cheeks.

Hopewell-Smith suggests that it is due to an extension, over the pulp, of proliferated epithelial cells of the gum, this proliferation being due to irritation from the jagged edges of a carious tooth.

Polypus of the pulp may undergo calcification.

Treatment:—

(i) Syringe with warm water to wash away any *débris*.

(ii) Protect the surrounding parts with rubber dam or cotton rolls.

(iii) Dry the growth and cavity with wool and warm air.

(iv) Paint the growth completely with tincture of iodine.

(v) Seal the cavity with calxine.

Repeat the above treatment every twenty-four hours until the growth is destroyed.

(vi) Devitalize and extirpate the pulp, treat, and fill the roots, &c.

Another method is to:—

(i) Syringe with warm water.

(ii) Protect with dam or cotton-wool rolls.

(iii) Dry.

(iv) Apply on a glass rod or platinum or gold wire

℞ Chromic acid ... ... 1 part.
Water ... ... 4 parts.

This application gradually destroys the growth.

(v) Devitalize and extirpate the pulp, treat the roots, &c.

Some operators adopt the following method:—

(i) Apply carbolic acid to the growth to obtund it.

(ii) Excise it.

(iii) Devitalize and extirpate the pulp, treat, and fill the roots, &c.

It is often necessary and advisable to extract the tooth instead of treating the polypus.

(*d*) Suppuration.

Definition.—A degeneration of the pulp tissue into pus through a slow progressive ulceration.

Causes.—Traumatism.

Too great force in regulating.

Too rapid separation of teeth.

Thermal influences on large metal fillings placed too near to the pulp.

Capping of unsuitable pulps.

Long continued exposure of pulp to irritating agents.

Symptoms.—There is sometimes no pain, and this

is usually so when there is a vent for the escape of the pus and gases.

There is usually, at first, a slight gnawing sensation, which gradually becomes pain of an excessive character.

In the early stages thermal changes cause pain, but later, *heat causes intense pain*, due to an expansion of the gases and therefore increased pressure, whilst *cold relieves*, due to a contrary effect.

Treatment.—Open up immediately to evacuate pus. This gives immediate relief.

If the whole pulp has not degenerated devitalize or anæsthetize the remaining portion.

Extirpate, treat, and fill the roots, &c.

(*e*) Degenerations.

Fibroid (senile or reticular atrophy).

Definition.—A degeneration, through fibrification, of all the elements of the pulp, *viz.*, nerves, blood-vessels, &c.

Cause.—Defective nutrition occurring in connection with senility and following on prolonged chronic pulpitis.

Macroscopically:—

Tooth retains its natural colour.

Pulp cavity free from products of decomposition.

Sensitivity of pulp diminished.

Pulp flattened, shrivelled, reddish or dark-brown, and often brittle.

Microscopically:—

Fibrous tissue copious and marked.

Disappearance of original cells of pulp.

Presence of many areolar spaces.

Shrunken or absent odontoblasts.

Fewer and enlarged blood-vessels.

Fatty or granular nerve-sheaths.

Symptoms.—Little or no pain unless the pulp or root of the tooth is exposed, when there is severe pain.

Treatment.—Devitalize or anæsthetize the pulp, extirpate it, and treat and fill the roots, &c.

Fatty.

This degeneration may occur in connection with:—

Senile pulps.

Pulps of deciduous teeth undergoing absorption.

Capped pulps.

Definition.—A condition in which the nerve-sheaths and the vessels of the pulp have undergone degeneration, the cells have become almost obliterated, and a fatty structure become conspicuous.

Cause.—Diminished vital activity following pulpitis.

Macroscopically:—

Pulp diminished, in volume, by about one half, cloudy, yellowish red or grey, is sometimes cheesy like and sometimes soft and greasy.

Microscopically:—

Degenerated, fatty odontoblasts.

Chains of fat globules occur along the lines of the vessels and nerves.

Degenerated nerve-sheaths.

Degenerated vessel walls.

Treatment.—Extirpate the pulp.

Treat and fill the canals, &c.

Calcareous.

This is a common form of degeneration, and may take the form of *secondary* or *adventitious dentine, pulp stones,* or *calcification of the tissue of the pulp.*

Secondary or adventitious dentine:—

This differs according to the conditions under which it is formed, *viz.*:—

(i) Age of pulp.

(ii) Severity of the inflammation.

Definition.—A secondary formation on the surface of the pulp.

Cause.—Injury to and consequent irritation of the distal ends of the dentinal fibrils, through

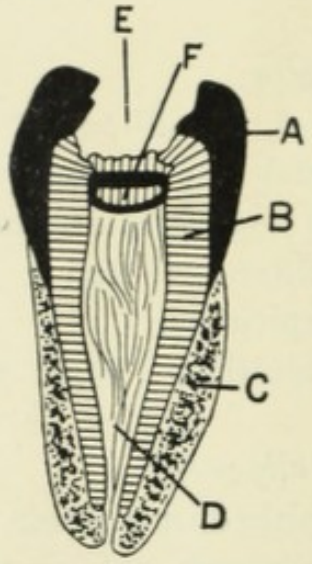

FIG. 56.—A, enamel; B, dentine; C, cementum; D, the pulp; E, cavity due to caries: caries may not be present, but the irritation may be due to erosion, abrasion, &c.; F, *secondary or adventitious dentine* produced through long-continued irritation of the distal ends of the dentinal fibrils.

caries, erosion, attrition, abrasion, &c., the new tissue being formed at the pulp ends (*Dentine repair of Salter*), fig. 56. It is situated opposite the lesion, is continuous with the normal dentine, and is not connected with the pulp.

Varieties:—

Areolar, which contains a large number of interglobular spaces. It is the commonest variety and is formed rapidly.

Fibrillar, which is like normal tubed dentine. There is a bend between the tubes of the normal dentine and the secondary, and the boundary zone between the two is well marked.

Hyaline, which is homogeneous, and is formed slowly.

Cellular, which has fusiform or round cells.

Laminar, which has a laminated form.

Symptoms.—Absence of pain.

Loss of enamel and dentine.

Presence of secondary dentine which is darker in colour than normal dentine.

Pulp chamber smaller than normal.

Pulp canals often constricted.

Treatment.—Leave alone, or fill or crown.

Pulp Stones (fig. 57):—

Definition.—A pulp stone is a nodule of calcified material developed in the substance and near to the periphery of the pulp.

Cause.—Obscure.

Probably some form of irritation due to chronic inflammation, senility, gout, abrasion, erosion, attrition, &c.

Hopewell-Smith thinks that they are due to a secretion from or a conversion of the small round cells of the pulp.

Situation.—In the substance of the pulp near to the periphery.

More common in adult teeth.

In any part of the pulp, but chiefly the coronal portion.

Sometimes found in the pulps of young teeth which show no signs of having been irritated.

It becomes embedded in any secondary dentine that eventually forms.

Symptoms.—Often pain of a neuralgic character.

Sometimes no pain.

The nodules are gritty to the touch.

Treatment.—Devitalize or anæsthetize the pulp.

Extirpate.

Treat and fill the roots, &c.

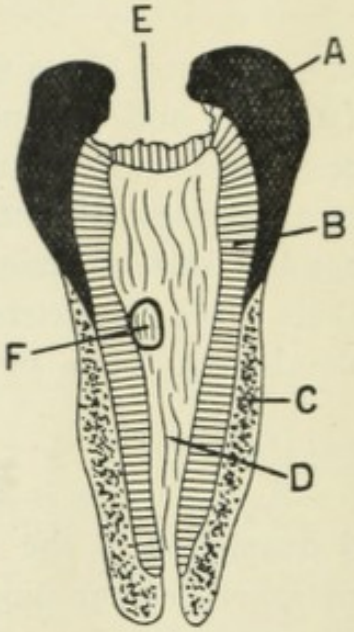

FIG. 57.—A, enamel; B, dentine; C, cementum; D, pulp; E, cavity due to caries. The presence of a pulp stone is not dependent upon this. It sometimes occurs in a sound tooth; F, pulp stone.

Calcification of the tissues of the pulp:—

In this degeneration the whole pulp is affected, small, calcified, gritty masses being seen throughout the tissues. Sometimes the structure is visible, sometimes granular, and sometimes the mass is structureless.

CHAPTER VIII.

THE TREATMENT AND FILLING OF ROOT CANALS.

The operation of root canal treatment is one of the most important in dental surgery, and a thorough knowledge of the anatomy of the roots of teeth is necessary for the operation to be, as far as is humanly possible, successful.

The following diagrams are intended to show the more common forms of pulp canals and the usual number of roots. Many other forms, however, may occur, and a tooth may have a root or roots above the normal number.

In the teeth of the young the canals are larger than

Fig. 58.

Fig. 59.

Fig. 60.

Fig. 58.—Root canal of a young upper central or lateral incisor, showing well marked cornua.

Fig. 59.—Root canal of an upper central or lateral incisor.

Fig. 60.—Transverse section through the root canal of an upper central or lateral incisor.

in those of the adult, whilst in old age they are smaller, due to the formation of adventitious dentine.

The foregoing are usually treated without difficulty, being comparatively large and easy of access. This applies also to figs. 64 and 66.

FIG. 61. FIG. 62. FIG. 63.

Fig. 61.—Normal root canal of a lower central or lateral incisor.
Fig. 62.—Bifurcation of the root canal of a lower central or lateral incisor.
Fig. 63.—Transverse section through the root canal of a lower central or lateral incisor, showing the lateral constriction.

The above are a little difficult to treat thoroughly on account of their lateral constriction. This applies also to figs. 65 and 67.

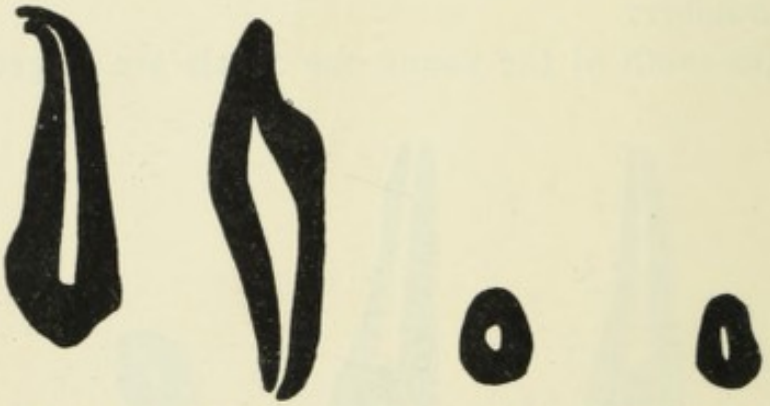

FIG. 64. FIG. 65. FIG. 66. FIG. 67.

Fig. 64.—Root canal of an upper canine or cuspid.
Fig. 65.—Root canal of a lower canine or cuspid. This tooth often has two roots, and, therefore, two canals.
Fig. 66.—Transverse section through the root canal of an upper canine or cuspid.
Fig. 67.—Transverse section through the root canal of a lower canine or cuspid, showing the lateral constriction.

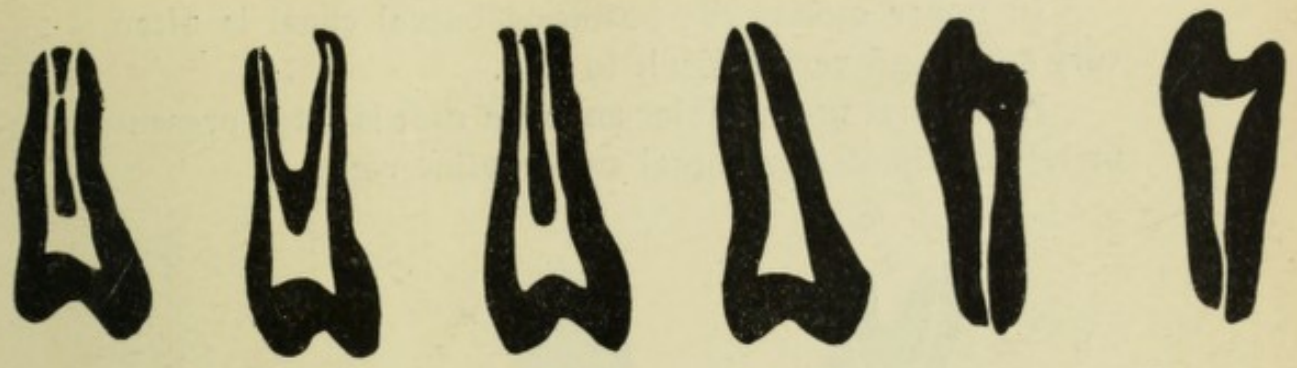

FIG. 68. FIG. 69. FIG. 70. FIG. 71. FIG. 72. FIG. 73.

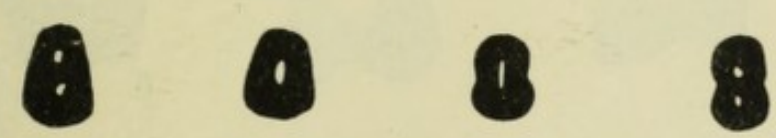

FIG. 74. FIG. 75. FIG. 76. FIG. 77.

Fig. 68.—Root canal of a first upper premolar.

Fig. 69.—Another form, showing the usual two roots and canals of a first upper premolar. Three roots are sometimes present.

Fig. 70.—Root canal of a second upper premolar.

Fig. 71.—A more common form of root canal of a second upper premolar. Two or three roots, and therefore canals, may be present.

Fig. 72.—Root canal of a first lower premolar. This is sometimes bifurcated, but very rarely.

Fig. 73.—Root canal of a second lower premolar.

Fig. 74.—Transverse section through a first upper premolar, showing the usual two canals.

Fig. 75.—Transverse section through the root of a second upper premolar, showing the usual one canal.

Fig. 76.—Transverse section through the root of a lower premolar, showing the usual one laterally constricted canal.

Fig. 77.—Transverse section through the root of a lower premolar, showing two canals.

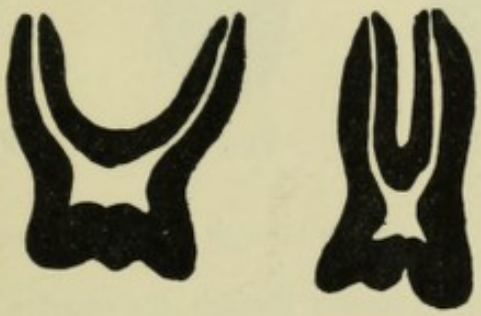

FIG. 78. FIG. 79.

Fig. 78.—Longitudinal section through the palatine and one buccal root of a first or second upper molar.

Fig 79.—Longitudinal section through the two buccal roots of a first or second upper molar.

In upper molars the posterior buccal canal is often very small and very difficult to find.

In the first upper molar an extra root is often present between the anterior buccal and palatine roots.

FIG. 80.

FIG. 81.

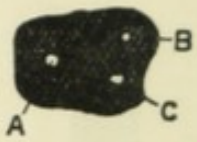

FIG. 82.

Fig. 80.—Longitudinal section through the palatine and one buccal root of a third upper molar.

Fig. 81.—Longitudinal section through the two buccal roots of a third upper molar.

Fig. 82.—Transverse section through a left upper molar showing (A) the palatine canal; (B) the anterior buccal canal; (C) the posterior buccal canal.

FIG. 83.

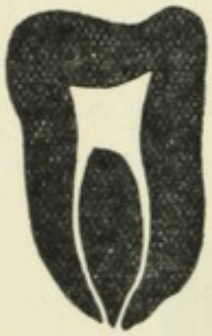

FIG. 84.

FIG. 85.

FIG. 86.

FIG. 87.

Fig. 83.—Longitudinal section through the anterior and posterior roots of a first or second lower molar.

Fig. 84.—Longitudinal section through the anterior root of a first or second lower molar, showing how it is often penetrated by two canals.

Fig. 85.—Longitudinal section through the anterior root of a first or second lower molar, showing a rare form of division.

Fig. 86.—One form of root canal of the third lower molar.

Fig. 87.—Another form of root canal of the third lower molar.

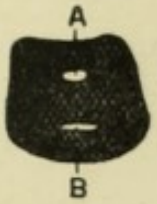

FIG. 88.

FIG. 89.

Fig. 88.—Transverse section through a lower molar, showing the usual two canals; A, the posterior canal; B, a constricted anterior canal.

Fig. 89.—Transverse section through a lower molar showing: A, the posterior canal; B,B, two anterior canals.

In connection with the first lower molar there is often a postero-internal third root.

The *condition* of root canals requiring treatment may be either—

(*a*) Septic (usual), or

(*b*) Aseptic. (For treatment of these, see under *Extirpation of the Pulp*.)

The treatment of *septic* root canals consists in rendering them *aseptic* and keeping them so.

Occasionally on opening up teeth containing septic pulps violent septic inflammation, accompanied by severe pain and swelling, results, due to the entrance of pathogenic micro-organisms from the air or the saliva. Strict antiseptic precautions must, therefore, always be adopted during treatment.

The author has adopted the following *treatment of septic roots* with much success. On the first visit—

(i) Apply the rubber dam.

(ii) Dry the cavity.

(iii) Open up into the pulp chamber in a direct line with the axis of the tooth, using a spear-pointed drill, and enlarge with a cross cut fissure bur. Leave the canals severely alone, and so avoid forcing septic matter through the apices of the roots.

(iv) Wash the *débris* away with warm water.

(v) Dry the cavity.

(vi) Insert into the pulp chamber a pledget of cotton-wool soaked in tricreso-formalin.

(vii) Seal in with calxine, and leave for twenty-four hours.

NOTE.—*Tricreso-formalin* is a refined mixture of meta-, ortho-, and para-cresols combined with formalin, which is a 40 per cent. solution of formaldehyde in water. Its action on septic pulps is two-fold.

(*a*) It acts as a powerful disinfectant, killing the various germs present.

(*b*) It acts chemically. The formaldehyde gas which is evolved unites with the gaseous by-products of putrefaction, rendering them inert. It combines with the ammonia, producing an antiseptic substance, *urotropin*, and splits up the hydrogen sulphide into sulphur and alcohol. The tricresol changes the fatty constituents into another antiseptic, *lysol*. It is absolutely essential to success that the solution should be fresh, that it should be used in sufficient quantity, and that it should be hermetically sealed in the cavity.

On the second visit—

(i) Apply the rubber dam.

(ii) Remove the calxine and the dressing.

(iii) Wash out with a 3 per cent. solution of carbolic acid.

(iv) Clean out the canals with a barbed broach.

(v) Dry the cavity and canals.

(vi) Insert on a smooth broach into each canal a wisp of cotton-wool soaked in tricreso-formalin. Care should be taken not to force any through the apices, as it is an irritant.

(vii) Seal with calxine and leave for seven days. Sometimes severe pain results from the use of tricreso-formalin. This is so if any live pulp remains. In such cases remove the dressing, and neutralize the drug with ammonia water.

On the third visit—

(i) Apply the rubber dam.

(ii) Remove the calxine and the dressing.

(iii) Insert on a smooth broach into each canal a wisp of cotton-wool soaked in peroxide of hydrogen.

Should there be any evolution of oxygen, which is detected by a bubbling or frothing, renew the tricreso-formalin dressings, seal with calxine, and wait for a day or two. Should, however, the canals be aseptic

(iv) Enlarge them if advisable.

The process, however, is rarely necessary, and is not recommended by the author except in certain cases of crowning, where it is essential

FIG. 90.

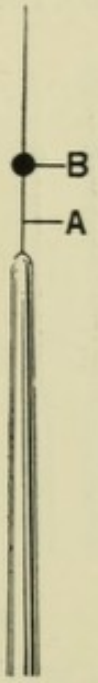

FIG. 91.

A, smooth broach for measuring length of canal; B, movable rubber disc for marking length of canal.

to use the canal for a pin or post, and in cases where the dentine has been impregnated and softened with septic material.

Very small and constricted canals may be left unfilled without harm.

Should enlarging, however, be decided upon, the process may be carried out by opening up the mouth of the canal with a special drill (fig. 90), measuring its length with a fine

smooth broach supplied with a small movable rubber disc for the purpose (fig. 91), and then carefully and without force using drills of various sizes, such as *Beutelrock's* (fig. 92), or *Gates Glidden's* (fig. 93), frequently removing them to free the *débris*, and noting particularly the length of the canal on the drill, whilst operating, by means of the movable rubber disc. Another method of enlarging is to introduce into the canal on a fine platino-iridium

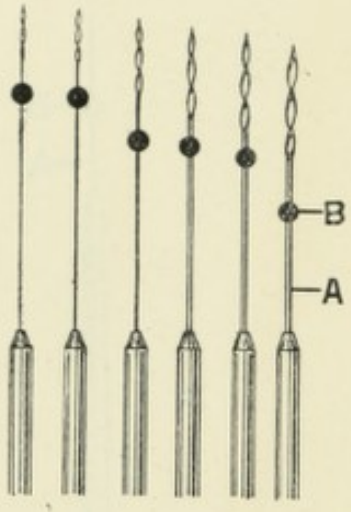

FIG. 92.

A, Beutelrock drill.
B, movable rubber disc.

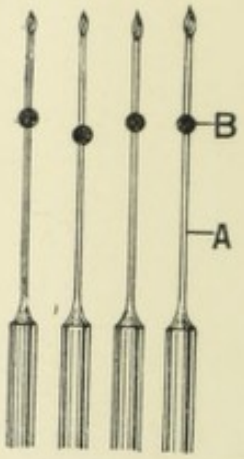

FIG. 93.

A, Gates Glidden drill.
B, movable rubber disc.

instrument a drop of a 25 to 50 per cent. solution of hydrochloric or sulphuric acid, saturate with a solution of sodium bicarbonate to neutralize, and then remove the softened portions of dentine with a pulp broach.

Sometimes a drill or broach is accidentally broken in a canal. It may be removed with special instruments such as those depicted in figs. 94 and 95.

Should these special instruments fail to effect

the desired result, the broken portion may be rusted out by introducing into the canal a dressing of tincture of iodine, sealing it in, and

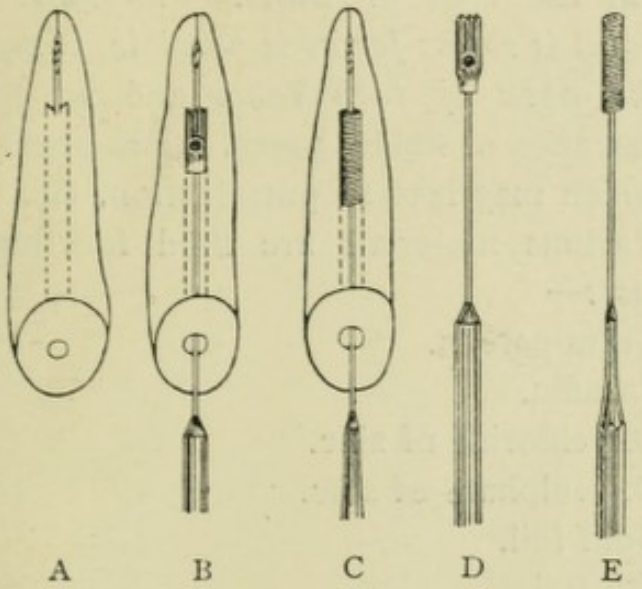

FIG. 94.

Beutelrock's Drill Extractors. (These are made in various sizes).

A, broken drill in the root of a tooth; B, instrument D enlarging canal; C, instrument E grasping drill; D, reamer for enlarging canal where necessary; E, spiral spring for grasping drill.

The reamer is rotated in the engine in the reverse way to the usual movement, whilst the spring is attached by rotating it in the ordinary way, at the same time using gentle pressure.

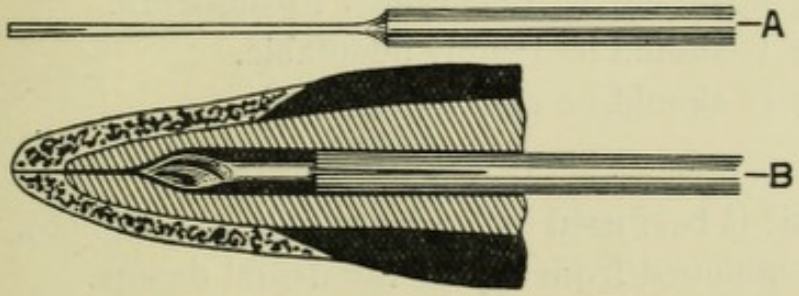

FIG. 95.

A, drill extractor having a split and threaded cone socket. Enlarge the canal if necessary, and gently push and turn the instrument into it until it grasps the drill and effects its removal; B, Drill extractor in canal grasping the broken instrument.

leaving it for some days. This treatment should be repeated until it is possible to remove the broken drill.

(v) Wash out the canals and cavity with a 3 per cent. solution of carbolic acid, and dry with alcohol and hot air.

(vi) Fill the root or roots. *The most important point is to, as far as is possible, completely seal the apex of each canal* and so prevent the entrance of fluids, gases, micro-organisms, &c., which may lead to putrefaction, &c.

Various materials are used for this purpose, *viz.*:—

Gutta-percha.
Paraffin.
Oxychloride of zinc.
Oxysulphate of zinc.
Gold foil.
Tin foil.
Hickory wood points.
Cotton-wool, &c.

The *essentials of a permanent root filling* are:—

(*a*) It should remain unchanged.
(*b*) It should be non-irritating.
(*c*) It should be impervious to moisture.
(*d*) It should be readily adaptable.
(*e*) It should be easy of removal.

Gutta-percha.

This is best used in the form of cones (fig. 96), which can be obtained from any of the dental depôts.

Process:—

(*a*) Measure the length of the canal and choose a suitable cone.

(*b*) Insert it in the canal, and should the point penetrate the apex, which would produce a slight twinge of pain, cut it off on removal.

(*c*) Moisten the canal with eucalyptol (which is less irritating than eucalyptus) and menthol.

(*d*) Introduce carefully a solution of chloro-percha into the canal up to the apex by means of a wisp of cotton-wool on a broach.

(*e*) Introduce the gutta-percha cone into the canal, and if necessary insert other cones alongside.

(*f*) Place a piece of base plate rubber over the cones, and by means of a round burnisher condense.

(*g*) Remove the base plate rubber, and seal in the root fillings with oxyphosphate of zinc.

(*h*) Fill the tooth.

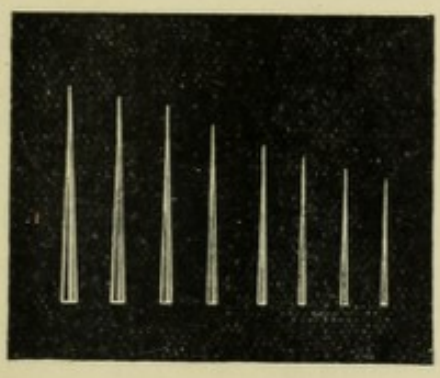

FIG. 96.

FIG. 97.

Gutta-percha cones are manufactured with strands of silk, or silver wires having loops (fig. 97) running through them, to facilitate their removal should it be necessary.

Another method of filling with rubber is to:—

(*a*) Moisten the canal with eucalyptol and menthol.

(*b*) Introduce chloro-percha into the canals.

(*c*) Pack very small pieces of base plate rubber into the canals.

(*d*) Place a large piece over these.

(*e*) Place a plug of cotton-wool over this.

(*f*) Press gradually upon this with a burnisher.

(*g*) Remove the cotton-wool and the large piece of base plate rubber.

(*h*) Seal the root-canals with oxyphosphate of zinc.

(*i*) Fill the cavity.

The *advantages* of gutta-percha as a permanent root filling are:—

It is non-irritating.

It is easily adapted.

It can be easily removed by softening with chloroform and using a barbed broach.

It is unchangeable if the canal has been rendered thoroughly aseptic.

Paraffin.

This must have a melting-point of not less than 135° F. If necessary it can be readily removed with hot air.

Process:—

(*a*) Place a piece of paraffin in the cavity over the canal.

(*b*) Pass a fine heated broach through the paraffin into the canal and repeat this on several occasions when the paraffin will have found its way into and have filled it.

Oxychloride of Zinc.

Process:—

A thin mixture is made and gradually and gently worked into the canal by means of a wisp of cotton-wool on a broach.

Its *disadvantages* are that it is an irritant, and care must be taken not to force it through the apex.

It is difficult of removal, but this may be effected by dissolving out with a 25 to 50 per cent. solution of sulphuric acid.

Its *advantages* are:—

It becomes very hard.

It remains antiseptic for a long time, due to the excess of zinc chloride contained in it.

It has a coagulating and mummifying effect on any remnants of pulp tissue in the canal.

It prevents decomposition.

Oxysulphate of Zinc.

This is not so irritating as the oxychloride. *Colyer* mentions the following as a good root filling: —

℞ Oxide of zinc, Sulphate of zinc, Loretin — āā 3ii.

Process.—As with oxychloride of zinc.

Gold and Tin Foil.

The great *disadvantages* of these materials are that they are very difficult to adapt properly or to remove.

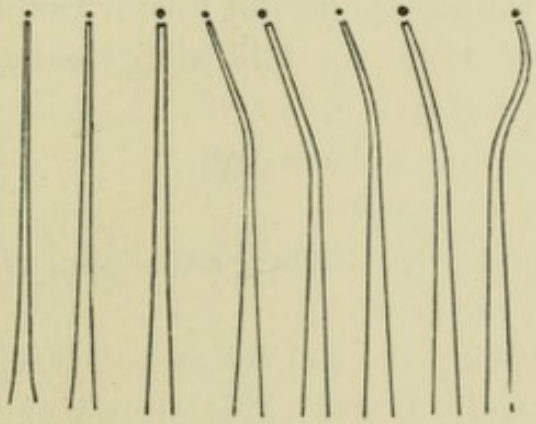

Fig. 98.

Process.—Narrow strips of the metal are introduced on special pluggers (fig. 98) into the canal, until the apex is hermetically sealed.

Hickory Wood Points.

These can be obtained from any dental depôt. They are difficult of removal.

Process:—

(*a*) Choose and adapt the point as with the rubber cones.

(*b*) Notch the point to mark the length required and to assist in breaking it in the canal.

(*c*) Pump into the canal a little oxychloride or oxysulphate of zinc.

(*d*) Insert the wood point and gradually and gently force it into position.

(*e*) Twist the point thus breaking it off and leaving it in the canal.

Cotton-wool.

This should never be used, except in connection with oxychloride of zinc, oxysulphate of zinc, or some similar root filling, as it becomes foul and septic.

As there is no known drug which can be hermetically sealed in a tooth having a putrescent pulp (except formaldehyde preparations) without the likelihood of pain supervening, those who do not favour the use of tricresoformalin should adopt the following treatment:—

On the first visit—

(i) Apply the rubber dam.

(ii) Dry the cavity.

(iii) Flood with a strong antiseptic, such as peroxide of hydrogen.

(iv) Open up the pulp-chamber, leaving the canals severely alone. This allows of the escape of gases and prevents the forcing of septic matter through the apices.

(v) Insert a loose cotton-wool dressing.

On the second visit—

(i) Apply the rubber dam.

(ii) Remove all carious tissue.

(iii) Prepare the cavity.

(iv) Wash out with warm water.

(v) Swab out the cavity with peroxide of hydrogen.

(vi) Clean out and enlarge the canals, and to prevent the passage of septic matter through the

apices *Colyer's* advice should be followed, "to proceed gradually from the orifices of the canals to the apices, frequently introducing sodium peroxide followed by peroxide of hydrogen to sterilize the pulp tissue."

(vii) Carefully introduce, into the canals, chloroform to remove the grease.

(viii) Dry thoroughly with hot air.

(ix) Treat each canal by carefully inserting (on several occasions) on a smooth broach and rotating it, a wisp of cotton-wool soaked in a drug which has germicidal, antiseptic and disinfectant properties, such as *mercuric chloride, creosote, oil of cloves, oil of cinnamon, oil of eucalyptus, carbolic acid, peroxide of hydrogen, sodium dioxide, &c.*, and then leaving it in.

NOTE.—Such drugs act by—

(*a*) Destroying pathogenic and saprophytic micro-organisms.

(*b*) Rendering inert or harmless their waste products, or

(*c*) Preventing the development of their spores.

Of the above agents, creosote, carbolic acid and mercuric chloride are *coagulants*, which according to some writers do not become diffused through the dentine on account of their coagulating effect upon albumin, and therefore should not be used in root treatment. Other writers, however, are not in agreement with this.

Creosote.—Use pure with an equal quantity of formalin.

Mercuric Chloride.—Use a 1—500 solution in alcohol. It was originally used in *immediate root treatment.* It stains the tissues.

Oil of Cinnamon.—It stains the tissues.

Oil of Eucalyptus.—Use in combination with iodol. It is a solvent of gutta-percha and should therefore be sealed in with calxine.

Carbolic Acid.—Use a 1—40 solution.

Peroxide of Hydrogen (20 vols.)—When in contact with putrescent matter oxygen is liberated, and this is detected by a bubbling. The liberated oxygen acts as the germicide.

Sodium Dioxide.—This is perhaps the best drug of those mentioned for the purpose of sterilizing root canals. It is introduced on asbestos fibre which has been dipped in alcohol. Oxygen is liberated whilst putrescent matter is present, and this gas acts as the germicide. Syringe with warm water and neutralize with a 10 per cent. solution of sulphuric acid.

(x) Dry the cavity thoroughly.

(xi) Seal with calxine.

On the third visit—

(i) Apply the rubber dam.

(ii) Remove the calxine and dressings.

(iii) If aseptic, fill the roots and cavity. Otherwise re-treat the canals and leave filling for a future occasion.

The *immediate root-filling process* of *septic canals* is not recommended by the author, but should it ever become necessary, proceed as follows:—

(i) Apply the rubber dam.

(ii) Remove carious tissue.

(iii) Prepare the cavity.

(iv) Wash out with warm water.

(v) Swab out the cavity with peroxide of hydrogen.

(vi) Open up the pulp-chamber and canals, taking the usual precautions.

(vii) Introduce chloroform into the canals to remove any grease.

(viii) Dry thoroughly with hot air.

(ix) Treat the canals with a 1—500 solution of mercuric chloride.

(x) When considered aseptic fill the roots and tooth.

CHAPTER IX.

DISEASES OF THE ALVEOLAR DENTAL PERIOSTEUM.

Pericementitis or Periodontitis.

Definition.—Inflammation of the alveolar dental periosteum.

Varieties:—

(*a*) Local.

(i) Acute.

(ii) Chronic.

(*b*) General.

(i) Acute.

(ii) Chronic.

(*a*) Local.

(i) *Local acute periodontitis:*—

Definition.—Inflammation of the alveolar dental periosteum of the root or roots of one tooth.

Causes:—

(i) Spread of inflammation from the pulp.

(ii) Passage of toxic products of *pyogenic organisms viâ* the apices of roots.

Note.—The above are the two most common causes.

(iii) Passage of toxic products *viâ* the gingival margin, gum, blood, or dentine.

(iv) Traumatism, *e.g.*, too much force and strain in regulating and separating teeth, in fitting and fixing bridges, and in malleting gold when filling.

Ill-fitting bands, dentures, and crowns.

Accidental perforation of a root-canal with an instrument.

Too vigorous use of a tooth-brush or floss silk.

Injury from a fish-bone, &c.

(v) Injudicious use of drugs, such as arsenic, carbolic acid, formalin, &c.

(vi) Calculus.

(vii) *Débris* of food wedged between teeth.

(viii) Badly finished fillings having rough and overhanging cervical margins.

(ix) Imperfect root filling.

(x) Presence of root filling beyond apical foramen.

(xi) Impacted lower third molar.

(xii) Cold.

Signs and Symptoms.—They are less severe in the traumatic than in the septic variety.

They take longer to develop in debilitated than in healthy subjects.

They are, at first:—

Discomfort and feeling of tension, due to an excess of blood in the part.

Tooth slightly loose.

Desire to grind the teeth together.

Pressure upon the tooth gives relief, due to the tension being relieved, the excess of blood being forced out of the vessels.

Red ring at margin of gums.

Then:—

Tooth still further raised, due to the inflammatory infiltration thickening the membrane.

Discomfort and feeling of tension increased.

Gums swollen, tender, and red or purple.

Pain deep, dull, and constant.

Pressure upon tooth causes pain, due to advancing stasis rendering it more difficult to relieve the tension.

Then later:—

Pressure upon tooth causes excruciating pain, due to increased tension through the thrombosed condition of the vessels and the impossibility of forcing away the excess of blood.

Throbbing and boring pain.

Difficulty in eating.

In some cases feverish symptoms.

And finally:—

Tooth becomes very loose.

Swelling becomes more defined, palpation producing fluctuation, due to the presence of pus (*acute dento-alveolar abscess*).

Pus increases and burrows through the bone.

Lymphatic glands in the submaxillary region may be enlarged.

Sometimes severe feverish symptoms.

Pus escapes into soft tissues.

Swelling increases greatly.

Pus perforates soft tissues and points.

Pain abates.

Swelling decreases.

Pathology.—As in inflammation (see p. 146).

There is:—

(i) Momentary *constriction* of the blood-vessels, then

(ii) Prolonged *dilatation*, then

(iii) *Acceleration* of the blood-flow, then

(iv) *Slackening* of the blood-flow, then

(v) *Oscillation*, then

(vi) *Stasis*. If the irritation is removed *resolution* can take place, otherwise

(vii) *Thrombosis* occurs, when *resolution* is impossible.

(viii) Leucocytes and embryonic cells break down into pus corpuscles.

(ix) Pus forms, through the digestive and peptonizing actions of *pyogenic organisms* upon the intracellular substance of the tissues.

(x) Pus strips the membrane from the root.

(xi) Through pressure the pus makes a cavity in and burrows through the bone in the direction of least resistance.

(xii) Pus escapes into the soft tissues and points.

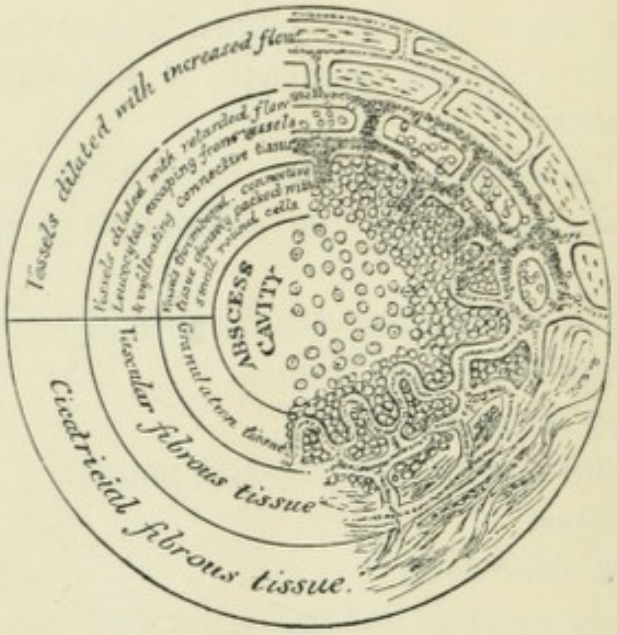

FIG. 99.

Streptococci, staphylococci, and sometimes *spirilla* and *spirochætes* are the organisms found in pus.

There is *no sac* with an acute dento-alveolar abscess.

The diagram (fig. 99) borrowed from *Walsham's* "Theory and Practice of Surgery," Sixth Edition, by permission of the publishers, *Messrs. J. and A. Churchill,* is a diagrammatic representation of the minute changes in the formation and healing of an abscess. The upper

half shows abscess enlarging; the lower half abscess healing.

NOTE.—The pus usually penetrates and discharges through the thin outer alveolar wall over the root of the affected tooth.
It may exude at the gingival margin.
It may discharge into the pulp canal.
It sometimes burrows along the fascia and muscles.
In connection with :—
The *maxillary incisors*, it may drain into the nasal fossæ.
The *maxillary lateral incisors*, it may burrow along the muco-periosteum of the hard palate and form a swelling at the junction of the hard and soft palates.
The *maxillary canine or cuspid*, it may point at the inner canthus of the eye.
The *maxillary first premolar*, it may point at the angle of the mouth.
The *maxillary second premolar*, it may burrow along the muco-periosteum of the hard palate and form a swelling at the junction of the hard and soft palates.
Any of the maxillary cheek teeth, it may penetrate the antrum of Highmore.
The *mandibular incisors*, it may penetrate under the chin.
The *mandibular canines*, it may penetrate under the chin.
The *third lower molar*, it may penetrate at the angle of the jaw.
Any of the mandibular teeth, it may penetrate the floor of the mouth, the cheek, between the layers of the deep cervical fascia, producing *angina Ludovici*, or under the chin.

Acute dento-alveolar abscess may occur in connection with living pulps, when it is possible that the organisms have gained access through the blood.

Treatment :—

(*a*) Radical (extraction).
(*b*) Conservative.
(1) Local.
(2) General.

(*a*) Radical—
Extraction should be carried out if pus has formed and has invaded the nasal fossa, the antrum of Highmore, or the fascia or muscles.

(*b*) Conservative—
(1) Local treatment.

In the early stages before suppuration has set in, and if not due to a septic pulp : —

(i) Remove cause, such as ill-fitting crown, denture, tartar, &c.

(ii) Apply a counter irritant to the gums, such as

℞ Tincture of aconite
,, iodine
Spirits of chloroform
,, camphor
āā fl. ʒi. Misce.

or capsicum plasters.

(iii) Scarification of the gums may prove useful should counter irritants be ineffective.

If due to a septic pulp—

(i) Support the tooth by moulding around it and between it and the adjacent teeth modelling composition, or a mixture of cotton-wool and ~~artificial dentine~~ cement.

(ii) Open up into the pulp chamber in a direct line with the axis of the tooth.

(iii) Apply a counter irritant to the gums over the affected tooth.

(iv) Relieve the bite by placing a temporary cap on a tooth on the opposite side or drying the crown surfaces of the teeth on the opposite side and flowing oxyphosphate of zinc over them.

(v) Send patient away for forty-eight hours.

(vi) Extirpate the pulp.

(vii) Treat the root or roots (see p. 119).

If suppuration has set in—

Get rid of the pus as soon as possible, and, where practicable, as, for example, in a single rooted tooth, to *save time, pain, and tissue*—

(i) Have an anæsthetic administered.

(ii) Make a triangular flap of gum over the affected root, with the apex directed towards the crown of the tooth (fig. 100).

(iii) Turn the flap of gum back from the bone.
(iv) Stop the hæmorrhage.
(v) Scrape away the periosteum beneath.

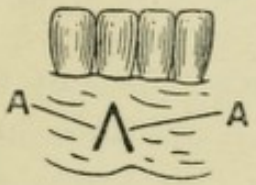

FIG. 100.

A, A, two incisions to form a triangular flap of gum.

(vi) Trephine, with a sterilized instrument, through the alveolar plate at the apical space (fig. 101).

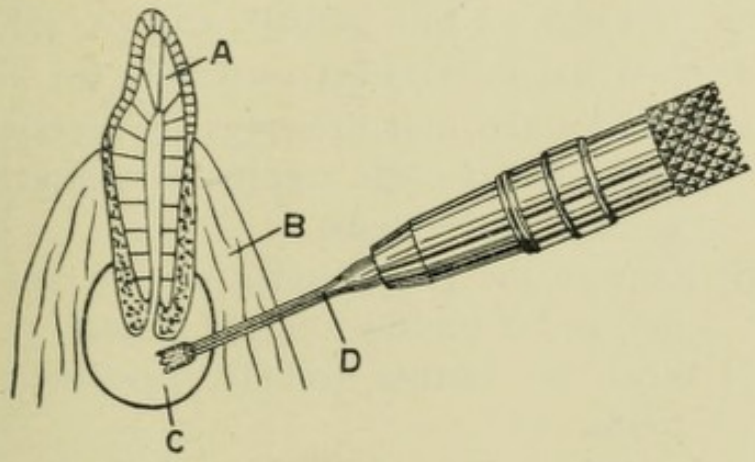

FIG. 101.

A, affected tooth; B, alveolus; C, collection of pus; D, trephine.

On the recovery of the patient from the anæsthetic—

(vii) Evacuate the pus and syringe out the opening with a warm antiseptic solution, such as carbolic acid (1—40).
(viii) Open up the root canal or canals.
(ix) Extirpate the pulp and thoroughly cleanse canals. by washing through sinus with antiseptic solution

(x) Fill each canal with an antiseptic, such as creosote or hydrogen peroxide solution. This may be effected with a hypodermic syringe, or by introducing the drug on wisps of cotton-wool.

(xi) Pack a large piece of base plate rubber into the cavity and press upon it with a tapping motion. This forces the drug through the apex into the external opening.

(xii) Pack each canal with a wisp of cotton-wool soaked in the drug used, and seal the cavity with calxine.

(xiii) Pack the external opening with an antiseptic gauze and leave for some hours.

(xiv) Remove the gauze from the external opening and syringe with carbolic acid (1—40).

NOTE.—This should be repeated several times a day.

(xv) Remove the root dressings, if necessary retreat, but if roots aseptic, fill permanently, and seal with calxine.

(xvi) Remove the gauze from the external opening, and if healthy allow it to heal.

(xvii) When no further trouble, permanently fill tooth.

If it is decided not to trephine through the bone—

(i) Promote the formation of pus and pointing by applying inside the mouth on the affected part hot fomentations such as figs, bread, &c.

(ii) Should the external skin be involved, protect it by applying a layer of cotton-wool painted over with collodion on the surface to be applied, but if it is impossible to prevent the pus pointing externally, it is wise to evacuate it by means of an incision made parallel to the fibres of the platysma.

(iii) When the pus has pointed, treat as in after trephining.

Should it be impossible to seal up the tooth without further trouble ensuing—

(i) Treat the roots as thoroughly as possible.

(ii) Fill the roots. In this case it is advisable to loosely pack crushed thymol into the canals.

(iii) Fill the tooth.

(iv) Make an oblique perforation into each canal beneath the gum (*Rhizodontrophy*). These are left open permanently, and act as vents for the escape of any gases that may form.

(2) General treatment:—

(i) Give a brisk purge.

(ii) Administer one of the following in 5 to 10 gr. doses:—

Quinine.
Phenacetin.
Antikamnia.

Sequelæ.—Local chronic periodontitis.
Empyæmia antri.
Necrosis.
Closure of the jaws (*trismus*).
Affected glands.
Septicæmia.
Pyæmia.
Angina Ludovici.

The symptoms of *angina Ludovici, streptococcal pharyngitis*, or *Ludwig's angina*, which is a very serious disease, often terminating in death, are:—

Rise in temperature.
Rigors.
Prostration.
Congested, œdematous, and swollen pharynx.
Floor of mouth swollen and pushed up, giving the appearance of a second tongue.

Œdema of larynx may supervene.

The parotid region, the orbit, or the thorax may be involved.

Suppuration spreads, and may cause death from septic bronchial pneumonia, pleurisy, and pericarditis.

The symptoms should be recognized immediately, and a surgeon should be called in without delay.

(ii) *Local chronic periodontitis.*

Definition.—An inflammation of the alveolar dental periosteum acting with less intensity and over a longer period than the acute.

Site.—(A) Near the apex of the root.

(1) *Productive or proliferative periodontitis* or *exostosis.*

(2) *Rarefying periodontitis or absorption.*

(3) *Chronic suppurative periodontitis without a sinus (chronic dento-alveolar abscess).*

(4) *Chronic suppurative periodontitis* or *chronic dento-alveolar abscess accompanied by a sinus (gumboil).*

(B) At the gingival margin.

(C) Affecting the whole root. [Either (A) or (B) may result in (C).]

(A) Near the apex of the root.

(1) *Productive or proliferative periodontitis or exostosis* (fig. 102).

Definition.—A pathological overgrowth of the cementum of a tooth.

Causes.—As in local acute periodontitis, the irritation, however, being slight but constant, *e.g.*—

Erosion.

Over-use of tooth.

Thermal changes.
Death of pulp.
Sometimes constitutional, the children of parents who have suffered from the disease being affected in the same way.

Pathology.—Proliferation of connective tissue.
Alveolar dental periosteum thickened.
Blood-vessels slightly enlarged.
Lacunæ of new cemental tissue are numerous, large, and coarse. The more rapid the formation the larger the lacunæ and the more numerous the canaliculi.

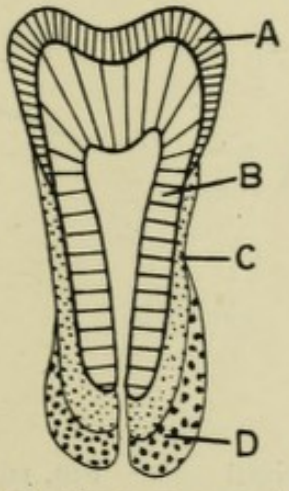

FIG. 102.—A, enamel.; B, dentine; C, cementum; D, exostosed tissue.

The incremental lines of *Salter* are well marked.
Sharpey's fibres are often well marked.
Vascular canals are sometimes present in the new cemental tissue.
Pathological gemination sometimes occurs.
There is absorption of the alveolus (*rarefying osteitis*) to make room for the new tissue. This is depicted in figs. 103 and 104, borrowed from *Colyer's* "Dental Surgery and Pathology" by permission of *Messrs. Longmans, Green and Co.*, the publishers.

Anchylosis between the tooth and the bone rarely occurs.

The new cemental tissue is smooth and regular if the formation is continuous. If formation periodic it is irregular.

Symptoms.—Sometimes none.

Usually there is nothing but neuralgic pain.

The following may sometimes be present:—

Tooth extruded.

Tooth loose.

Tooth tender to pressure.

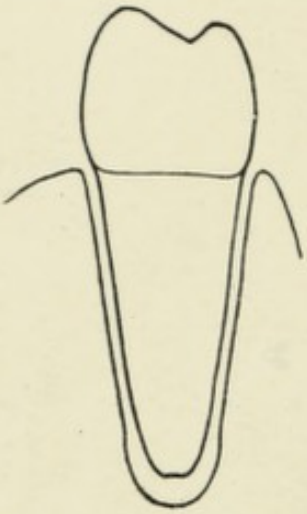

FIG. 103.—Showing normal relation of tooth to the alveolar process.

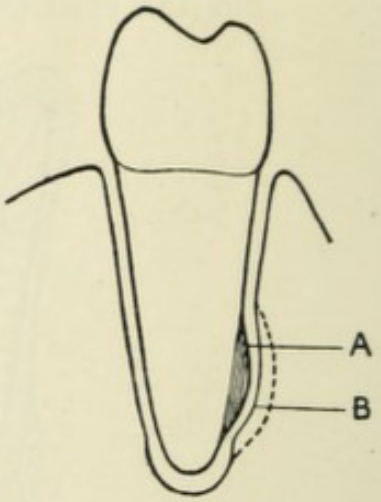

FIG. 104.—Showing changes which occur in productive periodontitis: A, new tissue; B, zone of rarefying osteitis.

Dull gnawing pain.

Absorption of gum.

With a vital pulp thermal changes cause pain.

Treatment.—Clear up, if doubtful, by means of X-rays.

Remove irritant.

Keep mouth thoroughly clean.

Apply counter irritants to the gum.

Should these means fail, extract. Even after extraction the pain does not subside for some time.

(2) *Rarefying periodontitis or absorption* (fig. 105)—

Definition.—An inflammation of the alveolar dental periosteum leading to a destruction of the dental tissues of the root or roots of teeth.

Causes.—As in local acute periodontitis, the irritation being comparatively severe, *e.g.*—

Presence of root filling beyond apical foramen.
Death of pulp.
Necrosis of alveolus.
Pressure from a displaced erupting tooth.
Loss of function (due to having no antagonist).

The process is much more rapid in pulpless teeth.

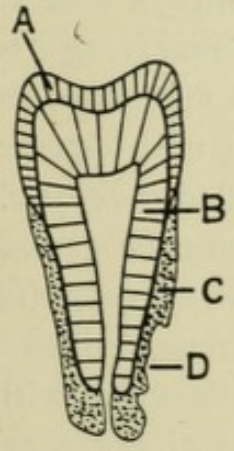

FIG. 105.—A, enamel; B, dentine; C, cementum; D, zone of destruction of cementum (*absorpion*).

Pathology.—Cell infiltration.
Cell proliferation.
Presence of multi-nucleated cells (*osteoclasts*), which absorb tooth structure.
Presence of cup-shaped depressions (*Howship's lacunæ*) in the hard tissue. Each of these contains an *osteoclast*.
Presence of cells similar to those found during the absorption of deciduous teeth.

Cementum goes first, then dentine. The portion of dentine in close proximity to the pulp is very resistant, and is attacked with difficulty.

Symptoms.—Discomfort.

Tenderness on percussion.

Looseness of tooth.

Pain to thermal changes if pulp vital.

Sometimes discoloration.

Sometimes no symptoms.

Treatment.—Clear up, if doubtful, by means of X-rays.

Remove cause.

Counter irritants.

It is nearly always wise to extract.

The processes of *rarefying periodontitis* and *productive periodontitis* may alternate, the cavity formed by the former being filled in with cemental tissue by the latter (fig. 106). This is called *inostosis*.

(3) *Chronic suppurative periodontitis without a sinus* (*chronic dento-alveolar abscess*) (fig. 107).

Definition.—A circumscribed collection of pus, due to inflammation, in connection with the root of a tooth.

Causes.—As in *local acute periodontitis*.

Symptoms.—Sometimes very little sign.

Often slight pain on pressure.

Gums often swollen and congested.

There is usually a sensitive swelling over the apex of the root of the tooth.

Pathology.—As in *acute dento-alveolar abscess*.

Pyogenic organisms often die after starting the trouble, and the pus is sterile.

Presence of abscess sac (fig. 107c), which is usually pyriform.

Walls of sac thick and fibrous, the fibres being longitudinal.

Inner lining is granulation tissue.

Treatment.—This may be carried out by—

(i) Applying rubber dam.
(ii) Opening up the canal.
(iii) Cleansing out canal very carefully with a barbed broach sufficiently to allow of drainage through canal.
(iv) Washing out with warm water.
(v) Treating with antiseptics every twenty-four hours until cured.

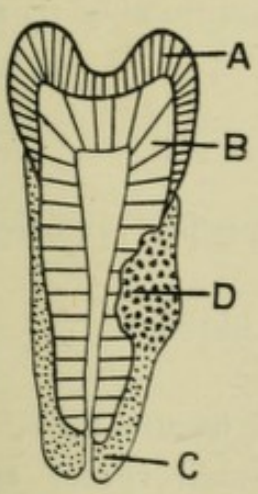

FIG. 106.—A, enamel; B. dentine; C, cementum.; D, cavity produced by *rarefying periodontitis* filled in with cemental tissue.

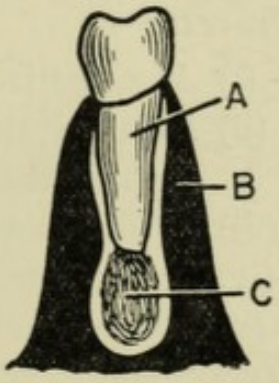

FIG. 107.—A, abscessed root; B, alveolus; C, abscess sac.

To cure completely, however—

(i) Cleanse mouth.
(ii) Treat the root canals, removing any decomposing tissue present by enlarging.
(iii) Fill the root canals when aseptic.
(iv) Temporarily fill the tooth.
(v) Anæsthetize the patient.
(vi) Trephine through the alveolar plate over the apex of the affected root as in acute dento-alveolar abscess (see page 141 and figs. 100 and 101).

(vii) Enlarge the opening.

(viii) Thoroughly curette the surface of the abscess cavity with a spoon excavator.

(ix) Wash away *débris* and blood.

(x) Syringe out with an antiseptic solution, such as 1-40 carbolic acid, and pack with an antiseptic gauze. This should be repeated until healing has taken place by granulation.

(xi) Permanently fill the tooth.

In obstinate cases, due to absorption, necrosis, or exostosis of the apex of the root roughening it, *excision of the apex* may be resorted to.

Process:—

(i) Anæsthetize the patient.

(ii) Enlarge the external opening sufficiently to expose the apex.

(iii) Syringe away *débris* and blood.

(iv) Stop hæmorrhage.

(v) Excise apex with a small fissure burr.

(vi) Smooth edges with a small round stone or finishing burr.

(vii) Syringe cavity with 1-40 carbolic acid.

(viii) Pack with antiseptic gauze and allow to heal by granulation.

Should the foregoing measures fail, *replantation*, which will be dealt with in a later chapter, or *extraction* may be resorted to.

(4) *Chronic suppurative periodontitis or chronic dento-alveolar abscess accompanied by a sinus* (fig. 108)—

Definition.—A circumscribed collection of pus, due to inflammation, communicating with a track having an external opening.

Causes.—As in *local acute periodontitis.*

Symptoms.—Presence of *gumboil* (fig. 108E), usually over the apex of the affected root.

Usually no pain if there is a free opening.

May be slight pain on percussion.

Swelling limited.

Gums swollen.

Pathology.—As in *chronic suppurative periodontitis without a sinus.*

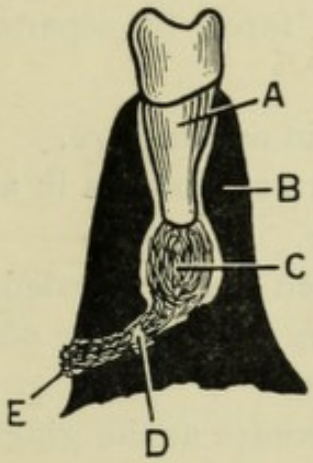

FIG. 108.—A, abscessed root; B, alveolus; C, abscess sac; D, sinus, connected with abscess sac; E, external opening or termination of sinus (*gumboil*).

Treatment:—

(i) Cleanse the mouth.

(ii) Treat the root canal, removing any decomposing tissue present by enlarging.

(iii) Pump some antiseptic, such as 1-40 carbolic acid, through the root canal into the sinus.

(iv) Fill the root canal when aseptic.

(v) Temporarily fill the tooth.

(vi) Anæsthetize the patient, enlarge the external opening, and then treat as in chronic suppurative periodontitis without a sinus after trephining.

Should the sinus be obstinate to treatment stimulate its walls by scraping, or painting them with a 10 per cent. solution of silver nitrate, a 1 per cent. solution of zinc chloride, or pure carbolic acid.

(B) At the gingival margin:—

Causes.—Ligatures.
Bands.
Salivary calculus.
Débris of food wedged between teeth.
Too much force in separating or regulating teeth.

Symptoms.—Pain on pressure.
Pain to thermal changes in a vital tooth.
Tooth loose.
Gums swollen and congested.
Tags of gum between the affected tooth and its neighbours.
Pus often exudes at the gingival margin.

Pathology.—As in inflammation.
Ulceration and destruction of portions of the membrane.
Inflammation may spread to pulp.

Treatment.—Remove cause.
Thoroughly cleanse mouth.
Syringe pockets with an antiseptic, such as peroxide of hydrogen.
Touch ulcerated surfaces with an escharotic, such as silver nitrate.

Sequelæ of local chronic periodontitis:—
(i) Necrosis of cementum.
(ii) Granuloma.
(iii) Dental cyst.
(iv) Anchylosis.

(i) Necrosis of Cementum.

Definition.—Death of cementum.

Cause.—Cutting off of blood supply following local chronic periodontitis.

Varieties:—

(*a*) Partial (common).

(*b*) Complete (rare).

Site:—

Usually at apex.

May be at any part of root.

Sometimes whole root affected.

Sometimes one root only of a multiple-rooted tooth affected.

Pathology.—Inflammation leading to *destruction* of the alveolar dental periosteum.

Symptoms.—History of periodontitis.

Absence (usually) of a vital pulp, when the tooth has a dull appearance.

If partial:—

Tooth usually loose.

Tooth painful to pressure.

Pus may exude at the gingival margin.

Gums may recede.

If pulp alive there is intermittent pain and thermal changes cause pain.

One root of a multiple-rooted tooth may be affected and bared of gum and bone. In this case the branch of the pulp in the necrosed root may be dead, whilst the branches in the other roots remain alive.

If complete:—

Gradual expulsion or exfoliation of tooth.

Inflammation, tenderness, and swelling of gums.

Affected tissues dark in colour.

Suppuration from gingival margin.

If gums have receded, symptoms are not so severe.

Treatment.—If necrosis is feared, treat as for periodontitis.

If it has occurred and is at the apex, excision of the apex may be resorted to (see page 150).

Should it have occurred on any other part of the root, *extraction* and *replantation* may be carried out.

Should one root only of a mutiple-rooted tooth be affected, the pulp of the tooth may be extirpated, the healthy roots treated and filled, and the necrosed root excised with a fine fissure burr.

When the disease has attacked the whole of the root or roots, extraction is advisable.

(ii) Granuloma (fig. 109).

Definition.—A new growth consisting of granulation tissue springing from the alveolar dental periosteum of the root or roots of teeth.

Causes.—Those of local chronic periodontitis.

Varieties.—Oscar Römer describes two:—

(*a*) Epithelial (more common).

(*b*) Endothelial or myxomatous.

Microscopical appearances:—

(*a*) Epithelial.

Granulation tissue.

A few epithelial cells.

(*b*) Endothelial.

Granulation tissue.

Endothelial, spindle-shaped, and myeloid cells. When the endothelial cells are numerous the growth is called an *epithelial root tumour.*

Presence of new blood-vessels.

Symptoms.—Tooth sometimes loose and extruded.
Difficult to diagnose until tooth extracted.
Usually no clinical signs.

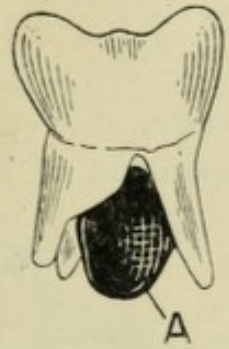

FIG. 109.—A, granuloma.

Treatment.—Extraction.

A *granuloma* may pass from an *epithelial root tumour* to

(iii) DENTAL CYST (fig. 110).

Definition.—A fluid swelling, containing a viscid, translucent, mucus-like fluid, arising from the alveolar dental periosteum.

Cause.—Irritation *viâ* apices of septic teeth or roots.

Origin.—There are two theories:—

(*a*) Mesoblastic—
Through the breaking down in the centre and the development at the periphery of a *granuloma;* or
Through the secretion of fluid by a *chronic abscess.*

(*b*) Epiblastic—
Through irritation and development of certain aggregations of epithelial cells found in the alveolar dental periosteum, the remains of the *epithelial sheath of Hertwig.*

Pathology.—Central cells degenerate into fluid containing cholesterin crystals, serum globulin, and serum albumin.

Absence of fats, mucus, and colloid in the fluid.

Peripheral cells proliferate.

Rarefying osteitis from within.

Deposition of bone externally.

Absorption is more rapid than deposition, eventually producing perforation of bone.

Epithelial lining, which may be squamous, stratified squamous, or ciliated columnar.

Fibrous tissue externally.

Treatment.—Remove septic tooth or root.

Cut out the outer wall of the cyst.

Drain cyst of contents.

Thoroughly scrape away all epithelial lining.

Swab out with pure carbolic acid.

Pack daily with iodoform gauze, allowing cavity to heal from base.

(iv) ANCHYLOSIS (Synostosis).

Definition.—Osseous union between a tooth and the alveolus.

Cause.—Local chronic periodontitis due to a general trouble, such as gout.

Pathological Process:—

First, Inflammation.
Then Suppuration.
,, Transformation of affected alveolar dental periosteum into granulation tissue.
,, Absorption of bone.
,, Transformation of affected bone into granulation tissue.
,, Union of both granulation tissues.
,, Fibrification of granulation tissue.
,, Ossification ,, ,, ,,

(*b*) General periodontitis.

(i) *General acute periodontitis:*—

Definition.—Inflammation of the alveolar dental periosteum of the root or roots of a number of teeth.

Causes.—Severe traumatism.
Drugs (mercury, &c.).
Gout, syphilis, rheumatism, exanthematous fevers, &c.

Symptoms.—As in local acute periodontitis, several teeth, however, being involved.

Pathology.—As in local acute periodontitis.

Treatment.—Local:—
Antiseptic mouth-washes.
Poppy-head fomentations.

NOTE.—Advise patient to crush five or six poppy capsules, boil them in a quart of water down to a pint, and frequently bathe the mouth with the hot wash, taking care not to swallow any.

Scarification.
Counter irritants.
General:—
Treat cause.
Purge.
Tonic.

(ii) *General chronic periodontitis commencing at the gingival margin* (PYORRHŒA ALVEOLARIS):—

Definition.—A progressive destruction of the alveolar process, accompanied by a discharge of pus at the gingival margin, and looseness of the teeth.

Causes.—(*a*) Local.
(*b*) General.

(*a*) Local.
(i) Salivary calculus.

(ii) *Débris* of food lodging and fermenting between teeth.

NOTE.—Want of cleanliness contributing to the above is probably the cause of the greater percentage of cases.

(iii) Ill-fitting dentures.

(iv) Ill-fitting bridges.

(v) Ill-fitting crowns and crowns with faulty contact points allowing food to be wedged between the teeth.

(vi) Badly made fillings with overhanging cervical margins or faulty contact points.

(vii) Irritation from chemicals, tobacco, spirits, &c.

(viii) Extraction of teeth, allowing other teeth to separate and food to be wedged in between them.

(ix) Injury from use of toothpick or floss silk.

(x) Injury from clamps and retained ligatures.

(xi) Irregularity and crowding of teeth, rendering it difficult to cleanse them.

(xii) Abnormality of bite putting excessive strain on certain teeth.

(xiii) Misuse of orthodontia appliances.

(xiv) Mouth-breathing, drying the gingival border of the gum and impairing circulation.

(xv) Insufficient use of teeth induced by soft food.

(xvi) Pyogenic organisms. (Probably not a cause, but a secondary factor.)

(*b*) General.

(i) Drugs (mercury, lead, &c.).

(ii) Gout, rheumatism, diabetes, nephritis, rickets, scurvy, &c.
(iii) Senility.
(iv) Uric acid diathesis.
(v) Malnutrition.

Signs and Symptoms:—

In the early stages there is—

No pain.
No pus.
Slight congestion of tags of gum between teeth.

In the later stages there is—

Usually no pain.
Increased congestion.
Bleeding of gums on slightest provocation.
Soreness of gums.
Gradual disappearance of tags of gum between teeth.
Separation of gums from teeth forming pockets.
Exudation of pus at the gingival margin on pressure.
Deposition of serumal and salivary calculus and *débris* of food in the pockets and on the roots of teeth.
Destruction of bone and periosteum.
Roots of teeth become exposed.
Extrusion of teeth.
Fœtid breath.
Looseness of teeth.
Often death of pulps, and alveolar abscesses.

In the last stages there is—

Constant hæmorrhage from gums.
Bad taste in the mornings.
Loss of teeth.

NOTE.—Disease clears up on loss of teeth, which fact tends to prove that the *primary cause* is *local* and not *constitutional*, and that it is not a bone disease.

Pathology.—Inflammation probably begins at the junction of the alveolar dental periosteum with the gum, starting in the former tissue.

Infiltration of alveolar dental periosteum and gum with leucocytes.

Ulceration of gum.

Formation of pus.

Deposition of serumal calculus on roots of teeth.

Presence of destroyed portions of alveolar dental periosteum.

Inflammation attacks edges of alveolus.

Alveolus loses its lime salts, and the lamellæ, lacunæ, and canaliculi disappear.

Osteoid and then fibrous tissue results.

Fibrous tissue becomes infiltrated with leucocytes.

The thicker alveolus below suffers from a lacunal absorption, osteoclasts and *Howship's* lacunæ being present.

NOTE.—The more compact the tissue the less rapid the process.

Organisms most commonly present in pus in pyorrhœa (Goadby):—

Micrococcus catarrhalis.
Bacillus coryza segmentosa or Bacillus septus.
Micrococcus pneumoniæ.
Bacillus necrodentalis.
Micrococcus citreus granulatus (*Freund*).
Streptococcus brevis.

Treatment:—

(i) Have mouth thoroughly rinsed with an antiseptic wash.

(ii) Make a thorough examination with a blunt-pointed instrument, taking care not to injure

the gums, of all the teeth and pockets, to determine extent of disease.

(iii) Remove all hopelessly loose teeth.

(iv) Remove any source of irritation, such as rough overhanging edges of fillings, *débris* of food, malocclusion, &c.

(v) Confine the treatment to several teeth at one time until improvement begins.

(vi) Inject into each pocket a local anæsthetic.

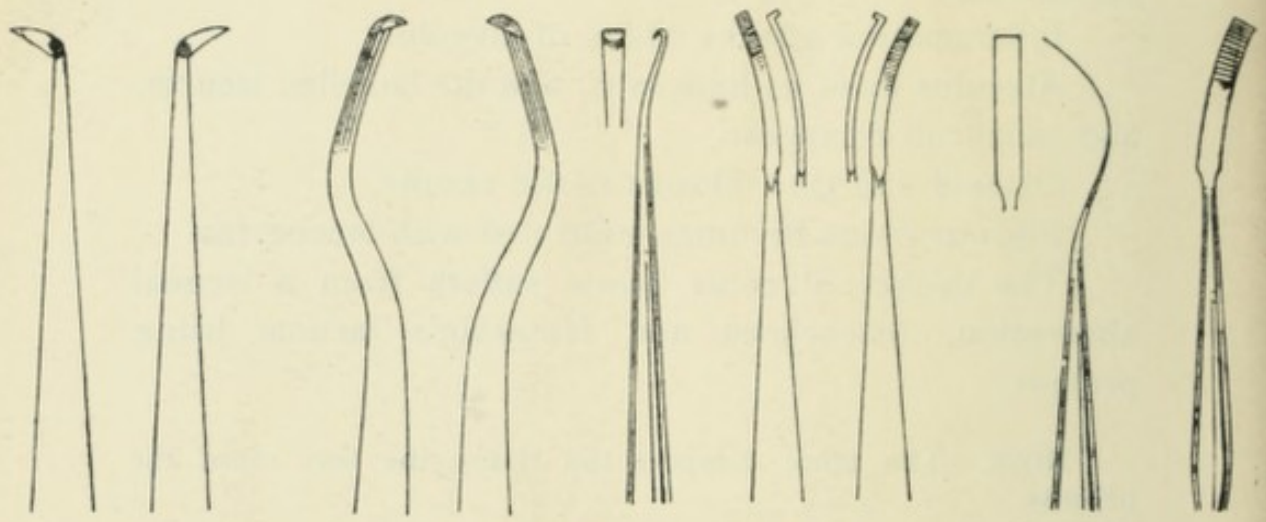

FIG. 111.

(vii) With suitable scalers, several varieties of which are shown in fig. 111, remove, as thoroughly as possible, all calculus, both salivary and serumal, and any destroyed membrane, allowing the patient to rinse the mouth at intervals with the following wash:—

℞	Hydronaphthol	...	...	gr. xx.
	Alcohol	...	...	... ℥j.
	Water ...	...	...	... ℥j.

NOTE.—A teaspoonful in a little water. The wash may be used with benefit three times a day for several days after the scaling is completed.

(viii) Dissolve any remaining deposit by placing pledgets of cotton-wool, dipped in the following, in each pocket, and leaving for thirty minutes.

℞ Aromatic sulphuric acid ... ʒiij.
Tinc. of capsicum ... ... ♏x.
Water ... ... ʒij.

A 15 per cent. solution of trichloracetic acid may be used, or fluoram crystals may be packed down the pockets with cotton-wool wound on a platinum probe.

(ix) Thoroughly smooth all rough surfaces on roots by means of wood points, &c.

(x) Forcibly wash out the pockets with warm water, followed by peroxide of hydrogen, using a syringe or atomizer.

(xi) Treat pockets with a germicide. This can be carried out by packing powdered copper sulphate into the pockets with a wood point, leaving it there for thirty minutes, and then washing out thoroughly with warm water.

Another method is to pump into the pockets with a wood point the following:—

℞ Iodine crystals ... ... gr. x.
Creosote ... ... ʒss.

Leave this for thirty seconds, and then introduce in the same way:—

℞ Tannic acid ... ... gr. x.
Glycerine ... ... ʒss.

Another method is to swab out the pockets with

℞ Perchloride of mercury ... gr. j.
Peroxide of hydrogen ... ʒj.

Still another method is to pack crystals of salicylic acid into the pockets by means of a wood point dipped into a 70 per cent. solution of alcohol.

Other drugs used for the purpose are:—

Acetozone.
Aseptol, 3 per cent. solution.
Resorcin, 10 per cent. solution.
Protargol, 8.3 per cent. solution.
Euthymol.

(xii) Extirpate pulps of teeth where thought advisable and fill roots.

(xiii) Ligature or apply a splint to any loose teeth for support, where considered advisable.

(xiv) Advise removal of artificial dentures except for mastication.

(xv) Advise massage of the gums from the apices of the teeth to the necks with a rubber brush or the finger.

(xvi) Advise an antiseptic mouth-wash, such as—

℞ Potassi chloratis ... ... ʒij.
Glycerini boracis ... ... ʒv.
Aq. rosæ ... ... ad. ℥x.
M. ft. lotio.

or

℞ Tinct. myrrhæ ... ... ℥ss.
,, krameriæ ... ... ℥ss.
,, cinchonæ ... ... ℥ss.
,, catechu ... ... ℥ss.
Eau de Cologne ... ... ℥j.
M.

NOTE.—A large tablespoonful in a wineglassful of water.

(xvii) To get rid of the pockets it is sometimes necessary to slit them open, cauterize them, and excise the tags of gum.

Ionization or Cataphoresis.

The treatment of pyorrhœa by *ionization* or *cataphoresis* has been recommended.

It consists in introducing certain drugs into the human body, through the tissues, by means of *electrolytic action*.

One *pole* or *electrode* of the battery is used for introducing the drug (the *active electrode*), and consists usually of a zinc needle (fig. 112).

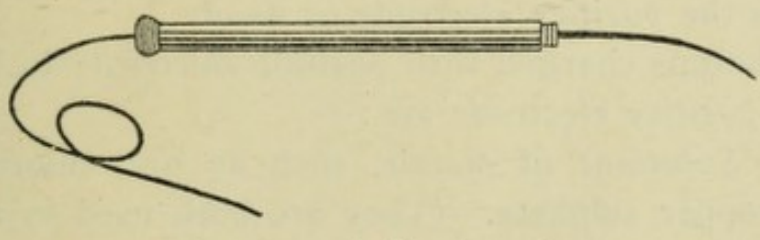

Fig. 112.—Active electrode.

The other *pole or electrode* (fig. 113), is held in the patient's hand or attached to the wrist, and is termed the *indifferent electrode*.

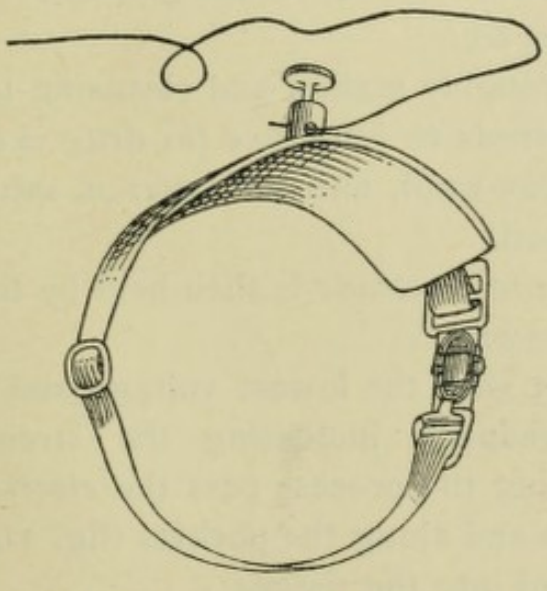

Fig. 113.—Indifferent electrode for wrist or arm.

Whether the *positive* or *negative electrode* is used as the *active electrode* depends upon the solution used.

Certain solutions which conduct electricity are used for the purpose, and are called *electrolytes*.

The same solutions contain what are called *ions*.

Certain of these *ions* are positive *ions* or *cations*, and others are *negative ions* or *anions*.

When a current of electricity is passed through the solution the *positive ions* or *cations* are repelled from the *positive electrode* or *anode* and travel towards the *negative electrode* or *cathode*, whilst the *negative ions* or *anions* are repelled from the *negative electrode* and travel towards the *positive electrode* or *anode*.

Solutions charged with *positive electricity* to be used on the *positive electrode* are:—

Solutions of *metals*, such as zinc chloride and copper sulphate. (They are both used in a 5 per cent. solution.)

Alkaloids, such as morphia, cocaine, or hydrogen.

Solutions charged with *negative electricity* to be used on the *negative electrode* are:—

Acid radicals, such as salicylic acid.

Iodine, &c.

After thoroughly scaling and cleansing the roots and teeth, the *electrode to introduce the drug* is covered with a wisp of cotton-wool, and the latter is saturated in the selected solution.

The *opposite electrode* is then held by the patient or attached to the wrist.

Then start with the lowest voltage and the weakest amperage, gradually increasing the strength of the current. During the process pass the *electrode carrying the drug* down and along the pockets (fig. 114), when the drug is repelled into the tissues.

Treat several teeth at each sitting of about thirty minutes' duration, keeping the current up for about two minutes at a time on each tooth treated, and occasionally

saturating the wool on the *active electrode* with the selected solution.

Turn off the current by degrees. Apply an astringent to the gums. Paint the gums with:—

℞ Tinct. of aconite ...
,, iodine ...
Spirits of chloroform
,, camphor ...
āā fl. ʒi. Misce.

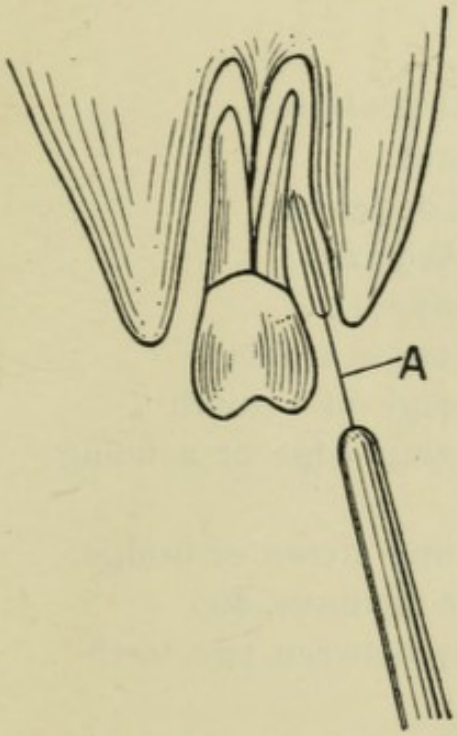

FIG. 114.—A, *active electrode* conveying wisp of wool, saturated with a selected drug, into a pyorrhœa pocket.

Repeat the treatment every forty-eight hours until the desired result is obtained.

VACCINE TREATMENT.

Vaccine treatment of pyorrhœa has also been recommended. It has been adopted with varying success, and consists in isolating what is supposed to be the predominating microbe found in the pus, usually a streptococcus or a bacillus, and making and injecting a vaccine from that organism.

CHAPTER X.

DISEASES OF THE GUMS AND MUCOUS MEMBRANES OF THE MOUTH.

Hypertrophy.

Definition.—An increase of the elements which compose the tissue of the gum resulting in an increase in its bulk.

Varieties:—

(i) Local (*Polypus*).

(ii) General.

(*a*) Congenital.

(*b*) Acquired.

(i) Local (*Polypus*).

Causes.—Irritation from:—

Jagged edge of a tooth.

Overhanging edge of a filling.

Calculus.

Badly fitting crown or bridge.

Clasps on dentures, &c.

Site.—Usually between two teeth.

Symptoms:—

Resembles normal gum.

Painful to pressure.

Bleeds readily.

Often pedunculated.

May invade cavity in a tooth, when it must be diagnosed from *polypus of the pulp.*

Polypus of Gum.	*Polypus of Pulp.*
Can be traced into continuity with gum.	Unconnected with gum.
Painful to pressure.	Not painful to pressure.
Bleeds readily on pressure.	Does not bleed readily on pressure.

Pathology:—

Springs from periosteum.
Fibrous stroma increased.
Enlarged surface papillæ.
Thin epithelial surface.
Cellular elements numerous.
Excess of blood in part.

Treatment:—

Remove cause.
Excise growth and scrape away periosteum from which it springs.
The actual or electric cautery may be used to remove growth, or it may be removed by the following method:—

(i) Protect surrounding parts with cotton-wool rolls.
(ii) Dry the growth.
(iii) Apply a little potassa cum calce to the growth and leave for several minutes.
(iv) Remove resulting jelly-like mass.
(v) Syringe.
(vi) Repeat treatment until growth removed.

(ii) General.

Causes.—It may be *acquired*, due to functional activity through inflammation from the presence of carious teeth or pyorrhœa alveolaris.

It is often coincident with the eruption of the teeth (*congenital*).

Symptoms:—

Growths almost covering teeth.
If mouth *aseptic*, new growths colour of normal gum,
Does not bleed readily,
Not painful,
Edges thick, smooth, and rounded.

If mouth *septic*, growths dark purple in colour,
Tags of gum between teeth,
Purulent discharge from between gums and teeth,
Gum bleeds readily,
Fœtid breath,
Itching sensation,
Often very painful.

NOTE.—It may occur in several members of the same family. Those affected are often abnormal in some other way (hair, &c.).

Pathology:—

Springs from periosteum.
Fibrous stroma increased.
Enlarged surface papillæ.
Thin epithelial surface.
Cellular elements numerous.
Excess of blood.
In some cases presence of saccharomyces.
Hypertrophy of bone.

Treatment.—Complete removal of growths and hypertrophied bone.

GINGIVITIS.

Definition.—Inflammation confined to the gums.

Varieties (Colyer):—

(*a*) Catarrhal (usually).
(*b*) Purulent.
(*c*) Chronic hypertrophic marginal.

Causes:—

(i) Local—

Want of cleanliness
Want of use
Irregularity of teeth
} Causing lodgment of *débris* of food between teeth and gums, and resulting putrefaction and fermentation.

Caries.
Calculus.
Ill-made fillings, crowns, &c.
Wrong use of regulating appliances.
Mouth-breathing.
Tobacco.
Drugs (arsenic, &c.)
Traumatism (injury from tooth-brush, &c.).

(ii) General—
Dyspepsia.
Gout.
Scurvy.
Syphilis.
Alcoholism, &c.

(iii) Medicinal—
Mercury, Iodides, &c. } Overdoses cause excretion of irritants from the blood.

Symptoms:—
Gum margins red, soft, swollen, and tender.
Gums bleed readily.
Very little pain.
Often tags of gum between teeth.
Sometimes purulent discharge at margin of gums.

Pathology:—
Hyperæmia.
Inflammation.

Treatment:—
Remove cause such as food, calculus, &c.
Advise an astringent mouth-wash, such as:—

℞ Tannic acid ... ... ʒij.
Rectified spirit ... ... ʒj.
Rosewater to ... ... ℥x.

Advise massage with the finger or a rubber brush from apices of roots of teeth to crowns.

Syringe out any pockets with peroxide of hydrogen.

Pack pockets with powdered copper sulphate, carried on a wood point, several times a week.

If any pain, apply

℞ Tinc. iodine, Spirits of camphor } Equal parts.

Excise any tags of gum.

Lead Poisoning affecting Gums (Lead Line).

Probable Cause.—Chemical action, between the lead brought by the blood to the gums and the sulphuretted hydrogen evolved from calculus or decomposing *débris* of food, resulting in the formation of lead sulphide.

Symptoms:—

Blue line at margin of gums.

Colic, Wrist drop (due to paralysis of extensor muscles) } Usually.

Metallic taste.

Constipation.

Site:—

Occurs in painters, compositors, &c.

Usually in lower jaw.

Usually in region of incisors.

No line is present in an edentulous jaw.

The *lead line* must be diagnosed from a similar appearance produced by any one of the following:—

Charcoal and soot used in cleansing teeth.

Carbon dust in miners.

Copper poisoning.

Bismuth poisoning.

Calculus under gums.

Treatment.—Remove patient from environment, when disease clears up in a few weeks.

Stomatitis.

Definition.—Inflammation of the mucous membrane of the mouth.

Varieties:—

Catarrhal or *Simple.*
Mercurial.
Ulcerative.
Gangrenous, Noma, or *Cancrum oris.*
Follicular.
Parasitic.
Aphthous.
Syphilitic.

Catarrhal or Simple.

Causes:—

(i) Local—
Want of cleanliness.
Caries.
Teething.
Mouth-breathing.
Tobacco.
Spiced foods.
Traumatism, &c.

(ii) General—
Gastritis.
Inflammations of throat and nose.
Gout.
Diabetes.
Dyspepsia.
Typhoid.
Alcoholism, &c.

(iii) Medicinal—
Mercury.
Iodides.

Symptoms:—

Mucous membrane inflamed and swollen.
Epithelium thickened.
In early stages gums dry, then excessive secretion.
Excessive secretion of mucus and saliva.
Sticky purulent discharge.
Margin of gums congested and tender.
Gums whitish and mottled. This appearance is due to the production of red patches through the rubbing off from the papillæ of the sodden epithelium.
Seldom any pain.
Fœtid breath.
Submaxillary lymphatic glands affected.
General debility.

Pathology:—

Inflammation usually starts at margin of gums.
Dilatation of vessels of submucous tissue.
Exudation and proliferation of cells.
Cells become sodden by exudation.
Inflammation may spread *deeply*, causing—
Ulceration of the alveolar dental periosteum,
The formation of pockets, and
A purulent discharge at margin of gums.
Inflammation may spread *superficially*, causing--
Catarrh of the mouth.

Treatment:—

Remove cause.
Stop use of drugs, such as mercury.
Stop use of tobacco and alcohol.
Order astringent and antiseptic mouth-washes.
Purge.
Tonic.

Mercurial.

Cause.—Excessive use of mercury combined with a septic mouth.

Symptoms.—Those of catarrhal stomatitis with:—

Red ring at margin of gums.
Ulceration and sloughing of gums.
Bleeding of gums.
Periodontitis.
Pain on mastication.
Profuse salivation (*ptyalism*), due to spread of inflammation to salivary glands.
Metallic taste.
Pronounced fœtor of breath.
Loosening of teeth.
Teeth fall out.
Necrosis.
Tongue may become swollen (*parenchymatous glossitis*).
Pain on opening the mouth.
Colic.
Diarrhœa.
General malaise.

Pathology.—Inflammation leading to necrosis and gangrene.

Treatment:—

Stop use of mercury.
Cleanse mouth.
Tincture of iodine to the gum.
Local depletion by incision may be carried out.
Antiseptic and astringent mouth-washes.
The following may be given internally:—

℞			
	Potassium iodide ...	...	gr. xl.
	Syrup of orange flower	...	ʒj.
	Spirits of chloroform	...	ʒij.
	Water to	...	℥viii.

Begin with 5-gr. doses, gradually increasing dose.

or

℞ Potassium chlorate ... ... gr. x.
Water ... ... ʒss.

To be given in four doses during the day.

On clearing up of disease, remove any hopelessly loose teeth.

Potassium permanganate may be used as a mouth-wash to correct fœtor of breath.

Ulcerative.

A disease of childhood in subjects debilitated through measles, typhoid, &c.

Does not occur in healthy and robust children.

Rarely seen in better-class children.

Average age at which it occurs is 6 years.

Sometimes occurs in schools, &c., as an epidemic.

Sometimes occurs in adults.

Sometimes coincident with menstruation and pregnancy.

Site:—

Usually in maxilla.

Usually in front of mouth.

Usually unilateral.

Causes:—

Malnutrition.

Unhygienic conditions.

General debility.

Infected milk.

Scurvy.

Administration of lead, mercury, or arsenic.

Digestive disturbances, &c.

Symptoms:—

Commences at free margin of gums.

Not much pain in early stages.

Heat, redness changing to blueness, and swelling.

Ulceration of gums, blood escaping from mouth during sleep.
Ulcers may be superficial or deep.
Pain later.
Excessive flow of saliva.
Characteristic fœtor of breath.
Bone laid bare.
Ulceration of alveolar dental periosteum.
Teeth become loose and tender, and may fall out.
Necrosis.
Ulceration may spread to cheeks, tonsils, and pharynx.
Sometimes fever and death.

Characteristics of ulcer:—

Edges.—Ragged, sharp, bluish in colour.
Base.—Inflamed and covered with a yellow slough.
Surroundings.—Red, tender, and swollen.

Bacteriology:—

Spirochæta dentium
Bacillus fusiformis
Saccharomyces albicans
} Always present in large numbers.

Treatment:—

Remove any tooth or teeth causing irritation or which are septic.
Antiseptic and astringent mouth-washes.
Give potassium chlorate internally in doses of from 3 to 10 gr.; 1 gr. for each year of the child's age. For an adult 15 to 20 gr.
Frequent overdoses of potassium chlorate may produce *hæmoglobinuria*, a disease, rare in *England*, in which the urine is red or brown.
Apply locally silver nitrate, 10 gr. to the ounce; or potassium chlorate with honey, in doses of from 3 to 5 gr., several times a week.

Order the following gargle:—

℞ Potassæ chloratis ... ... ℥j.
Acidi hydrochlor. dil. ... ℥ss.
Aquam. ... ... ad. Oj.

Purge.
Nourishing diet.
Tonic.
Fresh air.
If cheek is involved apply carbolized oil on lint to the part.

Gangrenous, Noma, or Cancrum Oris.

Cause:—

Probably due to a parasitic bacillus.
Occurs usually in children, between 2 and 6 years, due to malnutrition.
Unhygienic conditions.
General debility, &c.
Often coincident with convalescence from one of the exanthematous fevers.
It occurs more often in girls than in boys.
It sometimes occurs in adults suffering from diabetes and chronic nephritis.

Signs and Symptoms:—

Rapid development.
Sloughing ulcer on inside of cheek.
Surrounding parts become gangrenous.
Ulcer deepens.
On outside of cheek is a hard, dense, brawny, dull red swelling.
In the centre of this is a dark spot situated opposite to ulcer.
Perforation of cheek.
Sloughing

Teeth loosen and fall out.
Profuse salivation.
Gangrenous odour.
Absence of much pain.
Restlessness.
Delirium.
Septicæmia.
Death.

Pathology.—A capillary thrombosis.

Bacteriology.—The following have been found to be present:—

Diphtheria bacillus.
Bacillus fusiformis.
Bacillus necrosis.
Spirochæta dentium.

Treatment:—

Dry soft parts.
Scrape away all gangrenous tissue.
Cauterize with nitric acid.
Follow with chloride of lime.
Antiseptic dressings.
Antiseptic mouth-washes.
Nourishing diet.
Tonic.
Hygiene.
Plastic operation later.

Follicular.

Inflammation leading to ulceration of scattered follicles in the gum. Similar to a herpetic eruption.

Site:—

Frænum.
Under surface of tongue.
Sulcus between gum and lips.
More often in children than in adults.

Symptoms:—

Occurs at any age.
Isolated red spe
These break dow nto sharp-edged ulcers.
Base of ulcers yellow.
Zone of redness around.
Ulcers seldom single.
Ulcers painful.

Treatment:—

Cleanse teeth.
Astringent mouth-washes.
Wash ulcers with warm water.
Touch ulcers with silver nitrate or copper sulphate.

Parasitic (*Thrush*).

This is a contagious disease.

Varieties.—Colyer mentions two—

(*a*) Due to *Saccharomyces* or *Oïdium albicans* (more common).
(*b*) Due to *Aspergillus nigrescens*.

Causes:—

Unhygienic conditions Dirty feeding-bottles Debility Malnutrition Indigestion	In connection with children.
Associated with acute specific fevers and Phthisis	In connection with adults.

Symptoms:—

Occurs usually in children.
At first simple inflammation.
Mucous membrane much inflamed.
Mouth dry.

Mucous membrane covered with many white spots, each about the size of a pin's head, and circular.

Spots near angle of mouth and tongue.

Spots coalesce, forming large patches.

Spots may spread to œsophagus and pharynx.

Patches come away, leaving reddish surfaces.

Mastication difficult.

Enlargement of glands.

Red ring around anus due to acrid, green evacuations.

Drowsiness.

Torpor.

If in adults suffering from phthisis it is a sign of approaching death.

Pathology:—

Fungus develops in upper layers of mucosa.

Filaments of fungus form network among epithelial cells.

The patches consist of epithelium and fat with sporules of *Oïdium albicans*.

Treatment:—

Thoroughly cleanse mouth with lint, throwing used lint away.

Touch patches with

℞ Borax ... ... ʒiij.
Aqua ... ... ʒj.

or

℞ Sodii borat. ... ... ʒj.
Glycerinæ ... ... ʒij.
Aquæ ... ... ʒvj.
M.

Apply with a camel-hair pencil several times a day.

or

℞ Permanganate of potash.

Should these fail use as a mouth-wash:—

℞ Zinci sulph. ... ... gr. iij.
Aquæ rosæ ... ... ℥ij.
M.

or

℞ Silver nitrate ... ... gr. 1-10.
Water ... ... ℥j.

or

℞ Glycerinum acidi carbolici... ♏xv.
Aqua ... ... ℥j.

or

℞ Sulphurous acid ... ... 1 part.
Water ... ... 8 parts.

If the ulcers are foul use bicyanide of mercury 1-1,000.

See to cleanliness of feeding-bottles.

Nourishing diet.

Aperient.

Aphthous.

A contagious disease of childhood.

Rarely seen in adults.

Cause:—

Not known.

Seen during dentition in weakly and debilitated children.

Often coincident with rachitis.

Often accompanies digestive disturbances.

In debilitated adults it may be associated with menstruation or pregnancy.

Site:—

Inner surfaces of lips and cheeks.

Tongue.

Roof of mouth.

Gum.
Frænum linguæ.

Pathology:—

Inflammation leading to—
Thickening of membrane, and
Fibrous deposits on and under epithelium.
Epithelium sodden by exudation.

Bacteriology:—

Not certain.
Streptococcus fæcalis conspicuous.

Signs and Symptoms:—

Appears suddenly.
Small yellowish white patches on mucous membrane.
Singly or in groups.
Patches raised.
Pain.
Mastication difficult or impossible.
Heat.
Zone of inflammation surrounds each patch.
Patches spread and coalesce.
Hæmorrhage on removal of patches, which are very adherent.
Ulcers superficial, flat, hollowed out.
Profuse secretion of saliva.
Feverish symptoms.

Treatment:—

Cleanse mouth.
Potassium chlorate mouth-wash.
Boro-glycerine locally 1-20.
Should patches still persist touch them with silver nitrate.
Look after general health and digestion.

Syphilitic.

Primary Stage—

Hard chancres may occur anywhere in the mouth.

Extremely infectious.

Cause:—

The entrance of the specific organism of syphilis through a wound or scratch, due to

Kissing an infected part.

Using infected surgical instruments.

Using infected eating or drinking utensils.

Playing upon infected wind instruments.

Using infected pipe, &c.

Wet nurse may infect child or child the wet nurse.

Symptoms:—

Ulcer has indurated base.

Shotty glands.

No pain.

Clear yellow serum, containing a great number of the *Spirochæta pallida* (*Treponema pallidum*), exudes on pressure.

Secondary Stage—

Occurs from four to six weeks after the primary as *mucous tubercles*.

Mucous tubercles may undergo ulceration.

Extremely infectious.

Cause:—

Presence of virus in circulation.

Site:—

Gums.

Inner surface of lips.

Inner surface of cheeks.

Palate.

Tonsils.

Sides of tongue.
Anterior pillar of fauces.

Symptoms:—

Sore throat.
White patches.
Patches slightly raised.
Ulcers shallow and superficial and known as *snail track ulcers*.
Lymphatic glands often enlarged.

Tertiary Stage—

It may occur shortly or many years after the secondary.
It presents itself in the mouth as papules and large and small nodules (*gummata*).
These may ulcerate, or they may become sclerosed, causing *tertiary papular syphilide* (more common).

Site:—

Gum.
Cheek.
Tongue.
Palate.

Symptoms:—

Ulcers usually superficial.
Not painful.
Some discharge of pus.

EPULIS.

Definition:—

A new growth involving the gum.

Varieties:—

(i) *Fibrous* or *simple* (*Fibroma*).
(ii) *Myeloid*.

Cause:—

May be due to chronic irritation from a septic tooth or

A piece of bone.

Hopewell-Smith states that congenital influences may be concerned in the formation.

(i) *Fibrous or simple (Fibroma).—*

Site:—

Chiefly in region of maxillary incisors.

Chiefly in women.

Symptoms:—

Tag of gum between two teeth enlarged.

This increases in size.

May become size of walnut.

No heat.

No tenderness.

May be attached by small pedicle or broad base.

Firm.

Pedunculated.

Resembles normal gum in colour.

Becomes ulcerated if bitten upon.

Microscopically:—

Springs from periosteum or periodontal membrane.

Bundles of fibrous connective tissue.

Epithelial covering thick.

Enlarged papillæ.

Blood-vessels few.

Lymph spaces scanty.

May contain spicules of bone.

Treatment:—

Anæsthetize patient.

Completely excise growth and remove portion of alveolus involved.

Use actual cautery for hæmorrhage.

(ii) *Myeloid.*—

Symptoms:—

Tag of gum between two teeth enlarged.
Grows very rapidly.
Growth soft.
Dark purple in colour.
Hæmorrhage readily occurs.
Becomes ulcerated if bitten upon.

Microscopically:—

Springs from bone marrow of alveolar process.
Presence of myeloid or giant cells.

Treatment:—

Remove teeth in involved area.
Completely excise growth.
Remove affected alveolus.

PAPILLOMATA (*Analagous to warts*).

Definition:—

Hypertrophy of papillæ of mucous membrane of gum.

Symptoms:—

Elongated papillæ.
Slow growth.
No inflammatory symptoms.

Microscopically:—

Central portion mesoblastic tissue.
Epithelial covering thick.

Treatment:—

Leave alone.
If desired they may be removed by ligaturing them tightly at their junction with the gum, thus cutting off the blood supply and destroying growths, or
Excising them.
Cauterize to stop hæmorrhage.

Nævi.

Definition.—Tumours of blood-vessels.

Varieties:—

(i) Capillary.
(ii) Venous.
(iii) Arterial.

Site.—Usually incisor region.

Symptoms:—

Begin as small red spots.
These spread.
Vary in colour according to variety.
Surface smooth.
Swell readily.
Pressure forces blood out, when swelling becomes pale.
On removal of pressure, blood and original colour return.
Bleed readily when injured, *e.g.*, in brushing teeth.

Treatment:—

Cleanse mouth.
Order astringent mouth-wash.
Leave growths alone if not increasing in size.
Surgical methods, such as electrolysis or excision, may be necessary.

Scurvy.

Cause:—

Want of fresh food and fruit.
Sterilized milk.
Artificial diet.

Site:—

Usually in children.
Gums of edentulous jaws never affected.

Symptoms.—Gums are
Swollen,
Soft and spongy,
Turgid,
Dark in colour, and
Bleed readily at first.
There is constant oozing later.
Mucous membranes of lips and tongue pallid.
Ulceration of gums.
Loosening of teeth.
Fœtid breath.

Pathology:—
Capillary hæmorrhage due to change in the blood or blood-vessels.

Treatment:—
Cleanse mouth.
Astringent mouth-washes.
Fresh food.
Fresh unboiled milk.
Fruit.
Lime-juice.

Purpura.

Numerous small hæmorrhages from and into the gum.

Usually associated with purpura in other parts of the body.

Cause is unknown.

Pemphigus.

Mucous membrane covered with blebs.
Some blebs may bleed.
Some subside without bleeding.
Associated with general pemphigus.

Epithelioma and Sarcoma.

May occur in connection with the gums.

CHAPTER XI.

SALIVA AND SALIVARY CALCULUS.

Saliva.

Definition.—The mixture of the secretions of the salivary glands, occurring in the mouth.

Origin.—The parotid, sublingual, and submaxillary salivary glands. The buccal mucous glands.

Properties:—

Clear.
Tasteless.
Slightly opalescent.
Glairy.
Viscid.
Frothy.
Normally alkaline. (If acid, due probably to fermentation of food in mouth.)
Not antiseptic.

Specific gravity.—1,002 to 1,008.

Composition (Haliburton):—

Water			994.10
Solids—			
Soluble organic matter ...	Mucin		
	Ptyalin		
	Traces of proteid		
Epithelium			
Soluble inorganic matter ...	Sodium chloride ..		5.90
	Sodium carbonate		
	Calcium carbonate		
	Potassium chloride		
	Calcium phosphate		
	Magnesium ,,		
	Sodium ,,		
	Potassium sulpho-cyanide		

Urea (?)
Salivary corpuscles (leucocytes altered by action of saliva) from the tonsils and other adenoid tissues.
Micro-organisms, and
Food *débris*, are also found in the saliva.

Amount secreted:—

About two to three pints every twenty-four hours.
Two-thirds are secreted by the parotid gland.
One-twentieth is secreted by the submaxillary gland.

Functions:—

(*a*) *Mechanical—*

Lubricant aiding articulation.
Keeps mouth moist.
Softens food, aiding mastication and deglutition.
Dissolves food, aiding sense of taste.

(*b*) *Chemical—*

It changes starch food into dextrine and maltose, by the action of the ptyalin.

Conditions and diatheses altering saliva:—

After fasting (before breakfast, &c.)	Least alkaline.
During or immediately after a meal	Most alkaline.
Odour or thought of food Mastication ... ... Nausea	Increased secretion.
Xerestoma ... ... Small-pox (sometimes) ...	Decreased secretion.

Stomatitis	Increased secretion. Acidity.
Gingivitis	
Tonsillitis	
Pharyngitis	
Diphtheria	
Small-pox (sometimes) ...	
Dyspepsia	Acidity. Albuminous matter increased. Greatly increased mucous secretion.
Acute rheumatism	Sulphocyanides in excess.
Acute gout	
Bilious headache	
Early stages of inflammatory disorders	
General acute affections, *e.g.*—	Acidity.
Eruptive fevers	
Typhoid ...	
Malarial fever ...	
Pneumonia ...	
Use of mercury ...	
Use of potassium iodide	
General chronic affections, *e.g.*—	Increase of ptyalin. Excessive secretion of mucus.
Rheumatism ...	
Gout	
Dyspepsia ...	
Dysentery ...	
Pregnancy (sometimes) ...	Acidity.
Lactation (sometimes) ...	

Characters of parotid saliva:—

Clear.
Thin.
Mobile.
Not viscid.
Less alkaline than submaxillary saliva.
On standing becomes turbid, due to deposit of carbonate of lime.
Contains $\frac{1}{2}$ to $1\frac{3}{4}$ per cent. solids,
Sulphocyanate,
Ptyalin, and
Some globulin (?).
No mucin present.
No salivary corpuscles present.

Characters of submaxillary saliva:—

Clear.
Watery.
Mobile.
Always alkaline.
Becomes viscid on standing.
On standing deposits potassium and sodium chloride.
Contains 0.36 to 0.46 per cent. solids,
A lot of mucin,
A little ptyalin,
Salivary corpuscles, and
Proteid matter.

Characters of sublingual saliva:—

Clear.
Slimy.
Viscid.
More strongly alkaline than submaxillary saliva.
Contains 2.75 per cent. solids,
Diastatic ferment, and
Sulphocyanide.

Rich in ptyalin.
Rich in mucin.
Chief salt is phosphate of lime.

Characters of buccal mucous saliva:—

Alkaline.
Rich in albuminous matter.
Chief salt sodium phosphate.
Contains 1 per cent. solids.

Salivary Calculus.

Definition.—A deposition of salts from the saliva.

Site.—(i) On the teeth (*tartar*).
(ii) In a duct or a gland.

(i) On the teeth (*tartar*):—
The common situations are:—
The lingual side of lower incisors.
The buccal sides of upper molars.
This is on account of *Wharton's* and *Stenson's* ducts opening at these situations.

Varieties:—

(*a*) Hard.—This is dark in colour, is deposited slowly, and is difficult to remove.
(*b*) Soft.—This is light in colour, is deposited quickly, and is easy to remove.

Mode of formation:—

Saliva reaches mouth.
Carbon dioxide liberated.
Calcium carbonate and calcium phosphate precipitated.

Deposition of tartar is favoured by:—

Lack of cleanliness.
Pits and depressions in teeth.
Irregularities (crowding, &c.).
Want of use (unopposed and sensitive teeth, &c.).
Rough surfaces on teeth.

Clasps and bands on dentures.
Ill-fitting crowns.
Ill-made fillings with overhanging cervical margins.
Character of saliva.
General health. (The more perfect the health, the less the deposition of tartar.)

Composition.—Very variable.

Berzelius gives:—

Earthy phosphates	79.0
Salivary mucus	12.5
Ptyalin	1.0
Animal matter soluble in hydrochloric acid	7.5

Dr. Stevenson's analyses:—

	Water and Organic Matter		Inorganic Matter
Soft tartar ...	21.48	...	78.52
Hard tartar ...	17.51	...	82.49

The following are constituents of salivary calculus:—

Magnesium phosphate ...	
Calcium phosphate	
Calcium carbonate	
Calcium fluoride	78 to 82 per cent.
Sodium chloride	
Potassium chloride ...	
Sulphocyanide of potassium	
Salivary mucus	10 to 14 per cent.
Water	
Animal matter soluble in hydrochloric acid ...	
Epithelial cells	About 8 per cent.
Débris of food	
Micro-organisms	
Ptyalin	About 1 per cent.

Sequelæ :—

Gingivitis.
Inflammation of gums.
Suppuration of gums.
Hæmorrhage of gums.
Recession of gums.
Periodontitis.
Pyorrhœa alveolaris.
Absorption of alveolus.
Necrosis of alveolus.
Looseness and loss of teeth.
Fœtid breath.

Treatment :—

Thoroughly remove all deposits with suitable scalers (fig. 111).
Avoid injuring tooth structure.
Avoid injuring gums.
Syringe interdental spaces with atomizer and astringent antiseptic solution.
Polish tooth surfaces with wood points (fig. 115), rubber cups (fig. 116), brushes (fig. 117), and fine pumice powder.
Order an antiseptic astringent mouth-wash.

(ii) In a duct or a gland :—

Probable cause :—

Either—

(*a*) Deposition of the salts of the saliva around a foreign body.
(*b*) Inflammation of the lining membrane causing a precipitation of the salts of the saliva.

Site :—

More often in connection with a duct than a gland.
Chiefly in connection with the submaxillary.

Less often in connection with the sublingual.
Rare in connection with the parotid.

Composition:—

Chiefly phosphate and carbonate of lime and a little animal matter.

Symptoms:—

If in the substance of a gland—
Severe inflammatory trouble.
Obstruction of flow of saliva.

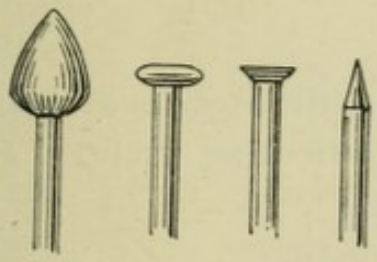

FIG. 115.
Wood points.

FIG. 116.
Rubber cup.

FIG. 117.
Brushes.

If in the duct of a gland—
Pain.
Distension (chiefly at meals).
Inflammation.

Sequelæ:—

Abscess.
Fistula.
Destruction of gland.

Treatment :—

If in connection with duct—

Locate and fix concretion.

Incise mucous membrane over concretion.

Remove.

If in connection with gland—

Removal of gland may be necessary.

Salivary Fistula.

Definition.—A sinus in the cheek communicating with the duct of a gland.

Site :—

Usually in connection with *Stenson's* duct.

Rarely in connection with other ducts.

Cause :—

Wounding duct in operating.

Salivary calculus resulting in abscess.

Symptoms :—

Opening on cheek.

Saliva flows down cheek.

Flow of saliva increased at mealtimes.

Treatment :—

Suture, if *Stenson's* duct divided.

Operations to divert flow of saliva into the mouth may be carried out in other cases.

CHAPTER XII.

ODONTOMES.

Definition.—An odontome is a neoplasm composed of dental tissues in varying proportions and different degrees of development arising from a tooth germ or a tooth still in its process of growth (*Bland-Sutton*).

Classification (Bland-Sutton):—

(*a*) Aberrations of the enamel organ.
Epithelial odontomes.

(*b*) Aberrations of the follicle.
Dentigerous cyst or follicular odontome.
Fibrous odontome.
Cementoma.
Compound follicular odontome.

(*c*) Aberrations of papilla.
Radicular odontome.

(*d*) Aberrations of whole tooth germ.
Composite odontome.

Epithelial Odontome, Multilocular Cyst, or Fibrocystic Disease of the Jaw (fig. 118).

Site:—

Lower jaw (more common).
Molar region.

Description:—

Masses of epithelium.
Numerous cysts with thin fibrous septa.
Septa between cysts often calcified.

Brown mucoid fluid inside.
Degeneration of cells internally.
Proliferation of cells externally.
Growing portions of cyst reddish.
Surface often lobulated, when growth may be mistaken for myeloid sarcoma.
Incompletely formed tooth in neighbourhood.
Cyst may become calcified, and is then called a *calcified epithelial odontome.*

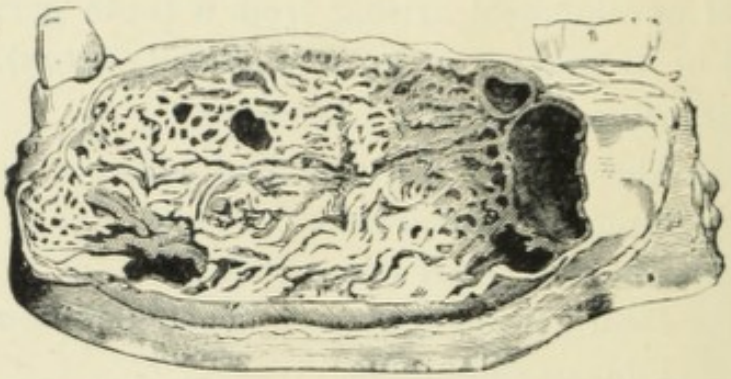

FIG. 118.

Epithelial odontome (R.C.S. Museum), after *Bland-Sutton.* (*Trans. Odont. Soc.*)

DENTIGEROUS CYST OR FOLLICULAR ODONTOME (fig. 119).

Site:—

Lower jaw (more common).
Molar and pre-molar region.
Usually in connection with permanent teeth.
May invade antrum.
Common in rickety children.

Cause:—

An excessive secretion of fluid, probably derived from the cells of the stellate reticulum, between *Nasmyth's* membrane or the enamel and the follicle wall.

Description:—

May grow to large size.
Contains thick, yellow, glairy fluid.
Fluid may contain cholesterin crystals.
Walls consist of fibrous connective tissue.
Epithelial lining usually.
Contains tooth which may be loose or embedded in the cyst wall.
Teeth devoid of *Nasmyth's* membrane.
Cyst unilocular.
May suppurate (seldom).
Walls may calcify.

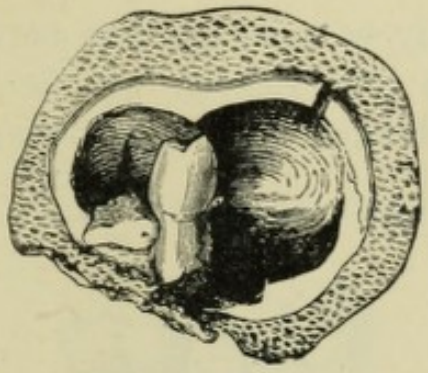

FIG. 119.
Follicular odontome (R.C.S. Museum), after *Bland-Sutton*. (*Trans. Odont. Soc.*)

FIBROUS ODONTOME (fig. 120).

Site:—

Rare in man.
May occur in rickety children.
Common in goats.

Description:—

If in man, unilateral.
If in animals, symmetrical.
Resembles fibroma.
Tooth surrounded by greatly thickened fibrous follicle.

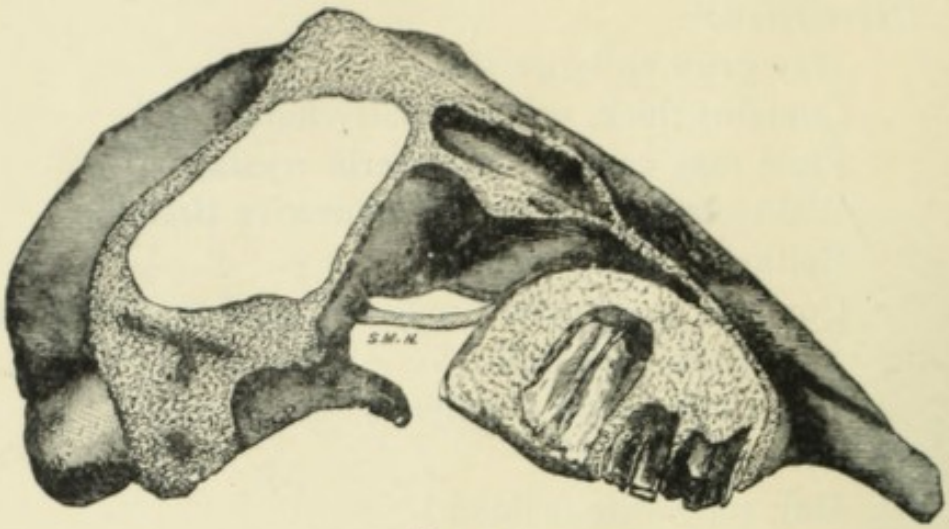

FIG. 120.

Mr. Sutton's specimen of a fibrous odontome in the jaw of a goat.

There may be some relation between this odontome and rickets, as the latter thickens membranes covering growing bones.

CEMENTOMA (fig. 121).

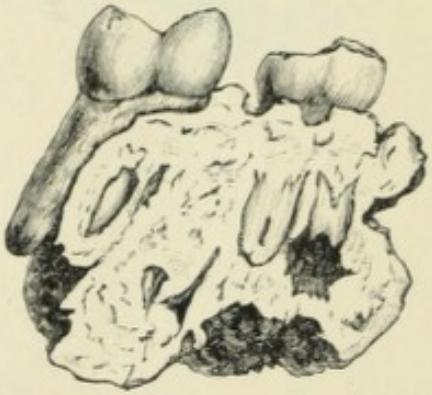

FIG. 121.

An odontome, probably a cementoma, removed by *Mr. John Murray.* (*Trans. Odont. Soc.*)

Site:—

Rare in man.

Common in horses and ruminants.

Description:—

Calcified fibrous odontomes.

Follicle walls become cemental tissue.

Compound Follicular (figs. 122 and 123).

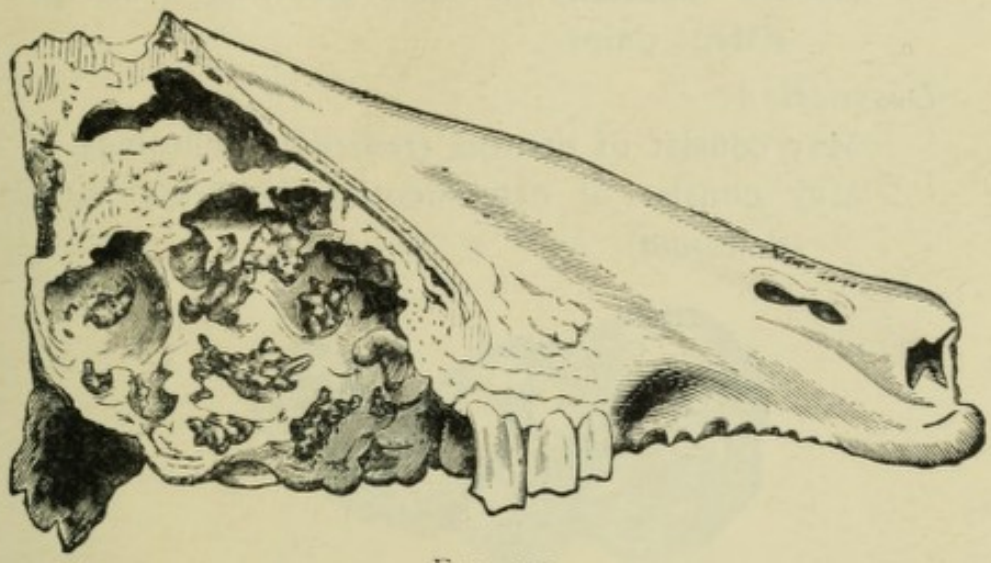

Fig. 122.
Follicular odontome in maxilla of a thar, after *Bland-Sutton*.

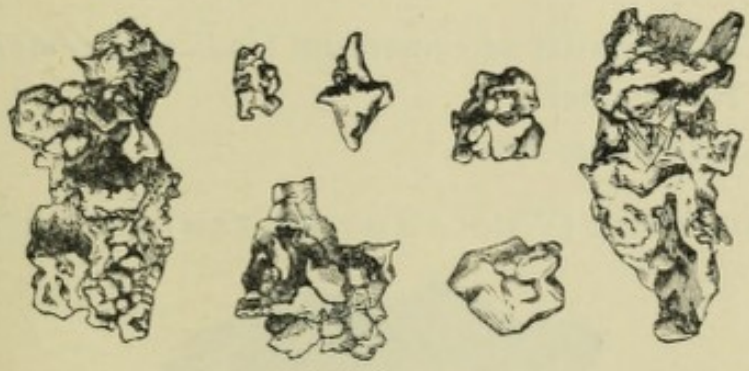

Fig. 123.
Seven denticles from the cyst shown in fig. 122.

Site :—

Rare in man.

Common in horses and goats.

Description :—

Like follicular, but containing more than one tooth or portions of teeth.

Radicular (fig. 124).

Site :—

Root of tooth only (odontome is formed after crown develops).

Common in lower animals.

Occurs especially in teeth growing from persistent pulps.

Description:—

May consist of dentine (*radicular dentoma*).

May consist of osteo-dentine (*radicular osteo-dentoma*).

FIG. 124.
Radicular odontome (R.C.S. Museum). (*Trans. Odont. Soc.*)

May consist of cementum (*radicular cementoma*).

No enamel.

Sometimes bone.

COMPOSITE (WHOLE TOOTH GERM) (fig. 125).

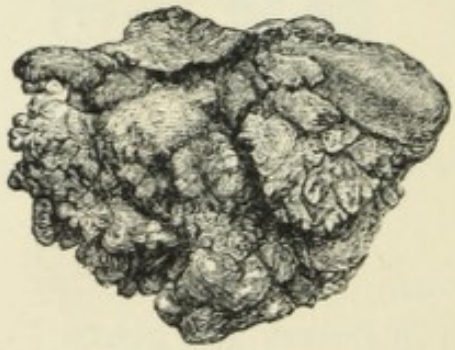

FIG. 125.
Mr. Christopher Heath's case of a composite odontome.

Site:—

Probably confined to man.

Description:—

Consists of masses of all the dental tissues.

May be of any shape.

Very rare.

Symptoms (generally) *of Odontomes:*—

Slow growth.

Not noticed (probably) until the 15th—20th year.

Painless (usually).

May be neuralgic pain.

Often pain with *radicular*.

Pain, if inflammation and suppuration occurs.

Absence of tooth (not always, as it may be connected with a supernumerary tooth).

Expansion of jawbone, due to absorption inside and deposition outside.

Treatment:—

Removal of growth.

If cystic—

Open up.

Scrape cyst walls.

Break down any loculi.

Evacuate contents.

Keep wound well drained.

Pack with antiseptic gauze.

Allow wound to heal from base.

CHAPTER XIII.

ODONTALGIA AND NEURALGIA.

Odontalgia.

Definition.—Pain in or around a tooth.

Varieties:—

(i) Local (more frequent). Pain in or around a tooth which is itself the cause.

(ii) Referred. Pain in or around a tooth which is not itself the cause.

Note.—Pain may be referred from an upper to a lower tooth on the same side, or *vice versa*, but as the nerves do not cross the median line pain can never be referred to the opposite side.

Causes:—

(i) Affections of the pulp.

(ii) Affections of the alveolar dental periosteum.

(iii) Affections of the periosteum.

(iv) Affections of the mucous membrane and submucous tissue.

(v) Irritation of the 5th cranial nerve, *e.g.*, impacted lower 3rd molar.

Symptoms.—Depend on cause.

Treatment.—Depends on cause.

Neuralgia (Trigeminal or Trifacial).

Definition.—Pain in the course of the 5th cranial nerve (fig. 126), or in the area of its distribution.

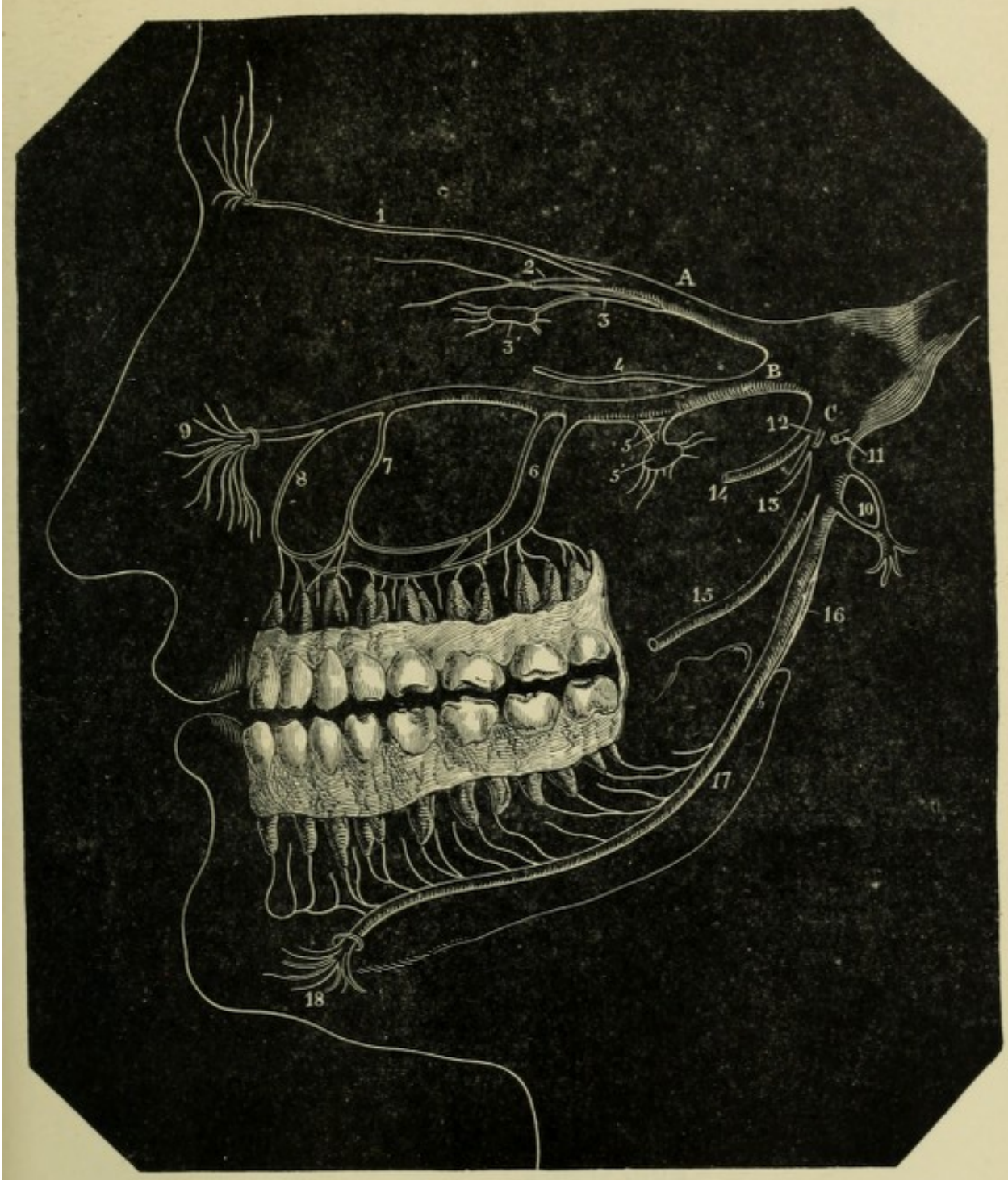

FIG. 126.

Diagram of the Distribution of the Branches of the 5th Nerve.

(From *Tomes's* "Lectures on Dental Physiology and Surgery"—drawn by the late *Mr. C. de Morgan.*)

A, Ophthalmic division: 1, frontal; 2, nasal and long ciliary; 3, branches to ciliary ganglion.

B, Superior maxillary division: 4, orbital $\left\{\begin{matrix}\text{temporal}\\\text{malar}\end{matrix}\right\}$ 5, spheno-palatine (Meckel's) ganglion; 6, posterior dental, passing down; 7, 8, anterior dental; 9, infra-orbital.

C, Inferior maxillary division; 10, auriculo-temporal; 11, masseteric; 12, deep temporal; 13, pterygoid; 14, buccal to buccinator, &c.; 15, gustatory; 16, myelo-hyoid branch; 17, inferior dental; 18, mental.

(From *Tomes's* "Dental Anatomy." Fifth edition. By permission of *Messrs. J. and A. Churchill*, the publishers.)

Varieties:—

(i) Minor (simple).
(ii) Major (tic douloureux, epileptiform neuralgia, or prosopalgia).

(i) Minor (simple).

NOTE.—This is not a disease but a symptom of some local disorder.

Age.—Occurs between ages of 20 and 60.

Sex.—More often in women.

Causes:—

Predisposing.

Neurosis.
Unhygienic surroundings.
Mental or physical overwork.
Impairment of general health.
Sedentary life.
Following convalescence from illness.
Worry and anxiety.
Senility, &c.

Exciting:—

(*a*) *Peripheral, e.g.,*

Caries.
Degenerations of pulp (usually chronic).
Secondary dentine.
Calcification of tissue of the pulp.
Pulp stones.
Difficult eruption, especially of 3rd molar.
Absorption of 2nd molar by erupting 3rd molar.
Exposure of cementum or dentine.
Abrasion.
Attrition.
Erosion.
Fracture.

Fillings too near to the pulp.
Morbid conditions of alveolar dental periosteum (especially suppurative).
Rarefying periodontitis.
Productive periodontitis.
Inostosis.
Morbid conditions of the gums.
Recession of gums exposing root.
Morbid conditions of periosteum, *e.g.*, periostitis of bony canals, causing pressure on nerve.
Morbid conditions of mucous membranes of mouth, nose, or antrum.
Affections of skin.
Affections of eye.
Affections of uterus, &c.

(*b*) *In the course of the nerve, e.g.*,
Injury in extracting a tooth.
Injury in excising apex of a root (especially in lower jaw).
Inflammation of nerve.
Degeneration of nerve.
Pressure on nerve, *e.g.*, tumour.
Cicatrix involving nerve, &c.

(*c*) *Systemic, e.g.*,
Bright's disease.
Locomotor ataxia.
Epilepsy.
Gout.
Anæmia.
Rheumatism.
Malaria.
Syphilis.
Influenza.
Lead poisoning, &c.

(*d*) *Cerebral, e.g.*,
Hysteria.
Degenerations.
Tumours.

(*e*) *Idiopathic* (when there is no discoverable cause).

Sites when due to Dental Cause:—

Upper incisors	... ...	supra-orbital area just to one side of median line.
Upper canine ... 1st upper pre-molar	... ...	infra-orbital region.
2nd upper pre-molar	... ...	temple. maxillary area.
1st upper molar	... ...	malar region.
2nd upper molar	... ...	mandibular area, chiefly over ascending ramus.
3rd upper molar	... ...	mandibular area, chiefly over ascending ramus. part of ear. occasionally hyoid region.
Lower incisors ... ,, canines ... 1st lower pre-molar	... ...	mental area.
2nd lower pre-molar	... ...	mental area. sometimes hyoid region.
1st and 2nd lower molars	... ...	ear. angle of jaw. hyoid region.
3rd lower molar	... ...	neck, in superior laryngeal region.

Symptoms:—

Pain often sharp, stabbing, and burning,
often agonizing,
sometimes dull,
often intermittent, sometimes persistent,
increases in intensity, and then decreases as attack ceases,
may be referred to breast, axilla, shoulder, arm, or other part of affected side.

Cold, noise, or draught of air may bring on pain.

There may be pain on palpation over bony canal.

Part hypersensitive.

May be tenderness, œdema, or hyperæmia of the part.

Skin may be red or swollen.

Often excessive secretion of saliva.

Often copious flow of tears.

In later stage the touching of the affected area with the blunt end of a pin discovers "*tender points.*"

(ii) Major (*tic douloureux, epileptiform neuralgia*, or *prosopalgia*).

NOTE.—This is probably a definite disease of the nervous system.

Age.—Occurs usually between the ages of 30 and 40.

Sex.—More often in males.

Cause.—Not known. May be degeneration of cells in Gasserian ganglion, due to dental troubles, such as sepsis in mouth, &c., or some other cause not dental, *e.g.*, tumour in neighbourhood of Gasserian ganglion, &c.

Symptoms:—

Usually begins in infra-orbital or inferior dental branch.

Pain very severe and paroxysmal.
Often sweatings of face,
muscular twitchings,
copious flow of tears,
and excessive secretion of saliva.
Characteristic emotionless facial expression due to fear of any movement bringing on pain.
Tongue coated.
Sordes covering teeth.
Fœtid breath.
(These last three symptoms are due to patient keeping muscles, &c., still.)
Symptoms usually confined to one side.
Occasionally pain in eyeball, ear, and centre of hard palate.

Diagnosis of Neuralgia:—

Discover site of pain.
Discover character of pain.
Look carefully with a probe and mirror for carious cavities.
Examine carefully all fillings, especially large metal fillings, crowns, and bridge work.
Test each tooth with a hot instrument, and with a small piece of cotton-wool soaked in cold water. With teeth containing inflamed and degenerated pulps acute paroxysms of pain are set up.
Test for periodontitis.
Look for exposed roots.
Use X-rays to examine filled roots, exostosed roots, &c.
Examine for troublesome erupting 3rd molar.
Examine for periostitis by palpating over orifices to bony canals.
Should there be no dental trouble suspect tumour, constitutional disturbance, &c.

Treatment of Neuralgia:—

Remove any dental cause.

General health must be improved.

Advise holiday.

Freedom from care and worry.

Good food.

Fresh air.

Should the cause not be dental, the patient's medical adviser should be consulted.

Tonics used in Treatment:—

℞ Chloride of ammonium ... ... ʒiij.
Tincture of lemons ... ... ʒiij.
Spirits of chloroform ... ... ʒiss.
Water to ... ... ʒvi.

Two tablespoonfuls every three hours.

Phenacetin in 5-10 gr. doses.

In *malaria*, quininæ sulphas in 1-10 gr. doses or liquor arsenicalis (*Fowler's* solution) in 7 minim doses.

In the *weak* and *debilitated*, cod-liver oil in 1-4 drachm doses directly after meals.

In *syphilis*, potassium iodide in 10-15 gr. doses every four to six hours.

In *anæmia*, liquor arsenicalis (*Fowler's* solution) in 7 minim doses.

or

℞ Liquor ferri perchloridi fortis ♏xv.
Aqua ... ... ʒj.

three times daily.

In *rheumatism:—*

℞ Potassium iodide ... ... gr. xlviij.
Sodium salicylate ... ... ʒxv.
Oil of gaultheria ... ... ʒij.
Acacia mixture q.s. ... ad. ʒvj.

A dessert-spoonful, well diluted, every three hours.

Local Applications:—

Menthol.

Cantharides.

Capsicum.

℞ Liniment of belladonna ... ʒvj.
Tincture of aconite ... ʒvj.
Chloroform ... ... ʒiv.

or

℞ Chloral hydrate ... ... ʒij.
Camphor ... ... ʒij.

Neuralgic Specifics:—

Tinc. gelsemium sempervirens, 10 minims every hour up to four hours.

Veratrina $\frac{1}{60}$th-$\frac{1}{20}$th of a grain.

Butyl chloral hydrate in 5 gr. doses every hour until 30 gr. taken.

Tincture of aconite in 1 minim doses every hour.

Bromidia in 1 drachm doses.

Hypodermic Injections:—

Morphia in $\frac{1}{3}$ to $\frac{1}{2}$ gr. doses.

Cocaine in $\frac{1}{2}$ to 1 gr. doses.

Osmic acid 1-2 drops of a 1 per cent. solution.

℞ Beta-eucaine ... ... gr. ij.
Absolute alcohol ... ... ʒvj.
Water to ... ... ʒj.

Should all the above means fail *electricity* may be adopted, or one of the following surgical operations may be necessary:—

Neurotomy (nerve stretching).

Neurectomy (excision of part of nerve).

Removal of the Gasserian ganglion.

Write this out. to page 109

CHAPTER XIV.

EMPYEMA OF THE MAXILLARY ANTRUM.

Definition:—

A collection of muco-pus pent up in the maxillary antrum (fig. 127).

Causes:—

(*a*) Infection from nose and frontal and ethmoidal sinuses.

(This may follow on exanthemata and influenza.)

(*b*) Infection from roots of teeth (alveolar abscesses, &c.).

According to *Underwood* the 3rd molar is always intimately related with the antrum, and the canine practically never.

Tomes mentions the canines, pre-molars, and the 1st and 2nd molars as being in close relation with the antrum. In the author's experience the 2nd pre-molars and 2nd molars are usually the cause, if dental.

(*c*) Infection from outside wound (rare).

(*d*) Infection from neighbouring fractured or necrosed bone following on traumatism, syphilis, &c.

Varieties:—

(i) Acute.

(ii) Chronic.

(i) Acute empyema.

Symptoms:—

Much pain in region of affected part, especially if *ostium maxillaire* is closed through swelling of mucous membrane.

Distension in zygomatic region.

Cheek swollen.

Cheek may be red and tender.

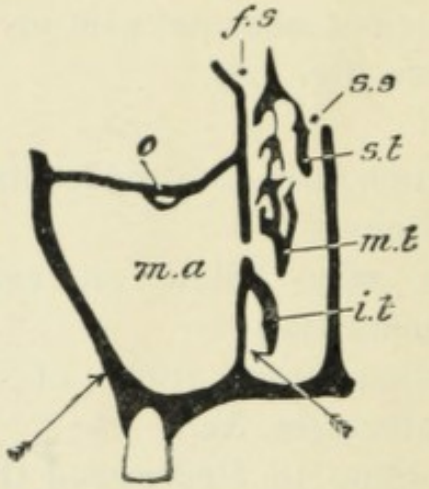

FIG. 127.

From *Spencer and Gask's* "The Practice of Surgery," by permission of *Messrs. J. and A. Churchill*, the publishers.

The Maxillary Antrum.

i.t, *m.t*, *s.t*, inferior, middle and superior turbinals; *m.a*, maxillary antrum opening into middle turbinal fossa; *f.s*, frontal sinus and infundibulum, into which open anterior ethmoidal cells; *s.s.* orifice of sphenoidal sinus; *o*, infra-orbital canal; arrows show the points at which the antrum is punctured.

Deep-seated pain.

Pain on pressure.

Throbbing pain after exertion or stooping.

Often supra-orbital neuralgia.

Copious discharge of pus into nose if *ostium maxillaire* not blocked.

Pus foul.

Patient may state that pus comes from throat on account of its passing back into naso-pharynx.

Floor of orbit may be pushed up and the eyeball be displaced.

Often feverish symptoms.

Diagnosis:—

Nasal speculum discovers pus in middle meatus.

Transillumination is often useful.

Place a small electric lamp in patient's mouth, have the lips closed, and darken the room. On the affected side, in comparison to the unaffected,

(i) the pupil is badly illuminated,

(ii) the light is not thoroughly appreciated,

(iii) there is not much light visible around lower edge of orbit, and

(iv) not much light passes through cheek.

Puncturing of antral wall from inferior meatus of nose discovers pus.

Treatment.—If due to dental cause:—

Extract tooth (the 1st molar for preference).

If all teeth sound open up through canine fossa.

Enlarge opening (if 1st molar removed, enlarge in region of anterior buccal socket).

Syringe twice daily with a warm normal saline solution containing a small quantity of sodium bicarbonate.

In the intervals close with a rubber plug.

Gradually shorten plug and eventually remove it, allowing the opening to heal by granulation.

If the cause is nasal—

Puncture from the inferior meatus through lower part of antral wall (fig. 127), and syringe antrum twice daily intra-nasally.

(ii) Chronic empyema.

Symptoms:—

Vague.
Scanty discharge of pus.
Pus swallowed.
Pus foul-smelling to patient.
Differs from *ozæna*, where odour is not noticed by patient.
Not much pain.
No bulging of antral walls.
One-sided frontal headache.
Fulness and tenderness over antrum.
Pus drains down nose when patient lies on opposite side.
Referred pains to frontal region, teeth, or over affected antrum.
Distension if *ostium maxillaire* is blocked.
If of long standing there is oral sepsis.

Diagnosis:—

As in acute.

Treatment:—

Remove tooth, or open up through canine fossa as in acute.
Enlarge opening sufficiently to allow of scraping the inflamed, thickened, and hypertrophied walls of the antrum.
Syringe with warm normal saline solution containing a little sodium bicarbonate.
Scrape walls of antrum.
Pack with lint dipped in 1-20 carbolic.
Syringe and pack daily.
When the packings are removed clean, dispense with them.
Insert rubber plug, and by degrees shorten and remove it, when the opening heals by granulation.

Caldwell Luc's operation consists in making a large opening through the canine fossa, removing the greater part of the naso-antral wall, including the anterior half of the inferior turbinate bone, scraping the walls of the antrum, suturing the bucco-antral wound, and syringing twice daily intra-nasally.

Sequelæ of Empyema Antri when Neglected:—

Bursts on cheek.

Necrosis of upper jaw.

Septic absorption and pyæmia.

Septic thrombosis of orbital veins producing proptosis or acute optic neuritis.

Thrombus may extend to cavernous sinus and set up meningitis.

Write out to 226.

CHAPTER XV.

NECROSIS OF THE JAWS. (FIG. 128).

Definition.—Death of bone of the jaw.

Site.—In compact tissue usually.

Usually in lower jaw, because:—

Its recuperative power is less.

It consists chiefly of compact tissue.

Its blood supply is less than in the upper jaw.

It is more exposed than the upper jaw.

Cause.—Cutting off of blood supply to the part through:—

(i) *Bacterial infection* from any of the following:—

Traumatism (fracture, &c.).

Disease of tooth (periodontitis, alveolar abscess, &c.).

Exanthematous fevers.

Scorbutis.

Syphilis.

Diseases of gums and mucous membranes (gangrenous stomatitis, ulcerative stomatitis, &c.).

Tubercle.

Pyæmia.

Actinomycosis.

(ii) *Chemical poisons:*—

Arsenic.

Mercury.

Phosphorus.

Symptoms:—

Those of inflammation.

May be slight, as in simple cases after extraction.

Pain in early stages simulates periostitis.

In severe cases the following are present:—

Thickening of gums.

Gums tumid and deep red.

Great swelling of surrounding parts.

Face swollen.

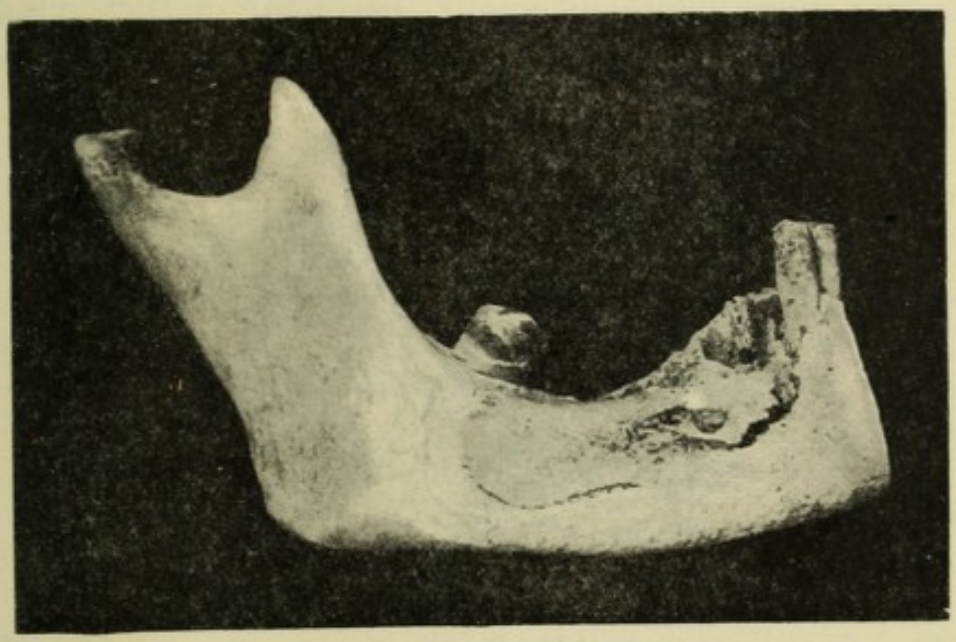

FIG. 128.

From *Colyer's* "Dental Surgery and Pathology," by permission of *Messrs. Longmans, Green and Co.*, the publishers.

Necrosis of the mandible involving the body of the bone. The necrosis was due to sepsis in connection with the canine.

Suppuration and formation of sinuses.

Oozing of pus from margins of gums.

Separation of gums from alveolus.

Exposure of bone.

Difficulty in opening mouth.

Loosening and loss of teeth.

Detachment of dead bone from living, forming a *sequestrum* or *exfoliation* (fig. 129).

Characteristic fœtid odour.
Feverish symptoms.
May terminate fatally.

Characters of necrosed bone:—

Hard.
Rough.
Porous.
Dull sound on tapping with a probe.
Lighter in weight than healthy bone.
First white in colour, becoming darker on exposure to the air and decomposing material.

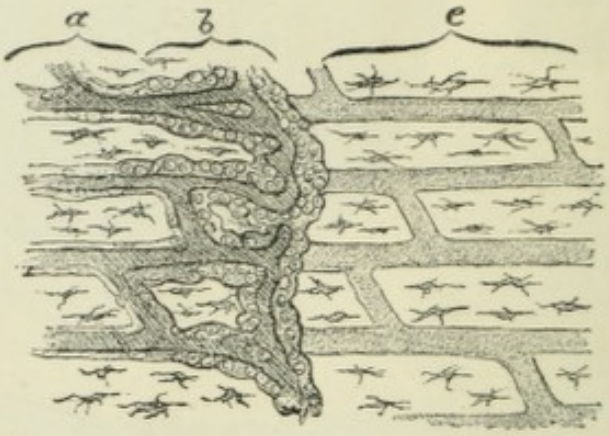

FIG. 129.

From *Spencer and Gask's* "The Practice of Surgery," by permission of *Messrs. J. and A. Churchill*, the publishers.

Diagram of the process of separation of dead bone: *a*, healthy living bone; *b*, inflamed living bone with formation of granulation tissue where it is in contact with the dead part; *c*, dead bone.

Pathology:—

Inflammation of gums.
Ulceration of gums.
This spreads to periosteum and bone.
Gum separated from bone.
Pus burrows under periosteum.
Periosteum raised up and blood supply cut off from bone.

Bone becomes necrosed.
Ulceration around necrosed bone.
Line of separation formed between dead and living bone.
This is filled in by granulation tissue (fig. 129).
Sequestrum forms.

Process of Reparation.—New bone (*involucrum*), developed from the periosteum, forms around *sequestrum*. It is poorly developed, being pierced by holes (*cloacæ*).

NOTE.—The lower jaw is readily repaired by a new formation of bone. The alveolar portions of the jaws and the upper jaw are not replaced by bone, but by fibrous tissue.

Treatment:—

Remove cause.
In early stages apply hot poppy-head fomentations inside the cheek.
Remove any hopelessly loose teeth and septic roots.
Incise freely down to the bone inside the mouth as soon as pus forms.

NOTE.—The upper jaw drains easily. This is not so with the lower jaw, and *Mr. Fairbank* therefore advises an external incision for free drainage in cases of necrosis of this bone.

Connect sinuses.
Irrigate sinuses with antiseptics.
Advise warm antiseptic mouth-washes, such as—
Dilute permanganate of potash.
Phenol sodique (one teaspoonful in a half glassful of water).
A 3 per cent. solution of peroxide of hydrogen or pyrozone, or

℞ Chloride of zinc ... ... gr. v.
Distilled water ... ... ℥j.

Wait for sequestrum to loosen and then remove.
Purge.
Give large doses of potassium iodide.
Nourishing diet.
Fresh air.
Tonics.

PHOSPHORUS NECROSIS (PHOSSY JAW).

Cause.—Exposure to the fumes of *yellow crystalline phosphorus*,
In match making,
When it is taken as a drug, &c., &c.
Several years of exposure to the fumes are necessary.
Healthy mouths are not affected.
The presence of a wound or a septic tooth is essential.
It is probably a local manifestation of general phosphorus poisoning.
Some authorities assert that the phosphorus lowers the general health and that the *tubercle bacillus* is the cause of the necrosis.
Red phosphorus, which is now used in match-making, is not poisonous.

Site:—
Body of jaw.

Symptoms:—
As in necrosis but more severe.
Not rapid.
Slight and then severe toothache.
Gums red and swollen.
Swelling increases greatly.
Excessive suppuration.
Severe constitutional symptoms.

Character of Bone:—

Characteristic pumice-like deposit on necrosing bone in mandible, but not in maxilla.
Haversian canals larger than in normal bone.
Haversian canals at right angles to general direction of bone.

Treatment:—

As in necrosis.
Remove from environment.
Dispense with yellow phosphorus.
Do not wait for *sequestrum* to form, but remove necrosed bone sub-periosteally.

Exanthematous Necrosis.

Occurs in children between the 4th and 10th years associated with:—

Scarlet fever (usually).
Measles (less often).
Small-pox (rarely).

Opinions differ as to whether it occurs during the acute stage of the fever or during convalescence.

Cause:—

Secondary infection with pyogenic organisms—
Through the blood,
through exposure of bone by ulceration, or
through injury to the gums in brushing the teeth or examining the throat with instruments.

Site:—

Alveolus.
Lower jaw.
Labial side of incisors. } Usually.

Buccal side of molars.
Buccal side of premolars. } Less frequent.

Symptoms:—

Insidious.
Begins as a periodontitis.
Usually no pain.
Very fœtid breath.
No swelling.
Teeth tender.
Pus oozes between gums and teeth.
Teeth loosen and may fall out.
Vertical stripping of gums from alveolus.

Treatment:—

As in necrosis.
Care and cleansing of temporary teeth.
Antiseptic mouth-washes.

CHAPTER XVI.

FRACTURES OF THE JAWS.

Definition.—A fracture is the sudden solution of continuity of a bone.

Causes.—(i) Traumatism.

(*a*) *Direct*, when the fracture occurs at the point where the violence is applied, *e.g.*:—

Blow.
Kick.
Extraction.
Gun-shot wound, &c., &c.

(*b*) *Indirect*, when the fracture occurs at a point away from the place where the violence is applied, *e.g.*:—

Falling from a height on to the heels.
Blow on the chin, &c., &c.

(ii) Disease or weakness of bone, *e.g.*:—

Necrosis.
Caries.
Former Fracture, &c., &c.

(iii) Muscular action, *e.g.*:—

Yawning.
Coughing, as in fracture of coronoid process.

Varieties:—

(i) Simple (skin not broken). Usually so in connection with ascending ramus.

(ii) Compound (open to air through wound in skin and soft parts). Usually so in connection with horizontal ramus.

(iii) According to line of fracture, *e.g.:—*
Transverse (in ascending ramus; sometimes alveolar portion and teeth separated from body of jaw).
Vertical (usually at symphysis in the young).
Oblique (usually in horizontal ramus).
Stellate (radiated or star-like).

(iv) According to condition, *e.g.:—*
Impacted (one piece forced and fixed into another).
Fissured (no displacement, but a crack runs through bone).
Depressed (one piece is forced below surface).
Punctured (perforated).
Splintered (small piece broken off).
Complicated (other part, such as nerve, artery, or joint implicated).

(v) According to extent, *e.g.:—*
Complete.
Incomplete or greenstick (bone partially broken and bent. Occurs in the young).
Comminuted (broken into several pieces. Produced by gun-shot wounds).
Multiple (two or more distinct fractures in same or different bones).

Sites.—Lower jaw usually.

Alveolus (common in tooth extraction).
Body of jaw.
Behind canine (due to depth of socket).
Angle.
Between first and second molars.
Where bone weakened through loss of teeth.
Where bone weakened by disease, *e.g.*, necrosis.
Where bone weakened by former fracture.
Symphysis, in the young (rare).
Ascending ramus (uncommon).
Neck of condyle (uncommon).
Coronoid process (very rare).

Upper jaw less often:—

Alveolus (common in tooth extraction).
Incisor region usually (when due to blow, &c.).
Nasal, ethmoid, and malar bones may be involved.

Signs and Symptoms:—

Only slight where portions of alveolus involved after extraction. In severe cases—
History of injury.
Knowledge of sharp, loud crack.
Patient usually tries to steady part with hand.
Speech often incoherent.
Pain often severe.
Pain on opening mouth.
Swelling.
Mucous membrane often torn.
Partial loss of function.
Hæmorrhage.
Salivation.
Undue mobility of part.
Line of teeth often altered.
Displacement and deformity.
Sometimes inability to close mouth.
Obscure when in ascending ramus.

Displacement:—

Position of fracture	Nature of displacement	Muscle causing displacement
Canine region	Smaller portion usually overlaps larger.	
	Broken portions usually displaced towards opposite sides	External pterygoids.
	The lesser portion is drawn up.....	Temporal and masseter.
	The larger portion is drawn down	Genio-hyoid and digastric.
Multiple fracture on both sides of symphysis	Middle portion is considerably depressed and drawn backwards	Genio-hyoid and digastric.
	Lateral portions are drawn forwards and outwards	Temporal and masseter.
Body of bone in front of angle	Posterior part of bone raised	Masseter.
Condyle	Condyle of injured side drawn upwards, forwards, and inwards. The bulk of the jaw is pushed towards the injured side. This must be diagnosed from unilateral dislocation where the jaw is pushed away from the seat of injury	External pterygoid.
Coronoid process	The small piece is probably drawn upwards and backwards, and is felt inside mouth...	Temporal.
Ascending ramus	Little displacement. Upper fragment may be drawn forwards...	Temporal.
Bone between masseter and internal pterygoid	No displacement, the pieces being held in apposition	Masseter and internal pterygoid.

Treatment:—

Wash out mouth with lotion of peroxide of hydrogen or permanganate of potash.
Stop hæmorrhage.
Remove any loose bone or teeth between fragments.
Remove septic or hopelessly loose teeth.
Scale teeth.
Reduce displacement as far as possible.
Carefully take an impression in soft wax.
Apply a gutta-percha splint (figs. 130 and 131), and, if necessary, a four-tailed bandage (figs. 132 and 133) temporarily.
Cast model.
Saw through model at point indicating line of fracture.
Join two halves together again in what is considered to be normal position.
Make and adjust splint.
Avoid movements of jaw.
Slop diet.
Keep mouth clean. Some surgeons advise feeding *via* anus for a few days on account of great difficulty of otherwise keeping mouth clean.
If external wound, keep clean with peroxide of hydrogen and antiseptic dressing.
Leave splint on for about six weeks.

Bandages and Splints used in Treatment of Fracture of Jaws.

Gutta-Percha Splint (fig. 130).

Four-tailed Bandage (fig. 132):—

Make a linen bandage 4 in. wide and 1 yd. long.
Slit it at each end (fig. 132A).
Leave 8 in. in the middle undivided (fig. 132B).

Make a slit in the middle of the undivided portion (fig. 132C) for the reception of the chin.

Lay on the chin.

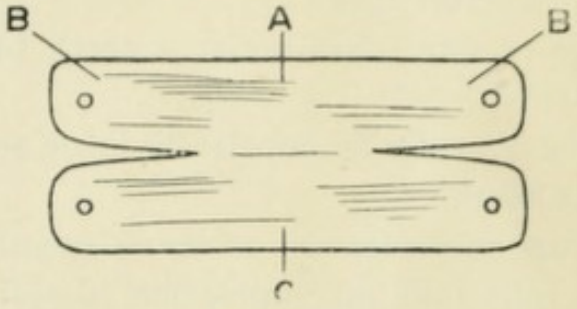

FIG. 130.

Gutta-percha splint. Shape gutta-percha as above, soften in hot water, and then place A under chin, bend B up over ascending ramus, and then bend C as in fig. 131.

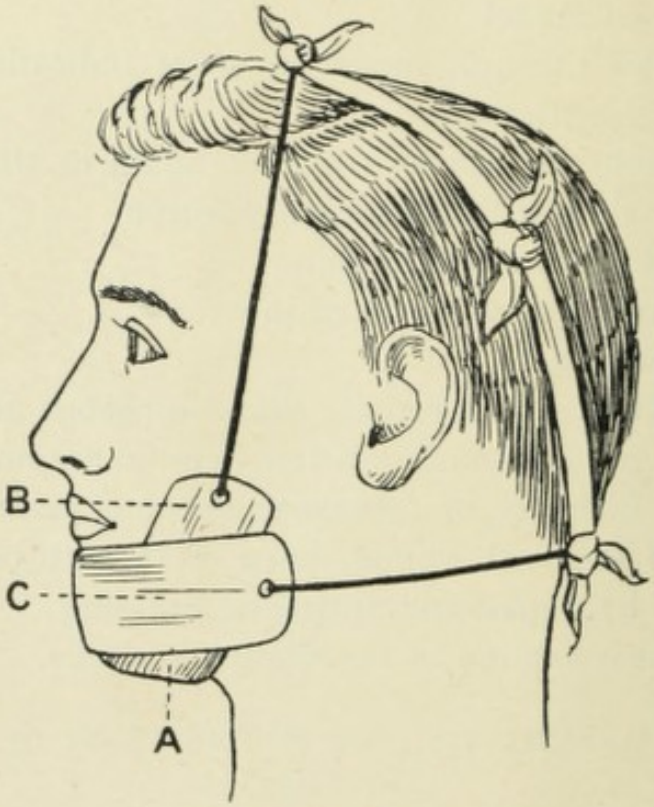

FIG. 131.

Gutta-percha splint applied. For explanation see fig. 130.

Tie the two lower over the head (fig. 133A).

Tie the two upper tails below the occiput (fig. 133B).

Knot the two tails together (fig. 133C).

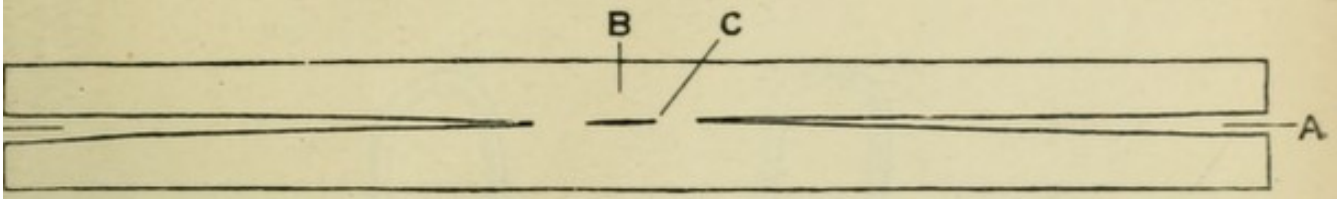

FIG. 132.

Four-tailed bandage. A, slit at each end; B, undivided eight inches; C, slit for chin.

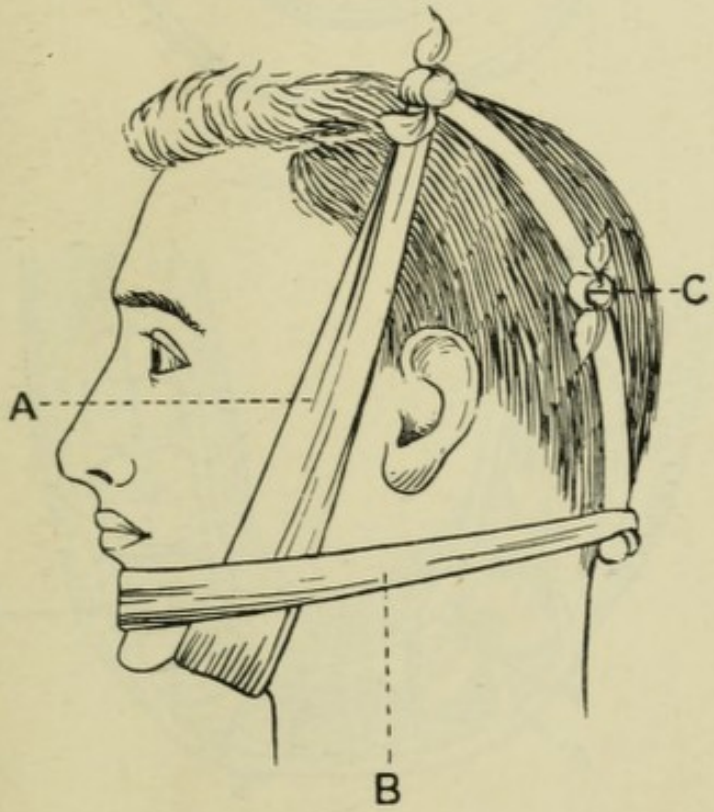

FIG. 133.

Four-tailed bandage applied. A, lower tail of bandage; B, upper tail of bandage; C, upper and lower tails tied together.

HAMMOND WIRE SPLINT (figs. 134 and 135).

Description:—

Made of soft iron or silver wire.

Adapted to both lingual and labial sides of teeth.

Fitted as accurately as possible to model.

Is fixed in the mouth with ordinary iron binding wire (fig. 135B).

NOTE.—Pass binding wire over outer bar, under inner bar, back over inner bar and under outer bar. Then twist the two ends and bend in to prevent irritation. Ligature, in this way, to several teeth in each fragment.

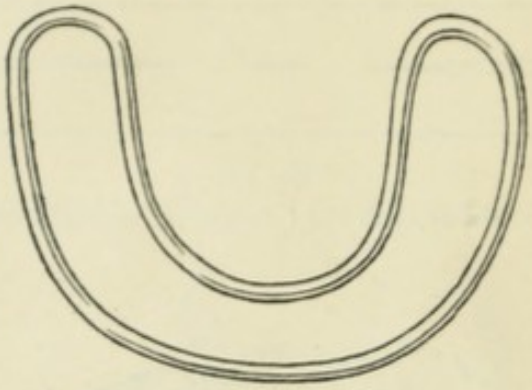

FIG. 134.
Hammond wire splint.

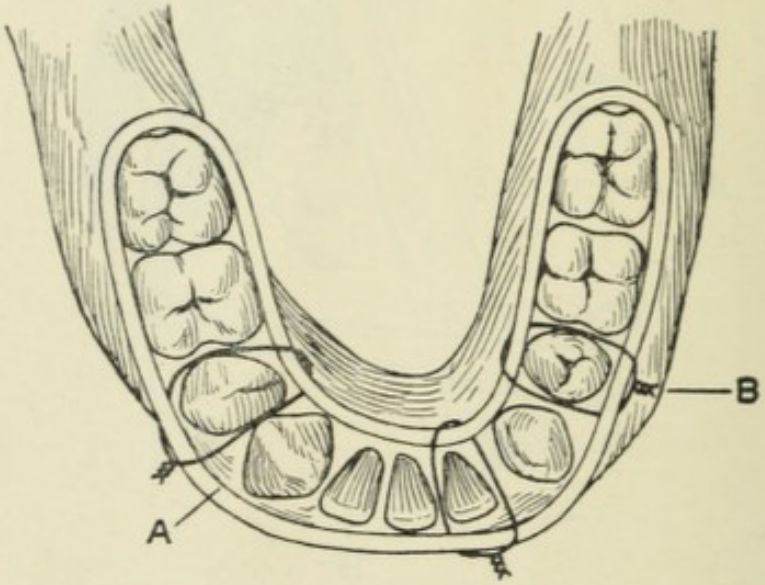

FIG. 135.
Hammond wire splint applied. A, wire splint; B, iron binding wire fixing splint to teeth.

Suitable cases for its use:—

Where firm teeth in each fragment.
Single fracture in front of molar region.

Advantages:—

Cleanly.
Not cumbersome.
Efficient.
Mastication and speech not greatly affected.

Disadvantages:—

Cannot be used in edentulous cases.
Cannot be used unless several firm teeth in each fragment.
Cannot be used where vertical displacement.

KINGSLEY OR HAYWARD SPLINT (fig. 136).

Description:—

A cap, to cover teeth, made of vulcanite.
It is lined with gutta-percha before applying.
Attached to the cap are two strong recurved iron wires supplied with points to secure bandage (fig. 136).

Advantages:—

Can be used in edentulous cases.
Can be used where few or loose teeth.
Can be used where there is vertical displacement.

Disadvantages:—

Uncomfortable; patient cannot lie on side.
Often displaced during sleep.
Uncleanly.
Mastication and speech affected.

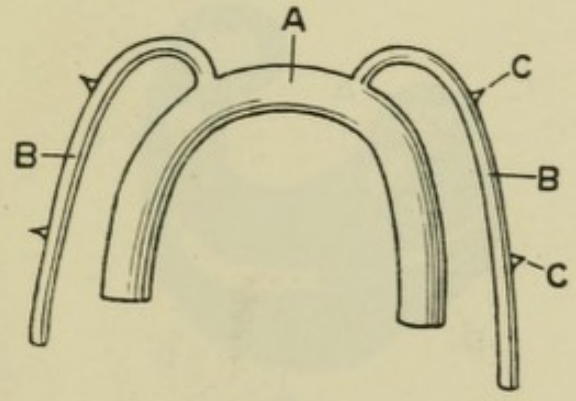

FIG. 136.

Hayward splint. A, vulcanite cap lined with gutta-percha to cover teeth; B B, recurved strong iron wires which pass out at angles of mouth; C C, hooks to hold bandage.

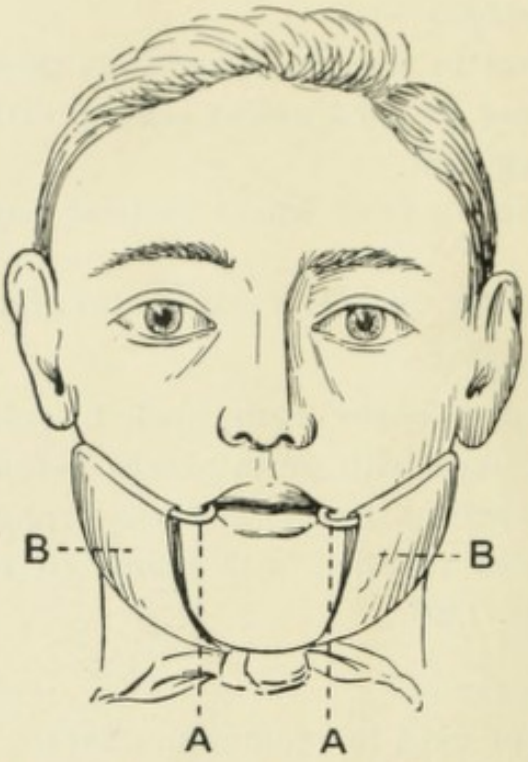

FIG. 137.

Hayward splint applied. A A, strong iron wires; B B, bandage tied under chin.

GUNNING SPLINT (fig. 138).

Description:—

Consists of upper and lower vulcanite cases joined together.

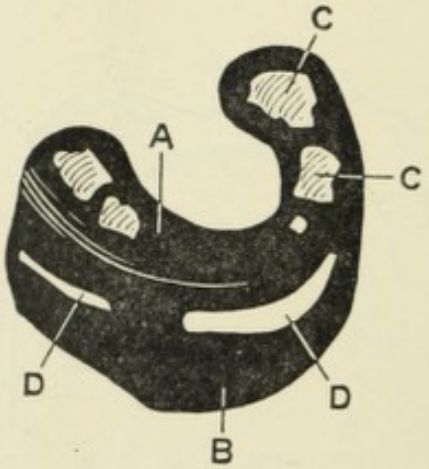

FIG. 138.

Gunning splint: A, vulcanite upper lined with gutta-percha, joined to B; B, vulcanite lower lined with gutta-percha; C C, impressions for upper teeth; D D, holes through which food is passed and mouth cleansed.

Spaces (fig. 138D) are left through which food can be passed and the mouth cleansed.

It is fixed with a four-tailed bandage.

Advantages:—

Useful in fractures of both jaws.

Useful in fractures behind last molar.

Useful in multiple fractures.

Useful in edentulous cases.

Useful where few or loose teeth.

Disadvantages:—

Difficult to adjust.

Difficult to keep mouth clean.

Difficult to remove.

Mastication and speech affected.

HERN SPLINT (fig. 139).

Description:—

A modification of *Gunning's*, consisting of a lower vulcanite plate only, with pillars for articulation with upper teeth.

It is perforated for cleansing purposes.

It is used in conjunction with a four-tailed bandage.

Advantages:—

Useful in fractures behind last molar.

Useful in multiple fracture of mandible.

Useful where few or loose teeth.

Useful in edentulous cases.

More cleanly than *Gunning's*.

More easily adapted and removed than *Gunning's*.

Not so cumbersome as *Gunning's*.

Upper teeth can be easily adjusted to pillars with the aid of gutta-percha.

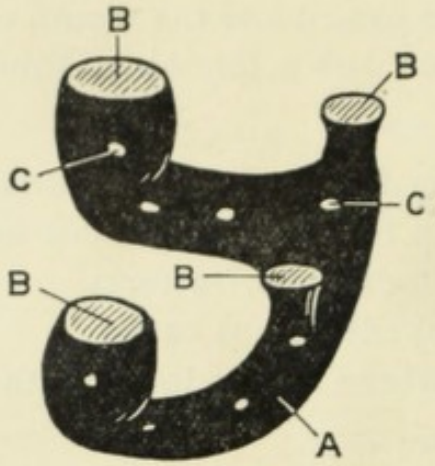

FIG. 139.

Hern splint: A, vulcanite splint; B B B B, anterior and posterior pillars with cups for gutta-percha for upper teeth; C_C, perforations for cleansing.

Disadvantages:—

Not easy to keep mouth clean.
Mastication and speech affected.

CRADLE SPLINT OF LEWIN PAYNE.

Description:—

Consists of two *Hammond's* splints, one for the upper and the other for the lower, joined together by upright wires. It can be used in place of *Gunning's*, and is more cleanly.

TOMES' SPLINT.

Description:—

A metal cap made to fit over several teeth on each side of fracture.
It is cemented into place.

ANGLE'S METHOD OF BANDING (fig. 140).

Complications:—

Dislocation of temporo-mandibular joint (rare).

Dislocation of teeth.

Fracture of teeth.

Severe hæmorrhage, through tearing of artery (rare).

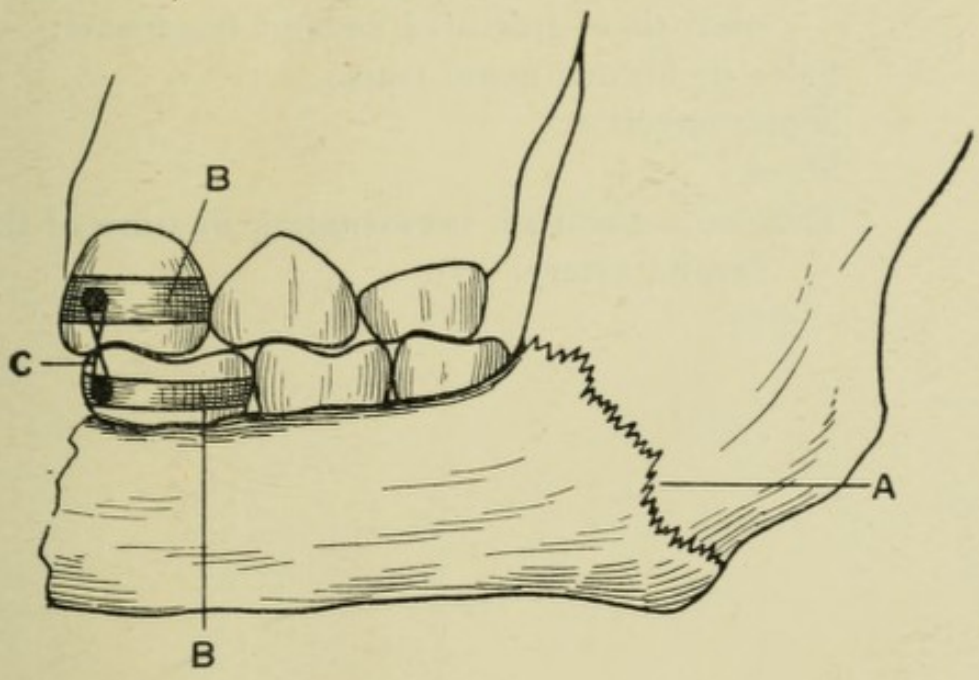

FIG. 140.

Angle's method of banding for fracture: A, fracture behind last molar; B, bands fixed around upper and lower teeth; C, wire between upper and lower bands preventing movement.

NOTE.—The general surgeon's method of repairing a fracture is to wire the fragments together.

Severe wounds of face, through gun-shot wound, kick of horse, &c.

Paralysis or neuralgia, through injury to inferior dental nerve, or its implication in or pressure from the callus, formed in the process of repair.

Necrosis of portions of alveolus or ends of fragments.

Abscess, due to splint, or following necrosis.

Salivary fistula, through abscess bursting externally.

Dyspnœa, through infiltration of blood into tissues about root of tongue.

Fracture of base of skull, through jaw being forcibly driven against it.

Non-union, through neglect of treatment, or necrosis of fractured ends of fragments.

False or fibrous union (rare).

Septic infection.

Shock.

Traumatic aneurism necessitating ligature of the carotid artery.

CHAPTER XVII.

DISLOCATION OF THE TEMPORO-MANDIBULAR JOINT (fig. 141).

Definition.—A partial or complete separation from one another of the articular surfaces of the bones entering into the formation of the temporo-mandibular joint.

Causes:—

(i) Traumatism.

Fall, blow, or kick on chin when mouth partially open.

Violent use of *Mason* or other gag in opening mouth.

Misplacement of prop in mouth for extraction during anæsthesia.

Too forcible extraction of lower teeth, &c.

(ii) Muscular action (spasmodic action of external pterygoid muscles).

Yawning.

Laughing heartily.

Shouting.

Vomiting.

Opening mouth widely as for impression taking, &c.

(iii) Predisposition.

Sex (more often in females).

Anæmic and weakly women.

Old edentulous women with atrophy of jaw.

Nature of displacement:—

Condyle and inter-articular cartilage drawn over eminentia articularis into zygomatic fossa.

Capsular ligament stretched, rarely torn.

Varieties:—

(i) Bilateral.

(ii) Unilateral.

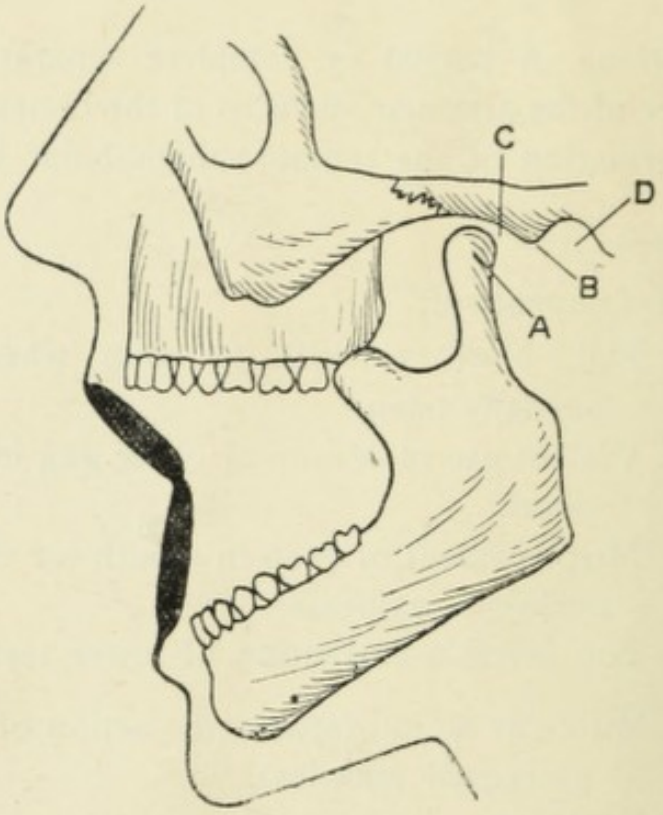

FIG. 141.

Dislocation of temporo-mandibular joint. A, condyle of lower jaw; B, eminentia articularis; C, zymogatic fossa; D, glenoid fossa in which the condyle naturally rests.

Signs and Symptoms:—

(i) Bilateral:—

Deformity.

Mouth open wide.

Jaw rigid.

Lower jaw protruded.

Severe dull pain.
Speech and deglutition affected.
Profuse and constant dribbling of saliva.
Hollows behind each condyle in positions usually occupied by the latter.
Condyles felt in abnormal positions in front of hollows.
Projections over zygomas due to constriction of temporal muscles.
Projections of coronoid processes below malar bones.
Displaced coronoid processes sometimes felt inside mouth.

(ii) Unilateral:—

Similar to above but confined to one side.
There is some amount of movement.
Symphysis is carried to opposite side.

Treatment:—

Place patient on a low chair.
Wrap your thumbs in napkins to prevent them being bitten.
Place thumbs on the last lower molars.
Place fingers under chin.
Press downwards and backwards with thumbs.
Press upwards with fingers.
Apply elastic four-tailed bandage for two weeks to prevent re-dislocation.
Warn patient to exercise care in opening mouth.
Another method is to—
Place cork between last molars on each side.
Forcibly elevate chin.
Apply bandage.

CHAPTER XVIII.

CLOSURE OF JAWS.

Definition.—An interference with the movements of the temporo-mandibular articulation.

NOTE.—*Trismus* is *spasm of the masticatory muscles*, closing jaws.

Causes:—

(i) Muscular spasm.
  (*a*) Reflex.
  (*b*) Disease, &c.
(ii) Mechanical.

(i) Muscular spasm:—

(*a*) Reflex.

Inflammatory conditions around jaw, *e.g.*, erupting 3rd molar, alveolar abscess, necrosis, &c. (*trismus*).

Inflammatory conditions of parotid gland, *e.g.*, specific parotitis (*mumps*), pyæmic abscesses, &c.

Synovitis or arthritis of temporo-mandibular joint.

Inflammatory conditions of mucous membranes of mouth, *e.g.*, ulceration from cancrum oris, syphilis, acute mercurial stomatitis, &c.

Inflammatory conditions of pharynx, *e.g.*, tonsillitis, quinsy, &c.

Inflammatory conditions of neck, *e.g.*, cellulitis, acute infection of upper cervical glands, &c.

Fracture of jaw.

(*b*) Disease, &c.

Tetanus.

Hysteria.

Hydrophobia.

Strychnine poisoning.

(ii) Mechanical:—

Swellings in neighbourhood, *e.g.*, odontomes, deep-seated tumours, extra buccal growths.

Rigidity of neighbouring tissues, *e.g.*, face cicatrices from burns, operations, &c., mouth cicatrices following ulcerations or severe operations, ossification of the pterygo-maxillary ligament, exostosis of the zygomatic arch, formation of bone in muscles (*myositis ossificans*).

Unreduced dislocation.

Hypertrophied or malformed condyle, the result of mal-united fracture or osteo-arthritis.

True anchylosis, bony or fibrous, the result of acute or chronic arthritis.

Symptoms:—

If dental cause:—

Inflammatory infiltration of neighbouring tissues.

Face hot, red, swollen, and painful.

Mouth can be opened slightly.

Suppuration around one or several cheek teeth.

Fœtid breath.

Muscles of mastication only involved.

If due to parotitis (mumps):—

Glands swollen in front of and below ear.
Both glands may be affected.
Tenderness to pressure.
Pain, sometimes very severe.
No redness.

If due to acute or chronic arthritis:—

Heat, redness, swelling, and dull aching pain.
Swelling over joint.
Tenderness over joint.
Opening mouth causes severe pain.
When acute, usually septic in origin.

If due to inflammation of pharynx:—

Difficulty in swallowing.
Glands behind angle of jaw swollen.
Slight swelling of face.

If tonsillitis:—

Tonsils red.
Tonsils swollen.
Neighbouring palate swollen and congested.
Neighbouring fauces swollen and congested.

If due to deep cellulitis:—

Œdema of tissues of neck.
Involvement of cellular tissue of neck later on.

If due to tetanus:—

At first, slight fever and difficulty in opening mouth.
Tonic or continuous spasms start first in muscles of mastication.
Spasms then spread down body, the patient assuming characteristic positions, *e.g.*, *opisthotonos* (bent backwards), *emprosthotonos* (bent forwards), *pleurothotonos* (bent sideways).

Angles of mouth contracted (*risus sardonicus*).
Great pain.
Mental faculties usually unimpaired.

If due to hydrophobia:—
History of dog bite.
Hallucinations.
Clonic or intermittent spasms.
Marked salivation or frothing.

If due to strychnine:—
History of having taken the drug.
Clonic or intermittent spasms all over body.

If due to osteo-arthritis:—
In early stages joint stiff.
Stiffness worse in mornings.
Clicking sound on movement of joint.
Stiffness gradually increases.
Joint becomes fixed, true anchylosis occurring.
Age at which it occurs usually over 40.

If due to true anchylosis:—
History of acute, chronic or osteo-arthritis following injury or inflammation.
If fibrous, joint slightly movable.
If osseous, jaw fixed.

If due to hypertrophy of the condyle:—
Swelling over joint.
Symphysis pushed away from affected side.
Slight movement of joint.

If due to a cicatrix:—
Presence of scar.
History of operation, injury, or ulceration.

If due to ossification of pterygo-maxillary ligament:—
Ligament stiff to the touch.

If due to external tumours:—

These are usually in connection with the parotid, submaxillary, or deep cervical glands.

If due to exostosis of the zygomatic arch:—

Plain on examination.

If due to deep-seated tumours:—

These may be suspected if not due to any of the foregoing causes.

Treatment:—

Remove cause.

If cause dental:—

Anæsthetize patient.

Carefully force open mouth with a *Mason* or screw gag.

Extract tooth causing trouble.

Advise poppy-head fomentations.

If cause acute or chronic arthritis:—

In early stages—

At night wedge a rubber block between molar teeth.

Draw up chin with four-tailed bandage.

This relieves joint.

Paint tincture of iodine over joint.

Purge.

In later stages—

The formation of a new joint will probably be necessary.

If cause osteo-arthritis:—

Protect joint from cold and damp.

Massage.

Rub sulphur ointment into the part.

Give sulphur and guaiacum internally.

The formation of a new joint will probably be necessary.

If cause anchylosis:—

Anæsthetize patient.

Divide adhesions.

If adhesions unite, the formation of a new joint will be necessary.

If cause cicatrices:—

Anæsthetize patient.

Divide cicatrices.

In the healing process new cicatrices and constrictions occur, and therefore a better treatment is to make a new joint.

If cause hypertrophy of condyle, exostosis of zygomatic arch, or ossification of pterygo-maxillary ligament:—

Make a new joint.

Operations:—

(i) Excision of condyle.

(Sometimes muscles hold parts together and reunion occurs.)

(ii) *Esmarch's* operation (fig. 142)—

(The removal of a wedge-shaped piece of bone from the angle of the lower jaw, the apex being at the alveolar border.)

Fibrous tissue replaces removed bone.

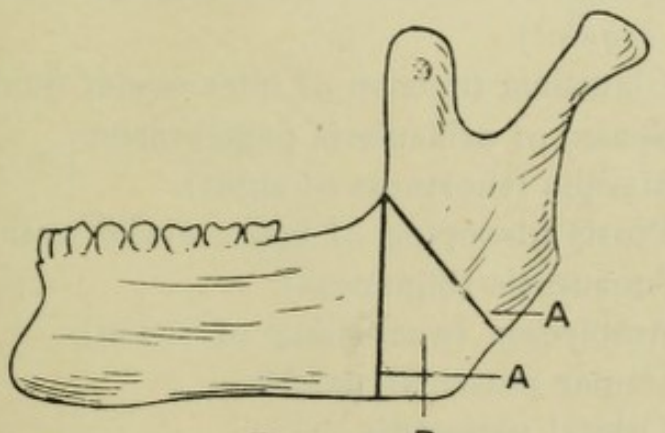

FIG. 142.

Esmarch's operation: A A, lines showing boundaries of piece of bone to be removed; B, portion of bone to be removed.

CHAPTER XIX.

AFFECTIONS WHICH MAY ARISE FROM DENTAL NEGLECT.

Of the mouth and neighbouring parts:—

Ulcerations.
Squamous-celled carcinoma.
Epithelioma.
Empyema of the maxillary antrum.
Necrosis.
Œdema of glottis.
Angina Ludovici.
Septic tonsillitis.
Post-pharyngeal abscess.
Cellulitis.

Of the eye:—

Lachrymation (flow of tears).
Spasm of internal rectus muscle (*strabismus* or *squint*).
Glaucoma (tension of intra-ocular fluid raised).
Spasm of orbicularis palpebrarum.
Myopia (shortness of sight).
Ptosis (drooping of upper eyelid over eye).
Amaurosis (blindness).
Amblyopia (weakening of vision).
Ocular muscular paresis.
Orbital periostitis.
Orbital cellulitis.
Acute retro-bulbar optic neuritis.
Exophthalmos (protrusion of eyeball).

Of the ear:—

Otalgia (earache).
Deafness.

Of the nervous system:—

Epilepsy.
Neuralgia.
Paralysis.
Chorea (St. Vitus's dance).
Septic neuritis.
Convulsive seizures.

Of the muscular system:—

Trismus.
Spasm of facial muscles.
Wry neck (spasm of sterno-mastoid).
Wasting palsy (progressive muscular atrophy).
Spasmodic closure of jaws.
Paralysis of arm.

Of the lymphatics:—

Acute lymphadenitis.
Chronic lymphadenitis.
Lymphatic abscess.

Of the joints:—

Osteo-arthritis.

Of the gastro-intestinal tract:—

Appendicitis.
Septic gastritis.
Indigestion.
Duodenitis.
Pancreatitis.
Jaundice.
Colitis.

Of the lungs:—

Septic bronchial pneumonia.

Of the skin:—

Herpes.

Of the brain:—

Meningitis.

Of the uterus:—

Uterine pain.

Of the endocardium:—

Suppurative endocarditis.

Septic diseases:—

Sapræmia.

Septicæmia.

Pyæmia.

Puerperal fever.

General:—

Chronic rheumatism.

Loss of sleep.

CHAPTER XX.

SWELLINGS OCCURRING IN AND ABOUT JAWS.

Classification into Fluid and Solid.

Fluid	Solid
Acute alveolar abscess	Fibrous odontome
Chronic ,, ,,	Cementoma
Dental cyst	Composite odontome
Dentigerous cyst or follicular odontome	Radicular ,,
Epithelial odontome	Enchondroma
Dermoid cyst	Fibroma
Aneurysm of descending palatine artery	Fibrous epulis
Angioma	Myeloid ,,
	Osteoma
	Myxoma
	Papilloma
	Adenoma
	Myeloma
	Granuloma
	Sarcoma
	Carcinoma

Diagnosis between Fluid and Solid.

	Fluid	Solid
Growth ..	Slow, with exception of abscess	Slow if innocent, rapid if malignant.
Alveolar border	Not affected	Slightly displaced.
Plates of alveolus	Outer plate more distended than inner	Plates distended symmetrically in innocent, asymmetrically in malignant.
Pain ...	Little or none except in abscess and suppuration, when pain may be severe	Little or none in innocent, severe in malignant.
To touch ...	May fluctuate. May get egg-shell crackling	No fluctuation. No egg-shell crackling.
External characters	Regular, smooth, globular	Irregular and not globular.
Radiography	Discovers fluid growth ...	Discovers solid growth.

IN CONNECTION WITH THE VARIOUS TISSUES.

Gum	Dental tissues	Alveolus	Body of jaw	Palate	Antrum
Papilloma	Dental cyst	Fibrous epulis	Osteoma	Chronic abscess	Cystic disease
Epithelioma	Granuloma	Myeloid ,,	Enchondroma	Dermoid cyst (soft palate)	Fibroma
Angioma	Acute alveolar abscess		Fibroma	Aneurysm of descending palatine artery (rare)	Encho droma
Sarcoma	Chronic ,, ,,		Myeloma	Follicular odontome	Osteoma
	Odontomes		Mucous cyst	Papilloma	Myxoma
			Carcinoma	Fibroma	Carcinoma
			Sarcoma	Adenoma	Sarcoma
				Osteoma	
				Sarcoma	
				Carcinoma	

DIAGNOSIS BETWEEN

	Alveolar abscess	Dental cyst	Dentigerous cyst
Cause ...	Inflammation in connection with root of tooth which is present	Irritation *via* apices of septic root of tooth which is usually present	Abnormal development of a tooth which is absent from the series.
Inflammation	Present ...	Absent unless suppuration	Absent unless suppuration.
Rate of growth	Comparatively rapid	Slow	Very slow.
Walls	Fibrous connective tissue	Fibrous connective tissue	Fibrous connective tissue.
Lining ...	Epithelium ...	Granulation tissue	Epithelial lining usually.
Contents ...	Pus	Glairy mucoid fluid containing cholesterin crystals	A tooth loose or embedded. A thick yellow, glairy fluid which may contain cholesterin crystals.

CLASSIFICATION ACCORDING AS TO WHETHER INNOCENT OR MALIGNANT.

Innocent	Malignant
Alveolar abscess	Myeloid epulis
Dental cyst	Carcinoma
Granuloma	Sarcoma
Odontomes	
Papilloma	
Angioma	
Fibroma	
Fibrous epulis	
Osteoma	
Enchondroma	
Myeloma	
Dermoid cyst	
Aneurysm of descending palatine artery	
Adenoma	
Myxoma	

DIAGNOSIS BETWEEN SOLID INNOCENT AND SOLID MALIGNANT.

	Solid innocent	Solid malignant
Growth ...	Slow	Rapid.
Pain ...	Little or none	Severe.
Alveolar border	Hardly affected	Irregular displacement.
Alveolar plates	Equally distended	One plate distended more than another.
Glands ...	Not affected...	Enlarged in carcinoma.
Face ...	No œdema	Œdema.
Hæmorrhage	Usually none	Attacks of bleeding if superficial.
General effect	None	Emaciation (cancerous cachexia).
Surroundings	Not affected, growth usually encapsuled	Infiltration of surrounding tissues, growth not encapsuled.
Relation to surroundings	Freely movable (usually) ...	Fixed and adherent.
Structure ...	Resemble tissues in which they grow	Differ from tissues in which they grow.
Recurrence	None if completely removed	Recurrence common.

CHAPTER XXI.

CHRONIC SUPERFICIAL GLOSSITIS AND ULCERS OF THE TONGUE.

Chronic Superficial Glossitis (Leukokeratosis).

Definition:—

A chronic inflammation of the mucous membrane of the tongue.

Causes:—

Tertiary syphilis.
Irritation from jagged edge of tooth, denture, crown, tartar, &c.
Excessive smoking.
Chewing tobacco and betel nut.
Excessive spirit drinking.
Condiments.
Chronic dental sepsis.

Description:—

Rare in women.
Occurs between ages of 40 and 60.
First hyperæmia and hypertrophy of papillæ forming raised red patches.
Then overgrowth of epithelium.
Epithelium opaque with horny cells.
Formation of whitish patches (*leucoplakia*).
Patches may coalesce (*psoriasis*).
Hypertrophied papillæ atrophy, leaving smooth red patches.
Surface of tongue may become cracked (*ichthyosis*).

Patches may also occur on palate, cheek and gums.

May terminate in epithelioma.

Treatment:—

Remove irritation.

Keep tongue clean.

Attend to teeth.

Give potassium iodide for syphilis.

Alkaline mouth-wash after each meal (5 per cent. solution of bicarbonate of soda).

Paint patches with 1 per cent. solution of chromic acid.

Ulcers of the Tongue.

(i) Simple—

Dental.

Dyspeptic.

(ii) Infective—

Syphilitic.

Tubercular.

Epitheliomatous.

Dental Ulcer.

Causes:—

Rough edge of tooth.

Rough edge of denture, crown, or band.

Calculus.

Site:—

Side of tongue.

Description:—

If superficial—

Painful.

Zone of inflammation around.

If deep—

Irregular.

Edges.—Irregular, raised, abrupt. May become indurated.

Base.—Depressed, sometimes sloughy, sometimes bathed in pus, and sometimes indurated.

Surroundings.—Inflamed.

Glands.—Not enlarged except from sepsis.

Treatment:—

Remove cause.

Dyspeptic Ulcer.

Cause:—

Gastric disorders.

Site:—

Near tip of dorsum of tongue.

Description:—

Comes quickly.
Goes quickly.
Multiple.
Shallow.
Circular.
Sensitive.
History of dyspepsia.
Edges.—Bright red.
Base.—Raw and soft.
Surroundings.—Inflamed.
Glands.—Not affected.

Treatment:—

Treat dyspepsia.
Potassium chlorate mouth-wash.

Syphilitic Ulcer.

Primary—

Site:—

Tip of tongue.
Edge of tongue.

Description:—

No pain.
Edges.—Raised.
Surroundings.—Not inflamed.
Glands.—Submaxillary lymphatics enlarged.

Secondary—

Site:—

Side of tongue.

Description:—

Oval or irregular.
Multiple.
Edges.—Sharply cut.
Base.—Ash grey.
Surroundings.—Inflamed.

Tertiary—

Site:—

Dorsum of tongue, far back.

Description:—

Indurated lump precedes ulceration.
Occurs usually under the 40th year of age.
Usually multiple.
Irregular and shallow.
Comparatively painless.
No profuse salivation.
Speech and mastication not affected.
No fixation of tongue.
Edges.—Indurated, sharply cut, smooth and overhanging.

Base.—Sloughy, firm, scanty discharge, and may have wet wash-leather-like appearance.

Glands.—Not affected unless septic infection.

Treatment:—

Potassium iodide internally.
Antiseptic mouth-wash.

Tubercular Ulcer.

Cause:—

Infection from sputum in phthisical patients.

Site:—

Under surface of tip of tongue.
Side of tongue.

Description:—

Rare.
Occurs usually in young adults.
Usually begins as a small nodule.
Superficial.
Often multiple.
May coalesce.
Very painful.
Edges.—Undermined, irregular and slightly raised.
Base.—Uneven, yellowish or greenish, thin and watery discharge.
Surroundings.—Inflamed.
Glands.—No enlargement.

Treatment:—

General treatment for tuberculosis.
Scrape ulcers.
Touch ulcers with pure carbolic acid.
Excision is the best treatment.

Epitheliomatous Ulcer.

Cause:—

Chronic irritation.

Site:—

Edges of tongue.

Description:—

Shallow.
Irregular.
Rapid development.
Very rarely multiple.
Severe radiating pain.
Sometimes no pain.
Profuse salivation.
Fœtid breath.
Speech and mastication affected.
Later fixation of tongue.
Occurs seldom in young, usually after the 40th year.
Occurs more often in the male sex.
Edges.—Hard, everted, sinuous and raised.
Base.—Irregular, indurated, greyish, sometimes red, bleeds readily, discharge excessive and offensive, sometimes scanty.
Surroundings.—Infiltrated and indurated.
Glands.—Submaxillary lymphatics enlarged.

Treatment:—

Excision.

Ulceration from *actinomycosis* and *rodent* and *lupoid* ulceration may, but rarely, occur.

CHAPTER XXII.

CONDITIONS UNFAVOURABLE TO TOOTH EXTRACTION.

Menstruation:—

Avoid operation if possible.

Patient susceptible to shock.

Vicarious menstruation from socket may occur after operation.

If operation unavoidable give a general anæsthetic.

Pregnancy:—

Avoid operation if possible.

Patient susceptible to shock.

Premature labour may occur, especially at third, fourth, or fifth month.

Congenital deformities may occur.

If operation unavoidable give a general anæsthetic. This may usually be done with safety up to the eighth month.

Lactation:—

Avoid operation if possible.

Patient susceptible to shock.

May be decreased lacteal secretion.

The author had a case where the secretion was stopped for nearly three days.

Lacteal fluid may be altered in composition and rendered unfit for child.

If operation unavoidable give a general anæsthetic.

General Debility:—

Give tonic to prepare patient.
Give a general anæsthetic.
Danger of shock and nervous depression.

Nervous Irritability:—

Give tonic to prepare patient.
Give sedatives.
Give a general anæsthetic.
Danger of severe shock, chorea, hysteria.

Heart Disease:—

Danger of shock.
Avoid operation if possible.
If operation be decided upon—
Have a cardiac stimulant administered an hour or two before, *e.g.*, brandy, strychnine, &c.
Have at hand a hypodermic syringe containing $\frac{1}{80}$ gr. of strychnine sulphate dissolved in 1 dr. of brandy.
Have a general anæsthetic administered.

The *symptoms of shock* are—

Dilated pupil.
Insensitive cornea.
Pallor of face.
Blanched lips.
Cold, moist skin.
Profuse perspiration.
Shallow breathing.
Feeble pulse.
Sometimes nausea and vomiting.
Low temperature (below 96° F. serious).

Treatment of shock.—

Withdraw anæsthetic.
Lower head and shoulders.
Elevate feet.

Give hypodermic injection of the strychnine and brandy.

Apply cold water to face and head.

Amyl nitrate or ammonia to nostrils.

Hot flannels to cardiac region.

If respiration impaired perform artificial respiration, pull tongue forward, and clear throat of mucus.

Should these methods not be successful any of the following may be adopted—

Give hot black coffee, or

Brandy up to 1 oz., in hot water, in sips, or

Thirty drops of aromatic spirits of ammonia in a little water, or

Inject a pint of water at 105° F., containing 1 oz. of brandy, into rectum.

When patient sufficiently recovered, advise bed, and mustard to feet.

Epilepsy:—

Before operation have large doses of potassium bromide administered.

Loosen all clothes about neck.

Danger of epileptic fit.

Should attack occur—

Place patient in horizontal position.

Force a piece of soft rubber or a cork between teeth to prevent patient injuring himself.

Nitrate of amyl or ammonia to nostrils.

Hæmorrhagic Diathesis (*Hæmophilia*):—

Avoid operation if possible.

Danger of serious hæmorrhage.

If operation unavoidable—

Have calcium lactate administered, with object of increasing coagulability of blood

(one tabloid of 10 gr. three times per day for ten days), or ergot in 20 drops to 1 fl. dr. every two or four hours on day before operation.

Plug socket after operation with a styptic (a solution of the dried extract or the fresh extract of thymus gland has been used with success). The use of adrenalin in hæmophilia is not advocated. For other styptics, *see* pages 278 and 279.

Apply a compress of cork, gutta-percha, or modelling composition.

Apply a four-tailed bandage.

Advise rest and upright position.

Advise administration of calcium chloride 10 to 15 gr. three or four times a day, or thymus gland up to 100 gr. daily.

An injection into the rectum of a 20 per cent. solution of gelatine up to 2 litres may prove beneficial.

CHAPTER XXIII.

DIFFICULTIES, COMPLICATIONS, ACCIDENTS, AND SEQUELÆ OF TOOTH EXTRACTION.

(i) *In connection with teeth.*
(ii) *In connection with jaw-bone.*
(iii) *In connection with soft tissues.*
(iv) *In connection with anæsthetics.*

(i) In connection with the teeth:—

Undue resistance, due to—

Teeth being misplaced.
Anchylosis of teeth with bone (rare).
Exostosis.
Gemination.
Dilaceration.
Curved, twisted, or abnormal number of roots.
Inferior dental nerve passing through gap in geminated roots.
Crowded condition of teeth.
Thickness and strength of alveolus, or
Character of teeth (*e.g.*, in bilious subjects, where the teeth are short and thick with yellow crowns).

Fracture, due to—

Use of wrong instruments.
Misapplied or too great force.
Undue haste in operating.

Wrong position of patient, either too high or too low.
Not penetrating far enough between tooth and alveolus, or
Curved, twisted, or abnormal number of roots.

NOTE.—A small aseptic piece of root may be left without harm. Should the fracture leave a live pulp exposed the latter may be cauterized with pure carbolic acid, or extirpated under an anæsthetic.

Accidental fracture of a neighbouring tooth, due to—

Wrong position of patient.
Undue or wrongly applied force.
Use of wrong instruments, or
Undue haste in operating.

Accidental removal or displacement of wrong tooth, due to—

Wrong diagnosis.
Wrong position of patient.
Undue or wrongly applied force.
Use of wrong instruments.
Wrong use of *Mason* or other gag or prop.
Part being obscured by blood.
Roots interlocking.
Gemination of two or more teeth, or
Anchylosis of tooth to bone.

Injury to or removal of a permanent tooth or tooth germ in extracting a temporary tooth, due to—

Crown of permanent tooth being enclosed and held by roots of temporary, *e.g.*, premolar by roots of temporary molar.
Undue force.

(ii) In connection with the bone of the jaw:—

Fracture of alveolus or body of jaw, due to—

Undue force.
Misapplied force.
Undue haste.
Applying forceps outside alveolus.
Disease of jaw.
Previous fracture of jaw.
Anchylosis of tooth to bone (rare).
Dense fibrous attachment of tooth to bone.
Wrong position of patient.

NOTE.—If the portions of fractured alveolus are small, remove; but if large, mould into position with finger and thumb and, if necessary, apply splint.

Dislocation of lower jaw, due to—

Undue force.
Misapplied force.
Anchylosis of tooth to bone (rare).
Dense fibrous attachment of tooth to alveolus.
Weakness of ligaments, or
Violent or improper use of *Mason* or other gag.

Perforation of antrum, due to—

Disease, such as necrosis, or
Undue force.

Forcing tooth into antrum, due to—

Disease, such as necrosis, or
Undue force.

Forcing tooth into abscess cavity, due to—

Diseased bone, or
Undue force.

Necrosis may follow on extraction, due to—

Bacterial infection.

(iii) In connection with the soft tissues:—

Laceration of gums, due to—

Applying forceps outside gums.

Adherence of gum to tooth. In this case stay operation and incise gum before proceeding further.

Wounding of tongue—

If lacerated, trim with scissors and order antiseptic mouth-washes.

If punctured, draw tongue forward and stitch.

If large branch of lingual artery involved, place finger on back of tongue, draw tongue forward (this presses lingual artery against hyoid bone), and twist involved vessel. Should this fail, cauterize, or the lingual artery may be tied.

Injuring lips, due to—

Rough use of *Mason* or other gag.

Pressure from handles of forceps.

Rough, sharp edges of teeth.

Injuring inferior dental nerve, due to—

Rough use of forceps.

Nerve passing through geminated roots.

Sloughing of gum
Cancrum oris
Tetanus
Spreading traumatic gangrene
Syphilis
Actinomycosis
} may result.

Suppuration in the socket may occur, due to—

A dirty mouth,

A dirty instrument,

Operator's dirty hands.

NOTE.—Before and after operation have the mouth cleansed with an antiseptic mouth-wash, such as:—

Sanitas
Condy's fluid
Permanganate of potash
} in weak solutions.

Or,

℞ Acidi. carbol. pur. ... ... ʒiv.
Liquor potassæ ... ... ʒvi.
Aquam ad ... ... ℥iv.
M. ft. lotio.
Add one teaspoonful to a half tumblerful of warm water and rinse every three hours after operation.

Pain may occur, due to—

(*a*) Leaving a portion of root in. If the piece is small and aseptic leave alone, but if large or septic remove.

(*b*) Injury to nerve. Here the pain will gradually subside without interference, providing the injury has not been severe.

(*c*) Sepsis and suppuration in socket.

(*d*) Fracture or straining of alveolus. In this case thoroughly cleanse, mould fractured or bent alveolus to place, and keep aseptic.

(*e*) Leaving exposed pulp. Here extirpate under a local or general anæsthetic, or cauterize with pure carbolic acid.

(*f*) Too rapid healing of external opening. Here incise, syringe out with an antiseptic, pack with iodoform gauze for some hours, and order an antiseptic mouth-wash.

(*g*) Necrosis of socket. Apply, locally, an anodyne, and order an anodyne mouth-wash.

(*h*) Periostitis.

(*i*) Alveolar abscess.

(*j*) Exostosis.

Hæmorrhage may occur.

Trismus may render operation difficult.

(iv) In connection with anæsthetics:—

With local anæsthetics—

Sloughing of gums—

Septic inflammation—

To prevent above as far as possible, cleanse mouth and give antiseptic wash before operating.

Use thoroughly sterilized instrument, and prepare drug freshly for each operation.

Severe after pains—

These may probably be prevented by

(*a*) Not using too strong a dose of drug.

(*b*) Not injecting into spongy and inflamed tissue.

Severe hæmorrhage.

Shock.

Toxic symptoms—

Should these supervene—

Place patient in recumbent position,

Loosen clothing to allow of free breathing.

Open windows at top to give as much fresh air as possible,

Keep legs and feet warm,

Give stimulants, and

If necessary perform artificial respiration.

Hallucinations—

It is always wise, especially with a female patient, to have a second party present during operation.

With general anæsthetics—

Arrest of circulation.

Arrest of respiration.

Accidental injury through misplaced prop or misuse of Mason gag.

Struggling patient (alcoholic or neurasthenic) may injure himself.

Sickness.

Headache.

Nervous prostration.

Shock.

Hallucinations.

Septic bronchial pneumonia—

This is liable to occur after operating on a septic mouth under ether. Keep the patient indoors for some time after operation.

Tongue may slip or be pushed down throat—

Symptoms:—

Difficult breathing.
Impending asphyxia.

Treatment:—

Pull tongue forward with tongue forceps.
Force head back.

A tooth, a filling, or a crown may be forced out with a gag or prop—

Avoid, if possible, placing gag on loose teeth or crowns.

Passage of tooth, a piece of broken instrument, blood clot, &c., to larynx.—

Symptoms:—

Spasmodic coughing, which may expel foreign body.
Excessive secretion of mucus.
Severe dyspnœa.
Dysphagia.
Sense of suffocation.
May be hæmorrhage.
May be vomiting.

May be asphyxiation and cyanosis.
Œdema may supervene.

Treatment:—

Bring head forward.
Immediately explore with finger, and by sweeping it round the pharynx attempt removal.
If this fails invert patient and slap back.
If this fails and obstruction incomplete the use of the laryngeal forceps, guided by the laryngoscope (an instrument used for seeing obstruction), may prove effective.
X-rays.
Otherwise, or if obstruction complete, immediate laryngotomy must be performed.
Tracheotomy may be necessary.

Passage of tooth, &c., to trachea.—

Symptoms:—

Violent spasmodic coughing.
Dyspnœa.
Impending asphyxia and cyanosis.

Treatment:—

Digital exploration.
Invert and slap on back.
If these fail X-rays and bronchoscope (an instrument used for seeing obstruction).
Otherwise tracheotomy.

Passage of Tooth, &c., to bronchus.—

More often in connection with right than left, due to—

(*a*) Position of inter-bronchial septum.
(*b*) Greater size of right bronchus.
(*c*) Right bronchus following more closely line of trachea.

Symptoms:—

Severe dyspnœa.
Violent irritable spasmodic cough.
Laryngeal spasm.
Signs of impending asphyxia and cyanosis.
May be collapse of lung or lungs.
Local bronchitis.
May be suppuration and dilatation of bronchus beyond obstruction (*bronchiectasis*).
Death may supervene from abscesses in lung.

Treatment:—

X-rays.
Examine chest by auscultation (determining condition by sense of hearing), and percussion.
Bronchoscope.
Low tracheotomy.

Passage of tooth, &c., to pharynx.—

Symptoms:—

May be dysphagia.
Pain, which is worse on swallowing.
May be dyspnœa.
Hacking cough.

Treatment:—

Digital exploration to back of pharynx, and if possible remove foreign body.
Invert and slap on back.
Laryngotomy.
After relief of dyspnœa remove foreign body with suitable forceps.
Pharyngotomy or extraction through *Brünings'* tubes may be necessary.

Passage of tooth, &c., to œsophagus.—

Symptoms:—

Dysphagia.
Usually constant pain.
If in upper part probable dyspnœa.
Gurgling sound on swallowing fluid.
If at pyloric opening gastric dilatation.

Treatment:—

Auscultation in region of œsophagus.
X-rays.
If in upper part, œsophagotomy.
If in cardiac end use bougie to push into stomach.
Gastrotomy.
If at pyloric opening, empty stomach and perform gastrotomy.

CHAPTER XXIV.

HÆMORRHAGE AFTER TOOTH EXTRACTION.

Varieties:—

(i) Primary.

Ceases within thirty minutes after extraction.

Stoppage is due to fall of blood-pressure, constriction of vessel walls, and formation of clot.

A few hours after stoppage blood-pressure rises and the clot may be forced out, giving rise to

(ii) Intermediate, reactionary, or recurrent.

Occurs within twenty-four hours of operation.

(iii) Secondary.

Occurs twenty-four or more hours after operation.

Is due to sepsis, which softens and weakens clot.

Varieties according to vessel involved:—

(i) Arterial.

Bright red colour.

Spurts out in jets.

Pressure on artery above wound stops it.

(ii) Venous.

Dark purplish-red colour.

Flows steadily.

(iii) Capillary.

Bright red colour.

Oozing from several points.

Causes:—

(i) Want of coagulability of blood.
(ii) Want of contractility of vessel walls.

Treatment:—

If slight—

Leave alone, as it relieves pain by reducing pressure in surrounding tissues.

If it does not stop—

(i) Remove any loose or sharp spicules of bone.
(ii) Press walls of socket together with thumb and fingers.
(iii) Apply cold water, or give ice, which is a physical styptic, to suck.

Sometimes water as hot as can be borne will prove effective.

Should these means fail—

(i) Clear away any clots.
(ii) Syringe out socket with a warm 1 in 40 carbolic acid solution.
(iii) Insert a pad of cotton-wool in socket.
(iv) After a minute or two remove pad and determine source of hæmorrhage.
(v) Plug socket tightly with a *styptic*.

NOTE.—It is wise to use a mechanical styptic, such as cotton-wool, gauze, modelling composition, gutta-percha, or wax, in combination with a chemical styptic, such as suprarenal extract (adrenalin chloride), perchloride of iron, tannin, turpentine, &c. Matico leaf is both a mechanical and chemical styptic.

Matico leaf:—

Soften leaf in hot water.
Cut into pieces as wide as socket is deep.
Keep rough side external and make into rolls.
Tightly plug socket with rolls.
Tomes states that he has never known this method fail when satisfactorily carried out.

Extract of suprarenal gland :—

Use *Parke, Davis and Co.'s* adrenalin chloride solution, 1 in 1,000.
Tightly pack wool plugs soaked in the solution into socket.
In place of the above *Parke, Davis and Co.'s* adrenalin gauze tape may be used.

Strong solution of ferric chloride :—

If used in excess this is very objectionable, due to its caustic action and resulting excessive sloughing and secondary hæmorrhage. If used—
Soak plug of wool in a mixture of one of the solution and three of water.
Squeeze out all excess.
Plug socket tightly.
The author strongly objects to the local use of this drug.
In some cases the internal administration of the drug in 15-minim doses every two hours will prove beneficial.

Tannin :—

Use a saturated solution of tannic acid and gun cotton in ether (*styptic colloid*).

Escharotics :—

These should not be used.
They destroy parts.
They extend wound.
Recurrence of hæmorrhage liable.

(vi) Apply a compress of modelling composition, cork, or gutta-percha.
(vii) Apply a four-tailed bandage.
(viii) Advise patient to observe the following conditions :—
Quietness.
Avoidance of stimulants.
Avoidance of hot drinks.
To take liquid food, *e.g.*, milk, *Horlick's* milk, beef extract.
Upright position, sleeping with elevated pillow at night.
If possible, keep feet in hot water.

(ix) Leave for twenty-four hours.

(x) Remove plug.

Should all these methods fail—

If the blood is thin and shows no tendency to coagulate, give calcium lactate in 10-gr. doses three times a day for five days.

If blood shows a tendency to coagulate give a hæmostatic (a drug capable of arresting hæmorrhage by internal administration), such as ergot, to contract the vessel walls.

℞ Liquid extract of ergot ... ʒiss.
Dilute sulphuric acid ... ... m.xl.
Water to ... ... ʒiv.

Two tablespoonfuls to be taken every half hour until bleeding ceases. It should not be given if patient pregnant.

In extreme cases where hæmorrhage persists one of the following methods may be necessary:—

(i) Actual cautery.

(ii) Trephine inferior dental canal, if from lower tooth, plug with a wood or ivory peg, compressing artery against inner plate of bone.

(iii) Pressure with finger on common carotid artery.

(iv) Tie common carotid artery.

If bleeding is from gums—

Have mouth rinsed with ice-cold water and alum.

Give ice to suck.

Tannic acid to gums.

Search for vessel and twist or compress.

If vessel partially divided (fig. 143) completely divide, as the partial division prevents the complete recession and

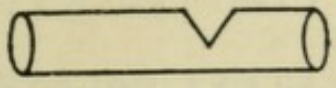

FIG. 143.—Partially divided blood-vessel.

constriction of the inner coat, which, in addition to clotting, is the natural method for arresting hæmorrhage (fig. 144).

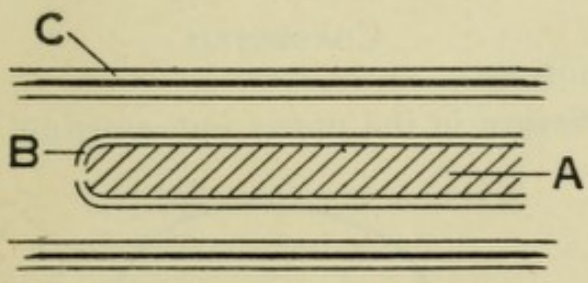

FIG. 144.—Cut blood-vessel. A, blood; B, cut end of inner coat which has receded and constricted; C, outer coat.

CHAPTER XXV.

CLEFT PALATE.

Definition:—

A fissure existing in the upper jaw.

Varieties:—

(i) Congenital.

(ii) Acquired (due to traumatism, syphilis, &c.).

Congenital.

Definition:—

A fissure in the upper jaw existing from birth.

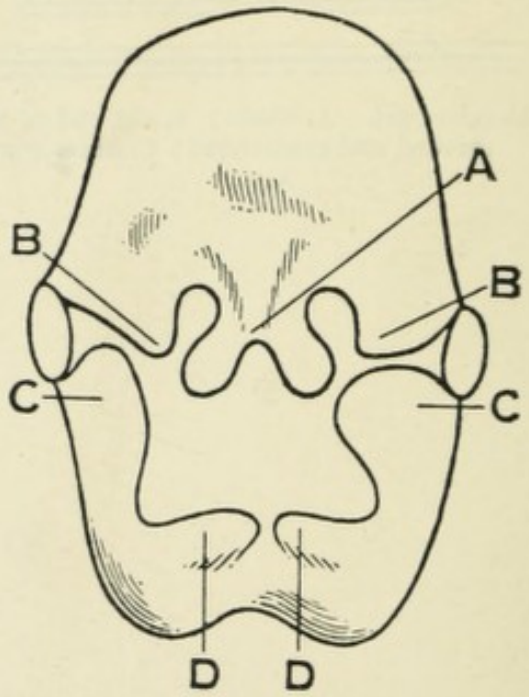

Fig. 145.—Embryonic head. A, median nasal process; BB, lateral nasal processes; CC, maxillary processes; DD, mandibular processes.

Cause:—

During development the nose and mouth are at first one cavity.

This then becomes divided off by a growth from the roof, the naso-frontal process (figs. 145,A, and 145,BB), and growths from the lateral boundaries, the maxillary processes (fig. 145,CC). It is due to an arrested development of, and a failure of normal union between these processes that cleft palate results.

Degrees of cleft:—

Bifid uvula (fig. 146).

Division of uvula and soft palate (fig. 147).

Division of soft palate and hard palate up to the anterior palatine foramen (fig. 148).

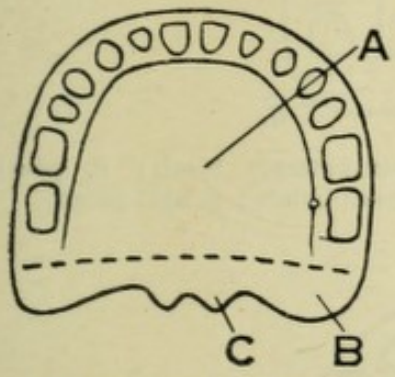

FIG. 146.—Cleft palate involving uvula only. A, hard palate; B, soft palate; C, divided uvula.

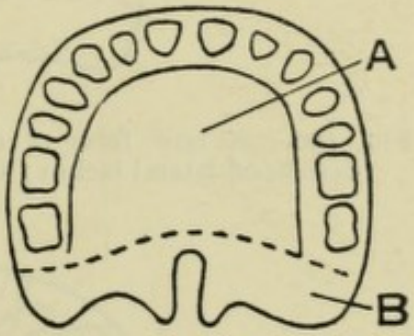

FIG. 147.—Cleft palate involving uvula and soft palate only. A, hard palate; B, soft palate.

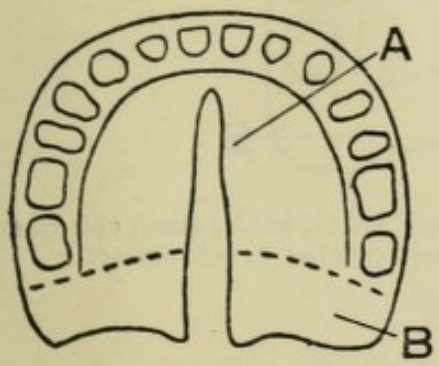

FIG. 148.—Cleft palate involving uvula, soft palate, and hard palate. A, hard palate; B, soft palate.

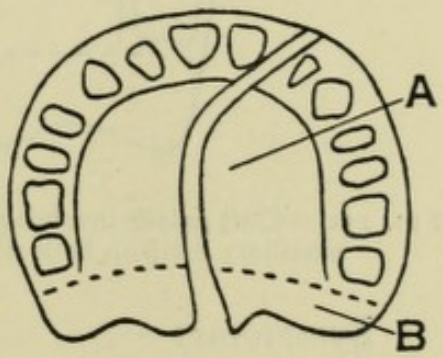

FIG. 149.—Cleft palate involving uvula, soft palate, hard palate, and inter-maxillary bone between central and lateral incisors. A, hard palate; B, soft palate.

Division of soft and hard palates and pre-maxillary bone, usually between the central and lateral incisors (fig. 149). In rare cases the division of the pre-maxillary bone may be as in fig. 150.

Division of hard and soft palates and pre-maxillary bone on both sides (fig. 151).

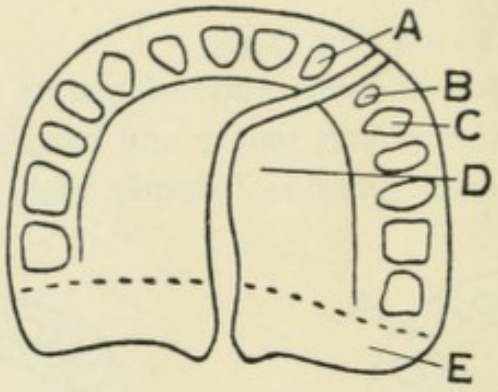

FIG. 150.—A rare form of cleft. A, supernumerary tooth; B, poorly developed lateral incisor; C, canine; D, hard palate; E, soft palate.

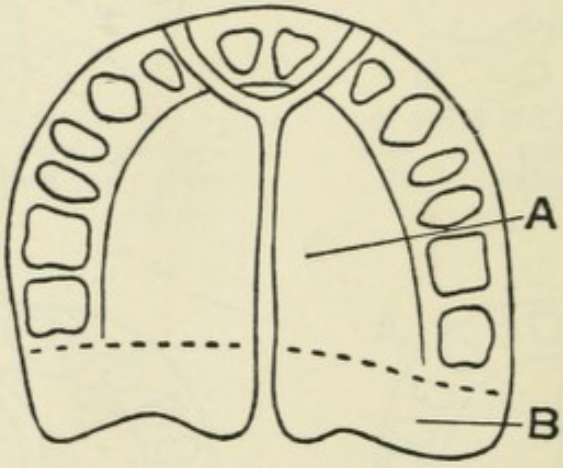

FIG. 151.—Cleft palate involving uvula, soft palate, hard palate, and inter-maxillary bone on both sides. A, hard palate; B, soft palate.

Symptoms:—

Sucking and swallowing difficult, sometimes impossible.

Tendency to adenoids.

Teeth irregular and liable to caries.
Subject liable to thrush and pulmonary inflammation.
Speech impaired (nasal intonation).
Defective articulation of teeth.
Hoarseness from laryngeal catarrh.
Sometimes death from wasting, diarrhœa, and broncho-pneumonia.

Treatment:—

(i) Operative.
(ii) Mechanical.
(i) Operative:—
(*a*) Staphylorrhaphy (suturing of soft palate).
(*b*) Uranoplasty (closure of cleft in hard palate).

NOTE.—Operate before child begins to speak. The best time is between six weeks and three months. The child must be healthy. The mouth must be clean.

OPERATION OF STAPHYLORRHAPHY.

Process:—

(i) Cleanse mouth.
(ii) Pare edges of cleft.

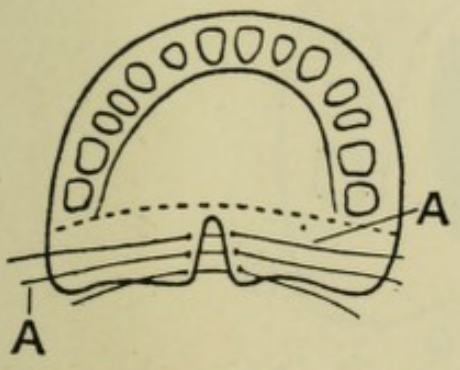

FIG. 152. — Staphylorrhaphy. AA, ligatures passed through each half in cleft soft palate.

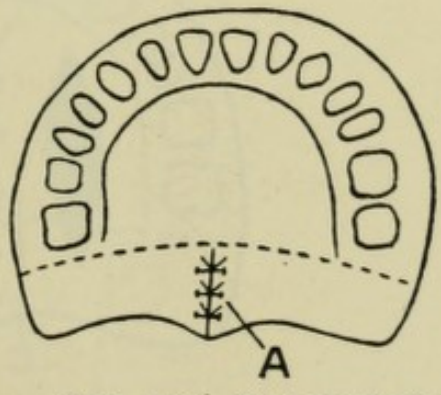

FIG. 153.—Staphylorrhaphy. A, ligatures tied, drawing together each half of cleft soft palate.

(iii) Pass sutures through soft palate (fig. 152,AA).

(iv) Divide muscles of soft palate to ensure rest and prevent separation of halves.

(v) Tie sutures (fig. 153,A) and leave for about seven days.

Operation of Uranoplasty.

Process:—

(i) Make two incisions down to the bone, one on each side of cleft about half an inch from the teeth and partly into the soft palate (fig. 154).

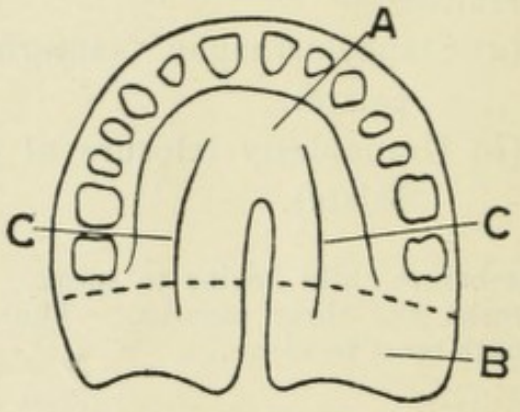

FIG. 154.—Uranoplasty. A, hard palate; B, soft palate; CC, incisions to bone.

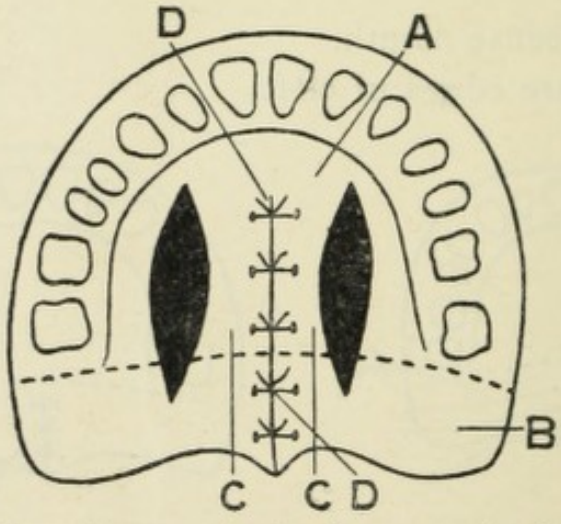

FIG. 155.—Uranoplasty. A, hard palate; B, soft palate; CC, two flaps of gum separated from the bone and brought into contact in median line; DD, ligatures of silver wire.

(ii) Pare edges of cleft.

(iii) With suitable instruments separate soft parts from bone.

(iv) Bring edges together in median line.

(v) Suture with separate pieces of silver wire in several places (fig. 155).

(vi) Stop hæmorrhage by means of pressure, &c., and leave for about seven days.

Mechanical:—

(i) Velum.

Advantages:—

Useful till speech improved.
Soft.
Elastic.
Follows all movements of muscles with which it comes in contact.

Varieties:—

(*a*) *Sercombe's* (fig. 156).

(*b*) *Kingsley's* (figs. 157 and 158).

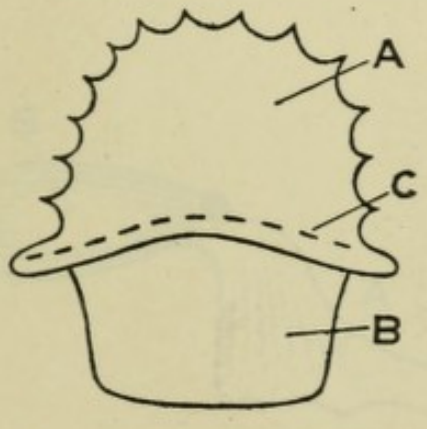

FIG. 156.—*Sercombe's* velum. A, vulcanite or metal plate; B, apron of soft rubber attached to posterior surface of plate extending to within quarter of an inch of the posterior wall of pharynx; C, sutures attaching soft rubber to plate.

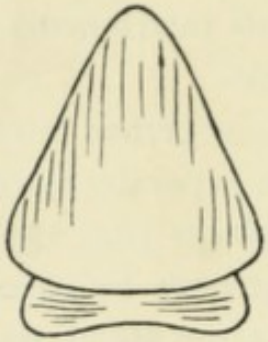

FIG. 157.—*Kingsley's* velum. A triangular double flap of rubber joined together and grooved for edges of cleft.

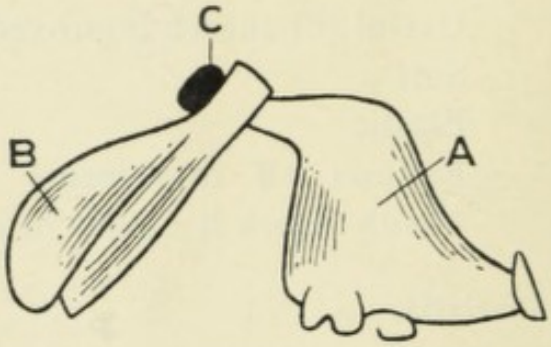

FIG. 158.—*Kingsley's* velum attached to plate. A, metal plate; B, triangular double flap of rubber; C, metal stud attached to plate to hold rubber flap.

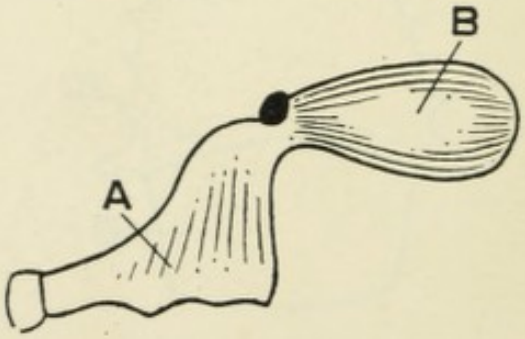

FIG. 159.—*Suerson's* obturator.—A, metal or vulcanite plate; B, solid plug of vulcanite.

(ii) Obturators.

These are plugs of hard vulcanite, solid or hollow, filling up to some extent the upper pharynx or posterior nares.

Varieties:—

(*a*) *Suerson's* (fig. 159).

Consists of a solid wedge-shaped plug of vulcanite with base upwards.

(*b*) *Kingsley's.*

Consists of a large hollow bulb of vulcanite.

CHAPTER XXVI.

DISCOLOURED TEETH AND BLEACHING.

Causes of Discoloration:—

(i) Pathological.

(*a*) *Death of pulp* from traumatism, &c., causes a disintegration of the red blood corpuscles and a penetration of the dentinal tubes by hæmoglobin.

(*b*) *Putrefactive decomposition* of the pulp is accompanied by an evolution of ammonia and sulphuretted hydrogen and the probable formation of ferrous sulphide with resulting staining (*Kirk*).

According to *Buckley*, ferrous and then ferric hydroxide is formed and causes the staining.

(ii) Chemical.

(*a*) *Medicinal substances*, e.g., *oil of cassia*, which contains *furfurol*, stains light brown.

Arsenic paste containing methylene, which is sometimes the case, stains bright blue.

Potassium permanganate, sometimes used in root-canal treatment, stains deep brown.

Mercuric chloride, sometimes used in root-canal treatment, stains slate blue, which becomes black.

(*b*) *Metals*, due to the formation of their soluble salts from the action of sulphuretted hydrogen in the dentinal tubes on metal fillings, or the use of metal instruments in bleaching or enlarging root canals, *e.g.*:—

Gold—

Use of gold instruments in bleaching.
Leaving gold filling in tooth prior to bleaching.
Contact of gold filling with nitro-hydrochloric acid, which is sometimes used for enlarging root canals.
The stain is at first pink and then black, and is due to action of chlorine on gold.

Iron—

Use of steel instruments in connection with iodine, chlorine, or mineral acids.
The stain is yellow, then black.

Copper—

Copper amalgam fillings.
The stain is bluish black.

Nickel—

In fillings.
The stain is at first grass green, then black.

Silver—

Obtunding teeth with silver nitrate.
The stain is jet black.

Mercury—

Mercuric chloride in root-canal treatment.

Stain is slate blue, which becomes black.

Manganese—

Potassium permanganate in root-canal treatment.

Stain is deep brown.

Superficial stains may occur from use of tobacco,
eating spinach,
eating black cherries, &c.

Treatment:—

Remove superficial stains by scrubbing with brush and pumice.

For the removal of stains due to death or decomposition of pulp two methods have been adopted.

(i) *Oxidation.*

The use of free oxygen liberated from oxygen compounds, *e.g.*,

Sodium dioxide, 25 per cent.

Perhydrol (Peroxide of hydrogen, 100 vols.).

Aluminium chloride.

Chlorinated lime and dilute acetic acid (*Truman's* method).

(ii) *Reduction.*

The abstraction of oxygen from the colour compound by a reducing agent, *e.g.*, sulphurous acid.

Preliminary treatment in all cases:—

(i) Remove all soft and carious tissue and all metal fillings.
(ii) Shape cavity.
(iii) Open up and treat root canal or canals.
(iv) Fill two-thirds of canal or canals.
(v) Swab with alcohol.
(vi) Dry with warm air.
(vii) Wash with distilled water and 10 per cent. borax, or a solution of bicarbonate of soda, to remove fat.
(viii) Wash with distilled water again.

Then—

If *sodium dioxide* used—

(i) Apply rubber dam.
(ii) Pack sodium dioxide powder into tooth with wood, ivory, vulcanite, or bone instrument.
(iii) Add a drop of distilled water.
(iv) Seal immediately.
(v) Leave for two or three days.
(vi) Re-treat if necessary.
(vii) Use a 5 to 10 per cent. solution of sulphuric or hydrochloric acid to remove yellow tint if desirable.
(viii) Wash with distilled water.
(ix) Dry with alcohol and warm air.
(x) Seal with gutta-percha and leave for some days.

If *perhydrol* used—

(i) Apply rubber dam.
(ii) Apply perhydrol on cotton-wool.
(iii) Evaporate water with warm air syringe. The application and evaporation should be carried out several times.

(iv) Seal with gutta-percha and leave for several days before filling permanently.

If *chlorinated lime and acetic acid* used—

(i) See that chlorinated lime is dry and of good quality.

(ii) Apply rubber dam.

(iii) Mix chlorinated lime with water into a stiff paste.

(iv) Remove as much moisture as possible from it with wool.

(v) Pick paste up on a wood point which has been dipped in a 50 per cent. solution of acetic acid and pack it rapidly in the cavity.

(vi) Seal.

(vii) Repeat treatment if necessary.

If *aluminium chloride* (*Dr. Harlan's* treatment) is adopted—

(i) Apply rubber dam.

(ii) Swab with peroxide of hydrogen.

(iii) Dry.

(iv) Pack aluminium chloride into cavity.

(v) Moisten with water.

(vi) Leave for five minutes.

(vii) Wash with distilled water.

(viii) Syringe with solution of bicarbonate of soda.

(ix) Dry.

(x) Paint with copal ether varnish.

(xi) Seal temporarily with gutta-percha.

Chlorine is liberated in this method.

If the *reduction process* is adopted—

(i) Desiccate separately 10 parts of sodium sulphite and 7 parts of boracic acid.

(ii) Grind them thoroughly together.
(iii) Keep in a dry place in an airtight stoppered bottle till required.
(iv) Apply rubber dam.
(v) Pack mixture into cavity.
(vi) Add a drop of water.
(vii) Seal immediately and leave for two or three days.
(viii) Re-treat if necessary.

In this process sulphurous acid is set free.

Bleaching of teeth by *ionic medication* has been advocated.

To remove *metallic* stains:—

In all cases apply rubber dam.

Gold, Iron, Copper, Nickel — Adopt *Truman's* method.

Silver — Adopt *Truman's* method, or
(i) Flood with tincture of iodine, and then
(ii) Apply a saturated solution of sodium hyposulphite.

Mercury —
(i) Apply ammoniacal solution of peroxide of hydrogen, and then
(ii) Apply a saturated solution of potassium iodide.

Manganese — Apply a concentrated solution of peroxide of hydrogen (*perhydrol*) saturated with oxalic acid.

In all cases *when the bleaching is accomplished*—

(i) Fill unfilled one-third of root with oxychloride of zinc as an antiseptic.
(ii) Line with oxyphosphate of zinc.
(iii) Fill permanently.

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