**Stomatology in general practice : a textbook of diseases of the teeth and mouth for students and practitioners / by H.P. Pickerill.**

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## IN GENERAL PRACTICE

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### A TEXTBOOK OF DISEASES OF THE TEETH AND MOUTH FOR STUDENTS AND PRACTITIONERS

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# PREFACE

*'He that keepeth his mouth keepeth his life'*

THERE is a somewhat ill-defined territory lying between medicine and dentistry, which to many practitioners is to a certain extent a *terra incognita*.

There is also practically the whole field of dental disease and its treatment which is certainly the same to the general practitioner of medicine. It is from a firm conviction that these fields should be recognized and well known that this book has been written. An attempt has been made to throw a beam of light *across* this territory, so that its main features and important points will stand out in bold relief; it has not been my object to try to illuminate the minute details of the whole area to be covered.

It should be understood that this book is not meant to replace what will be found in the usual textbooks on medicine, surgery, and dental surgery, it is rather meant to correlate, amplify, and elucidate much of the information there given.

To expect a medical practitioner or student to read a modern textbook on dental surgery would be futile; the information which would be of value to him would be so scattered and confused with technicalities as to occasion only a waste of valuable time. Yet no one can doubt at the present time that it is *absolutely essential* that practitioners of medicine should be familiar with the pathology and means of treatment of the more common diseases arising in connexion with the oral mucous membrane, teeth, and jaws.

Again, dental students and dentists should have a clear conception of the cause, diagnosis and course of all those oral lesions which are so intimately associated with dental or peri-odontal tissues, and it is hoped that this book will form a useful complement to the increasing number of dental publications dealing exclusively with operative dentistry.

The part played by chronic infections of the teeth and buccal tissues in giving rise to grave secondary systemic and local disorders is rapidly becoming more generally recognized, and it is not sufficient that a practitioner should be content with advising the patient to 'get his teeth seen to' or 'to go to the dentist', and thereby absolve himself of all responsibility. In view of the extent to which dentistry is at present practised by unqualified, untrained, and illiterate individuals, it is eminently desirable or even necessary that every medical practitioner should be in a position to be able to tell a patient exactly what the oral condition is, to see that the seriousness of the condition is recognized, to give sound advice as to treatment, and to see that the latter is carried out.

Very valuable indications, too, from a diagnostic point of view, may be frequently gained from a critical examination of other tissues in the mouth than the mucous membrane of the tongue. Not only so, but in a large number of cases a medical practitioner is obliged by circumstances to undertake treatment—to give only two instances: 'toothache' is treated by extraction, and fracture of the jaw by a four-tailed bandage—typical examples of simple, empirical, but frequently eminently bad methods of treatment resulting entirely from lack of training or teaching.

There should be no lack of training or teaching, it should be compulsory for every medical student to attend a short class on dental surgery or stomatology before his final examination, in order to ensure that he at least has some knowledge of diseases which will be confronting him

and complicating his work every day of his professional life. Such a course is obligatory in this university, and the present publication is a somewhat amplified form of lectures which have been delivered during the past year to a post-graduate class of medical practitioners and also to medical students.

As regards the general scope of the work, diseases of the mucous membrane and the jaws have been described rather from an ætiological point of view, and the treatment indicated. Tumours of the jaws arising from dental tissues have been dealt with more fully, because this subject is usually very inadequately dealt with in general textbooks, yet such cases require early diagnosis in view of their tendency to malignancy. Fractures of the jaws and the indications for the various kinds of splints have been described somewhat fully, since I have found this subject to be generally but imperfectly grasped.

The pathology, preventive and remedial treatment of dental disease, have been discussed in some detail with especial reference to the physiological means of immunity ; surgical methods of treatment have been described to such an extent as will enable the reader to adopt rational and correct methods from a palliative point of view chiefly, and to this end a short list of necessary instruments recommended for the purpose has been added in an appendix.

Special chapters have been devoted to the effects of systemic diseases upon the oral tissues, and to the effects of oral sepsis upon systemic conditions.

A rapid but effective method of recording the examination of school children's teeth has been described in an appendix.

No particular reference has been made to diseases of the tongue, these are generally sufficiently well recognized, and in any case lie outside the scope of this book. The original matter in the work includes the combined surgical and

mechanical method of treating cleft palate; the preventive treatment of dental caries; surgical treatment of dental arthritis; and a simple method of treating fractures of the jaw.

It has not been thought necessary to include a bibliography in a book of this kind, but most of the standard works have been consulted and reference has been made to current literature.

The majority of the illustrations are original, for a few I am indebted to the kindness and courtesy of others; acknowledgement of these is made in the text.

H. P. P.

University of Otago
1912

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## CHAPTER I

### HISTORICAL

Two things are obvious from a study of the earliest records of the practice of Medicine; the one that even in those remote times diseases of the buccal tissues and the teeth were not infrequent, the other that the treatment of these diseases was carried out by the practitioners of Medicine. In fact, it is only in comparatively recent times that medical practitioners have ceased to pay the same attention to such diseases as they formerly observed.

The origin of any systematized practice of medicine is found to be coeval with the beginning of a condition of higher civilization, doubtless being a case of demand and supply, that is to say, that as man receded from a purely natural mode of life and his environment became more artificial, so doubtless fresh diseases sprang up demanding the increasing care and thought of the more mentally trained of the community, i.e. the priests. Among diseases which thus arose were pathological conditions of the teeth and oral mucous membrane, due most probably in great measure to those dietetic changes which are inseparably connected with the social evolution of the human race.

One of the first nations to undergo such evolution was undoubtedly the Egyptian, and of this race we have records which date back to about thirty-seven centuries before the Christian era. The 'Ebers' papyrus, the compilation of

which was probably started about that time, gives detailed descriptions of the treatment then in vogue by the priests for many maladies, and amongst them a large number of prescriptions for the treatment of diseased buccal tissues. Curiously, nearly all these contain honey—probably to cover the taste of the other obnoxious ingredients. There are no indications that the Egyptians of this period practised surgery of the teeth in any form (apart from the use of applications), and although it has been stated that mummies had been found with teeth filled with gold this has so far lacked actual proof.

Asklepios, the god of Medicine (twelfth to thirteenth centuries B.C.), is believed to have been the first to introduce the practice of extracting teeth.

Hippocrates (460 B.C.), the 'father of Medicine', and himself one of the Asklepiadi, was the first to make any systematic study of the teeth, their development, diseases, and treatment. He wrote a book *De dentitione*, concerning diseases accompanying the eruption of the deciduous teeth, and described the intra-uterine formation of teeth.

It is very probable that the term 'milk teeth' dates from this time, for Hippocrates says in *De carnibus*, 'the first teeth are formed by the nourishment of the fœtus in the womb, and after birth by the mother's *milk*. Those that come forth after these are shed are formed by food and drink.'

He recommended counter-irritation for painful teeth and extraction for loose teeth, and also prescribed many fanciful dentifrices and mouth-washes for the preservation of the teeth. That an ulceration of the tongue might be caused by a rough or sharp tooth was first noted by him, and he taught too that many lesions of other organs were associated with oral disease. He introduced the wiring together of the teeth in cases of fracture of the jaws—a method which has recently been 'rediscovered'.

Aristotle (384 B.C.) made many remarkable observations on the comparative anatomy of the teeth, but also seems to have originated the error that men have more teeth than women; he speaks of iron forceps being used to loosen teeth, the latter being afterwards extracted with the fingers.

Celsus (30 B.C.), who, if he was not a great physician himself, by his writings gave a very good account of the practice of medicine in Rome in his time. He described various forms of stomatitis, including probably what is now known as *Cancrum oris*, since he says that the ulceration might spread extensively and lead to the death of the child. He advised the extraction of teeth in certain cases, but only after a large number of sedative applications had first been made and failed. He also devoted considerable attention to fractures and dislocations of the lower jaw, and described a 'four-tailed' bandage.

Another writer of the same period, Scribonius Largus, who is said to have accompanied the Emperor Claudius to England in A.D. 43, is of some interest to us in that he inveighed strongly against the growing tendency to specialization. He treats of the cure of odontalgia by fumigations and masticatories, and relates that the Emperor's wife owed the whiteness of her teeth to the use of ammoniacal tooth-powders. With this writer seems to have originated the theory, not yet extinct, that caries of the teeth is due to the action of small worms, and that these can be expelled by suitable fumigation.

In Galen we have another name inseparably connected with the history of Medicine, and, if one may judge from his writings, no small part of his work related to the teeth. He first noted that teeth were supplied with nerves, and he considered that caries was due to a corroding action from within—similar to ulceration. He enumerated a large number of remedies for odontalgia, but seems to have been somewhat afraid of extraction and its after effects. He

appears to have been the first to distinguish between dental myelitis and periodontitis, though he likewise promulgated an error, which still exists, that human teeth are capable of growth after complete formation in order to compensate for physiological attrition.

In the early Middle Ages some of the most eminent exponents of the art and science of Medicine were the Arabians. Of these, RHAZES (circa A.D. 850), AVICENNA (A.D. 980), and ABUCLASIS (A.D. 1050) were the most famous. These all included the treatment of dental and other oral disorders in their practice ; they invented quite a large number of instruments for removing calculus, loosening teeth (previous to extraction), regulating teeth, and for the performance of actual cautery. Abuclasis has the merit of having applied transillumination for the diagnosis of oral tumours. The sun was the illuminant, and only swellings which freely transmitted light were to be operated upon, tumours opaque to sunlight were to be avoided. The same surgeon also advocated the re-implantation of teeth which had been dislocated by trauma, and ligatured them in position with fine gold wire.

Passing on now to more modern times, namely the sixteenth century, we find such men as VESALIUS, EUSTACHIUS, and FALLOPIUS all paying considerable attention to the jaws and teeth, more especially, of course, from an anatomical point of view. Vesalius, for instance, described accurately for the first time the anatomy of the roots and pulp-cavities of the teeth ; he noted also that the ' wisdom ' teeth, although completely formed, frequently did not erupt at all. Fallopius paid considerable attention to the development of the teeth, and, considering that of course the microscope was unknown to him, his descriptions are wonderfully accurate ; whilst to Eustachius belongs the distinction of publishing the first book on the anatomy of the teeth (*Libellus de dentibus*, 1563).

Ambroise Paré, who flourished in this period, was essentially a surgeon; he was neither anatomist nor theorist, but confined himself almost entirely to the practice of Surgery, and in this the surgery of the mouth was an important part. He devised and used obturators for the treatment of acquired clefts of the palate; for fractures of the jaw and dislocation of the teeth he used practically the same method as Hippocrates; whilst for the extraction of teeth he had an elaborate array of formidable-looking instruments called pelicans, besides files, lancets, levers, &c., for other operations connected with the teeth.

During the seventeenth century Highmore described the Maxillary antrum, but although he recognized that diseased teeth might be associated with a continuous discharge from the antrum, he did not regard this as pathological but as quite a natural or normal occurrence, and did not even suggest that the condition should be treated. In fact no form of treatment was adopted for empyema of the antrum until the beginning of the next century, when William Cowper showed that by extracting the first molar tooth and perforating the antrum through its socket, the sinus could be cleared out, irrigated, and drained.

During the same period the microscope was devised by Leeuwenhoek, and the minute investigation of human tissues received a fresh impetus; probably the first bacteria ever seen were described by Leeuwenhoek as 'animalculæ', found in the deposits removed from interdental spaces in the mouth.

Somewhere about the year A.D. 1700 the treatment of the teeth began to be recognized as a speciality, for Pierre Fauchard[1] says 'that about that time in Paris it became necessary to pass an examination to qualify as a dentist'. Pierre Fauchard was himself a surgeon practising oral and dental surgery, and had been 'the disciple of Alexandre

[1] *Le Chirurgien Dentiste*, 1746.

Poteleret, Surgeon-in-chief to His Majesty's Ships, who had great experience in diseases of the mouth'.

The great JOHN HUNTER paid a considerable amount of attention to the anatomy, pathology, and surgery of the teeth. His two books—*The Natural History of the Human Teeth* (1771) and *Practical Treatise on the Diseases of the Teeth* (1778)—placed the subjects for the first time in England upon a rational and scientific basis. Hunter was a great exponent of the operation of implantation and transplantation of teeth, and strangely enough, although he knew naught of bacteria, insisted upon the necessity of boiling the tooth which was to be used. Despite this, however, the operation fell into disrepute owing to the cases of syphilis which were reported to have been thereby transmitted.

THOMAS BERDMORE, dental surgeon to George III, and who was contemporaneous with Hunter, recognized the necessity of medical students knowing something concerning the diseases and surgery of the teeth, and so held classes for that purpose, as also did FOX, a surgeon on the staff of Guy's Hospital at the beginning of the last century.

Thus we have briefly traced the origin and course of 'Stomatology' from the earliest records down to within measurable distance of the present time. We have seen that all those whose names are inseparably connected with the history of general medicine have also materially assisted in the growth and development of this special subject, and all that we know we owe to the gradual but persistent accumulation of facts and data by these and such-like men.

# CHAPTER II

## THE DEVELOPMENT, ANATOMY, AND PHYSIOLOGY OF THE TEETH AND ADJACENT STRUCTURES

Teeth are commonly said to be dermal appendages and to be analogous to hairs and nails. This is very misleading, and indicates only a small part of the truth. By far the greater part of a tooth—the part which gives it its characteristic shape and structure—is mesoblastic in origin; that is to say, the dentine, pulp, and cement are derived from mesoblast, whilst the cap of enamel covering the crown is the only part which arises from the epiblast. It does not accord with our present purpose to give a *detailed* description of the development of the teeth, but yet it is essential that there should be a clear conception of the salient points in such development.

The accompanying diagrams (Fig. 1) illustrate graphically the various stages in the development of the lip, the labio-dental sulcus, and the teeth; for a fuller description readers should refer to other works.[1]

Points which should be noticed are (i) the very early period at which development commences; (ii) the condensation of mesoblastic tissue before the advancing epithelial downgrowth (*Zahnleiste*)—'barring' as it were its further advance. It is essential that this should occur; if it does not form in this manner a kind of odontoma results, to which we shall refer later. (iii) The very early presence

[1] Cunningham, *Anatomy*, 3rd edition; or Tomes, *Dental Anatomy*, sixth edition.

and proximity of the *permanent* to the deciduous tooth-germ on the lingual side of the jaw.

Calcification commences at the line of junction of the epithelium and mesoblastic cells, in the tooth-germ.

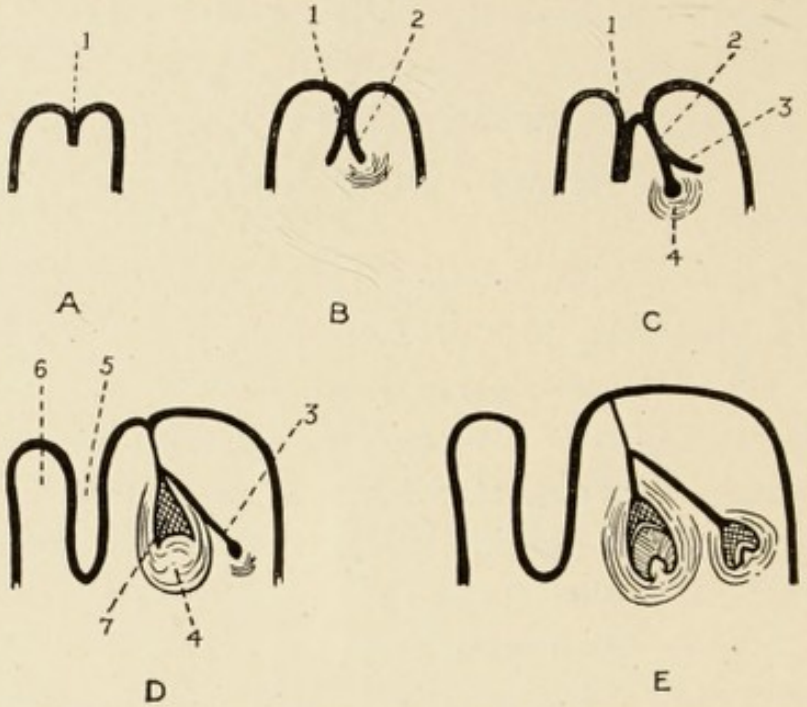



FIG. 1. Diagrammatic representation of the development of the teeth and lips. A, Medial section of the mandibular arch, about the fortieth day, (1) downgrowth of epiblast. B, Division of epiblastic downgrowth into (1) *Lippenfurche* and (2) *Zahnleiste*. C, Further development of B, *Lippenfurche* much thickened. Bulbous extremity to *Zahnleiste* and commencing division at 3. Condensation of mesoblast at 4. D, Central cells of *Lippenfurche* atrophy and disappear, thus labio-dental sulcus (5) and lip (6) is formed. Epiblast at end of *Zahnleiste* has become differentiated into enamel organ (7). Condensation of mesoblast (4) is forming dentine papilla and tooth-sac (4). The offshoot of the *Zahnleiste* towards the *lingual* side is beginning to form the enamel bud for the *permanent* tooth-germ (3). E, Stage at which calcification of deciduous tooth is well advanced and permanent tooth-germ fully formed and to the lingual side. Condition at birth or shortly afterwards.

Enamel is formed on the lower surface of the epithelium, develops upwards and outwards, and its developing margin *increases* in circumference until completion.

The enamel organ gradually disappears ; part of it, however, remains as a thin pellicle of hardened epithelial cells on

the surface of the enamel after eruption (literally, the skin of one's teeth), and is known as Nasmyth's membrane. The enamel, therefore, when it is completed, is without the pale of nourishment as derived from the blood-supply to a tooth.

Dentine is formed on the upper surface of the 'dentine papilla', grows inwards and downwards, and its developing margin *decreases* in size.

Calcification on the surface of the dentine papilla ceases after a time (from two and a half to three years after eruption), and the uncalcified central portion becomes the 'pulp' of the tooth.

The precise nature of the union effected between the enamel and the dentine is not known. (In fully developed teeth the cap of enamel can be suddenly split off a tooth by the application of heat.)

During development the tooth is surrounded by a connective-tissue sac, fibrous in its outer and cellular in its inner portion; from this is developed internally the cement which covers the dentine of the root, and externally the periodontal ligament which is the bond of union between the tooth and the alveolar part of the jaw-bone.

The alveolus is developed synchronously with the teeth. It is largely absorbed when the deciduous teeth are shed, it redevelops with the eruption of the permanent teeth, and undergoes more or less complete atrophy upon loss of the latter.

In the manner thus indicated, the ten deciduous teeth and their permanent successors are developed, the permanent molars are developed in a precisely similar manner by backward prolongations of the *Zahnleiste*, as the horizontal ramus of the jaw develops in a backward direction.

The skiagram shown in Fig. 2 illustrates the condition of affairs in an infant of one month. It is important to notice, firstly, how near to the surface are the enamel organs of the deciduous teeth, and therefore, that local

lesions of the oral mucous membrane, i.e. stomatitis either bacterial in origin or caused by too hot food, may injuriously affect the teeth by causing an irregular cessation of function of the enamel-forming cells.

Secondly, the existence of permanent tooth-germs should be remembered, and the commencing calcification of the

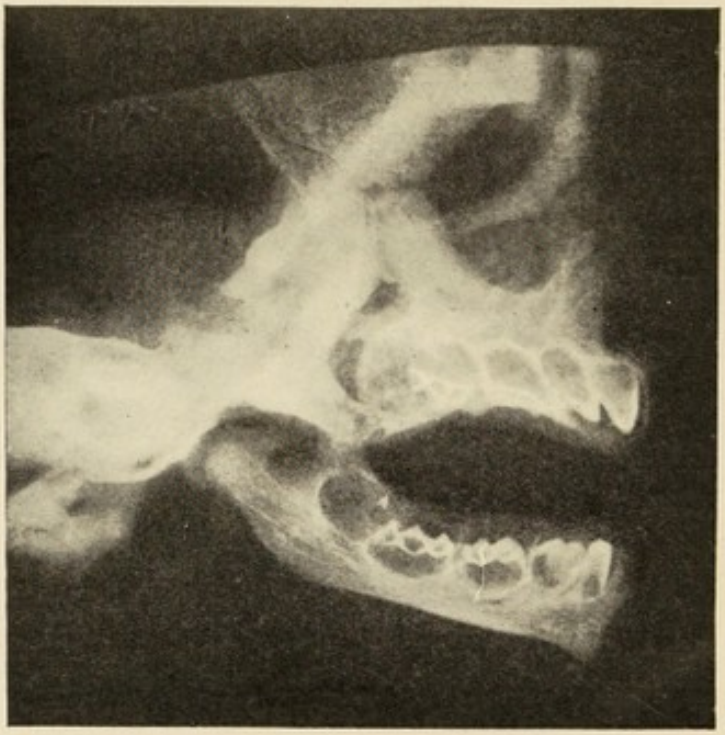


Fig. 2. Skiagram of the jaws of an infant one month old. Note the commencing calcification in the first *permanent* molar. (From Symington and Rankin's *Atlas of Skiagrams*, by permission.)

first permanent molar is obvious in Fig. 2. So that even at this early age the occurrence of systemic disorders may cause the teeth, the enamel especially, to be hypoplasic (i. e. pitted or honeycombed).

## Anatomy and Physiology

For purposes of description teeth are divided into three portions—crown, cervix (or neck), and root. The crown has five surfaces, which are named respectively medial and distal, labial (or buccal) and lingual, and occlusal (or

incisive). The medial surface is that which is towards the centre-line of the mouth, supposing all the teeth were spread out in a straight line from the incisors. The other surfaces are self-explanatory, and it will not be here necessary to describe the features of these in more detail,

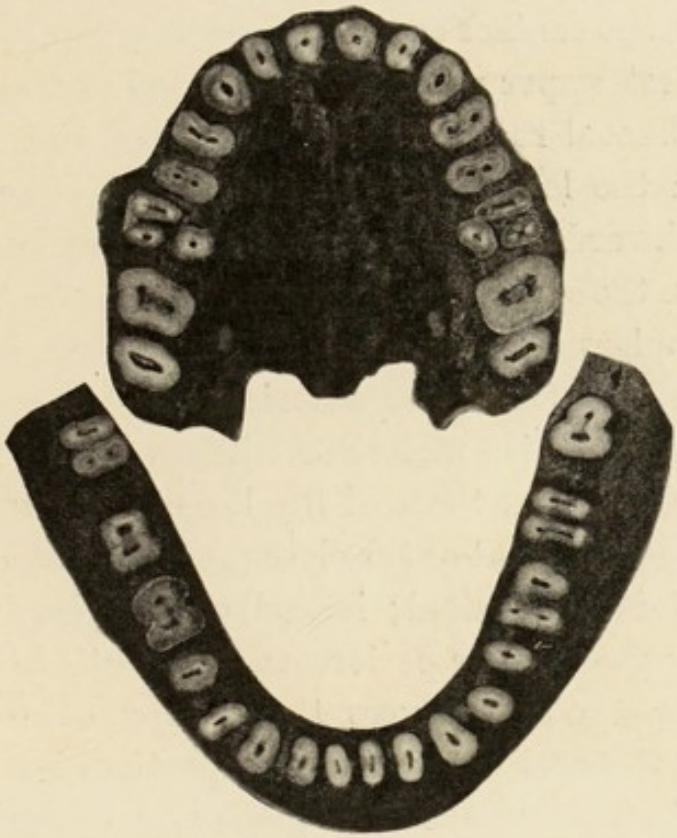


FIG. 3. Upper and lower jaws, showing sections of the teeth *in situ*. All the molar teeth have not been cut at quite the same level. For instance, the second upper molars have been cut through a little higher than the first molars, and hence do not show the division into three separate roots. The variation in the position and number of the root canals, especially in the lower molars, is also shown.

except to point out that the medial and distal surfaces of adjoining teeth are always convex from above downwards, and from side to side, and should touch each other only at the 'point of contact' about two-thirds of the distance between the cervix and the occlusal surface.

Any deviation from this normal contact conduces to the occurrence of disease.

It is essential, if only for the operation of extraction, that the number and disposition of the roots of the teeth should be quite familiar to the operator. For instance, the details of the accompanying illustration (Fig. 3) should be able to be visualized at any moment.

Notice particularly that in the upper jaw the incisor roots are circular, the canines oval to triangular, and the premolars oval (the first upper premolar as a rule has two roots), the two small buccal roots, and the large palatine root of the molars : in the lower jaw the incisor roots are oval from before backwards ; the premolar roots are less oval and shorter than the corresponding upper teeth ; the two roots of the lower molars are very much flattened from before backwards, and are placed one anteriorly and one posteriorly. The roots of the third molars or wisdom teeth in both jaws tend to coalesce, and those of the lower teeth to be curved backwards, on account of the close proximity of the contents of the inferior dental canal ; indeed, the inferior dental nerve occasionally grooves, or perforates, the root of this tooth.

**The Enamel** which covers the crowns of the teeth is thickest in the occlusal or incisive portion, having here a *thickness of about* 2·5 mm. and gradually becoming thinner towards the cervix, where it terminates. It is highly important to realize that the enamel is not a ' veneer ' merely, but is of substantial thickness, and being one of the hardest organized substances in nature is eminently adapted for cutting and *grinding* even the hardest of food. Tested in the dynamometer between plates of hard vulcanite, the cusps of teeth will withstand, without fracture, a pressure of 350 lb. suddenly applied—a force considerably greater than anything which is likely to be applied during mastication.

In minute structure, enamel is composed of hexagonal prisms which are somewhat flexuous in outline, but in a general direction run inwards *at right angles to the surface*,

and it is in such direction that enamel is most readily fractured or cleaved. Although enamel is said to contain no organic matter, yet occasionally painful sensations are conveyed from stimulation of its deeper portions; this is due to the presence in that situation of small ovoid spaces, 'enamel spindles,' filled with protoplasm and connected with the dentinal fibrils.

The surface of the enamel, as the author has shown,[1] is normally traversed by minute corrugations, or *imbrications*, running at right angles to the long axis of the tooth; these are more developed in civilized than in uncivilized races, and are a factor in the retention of food-stuffs.

Enamel is of course in a plastic state when first laid down, and subsequently becomes intensely hard; this hardening process is, though, as I have elsewhere shown, not complete when the tooth is erupted, but should continue for a considerable time afterwards, being probably aided by the infiltration and deposition of lime salts from the saliva. Teeth in which this has taken place to a maximum extent have a yellowish dense appearance, and I have termed them 'sclerotic' teeth; teeth in which it has not occurred have a whitish opaque appearance, and these I have classified as 'malacotic' teeth (*μαλακός* = soft).

Enamel of the latter class I have shown to be exceedingly permeable to a stain like silver nitrate, whereas sclerotic enamel is scarcely permeable at all. Sclerotic enamel shows a greater degree of hardness when tested by a diamond point in the sclerometer, and a greater resistance to decalcification by acids than malacotic enamel.

**The Dentine** is composed of a calcified matrix of collagen through which run tubules, converging from the periphery to the pulp chamber, and containing the sentient distal processes of the odontoblast cells of the pulp. Thus, in transverse section, dentine is seen to be perforated by numbers of minute

[1] *Prevention of Dental Caries and Oral Sepsis*, London, 1911.

'holes'—in the living condition filled by the 'dentinal fibrils', in the devitalized state by air or organisms; in any case dentine is to be regarded as a *porous material*, capable of absorbing and transmitting substances either beneficial or deleterious (Fig. 4). The fact that dentine contains normally a considerable proportion of sentient, soft, 'living' tissue has also to be remembered when operating upon it. It is now believed that dentine is laid down in a laminated manner at right angles to the direction of the tubules, and in this direction, when it is decalcified either artificially or by disease, it is most readily cut and removed.

Further, Mummery has adduced considerable evidence to prove that the 'odontoblasts' are not the cells which form the dentine, but osteoblasts which are interspersed between the large cells called 'odontoblasts'.

Dentine is in any case only a modified bone, and this view of its development, which is generally accepted at the present time, shows its formation to be exactly analogous to bone.

The teeth correspond to other bones in having a central medulla, modified certainly, but analogous. The chief point of difference between the teeth and other bones is the fact that the integument in this situation has become highly calcified and the greater portion of its organic nature has disappeared. If this integument had not become so calcified it would probably not occur to any one to classify the teeth as other than bones.

**The Pulp** is composed of a stroma of delicate connective tissue imbedded in which are blood-vessels, nerves, and specialized cells. A capillary network is formed at the periphery, and just beneath its surface the nerves break up into the plexus of Raschkow; thus the pulp is most vascular and most sensitive at the periphery.

The specialized odontoblasts form a single layer of epithelioid cells on the surface of the pulp, and because they adhered to the dentine rather than to the pulp when the latter was

forcibly removed they are termed the 'membrana eboris'. No nerves have been proved to enter the dentine, and the extreme sensitiveness of the latter has been accounted for by the fact that the terminations of the nerves in the plexus of Raschkow arborize around the odontoblasts and their proximal processes.[1]

The medulla, or pulp, in shape is a small replica of the tooth, a small cornu being situated under each cusp, and

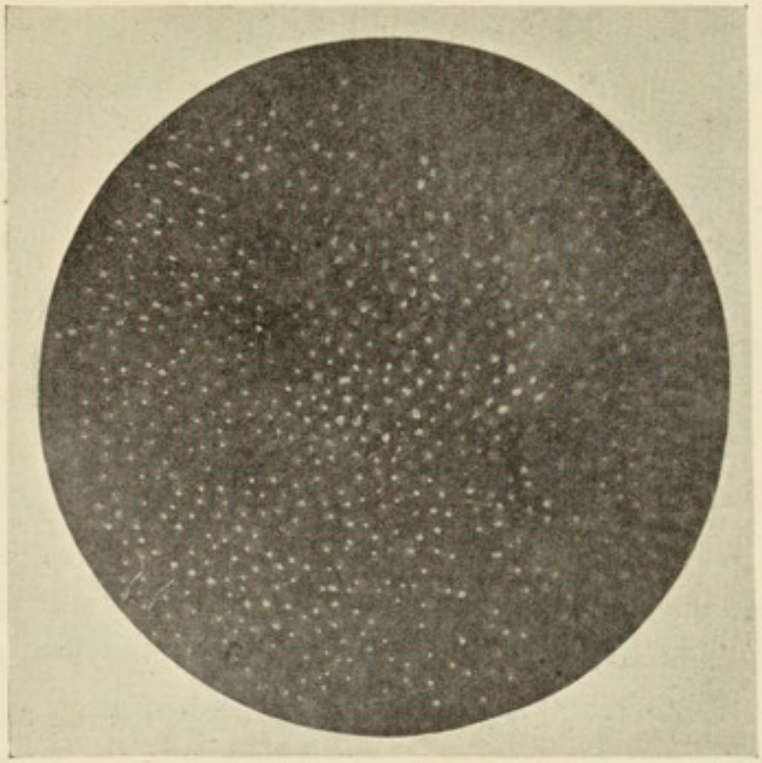


FIG. 4. Transverse section of dentine.

each root contains a central core of pulp tissue. In old age, and under the stimulus of chronic irritation, the pulp may resume its developmental function, and become entirely obliterated by the formation of adventitious or 'secondary' dentine.

The *cement*, which normally covers the root in a thin layer, is structureless in the cervical region, but practically

[1] Retzius, *Biologische Untersuchungen*, N.F. iv, v, vi (1892–4); Hopewell Smith, *Histology and Pathohistology of the Teeth*, p. 166.

always in the region of the apex contains lacunæ and canaliculi, but no Haversian systems.

When the cement is thicker, and contains dense masses of lacunæ, it is pathological, and is the effect of chronic inflammation.

Sharpey's fibres enter the surface of the cement and serve to connect it to the peri-odontal ligament.

**The Peri-odontal Ligament** is circular or conically shaped, and serves to sling the tooth, as it were hammock fashion, in its socket. The ligament is composed of connective tissue and fibres, between which run blood-vessels and nerves. The fibres are of the non-elastic white variety, and are continuous with the Sharpey's fibres in the cement on the one side, and with the periosteum of the alveolus on the other; they are very scanty in the apical region, and the arrangement of these fibres allows the tooth a certain amount of upward, downward, and lateral movement in the socket; they also help considerably to minimize the force of mastication being transmitted to the jaw-bones. There are also found in this situation scattered clumps of epithelial cells—the epithelial sheath of Hertwig—remnants of the *Zahnleiste.* Normally these cells remain quiescent, but occasionally they 'revive', proliferate, and cause neoplasms.

It is important to realize that teeth[1] are far more analogous to separate and individual bones than to hairs or nails, and that their connexion with other bones is by means of

[1] The fact that the teeth of the dogfish are very similar to, and in early life continuous with, the dermal spines on the rest of the body, does not prove that all teeth are dermal appendages. The dermal spines of the dogfish are particularly specialized structures, are exoskeletal and largely mesoblastic. Moreover, as has been pointed out by Ryder, it is possible to have teeth formed apart from the epiblast altogether, namely on the pharyngeal bones of certain fishes. 'Teeth' which are truly skin structures are the horny plates which do duty for teeth in the ornithorhynchus.

distinct *joints*, usually classified under synarthrodia; but this is not strictly correct, since a quite appreciable amount of movement is normally present.

To appreciate this one only has to observe the considerable facets which are present on the contiguous surfaces of the teeth of native races—produced by the movement of one tooth against the other.

(The pathology of this region falls more into line with the pathology of joints in general, and is much more intelligible when viewed thus than when the peri-odontal ligament is regarded as a 'membrane'.

Again, when this normal movement of the teeth is artificially prevented, as it is occasionally by the fitting of some forms of 'bridge work', the 'joints' become sooner or later disorganized and suppurate.)

**The Alveolus**, as we have already pointed out, is developed with the teeth and disappears with them. It is composed of a very thin layer of compact bone externally, and of spongy cancellous bone internally. Its function is to form sockets for the teeth, to afford attachment for the peri-odontal ligaments, and to lessen the shock of mastication. The outer plate is usually thinner than the inner; it is therefore, as a rule, fractured in the removal of teeth, and it also offers less resistance to the exit of inflammatory products.

**The Muco-periosteum** covering the jaws calls for no particular description; it is simply a mucous membrane externally and a periosteum internally. Its free margin, or the 'gum', should normally end in *thin delicate pink* festoons following closely the termination of the enamel upon the teeth; the surface is as a rule a little stippled. Any departure from these characteristics is to be regarded as abnormal and pathological.

### Eruption of the Teeth

No entirely satisfactory theory has yet been advanced to account for the mechanism causing the teeth to erupt, therefore we cannot discuss the ills which are said to be attendant upon 'teething' from a rational point of view—'teething' has been for centuries and still continues to be a most convenient stand-by for mothers and practitioners seeking a *fons et origo* for any of the ills attendant on infancy. Although there is obvious reason to believe that the cutting of the teeth is accompanied in many cases by pain, and that such stimuli may in an unstable nervous system be reflected along abnormal paths and thus give rise to reflex symptoms more or less remote, there is no reason to associate other organic lesions with the eruption of the teeth.

It is of advantage, however, to know approximately when both the deciduous and permanent teeth should erupt; it is often a valuable aid to a practitioner, when examining a child, to be able to approximately tell a child's age from an examination of the teeth (besides which it inspires the confidence of the parents); in addition, it is exceedingly necessary to know when a deciduous tooth either should be or may be extracted.

The ages, therefore, at which a given tooth should erupt must be quite familiar, and the author would suggest that the following numbers should be committed to memory, i. e.

**6, 9, 18, 13, 26.** *Months at which the deciduous teeth erupt.*

And—

**7, 8, 11, 10, 11, 7, 13, 21.** *Years at which the permanent teeth erupt.*

The numbers apply to the teeth in order from the central incisor round to the last molar, on one side of one jaw only. Lower teeth as a rule erupt a little before the corresponding upper teeth.

These numbers do not pretend to be the absolute exact ages at which all teeth should erupt—individual children vary, of course—but they will be found to be sufficiently exact for practical purposes, and it will be found also that they are easy to remember.

When the deciduous teeth have remained in position some few years their apices commence to be absorbed to make room for the subjacent developing permanent tooth; such absorption starts at from *two and a half to three years before* the permanent teeth erupt, and continues until the whole of the root has been absorbed, when the tooth is or should be shed.

When the permanent teeth erupt, their roots are not fully formed, and the apical foramina are large and patent; absorption of toxins, bacteria, and dangerous drugs like arsenic is very likely to occur, if they gain access to or are applied to the pulp during the stage of open apices, either in deciduous or permanent teeth. The ages at which the apices are 'closed' are from *two and a half years to three years after the eruption* (except the canine teeth, which are nearly complete at eruption).

## The Articulations of the Teeth

The lower teeth articulate with the upper teeth by means of a double series of inclined planes, at least from the canine teeth back to the last molars. (See Figs. 5 and 7.)

The one set of inclined planes is antero-posterior in direction and the other lateral. By means of these each tooth is guided into position and there becomes 'locked'.

The force of mastication falling upon these inclined planes determines, when a tooth is erupting, what position it shall take up. In a normal condition the result is perfect equilibrium and adaptability to function, but should a tooth, during eruption, for some reason not come into contact with the correct inclined plane, it will fall on some other, and will

be thus driven out of line by the force of mastication, and in being itself displaced will tend to displace adjoining

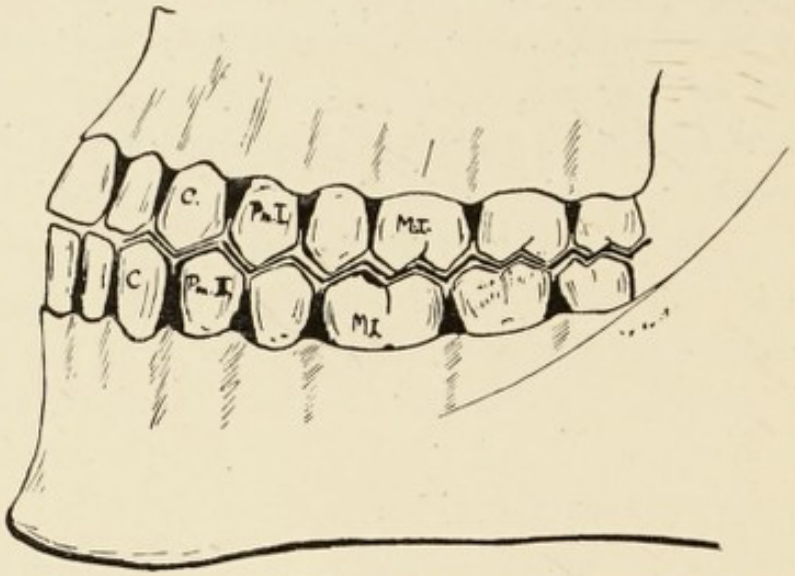



FIG. 5. Antero-posterior locking of lower and upper teeth by means of inclined planes. See also Fig. 7.

teeth also. From this point of view alone the period of the eruption of the teeth from 7 to 14 years of age is a most critical one, and is the period during which a child should be particularly under frequent inspection.

# CHAPTER III

## DEFORMITIES OF THE TEETH AND JAWS

Deformities of the teeth themselves are very frequent, due either to aberrations of the dental germs, or to sudden or continued pressure brought to bear upon the tooth during its development. Probably the most frequent abnormality in teeth is the presence of an extra root or an extra cusp, and this being so, the possibility of more roots than normal has to be remembered particularly in the treatment of lower canine and molar teeth.

More important, however, than deformity of individual teeth is deformity of the whole dental arch and jaws due to the abnormal position of teeth.

Only the teeth in the anterior region may be irregularly arranged and the remaining teeth and jaws not much affected, or the whole of the lower jaw may be either too far back or too far forward in relationship to the upper teeth. When the lower teeth are too far forward the patient is said to have an 'underhung' bite, the mandible is usually too large, and the mental process over-developed, giving rise to a somewhat 'bull-dog' appearance. When the lower jaw articulates too far back there is usually considerable crowding of teeth in the incisor region of the mandible, both jaws are much too narrow, the palate is very frequently 'gothic' in shape, and the upper incisors protruding and perhaps procumbent.

An illustration of this latter deformity, which is becoming increasingly common, is shown in Fig. 6.

It will be observed that the lateral incisors are placed

somewhat behind the central incisors, thus giving rise to what is popularly called a 'double-row' of teeth; this and the constriction of the jaws causes a very rodent-like appearance.

It is scarcely necessary to point out that the presence of such deformities interferes considerably with the exercise

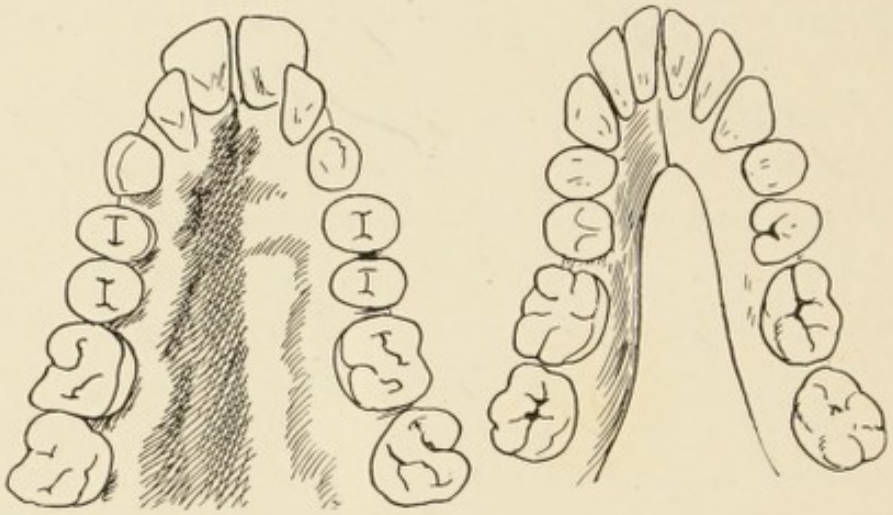


Fig. 6. Narrow rodent-like jaws and 'gothic' palate, a very common form of deformity.

of the function of the teeth, and leads also to the development of other diseases.

### Causes of Deformities

1. Strange as it may appear, it is becoming increasingly necessary to call attention to the fact that the function of the teeth and jaws is primarily to masticate food, and that an organ which does not fulfil its function does not, and cannot, develop to a normal extent.

It is becoming increasingly prevalent to relegate the trituration of food to machines and other devices, previous to its presentation as an article of diet.

In some instances, undoubtedly, the condition arises through *heredity* pure and simple, a child inheriting the large teeth of one parent with the small jaws and features of the other, but in the larger percentage of cases there is no reason

to suspect this cause, and the jaws are too small for the teeth because they have not developed to their full dimensions, and they have not developed because they have not been used to their full physiological extent.

It may be stated as a physiological axiom, that the bones of those individuals which possess the strongest muscles are much better developed than those of persons with weak muscles. The full development of the bones is dependent upon the musculature, and the latter of course upon exercise of function and the amount of resistance to the latter. Now the exercise which the muscles attached to the mandible are called upon to perform is chiefly that of the mastication of food. If the food be semi-liquid and soft, very little muscular effort will be called forth, the muscles will not develop, and the mandible to which they are attached will likewise remain undeveloped; whilst, on the other hand, a harder diet will stimulate greater muscular and osseous development.

There is another aggregation of muscles too, the tongue, which is, or should be, largely used in the act of mastication, and, as is well known, the tongue has a distinct influence over the lower jaw in expanding it outwards and forwards.

If the mandible fails to develop in the manner indicated, and it remains shorter and narrower than it should be, the upper jaw is also affected, and a condition of V-shaped arches and palate supervenes, with, as a rule, considerable crowding of the teeth. Reference to Fig. 7 will show that the lower teeth articulate with the upper teeth by means of inclined planes from within outwards, and that any outward movement of the lower teeth is bound to be followed by a corresponding movement in the upper teeth, and if this does not take place neither can the upper teeth move outwards. In this connexion it is to be remembered that it is the lower teeth which erupt first, and to a large extent thus determine the position of the corresponding upper teeth.

Comparatively few muscles are attached to the upper jaw; it is not movable, and its stimulus to growth and development must come largely from the impact and grinding of the lower teeth against the upper in the act of mastication.

It is a fact which has been written of so often as to scarcely need now any proof, that the diet of modern civilized races is becoming softer and more 'pappy'. It seems to be one of the accompaniments, the refinements, the luxuries, which have always attended that phase of social evolution known as higher civilization.

There seems to be inbred in all animals and races of mankind an instinct or almost unconscious tendency to procure food in the most nutritious and assimilable form; this of course originally was but fitting and right, and no doubt has played a most important part in evolution and the survival of the fittest. But unfortunately, now that civilized man has evolved to a position where the obedience to such natural instincts seems to be no longer necessary for his survival, he still retains most strongly this craving for the maximum of nourishment in the minimum of space, with the result that, in order to satisfy the modern taste in diet, all that is fibrous, all that is 'coarse' or apparently 'inert' is most carefully eliminated from all articles of food, firstly by the grower, by a process of natural selection, then by an elaborate system of mills and refineries, and finally by the cook. Anything that can by any possibility be termed 'tough' is anathema.

In this manner and by such means are the teeth and jaws robbed of their normal physiological functions. A large part of the energy which is developed by the enormous machinery in modern mills and refineries represents work which should be being done by the human muscles of mastication.

The effect is obvious and irresistible: the muscles and the bones dependent on them fail to develop to their normal

extent. It can be shown by means of the gnatho-dynamometer that the force of the bite, or strength of the masticatory muscles, may in favourable individuals be equal to 300 lb., but that in people whose diet is habitually a soft one the maximum pressure capable of being exerted may be as low as 50 lb., and once this habit has been established it tends to increase, since the peri-odontal ligaments become tender from lack of pressure, and any increase of force is resented by the teeth.

One might as well expect the bones of the arm to develop to their full and normal extent by keeping the arm constantly in a sling from infancy, as to expect the teeth and jaws to develop normally when their natural function is kept in abeyance as it is by the prevalent and habitual consumption of pretriturated and predigested food.

The jaws therefore become too small for the teeth when erupted (and also probably before eruption). The teeth take up irregular and crowded positions, and thus their resistance to caries is very considerably lessened because of the ease with which food-stuffs lodge between them, and the increased difficulty of its removal either by natural or artificial means before it has undergone fermentation.

Other causes of deformities are:

(*a*) The retention too long or the extraction too soon of deciduous teeth. Under some conditions, chiefly when the pulp has become gangrenous, the deciduous tooth is not absorbed as it should be; consequently the permanent tooth is either delayed in its eruption or it is deflected from its course. It then comes into contact with a wrong set of inclined planes and the deformity is exaggerated and perpetuated. (Not improbably, also, the enamel of the permanent tooth will be pitted and mal-developed, owing to the action of the absorbed toxins on the enamel organ.)

On the other hand, if deciduous teeth are extracted too

soon, the space which they should retain for their successors closes up, and again the permanent tooth is forced to take up an abnormal position. Particularly is this the case if either of the deciduous molars is extracted before the first permanent molars erupt, for the latter in erupting always tend to come forward if there is less resistance, instead of by their presence stimulating the jaw to growth in a posterior direction.

(*b*) Too early extraction of the first permanent molars, by allowing the force of mastication to fall in undue proportion upon the upper incisor teeth, causes the latter to become procumbent—a condition called 'superior protrusion'.

(*c*) Habits such as thumb-sucking and sucking the lower lip, mouth-breathing, &c., all have a similar effect.

(*d*) The presence of supernumerary teeth (as a rule stunted and conical in shape) causes a deformity in the dental arches, owing to deflection of the normal teeth and the bringing into play of a wrong set of inclined planes.

Practically all these deformities appear between the *seventh and fourteenth years*, and it is therefore during that period that a mouth showing any tendency to such deformities should be kept under close observation. Any deformities of the teeth or jaws appearing after the sixteenth year should be regarded with grave suspicion, in that it not infrequently indicates the presence of *malignant disease*.

### The Effects of such Deformities

One of the most immediate effects of such deformities is an increased tendency to dental caries. It is obvious that when the teeth are irregularly arranged it is exceedingly difficult, if not impossible, to keep them free from food débris, and, as we shall show, this is the important factor in the causation of caries.

The relationship between the occurrence of tonsils and adenoids and constricted jaws, too, is an established fact—

though as to which is cause and which effect some difference of opinion exists. It is quite clear that tonsils and adenoids may arise apart from any oral deformity, and when they do become so established they may tend to promote some irregularity of the teeth by reason of the mouth-breathing induced. But where the conditions of vaulted gothic palate, constricted jaws, and post-nasal growths are coexistent, the author is strongly of the opinion that the oral condition is the primary one. It has already been shown that lack

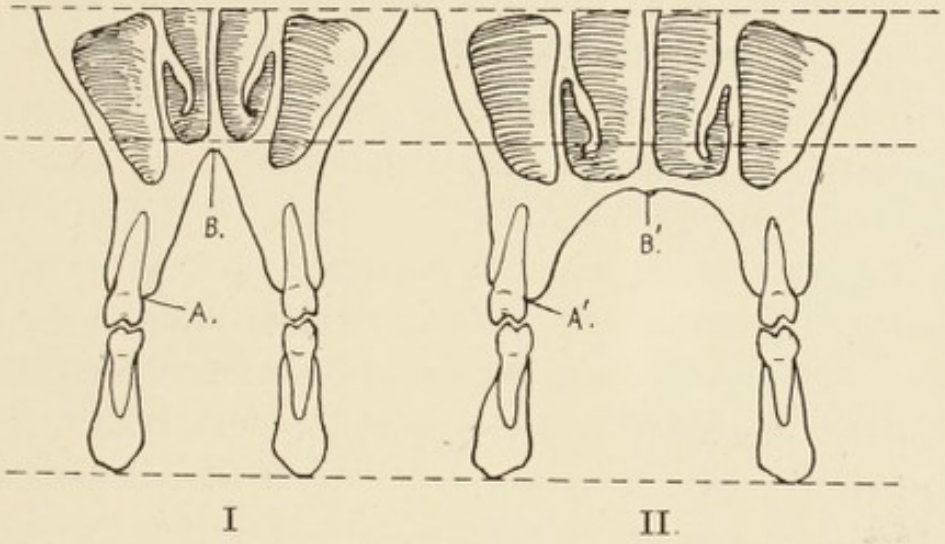



FIG. 7. Diagrammatic section of 'gothic' palate (I) compared with normal palate (II). The articulation of the lower with the upper teeth by means of lateral inclined planes is also illustrated.

of function causes a diminution in the width of the mandible, and by reason of the nature of the articulation of the lower with the upper teeth (by means of inclined planes) a narrowing of the upper jaw passively follows, and it also becomes Λ-shaped, as shown in Fig 7, No. I. No. II shows what should be the normal shape of the oral and nasal cavities. This Λ-shaped jaw does not arise from lack of development of the palatal processes, but from insufficient expansion, for it has been shown by measurements of these cases that the distance between A B and A′ B′ remains the same; the effect on the nasal cavities is obvious, their vertical and horizontal measurements are greatly diminished; further, if

the nasal septum has an inherent tendency to growth, this can only occur by its becoming deflected. The result is that nasal breathing becomes more difficult, and in any slight inflammation of the mucous membrane impossible; the patient acquires the habit of mouth-breathing, adenoids develop in the stagnating cavity, and the tonsils enlarge.

A vicious circle is thus established, for persistent mouth-breathing leads to further deformity of the upper jaw in the direction of inefficient expansion.

### Treatment of such Deformities

The most rational method of procedure is of course to obviate the occurrence of these deformities by eliminating the causal factors. What these are and how it may be done has already been sufficiently indicated, and it is by such means as these that the general medical practitioner may accomplish a vast deal in the suppression of these unsightly irregularities. The necessity of children masticating food which requires the exercise of some force should be pointed out, and if necessary suitable diets arranged for particular cases. Great care should be taken of the deciduous teeth, that these do not become so diseased as to necessitate their extraction before the right age. Should they, however, become *hopelessly septic* they *must be extracted*, since the risk of septic absorption, for perhaps a year or two, far outweighs the evil effect of crowding the permanent teeth.

Deciduous teeth retained beyond the normal age should be extracted if there is any sign of the presence of the permanent tooth—usually to be detected as a bulging of the bone upon palpation immediately below the deciduous tooth. Sometimes a difficulty may arise in deciding *whether a particular tooth is a deciduous or permanent one.*

This may be determined by drawing a fine-pointed probe over the termination of the enamel at the cervix: in

deciduous teeth it terminates in a distinct ridge, in permanent teeth its termination is not noticeable.

In the event, however, of a deformity becoming established it should be treated as early as possible by orthodontic methods. Such methods are of three classes. (i) *Immediate surgical treatment whereby the misplaced teeth are forcibly moved into position and retained there by means of a splint.* The author is at present developing a method of immediate surgical treatment which is giving satisfactory results.[1] The misplaced and irregular teeth are extracted and placed in sterile normal saline solution; fresh sockets are then drilled in the jaw-bone, and the teeth (or some of them) are 'reimplanted' in the fresh sockets, retention being obtained by means of a splint cemented to the remaining teeth, and left in position for some four or five months. The operation is conducted under the strictest aseptic precautions, the patients being in a 'general' hospital, and chloroform anæsthesia is essential. The following are examples of such cases recently treated:—

1. Girl, aged 12. Right upper incisors and canine teeth unerupted, i.e. several years overdue. A skiagram showed them to be 'impacted', i.e. the crowns of all three having met at a point. A metal splint was made to retain the teeth in their normal position. Under the above conditions the three teeth were extracted—the canine was discarded, and the incisors reimplanted. Two years afterwards the teeth are in good position, healthy, and performing their function well.

2. Boy, aged 8 (cleft palate closed by surgical operation). Two central incisors erupting and pointing down the throat. Left incisor moved forcibly forwards by fracturing anterior alveolar wall. Right incisor extracted, and socket formed in line of cleft; there was no bone present, and a flap of muco-periosteum had to be raised and sutured round the

[1] Hitherto unpublished.

distal portion of the root of the tooth in order to form a 'socket'. Retention by splint for the usual time. One year and six months afterwards both teeth are quite *firm* and in good position. (This result was somewhat unexpected, and was contrary to the view expressed at the time of the operation.)

3. Girl, aged 11. 'Petite' features and 'double row of teeth' i. e. central incisors very markedly protruding, and the lateral incisors almost immediately behind them. Circumstances and the *facial type* of the child both contra-indicated mechanical expansion. Deciduous canine teeth present, permanent canines unerupted. In this case *all* the above-mentioned teeth were first extracted, the four canine teeth were discarded, the orifices of the sockets of the central incisor teeth were closed by sutures, and four fresh sockets drilled in correct alinement.

The apices of the four incisors were excised, and the teeth inserted and retained by means of a splint. One year afterwards the teeth were in good position, there was not the slightest pain or tenderness, and the patient used them in a normal manner.

This method may not be justifiable or desirable in many cases of deformity of the teeth and jaws, but there are other cases where the ordinary slower and much more tedious orthodontic methods are impossible of application, either for social, physical, physiological, or pathological reasons.

Resection of portions of the mandible has been performed on a few occasions for marked 'inferior protrusion'.

(ii) Treatment by means of removable appliances, made usually of vulcanite, to which springs, screws, or wedges of wood are attached, which are made to expand the jaws and gradually move the teeth in the desired direction.

(iii) Treatment by means of fixed appliances, whereby the molar teeth are banded, and springy metal arches pass round the outside (and sometimes inside also) of the teeth.

The latter are ligatured to the arch, and thus expansion is brought about; torsion and retraction are obtained by the suitable use of rubber wedges and bands.

Practically all cases are amenable to one of these forms of treatment, and it is exceedingly important that whilst such orthodontic appliances are in use, both they and the teeth should be kept scrupulously clean; care must also be taken that the prolonged presence of such appliances does not interfere with the general nutrition of the child.

A form of treatment which must be adopted only after the most careful consideration of the case is that of extraction, either of the deciduous or permanent teeth.

Parents often request a deciduous tooth to be extracted in order to enable an obviously misplaced tooth to 'come into place'. The rule to be observed is that *no deciduous tooth should be extracted to make room for any other tooth than its own permanent successor.*

## Cleft Palate

It is not proposed here to enumerate either the causes or ordinary surgical treatment of cleft palate; these will be sufficiently familiar to the reader.

There are a number of cases of cleft palate, however, which are not suitable for ordinary surgical treatment, such as:

(i) Acquired clefts due to disease or extensive traumatism.

(ii) Clefts which have missed operation in childhood, and in which the vela are much atrophied.

(iii) Clefts which have been operated upon unsuccessfully.

Such cases may be treated by means of mechanical appliances; these are of two varieties, 'obturators' and 'artificial vela'.

(i) **The Obturator** (Fig. 8) is made similarly to an artificial denture, with a fixed extension posteriorly which fills in the space between the two halves of the soft palate.

The ' plate ' part covers the cleft in the hard palate, and is all that is necessary. The unsatisfactory part is the posterior extension; this being fixed, and necessarily large and somewhat clumsy, does not pretend to reproduce the parts according to nature. The rationale of its construction is to provide a body round which the muscles of the palate and pharynx and posterior pillars of the fauces can close, and thus shut off the nasal cavity when necessary.

The difficulty arises when the patient attempts to raise, or make tense, the soft palate; the vela, not being joined

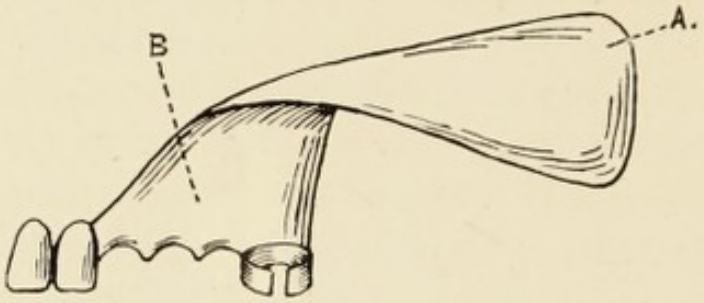



FIG. 8. An ' Obturator '. A, Solid or hollow mass of vulcanite filling up cleft; B, Ordinary dental plate.

in the centre, diverge more than ever, and leave a space between themselves and the obturator.

In some cases, however, of extensive loss of tissue it is the only method of treatment available.

(Obturators of a somewhat different form are frequently required after resection of the upper jaw. In such cases they should either be inserted at the time of the operation or as soon after as possible—within a week or ten days—in order to keep the soft tissues distended and prevent unsightly cicatrization.

In the case of the lower jaw, a stout piece of heavily gilded german-silver wire may be fixed into the divided ends of the bone, in order to maintain their relative positions and to prevent the chin falling backwards.)

(ii) **An Artificial Velum** is a somewhat similar appliance to an obturator, except that the posterior extension for the

cleft of the soft palate is movable and made frequently of soft vulcanized rubber (Fig. 9).

The object of such appliances is to reproduce the natural parts and to provide for voluntary movement and control of the artificial soft palate.

There are a variety of different forms. All coincide in being attached by a form of hinge to a denture fitting over the hard palate, and in having usually two flanges which fit above and below the sides of the cleft, and by means of which the movements of the natural velum is intended to be transmitted to the artificial velum in the centre.

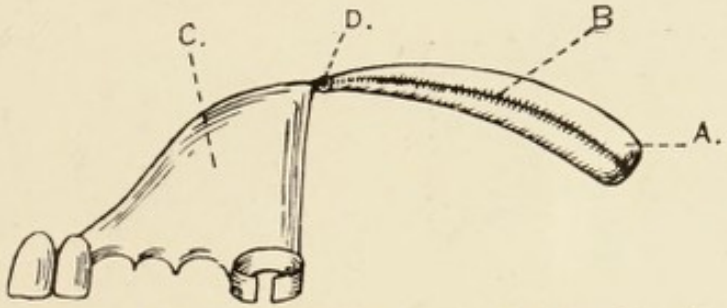



FIG. 9. Diagram of an artificial velum. A, Movable flap of soft vulcanized rubber having lateral grooves at B, into which the margins of the cleft should fit; D, hinge which secures the velum to the denture C.

The weak point in such appliances, however, is the very poor grip and feeble control of the rubber velum by the sides of the cleft. It is obvious that when the levator palati or palato-pharyngeus muscles contract on both sides since they are not joined in the centre, more *divergence* than elevation of the two sides of the palate must occur. Moreover, the soft rubber tends to rapidly deteriorate in the mouth, and requires frequent renewal, which is always a tedious undertaking.

(iii) **The Author's Method** of treating this class of case is by a *combination of surgical and mechanical means*, the object being to make a bar of soft tissue across the cleft posteriorly, to which a hinged artificial velum can be secured, and thus be under complete control.

The technique of the operation is as follows : the posterior parts of the soft palate and uvula are utilized to form the bar in the method shown in Fig. 10. The posterior edge of one half of the velum is pared (A) and the lateral edge of the other (B); both vela are then freed by lateral incisions at their posterior and lateral margins (C). The two prepared halves of the velum are then brought across so that their

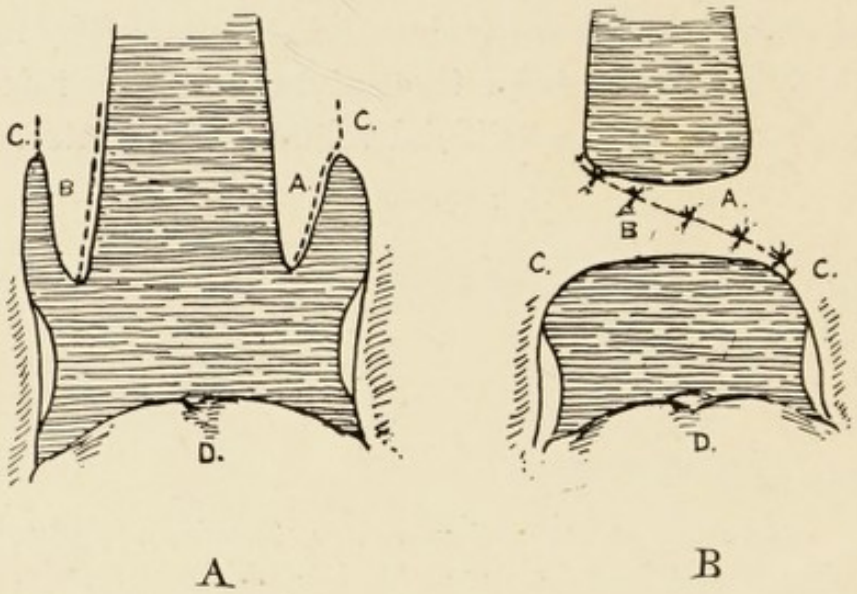



FIG. 10. Diagrams showing author's operation for making the muscular bar in the combined surgical and mechanical treatment of cleft palate. A shows position of parts prior to operation and the margins which are pared. B shows the two halves of the velum brought across and sutured in position.

pared edges are contiguous, and are sutured in this position, all tension being relieved by further lateral incisions if necessary. Fig. 11 shows a cast of a case after the operation. In about three weeks' time an appliance is constructed (Fig. 12) having a hinged artificial velum of hard polished vulcanite accurately moulded to the cleft, and to the posterior wall of the pharynx (in contraction) this passes on the lower side of the bar. Just in front of the bar a broad smooth hook-like process is attached to the vulcanite velum, and passes over the upper surface of the bar, thus anchoring the

vulcanite velum securely to the bar; the whole thus moves up and down rapidly and naturally during speech and deglutition. This method has the advantage of being quite

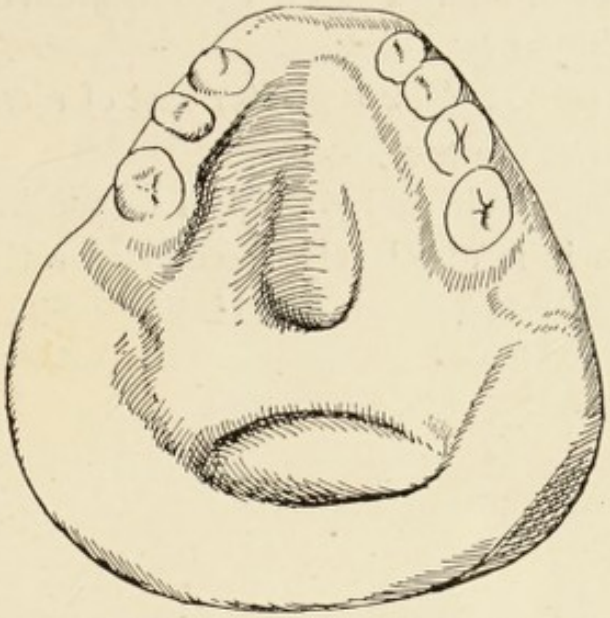


FIG. 11. Plaster cast of cleft after operation. Shows the bar made of the muscular tissue of the soft palate.

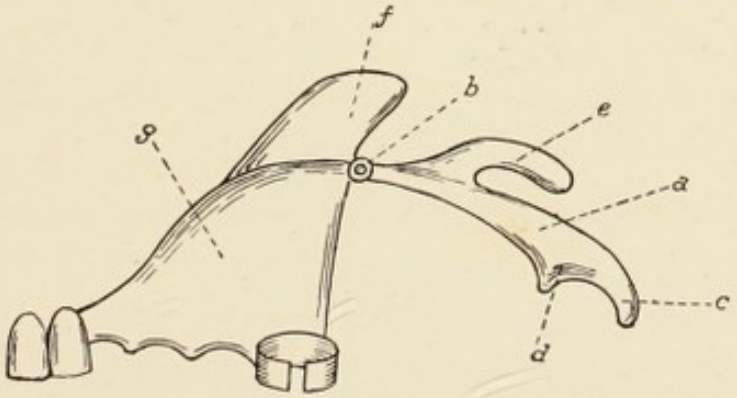



FIG. 12. Author's appliance. *a*, Velum of hard polished vulcanite; *b*, Hinge securing *a* to *g*; *c*, Termination pointed downwards and flattened posteriorly, representing uvula; *d*, Lateral processes effecting junction with anterior pillars of fauces; *e*, Hook-like process passing over 'bar'; *f*, Vertical process of vulcanite representing vomer; *g*, Artificial denture.

simple and rational, and in the author's hands has yielded excellent results.

There is this to be said in its favour, too, that by its means the uvula and its function can be reproduced, which is very

rarely done by surgical procedure alone, the palate usually ending in a more or less tense straight line, and being incapable of effecting a junction with the posterior wall of the pharynx in order to cut off the nasal cavity whilst the patient is eating or speaking.

Figures 11 and 12 show a plaster cast of a case so treated and a diagram of the appliance.

After treatment either by surgical or mechanical means in these cases it is absolutely necessary for the patient to undergo a proper course of enunciation and elocution and to endeavour to forget his former method of pronouncing letters and words. This should be commenced by learning the alphabet afresh and by speaking very slowly and deliberately.

# CHAPTER IV

## INFLAMMATORY CONDITIONS OF THE MOUTH

### ACUTE AND CHRONIC STOMATITIS—PERIOSTITIS—NECROSIS

#### Acute Stomatitis

A very large number of forms of inflammation of the buccal mucous membrane and deeper tissues are usually described, depending for their classification upon the form, extent, and *appearance* of the inflamed tissues; a full description of the clinical features of such lesions is included in most textbooks on medicine. It would, therefore, not be consistent with our object to repeat such information in a stereotyped manner here.

It is, therefore, proposed to discuss the subject somewhat briefly from a causative point of view. This is not possible entirely, because the bacteriology of the conditions has not been fully worked out nor the causal organisms of each form definitely ascertained, but much has been done, and undoubtedly when this is complete the old nomenclature of 'exudative,' 'parasitic,' 'ulcerative,' &c., will be replaced.

The following schema indicates the possible source or causes of an acute stomatitis.

Acute Stomatitis
- 1. *Traumatic*—Mechanical, thermal, or chemical.
- 2. *Excretion of Drugs.*
  - Mercury
  - Potassium Iodide
  - Lead.
- 3. *Organismal*, due to infection by:—
  - (*a*) Mixed pathogenic organisms, streptococci, staphylococci, &c.
  - (*b*) Diphtheria and pseudo-diphtheria bacilli.
  - (*c*) Bacillus fusiformis (Vincent's angina).
  - (*d*) Pneumococci.
  - (*e*) Gonococci.
  - (*f*) Tubercle bacilli.
  - (*g*) Saccharomyces albicans.
  - (*h*) Epizoötic Virus (?).
- 4. *Nervous.*

1. **Traumatic.** Under this heading are included such mechanical causes as ill-fitting, unhygienic, artificial dentures, appliances for the regulation of teeth, rough or sharp edges of carious teeth.

Chemical causes are such as excessive smoking, the drinking of corrosive fluids (including alcohol too concentrated) or the escape of an ' arsenical ' dressing from a tooth.

Thermal causes, from the eating or drinking of substances too hot. These give rise to a simple acute stomatitis which is usually superficial and may be localised or general.

An ill-fitting dental plate may cause a stomatitis of the whole of the surface covered by it; this may be due (i) to the friction of the plate, (ii) to the chemical irritation of the mercurial pigment contained in the vulcanite, (iii) to the chemical irritation of fermenting food detritus. The edges of such a denture may also cut and ulcerate the tissues in the buccal sulcus.[1]

A hypertrophic form of stomatitis frequently results in children from the wearing of some form of appliance for regulating the teeth.

2. **Excretion of Drugs.** The fact that mercury in excess may cause a stomatitis is of course well known; this may be simple, and may be confined to the 'gums', or it may involve the buccal mucous membrane and may *ulcerate.*

The severer forms are usually seen in mouths which have already some septic focus present, and the ulceration which occurs is probably due to the combined effect of the mercury on the tissues and the invasion of the damaged tissues by pathogenic organisms. Mercury brings about its effect by being deposited in the capillaries of the mucous membrane, mercuric sulphide being formed by the action of the sulphuretted hydrogen present in unclean mouths. The

[1] I have known patients apply for treatment to their medical attendant for an ulcerative stomatitis caused by their dentures, but to leave the latter out of their mouth and say nothing about them.

sulphide is carried out into the tissues by the leucocytes, and there gives rise to irritation and a localised necrosis of the cells.

Mercurial stomatitis is more frequently seen in children nowadays who are given soothing powders containing mercury, and in cases of obstinate ' stomatitis ' which have proceeded to ulceration this source should not be forgotten, and some of the powders obtained and tested for mercury. Professor Gaucher is of the opinion that the stomatitis caused by mercury is of a special and peculiar form, commencing as a general stomatitis, then becoming localised, causing necrosis of the bone and *subsequent* ulceration of the surface, and he states that cases are more frequent on the right side.

*Potassium Iodide* is excreted by the salivary glands, and when given in large doses, and continuously, is liable to cause a stomatitis which is located chiefly in the gums and fauces. It is probably caused through the liberation of free iodine from the iodine by the action of small quantities of nitrites contained in the saliva. The inflammation is usually attended with considerable pain and discomfort but does not proceed to ulceration.

Lead, arsenic and bismuth may cause a stomatitis in a somewhat similar manner, and ulceration may occur.

3. **Organismal.** This includes by far the largest number of cases of stomatitis. The character and extent of the lesion depends to some extent upon causal organisms; in some cases the inflammation remains simple, or an exudate may be formed, whilst in others the ulceration occurs which may become gangrenous. On the other hand, the intensity of the inflammation and the amount of destruction of tissue may vary considerably with the same organism, depending upon the tissue resistance of the patient. The tissues of the mouth have normally a very high power of resistance, and it is chiefly in weak debilitated children

in unhygienic environments that the organisms have so much in their favour that rapid tissue necrosis is brought about. The clinical picture may vary considerably, but the difference is essentially one of degree—depending upon the extent and rapidity of tissue destruction.

(*a*) *Infection by Mixed Pathogenic Organisms.* Nearly all cases of stomatitis are of necessity a ' mixed ' infection, but there frequently is a predominating organism. When this is not so, the inflammation is usually simple, diffuse, and general, and under this heading at present must be included the stomatitis seen in the course of exanthemata, in infants from the sucking of unclean ' dummies ' and in adult patients who are considerably debilitated by the presence of some other disease.

Cases of ulceration of the buccal mucous membrane are frequently associated with contiguous septic teeth—usually deciduous molars in children. Many of these cases seem to arise by an initial abrasion of the surface, by the sharp edge of a carious cavity, and then, owing to the low vitality of the patient, the organisms of the mouth gain an entrance and not only prevent healing, but cause extension of the lesion.

(*b*) *Diphtheritic Stomatitis.* Ulceration of the buccal mucous membrane may occur during the course of diphtheria, and be due to the diphtheria bacillus, but, quite apart from such conditions, severe stomatitis of the cheeks particularly has been found in children to be due in many cases to infection with the Klebs-Loffler bacillus, and the Hoffmann bacillus is sometimes found so predominating as to suggest its being the cause. Such inflammation usually is more or less deeply ulcerated, covered with a yellowish slough which does not resemble the typical diphtheria membrane; the margins are deeply injected, slightly raised, and sharply cut. In poorly nourished children the ulceration may suddenly 'fulminate', infection spreads deeply and widely, necrosis and gangrene occur, and ' cancrum oris ' is said to be present.

(The writer has seen at least one such case from which a *pure* culture of the Hoffmann bacillus was obtained, and which when injected into a guinea-pig caused death, the organism being recovered from the heart blood of the animal; and also several cases of severe and deep ulceration, where the diphtheria bacillus was present in very large numbers.)

It is important to recognize that many of these fulminating cases have an initial stage of simple ulceration lasting several days, during which the parents frequently say that the child could not eat properly, and complained of 'soreness', but that no further notice was taken of it. It is usually the 'feverishness' and the foulness of the breath which first calls serious attention to the condition.

(*c*) The *B. fusiformis* gives rise to a characteristic form of stomatitis, and with it is usually associated a spirochæte (or spirillum), and it is stated that in severe forms the latter organism predominates.[1] The inflammation usually commences in the mucous membrane around septic teeth, and soon spreads either along the gum margins all round the jaws, or to the contiguous surface of the cheek. In mild cases the inflammation is intense, but there is not much tissue loss; in the more serious cases necrosis of the superficial mucous membrane quickly supervenes, the free margins of the gums are destroyed, and the alveolar bone is exposed. In the cheek ulceration extends deeply, and may spread superficially backwards on to the soft palate and fauces. The ulcers show an irregular crater-like edge, and are covered with a greyish white slough. Such cases also may, if the circumstances be favourable, progress very rapidly, when, instead of a slow cellular necrosis with a surrounding reaction on the part of the tissues limiting extension, gangrene of the whole infected area quickly occurs, with little tissue reaction; again a condition of 'cancrum oris' or gangrenous stomatitis is present.

[1] Vincent and Plant.

The mild cases clear up but may become somewhat intractable; in the severer forms the prognosis of course is bad.

(*d*) *Pneumococcal Stomatitis* occurs occasionally as a primary infection; its recognition depends largely upon bacteriological examination. The muco-periosteum of the alveolus is deeply injected, and there is a marked tendency to overgrowth, so that the gums appear to be elongated. Multiple ulcers may be present and the peri-odontal ligaments may be involved so that the teeth become quite loose.

(*e*) *Gonorrhœal Stomatitis* may occur either in infants or adults. In the first case it arises from infection during parturition in a similar manner to ophthalmia neonatorum.

In adults the condition may assume more serious form. Infection reaches the mouth probably by external agency in most cases, although metastatic infection has been suggested, being thus analogous to the gonorrhœal infection of joints elsewhere seen frequently in the subacute stage.

The stomatitis usually commences at the lips, and rapidly involves the whole of the mucous membrane, which becomes swollen and granular; the tongue and gums are included, and the latter readily bleed; the peri-odontal ligaments are affected and the teeth may become loose.

The surface becomes covered with a sticky exudate, and in severe cases there is a muco-purulent and sometimes blood-stained discharge. Small multiple ulcers may be present covered with typical yellow pus. Salivation may or may not be present.

(*f*) *Tubercular Stomatitis* is usually secondary to some tubercular focus elsewhere, but primary cases have been recorded occurring probably by inoculation of an abrasion or suppurative lesion already present. The condition may be either chronic or acute, the former being more usual. Ulceration usually occurs, and is apt to become chronic, the ulcers are sinuous and superficial, and situated more fre-

quently on the under surface of the tongue, though the muco-periosteum or cheeks may be attacked.

The acute form is not infrequently secondary to lupus of the face, the muco-periosteum in the front of the mouth is often the seat of the infection. The surface becomes red glazed and 'granular' due to the development of minute

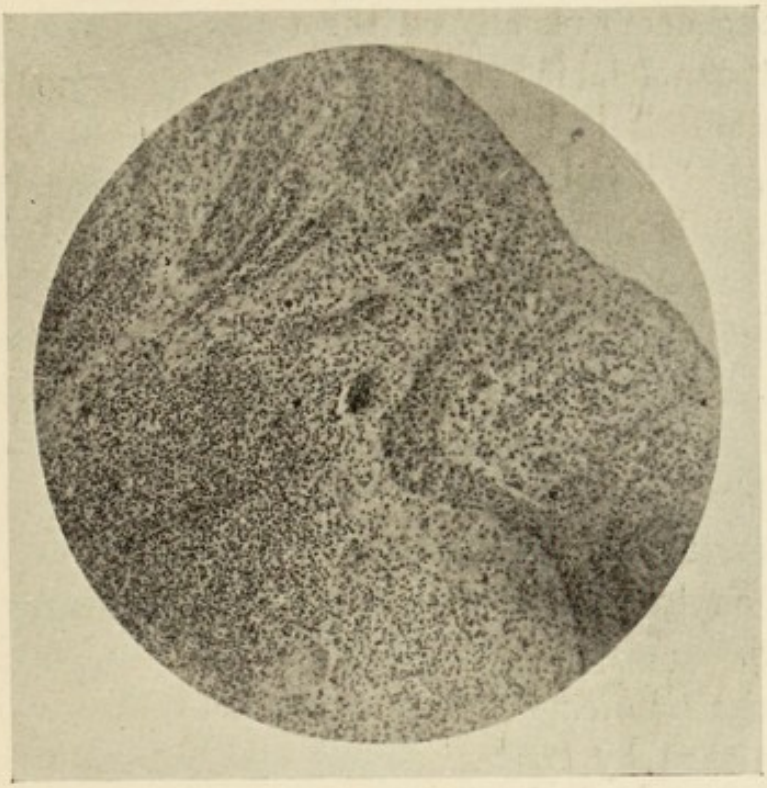


Fig. 13. Tubercular Stomatitis.

tubercles, which may break down and ulcerate, the surface is very sensitive and painful, and if the peri-odontal ligaments become involved as they sometimes do, the pain becomes severe owing to the periodontitis set up. The patient complains of severe 'toothache', which if the teeth are very carious one may be tempted to relieve by extraction; in the writer's experience, however, this is a mistake, and only leads to considerable aggravation of the condition. Fig. 13 shows a section of the mucous membrane from a case of acute tubercular stomatitis.

(*g*) *Saccharomyces albicans* gives rise to the well recognized

condition called ' thrush '. It usually affects ill-nourished infants in unhealthy surroundings, and also old people the subject of a wasting disease, it is then supposed to be serious and to cause death. Whilst this may be true to a certain extent, it is more probable that the organisms are enabled to get established because the end is approaching.

The typical whitish spots or ' raised patches ' are well known and occur usually on the tongue, palate, or cheeks. The white material is composed of the organisms, epithelium and fat, and it is usually inferred that it may be easily removed, but this is by no means always the case; when, however, it is removed a raw granulating surface is seen beneath.

The author has obtained cultures from such cases as these in which the organism resembled the ordinary yeast form much more than the usual short and long rod-like form of the *S. albicans.*

Another organism of this class, the *S. neoformans,* has been found in acute inflammations of the gums and peri-odontal ligaments.

(*h*) *Epizoötic Stomatitis* (foot and mouth disease of cattle) occurs in men by contagion from infected animals. It is usually epidemic, and a history of infection can usually be traced; it is stated that during epidemics in Berlin in 1888 and 1891, no less than 3,000 people were affected.

There is an incubation period of 5 to 10 days, and constitutional symptoms appear before the oral lesions.

The mucous membrane of the mouth becomes hyperæmic and swollen. Small yellow vesicles appear on the gums, lips, and tongue, these become pustular and rupture, leaving small ulcerated surfaces.

The condition usually lasts several weeks and clears up completely, but deaths have occurred.

This stomatitis may be confused with other vesicular forms, and the diagnosis must rest upon the synchronous appear-

ance of similar lesions on the hands or feet or other parts of the skin, and upon the history, which can usually be obtained, of contagion with some affected person or animal.

4. **Stomatitis of Nervous Origin.** Under this heading is classed herpes of the lips or buccal mucous membrane, seen either during acute pneumonia, influenza, or coryza, or associated with reflex irritation from diseased teeth. The eruption occurs in clusters on one side of the mouth, the blebs soon rupture and leave only a red ring with reddish yellow centre. Considerable distress may be caused during eating, but the condition tends to clear up in a few days.

Pemphigus may be included here also, since bullæ which soon burst are found on the palate and cheek, the condition in old people frequently becomes either chronic or very recurrent.

A form of neurotic stomatitis dependent upon mental worry has been described, it appears to take the form of a trophoneurosis, whereby crops of small vesicles and ulcers are developed, and which tend to be recurrent.

**Treatment.** In all forms of stomatitis an endeavour should *first* be made to ascertain both the exciting and the predisposing causes in each case, and both should be treated concurrently.

In stomatitis, due to any form of trauma or the excretion of drugs, the removal of the cause is always possible and efficacious. In order to protect a localised stomatitis, and to prevent pain, it may be painted with a tragacanth application (Appendix II, no. 6.)

Treatment of the many and various forms due to bacterial infection differs according to the nature of the causal organism, and to the extent of the lesion.

In *mild cases* treatment of the general condition should be by means of fresh air, change of surroundings, nourishing liquid food, saline aperients, and later, tonics. These

combined with antiseptic mouth-washes should be sufficient to clear up the condition.

When *ulceration* has occurred all possible sources of septic infection both within the mouth and without should be carefully searched for, and if possible eradicated or at least negatived by precautions. Particularly is it necessary to treat septic teeth which may be present, either by filling, extraction, or the removal of salivary calculus.[1]

As a general rule the most useful remedy is chlorate of potash, given as 3-grain lozenges frequently, in this way a prolonged local action is obtained since it is returned to the mouth again by the saliva. In children who cannot suck it may be given internally as a mixture with good results.

The whole mouth, and particularly the surface of the ulcers, should be frequently and carefully cleaned with soft swabs of peroxide of hydrogen, in full strength (10 volumes) if it can be tolerated, if not it may be diluted with water. If the ulcer appears to be intractable or inclined to penetrate deeply, the surface should be well painted with silver nitrate, 20 grs. to the ounce, and the mouth afterwards washed out with a solution of common salt.

If the condition does not quickly yield to such treatment, any ill effects of the potassium chlorate should be carefully watched for (by reason of its forming methæmoglobin in the blood) and the urine examined. If necessary it should be discontinued, and formamint lozenges substituted. Stronger antiseptic mouth-washes should be used for the more serious cases, such as perchloride of mercury 1 in 2,000, or a wash which evolves chlorine (Appendix no. 9).

[1] A case illustrative of this was that of a girl aged 9 with a fairly extensive stomatitis of the right cheek which had become somewhat deeply ulcerated, and had resisted treatment with antiseptic mouth-washes for a fortnight, and was looking serious. Two upper deciduous molars which were septic and covered with calculus were then removed, and chlorate of potash substituted for the mouth-wash, with the result that the condition had cleared up in a few days.

When the ulceration has become *gangrenous* the condition is most serious, the prognosis is very bad, and prompt and energetic treatment is necessary. Scraping the slough away and cauterizing the wound either by the actual cautery, or with fuming nitric acid, or zinc chloride is recommended, but the writer has seen the best results to follow from early excision of the whole area. The operation is drastic, but the peril is immanent. Even if it is not performed perforation of the cheek may occur, and from the use of strong caustics like nitric acid, dense contractions occur which frequently lead later on to permanent closure of the jaws. A plastic operation may afterwards be done to replace somewhat the lost tissue.

Small *follicular* ulcers yield at once to touching them with solid silver nitrate, and '*thrush*' may usually be cured by attention to the general health, avoiding sources of reinfection, and cleaning out the mouth frequently with glycerinum boracis, to which may be added in severe cases sulphate of zinc in the proportion of 8 grains to each ounce. Plenty of fresh air and pure milk are absolutely necessary in cases of infants.

Acute *tubercular* stomatitis may be treated, if in the front of the mouth, by exposure to Finsen light with excellent results.

*Prolonged* and subacute cases of stomatitis may be suitably treated by vaccines after the causal organism has been determined.

### Chronic Stomatitis

A chronic catarrhal stomatitis frequently results from the immoderate use of tobacco and alcohol, or from the presence of many septic teeth, and also in children and young girls from the immoderate consumption of sweets.

The remedy lies in removing the cause, and in general oral hygiene.

**Leucoplakia, Icthyosis, Leucoma.** These conditions are characterized by the formation of either milky or dense white patches situated on the mucous membrane of the cheeks, or on the muco-periosteum of the jaws. The areas may be quite small and multiple and scarcely raised above the surface, or they may become raised and confluent in old standing or progressive cases.

Occasionally the growth is very dense and cauliflower-like, as in one of the author's cases,[1] see Fig. 14, coloured plate (a section of the same growth is shown in Fig. 15). In this case the patient thought that the roughness was due to the presence of teeth (or portions of them).

The condition as a rule causes no pain or discomfort, but it is essential that it should be recognized early and treated seriously, since about 30 per cent. of these cases are believed to become malignant.

Cases of warty growths in the mouth which have been described as papillomata, should probably have been classed as leucoma. The latter is essentially a condition in which the epithelium alone is concerned. A papilloma, on the other hand, always has a central core of submucous or subepithelial tissue. The cause is supposed to be chronic irritation of the mucous membrane due to smoking, and according to Bruck to the effect of prolonged mercurial treatment. It is certainly seen in syphilitics during the tertiary period, but whether as a result of the infection or of the antisyphilitic treatment is at least disputed. The author has, however, seen cases in women, one a very marked case, where a history neither of smoking, syphilis, nor mercury, could possibly be obtained. In the case mentioned above, too, there was no other sign and no history of syphilis, and the man smoked quite a moderate amount of tobacco ; there was, however, a history of previous suppurative dental arthritis with the deposition of calculus, and it does not

[1] Reported in the *British Dental Journal*, July 15, 1909.

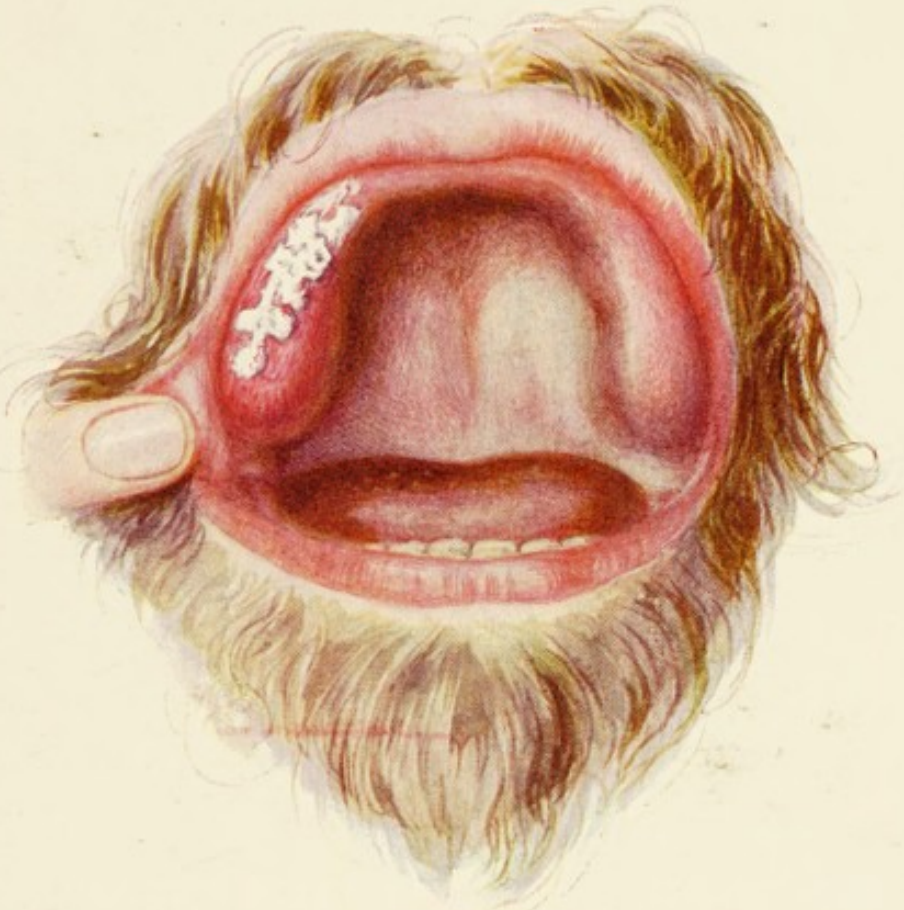


Fig. 14. Leucoma of the upper jaw.

seem unlikely that such chronic irritation should have originated the leucoplakia leading later to the leucoma.

**Treatment.** All possible sources of irritation must be strictly avoided or removed. The areas may be painted with very weak solutions of silver nitrate or chromic acid, or with lactic acid 25 per cent., or salicylic acid 10 per cent. in glycerine. It does not seem wise in view of the etiology

FIG. 15. Leucoma (or icthyosis) of the muco-periosteum of the upper jaw.

and possible malignancy of the condition either to use strong solutions, caustics, or irritants, or to use even weak ones for a prolonged period. If the condition does not yield to such treatment, and also in cases which are very dense and white when first seen, the writer prefers to excise the area. This can be done quite simply in many cases under cocaine, but the submucous tissue or the periosteum must also be removed or the condition tends to recur.

**Actinomycosis** is a condition which affects chiefly the region of the lower jaw, it begins insidiously, and may

remain quiescent for some time. The infection with the streptothrix actinomyces usually occurs within the mouth, but as the lesions develop and small abscesses form, they track outwards towards the angle of the jaw, and there give rise to a characteristic puckered appearance. The organism may—as in a case under the author's treatment—gain entrance through a carious tooth, and thus affect the interior of the bone first of all. In all suspicious cases the pus should be carefully examined for the typical yellow nodular masses and also microscopically for the clubs and filaments (the latter stain by Gram's method the former do not).

Treatment consists in removing the source of the infection and curetting the sinuses and pockets, and in giving large doses of potassium iodide (20–50 grs. three times a day). Treatment should be prompt and energetic since secondary infection of the alimentary organs may occur.

### Periostitis and Necrosis of the Jaws

Acute inflammation of the periosteum and death of the subjacent bone are so frequently associated, especially in the jaws, that they can be most conveniently described together. Although it is possible to have one condition present without the other, as a rule they have a common cause and origin, the periostitis being most commonly antecedent to the necrosis. The causes of the condition may be grouped in a similar manner to those of stomatitis:

I. *Trauma* { Fractures—blows.
Extraction of teeth.

II. *Drugs* { (*a*) From without—P. As.
(*b*) From within—Hg.

III. *Bacteria* { Infection due to—
(*a*) Dental lesions.
(*b*) Subsequent to specific infectious diseases.
(*c*) Syphilis.
(*d*) Extension of Stomatitis.

The lower jaw is more frequently attacked than the upper, the latter being more vascular. The periostitis or necrosis may be quite localised, or it may involve the major part of either jaw,[1] more commonly it is the alveolar portion which is necrosed, but a number of cases are recorded where the body of the lower jaw became necrotic, leaving the teeth firmly imbedded in the alveolar portion. Again, the outer alveolar plate may be alone involved.

1. **Traumatic Necrosis** is usually localised, and may follow comminuted fractures or those horizontal fractures which involve the teeth only, more especially when immobilization is not quickly secured. It may follow extraction of teeth, in which case it may be caused in several ways, some of which will be described under subsequent headings.

(i) Necrosis may be caused by excessive force, laceration of tissues, and general unskilfulness in extracting teeth, and when such cases do occur they are often extensive, because persons who perform operations in such a manner do not usually sterilize their instruments, so that to severe injury is added bacterial infection.

(ii) Cases of acute periostitis and necrosis are not uncommon after the injection of local anæsthetics into inflamed tissues. Such necrosis is caused partly by the vaso-constricting effect of such drugs as adrenalin chloride, and partly by force of the injection driving inflammatory products deeply into the bone. Again, in many cases (now that such operations are so frequently performed by untrained and illiterate persons) the condition is complicated by the use of a septic syringe or decomposed drugs.

(iii) In some cases, however, necrosis may follow extraction even when proper precautions are taken, if the patient is debilitated or suffering from exhaustion from over work,

[1] A number of specimens are extant showing sequestra of almost complete 'jaws', two such cases there are in the museum of this University.

but the risk of this may be reduced to a minimum by the use of suitable antiseptic mouth-washes *prior* to the operation.

2. **Drugs.** (*a*) (i) The access of the fumes of *yellow phosphorus* to the bone of the jaw causes almost certainly an extensive necrosis. The phosphorus as a rule gains entrance through a carious tooth, so that the centre of the bone is attacked first of all, it is therefore probable in these cases that some necrosis precedes periostitis (and this may possibly have something to do with the peculiar 'pumice stone' deposit of bone seen on the surface of the sequestrum).

(ii) The escape of *arsenious acid* from a dressing in a tooth in order to devitalize the pulp, may cause extensive necrosis. The escape may occur at the gum margin when a localised periostitis and necrosis result, or it may escape from the apical foramen in children with 'open apices'; in such cases the damage done may be very severe, since the centre of the bone is attacked first and the necrosis shows a marked tendency to spread. It usually results in the loss of several teeth with the alveolus as a sequestrum. Arsenic may escape in another way, through a perforation in the floor of the pulp cavity which has been mistaken for an exposed pulp, necrosis is usually limited in such cases.

(*b*) *Mercury* may cause, as previously stated, a stomatitis; this may extend deeply and cause perostitis and necrosis, fortunately rarely met with at the present time, now that mercury is not pushed to the stage of 'touching the gums' as it was formerly. Marshall[1] records two deaths from this cause in which the alveolar portions of both jaws were affected. Gaucher[2] has observed six fatal cases following the injection of 'grey oil'.

3. **Bacterial Infection.** This is the most frequent cause of periostitis and necrosis, and in the larger proportion of the cases infection occurs through some dental lesion.

[1] *Injuries and Surgical Diseases of Face, Mouth, and Jaws*, p. 274.

[2] *Bulletins de la Société Médicale des Hôpitaux*, March 1908, p. 477.

(*a*) This class of case is most usual in children, and arises commonly from alveolar abscesses in connexion with deciduous molars. When pus forms in these cases it may, instead of pointing in the buccal sulcus, track beneath the muco-periosteum, raising it from the bone, and necrosis of the latter then occurs.

It is liable to occur also either when a large number of virulent organisms gain access to the centre of the bone through a septic tooth with an open apex, or from the injudicious treatment of such a tooth, i.e. the sealing in of a dressing, thus forcing inflammatory products deeply into the jawbone. The author has seen necrosis extending from the first molar to the canine tooth from such a cause in connexion with a second lower premolar.

In adults severe periostitis and some necrosis in the region of the angle may follow the difficult eruption of a wisdom tooth when the latter is impacted (see p. 155). Periostitis and necrosis of the jaws may follow most of the acute fevers such as scarlet fever, measles, small-pox, and typhoid fever. In such cases the condition is probably an extension from a superficial stomatitis. This may be due to the (?) specific organisms concerned, or to the increased virulence and number of the ordinary organisms in an unclean and *stagnant* mouth.

In some cases, however, it has seemed that infection must have occurred via the blood-stream, and in all cases of course the patient's tissue resistance is low.

The author has recently seen two cases of tuberculous necrosis of the mandible in which it seemed indubitable that infection occurred immediately after the extraction of teeth.

(*b*) The necrosis is usually alveolar in children after exanthemata. A number of cases of severe necrosis of the lower jaw has been recorded after typhoid, and one case of death from necrosis of the jaw following small-pox.

(*c*) Necrosis of the jaws during the tertiary stage of *syphilis* is well known. It is preceded by a gummatous periostitis, and most frequently attacks the hard palate[1] and may cause the loss of the whole of that structure, or it may be arrested when only a small amount of damage has been done.

(*d*) *Extension* of one of the more serious forms of *stomatitis* may give rise to periostitis and necrosis of the surrounding bone. It is seen in some of those severe and fulminating cases known as 'cancrum oris', and when it has occurred it is an extremely bad sign, for such cases are usually fatal. A marginal necrosis of the alveoli sometimes occurs in the infection by the *B. fusiformis*, and a more chronic form of necrosis may follow tubercular ulceration of this muco-periosteum.

Necrosis is occasionally seen following scurvy, and it may be associated with the ulceration of malignant growths.

**Symptoms.** The clinical aspect varies naturally with the cause and severity of the case, but there is much similarity about all cases. There is usually a considerable amount of preliminary pain, associated usually with tenderness of the teeth, hyperæmia and swelling of the muco-periosteum, and œdema of the cheeks, accompanied by pyrexia. The breath is heavy and foul, tongue furred and swollen, there is anorexia and constipation, if diarrhœa is present it is usually a bad sign. Soon pus forms and points usually in several situations. If the patient does not succumb in the acute stage the condition now becomes somewhat 'chronic'; pus continues to discharge for weeks through the cloacæ whilst separation of the dead from the living bone is taking place. A considerable amount of toxæmia usually occurs

[1] I have seen one case where the lower jaw was *perforated in a vertical direction*, and it seemed as if the bone must fracture; unfortunately the patient disappeared, so I am unable to say what happened.

during this stage, but eventually the sequestrum may be felt to be more or less loose with the involucrum around it.

**Treatment.** The removal of the cause wherever possible is the *first* essential, then in early cases free incisions should be made down to the bone within the mouth, and hot antiseptic mouth-washes prescribed. By these means necrosis may be prevented; if not, and when the cloacæ are established, they should be syringed frequently with an antiseptic lotion such as 10 per cent. peroxide of hydrogen, and the patient should use a mouth-wash of permanganate of potash. Active treatment is usually postponed until the sequestrum is quite separated, when it should be removed through an incision within the mouth if possible. General treatment should be directed towards keeping up the patient's strength and tissue resistance by means of a nourishing and liberal diet (which must be fluid at first), fresh air, tonics, and saline aperients. Repair in the lower jaw is usually good, in some instances practically a new jaw has formed, which in one case recorded was wider than the original; but the bone is usually a narrow ridge, sufficient however to carry an artificial denture if such be *inserted as soon as possible* in order to obtain the stimulus of function, otherwise some of the bone may reabsorb.

Repair in the upper jaw is less good, a fibrous bridge or bar may form, but more often there is complete loss of tissue.

# CHAPTER V

## GINGIVITIS—DENTAL ARTHRITIS

### 'Pyorrhœa Alveolaris'

These conditions are grouped together because, although they may be quite distinct lesions occurring either one by itself, yet they are perhaps more frequently most intimately associated; they may be present at one and the same time, or the one may pass gradually into the other. These conditions (under these or other names) are frequently described in textbooks as distinct and separate pathological entities; the student is thereby confused by a multiplicity of names and diseases, and when examining a case is extremely confused as to a diagnosis because signs and symptoms of several diseases appear to be present at the same time.

Inflammation of the gum margins may be acute or chronic and either localised or general.

### Acute Gingivitis

Acute gingivitis, when it is general, may arise as the result of the administration of mercury, or during specific fevers, or owing to infection by specific organisms such as the treponema pallidum, or gonococcus, or tubercle bacillus. It not infrequently arises apparently through infection of the gingival tissues by the normal mouth bacteria, when the patient is subjected to particularly heavy stress of work rather mental than physical, or when the surroundings are unhygienic.

The localised form is usually associated with some kind

of mechanical irritation in connexion with the teeth such as wires, clasps, or bands used to retain artificial dentures or to ' regulate ' teeth, or to the overhanging cervical edges of crowns or fillings. The unintentional retention of a silk ligature around the neck of a tooth and beneath the gum occasionally explains an acute and painful gingivitis.

The deposition of salivary calculus always causes a chronic gingivitis, and exacerbations of this may occur giving rise to an acute inflammation. In cases which arise through infection pure and simple without any mechanical irritation, and which do not subside rapidly, it is extremely important to notice that a deposition of hard dark calculus occurs around the neck of the tooth, and thus we have the first step in the establishment of a vicious circle.

**Signs and Symptoms.** The 'gums' become red, tender and swollen, the patient is unable to eat hard substances, avoids using a tooth-brush, and thus the condition is aggravated by the accumulation of débris, food, epithelium, and organisms, and there is a considerable *fœtor ex ore.* Absence of friction, too, leads to the slower desquamation of epithelium from the gums, which are thus covered by a milky film ; if this is removed the gums are seen to be intensely hyperæmic and readily bleed. In severe and generalized cases suppuration occurs and pus is seen between the ' gums ' and the teeth.

It is extremely rare that such a condition is confined to the 'gums' proper, the joints between the teeth and the jaw-bones become involved by the upper part of the peri-odontal ligament becoming infected. Thus a Dental Arthritis is set up, which may be simple at first but may become later suppurative. The condition then may be called an *acute 'pyorrhœa alveolaris'.*

It is characterized by the above symptoms together with considerable tenderness of the teeth, that is to say all the teeth become tender to percussion.

There are general symptoms of pyrexia, with rigors occasionally, loss of appetite, foul tongue and breath, constipation, and pains in the limbs and back.

**Treatment.** It is absolutely necessary that these cases should be treated energetically and promptly; not that there is much risk of life from the disease itself or its general symptoms (if the cause of these general symptoms has indeed been correctly located), but the urgency lies in the danger of the acute phase drifting into a chronic condition—the well-known 'intractable' disease generally known by the name 'pyorrhœa alveolaris', the disastrous secondary effects of which will be referred to later. It is one of those conditions which most essentially must not be 'left to nature', for if so then most assuredly the vicious circle above mentioned will be set up.

The mouth should first be washed out with a weak solution of borax or normal saline, then, with a small piece of cotton-wool in a dressing forceps, the gums and teeth should be gently swabbed with peroxide of hydrogen. The patient must not be left until the mouth is comparatively 'clean' and the fœtor has considerably diminished, and this treatment should be repeated daily. A sharp saline purgative should be administered at once (mercury is to be avoided), and to relieve the pain, a *hot* carbolic [1] or poppy-head [2] mouth-wash may be ordered. In severe cases the patient must be confined to bed, kept on milk diet, and instructed how to use the swabs of peroxide of hydrogen after each feed. Under such treatment the symptoms should subside in a few days, and when the gums have become less tender they may be swabbed with tincture of iodine after the peroxide. The milk diet should be discontinued as soon as possible, and the patient instructed to massage the gums with the finger. When the attack has completely subsided, the patient should be sent to a dental surgeon in order that any trace

[1] See Appendix II, No. 14. [2] Ibid., No. 13.

of calculus deposits around the necks of the teeth may be removed without delay. At the same time the patient's environment should be changed, tonics should be given, and a liberal mixed diet of which fresh fruit forms an important part should be ordered.

It cannot be too strongly urged that in these suppurative cases the mere sucking of any form of lozenges in such cases is by itself only courting the establishment of a chronic condition—the constant swabbing and mechanical removal of the detritus which collects and covers over the focus of suppuration is the most essential form of treatment.

Cases of simple gingivitis either local or general may be treated by removing the cause, and by giving an aperient and formamint lozenges.

### Chronic Gingivitis and Arthritis

Chronic gingivitis, when it is present by itself, is usually hypertrophic in form; the mucous membrane, instead of ending in a pale pink margin at the cervix of the tooth and in similar delicate 'papillæ' between the teeth, is of a deepened colour, the margins are thickened, they more than fill the interdental spaces and encroach upon the crown of the tooth. This form of hypertrophic inflammation verges in pronounced cases into a diffuse neoplasm, when the teeth become almost buried in redundant masses of gingival tissue. The condition is frequently quite localised, when it is usually due to some form of mechanical irritation, such as a carious cavity, the lodgement of food particles, or some mechanical appliance.

As regards *treatment*, it is best to inject cocaine and remove the hypertrophied tissue and to prescribe an astringent mouth-wash.

Chronic gingivitis is frequently associated with dental arthritis, and it may then be either atrophic, hypertrophic, or suppurative.

The atrophic variety is frequently seen in patients of a gouty diathesis—there is very little sign of inflammation except excessive tenderness and pain of a neuralgic type. Both the gingival fold and the peri-odontal ligament atrophy and the root of the tooth becomes exposed. As a rule the condition is not general, but occurs in the neighbourhood of one or two teeth ; it may, though, pass from one group of teeth to another. During an acute exacerbation of this condition the muco-periosteum may swell up and a mucous glairy fluid may be evacuated.

There is rarely any salivary calculus, at least in the usual situation; deposits of urates are occasionally found high up on the root of the tooth, but this is not universally the case. The ligament and gum having once been lost it is never repaired, and the condition is usually slowly progressive until the joint becomes so weakened that the tooth is lost.

As regards *treatment*, sedative and analgesic applications may be made to the gums and any observable calculus should be removed, but most attention should be paid to the patient's general condition ; diet should be regulated and if gout be present it should be suitably treated. Many of these cases seem to originate as actual attacks of gout in the peri-odontal ligament, and the author is of the opinion that this is sometimes the only local manifestation of a gouty diathesis.

When the hypertrophic and suppurative forms of gingivitis are associated with dental arthritis the latter is or rapidly becomes suppurative also ; we have then present the disease long known as Rigg's Disease or *pyorrhœa alveolaris* (alveolar osteitis or suppurative marginal periodontitis), for this is essentially a septic infection of the upper part of the peri-odontal ligament and gingival fold of muco-periosteum, involving the alveolar bone secondarily and leading later to total disorganization of the joint. It is of great importance

that it should be well recognized, since it forms by far the most frequent source of ' oral sepsis '.

**Ætiology.** This form of septic dental arthritis (' pyorrhœa alveolaris ') is of extremely common occurrence ; it is usually seen in adults and perhaps more frequently in males than females ; it may, however, be seen in children of 10 or 15 years of age. Epidemics have been recorded. It is found in native races occasionally, and some skulls of ancient races exhibit marked traces of its effects. Domestic animals, horses, dogs, cats,[1] (and monkeys), are occasionally affected by it. It may be localised around one particular group of teeth, such as the upper or lower incisors or the molars on one side only, or it may involve the whole of the alveolar processes. Colyer is of the opinion that it is frequently associated with mouth-breathing.

The direct cause in all cases is infection by pathogenic organisms.

It is now known that no single organism is responsible for the production of the disease, but that the infection is always an extremely mixed one. The organisms most frequently isolated from this disease are[2] : micrococcus catarrhalis, streptococci, bacillus septus, bacillus necrodentalis, staphylococci and the pneumococcus. These organisms are or may be of course present ordinarily in a healthy mouth without giving rise to any local lesion. The factor which determines their entrance into the periodontal ligament is either an increase in their virulence or

[1] I once removed the submaxillary glands of a cat for experimental purposes—under strict aseptic precautions. But I omitted to examine the mouth and I did not ligature the distal end of Wharton's duct. The cat was affected with septic dental arthritis in an advanced stage, infection travelled down the ducts—intense Ludwig's angina and sloughing of the neck occurred—and died on the fourth day.

[2] See Goadby, *Erasmus Wilson Lecture*, 1907 ; id., *Proc. Roy. Soc. Med., Odonto. Sec.*, January 1910 ; Eyre and Payne, *Proc. Roy. Soc. Med., Odonto. Sec.*, November 1909.

a decreased tissue resistance on the part of the patient. This latter may be brought about either through local or general causes.

The most frequent local factor which upsets the balance between the resistance of the ligaments and the organisms is the deposition and accumulation of salivary calculus around the necks of the teeth.

This acts as a chronic mechanical irritant and affords a mechanical focus where *stagnation* can occur, and therefore also a situation for the accumulation and proliferation of bacteria and their products.

The patient's tissue resistance may also be lowered by reason of the presence of some chronic systemic disease, such as diabetes, nephritis, syphilis, chronic heart disease, alcoholism, or plumbism ; or after some prolonged acute disease such as typhoid fever or one of the exanthemata.

**Signs and Symptoms.** The condition commonly arises insidiously and is more or less ' chronic ' when first recognized. In the early stages the gingival folds are enlarged, thickened, have a rolled instead of a fine thin edge, and are ' congested ' in appearance (see coloured plate, fig. 16). The actual focus of the inflammation is on the inner surface of the gingival fold and on the upper surface of the ligament, *pus collects here and may be expressed by gentle pressure.* In chronic cases calculus is nearly always present on the teeth either in visible and appreciable masses or of the hard dark variety deposited below the neck of the tooth. The inflammatory process spreads downwards, the amount of calculus increases, the upper part of the peri-odontal ligament is destroyed, and a space or ' pocket ' is formed in which pus collects (this pocket is virtually an abscess cavity with a sinus). Infection next spreads to the bone of the socket and a rarefying osteitis or osteoporosis is set up, the bone thus gradually is converted into osteoid tissue and finally is replaced by inflammatory tissue which breaks down and suppurates.

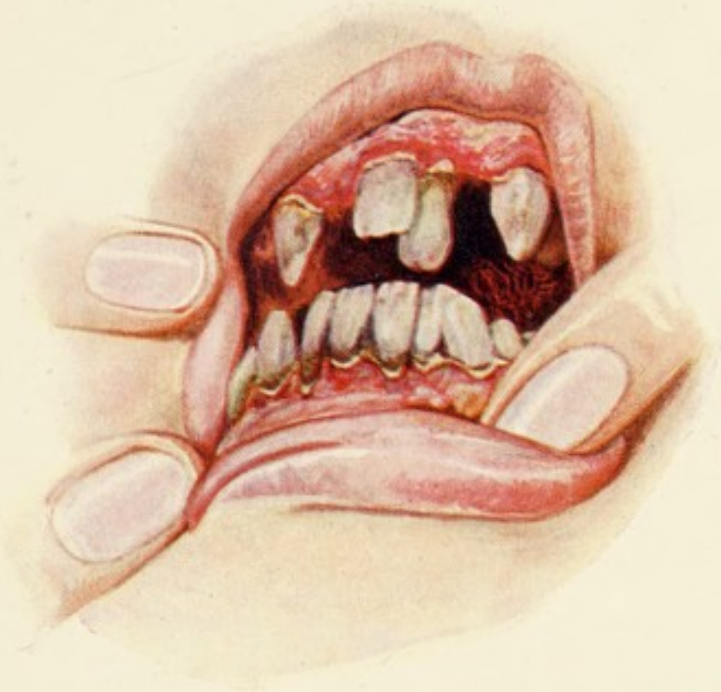
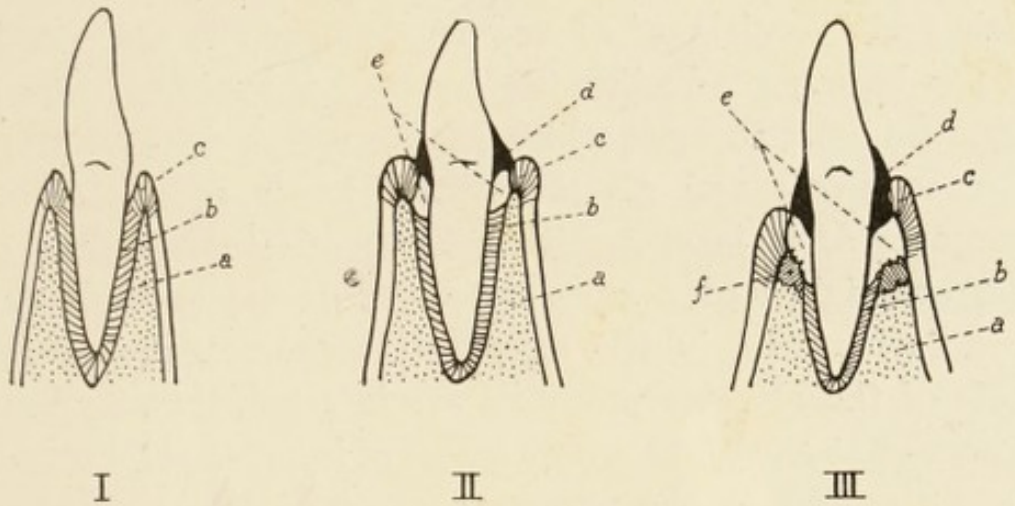


FIG. 16. Pyorrhœa alveolaris.

The result of this is that the pockets become much deeper, much more pus is formed, the teeth become increasingly loose, and calculus is deposited further down the roots of the teeth. Very little pain or discomfort is experienced by the patient up to this stage. The only symptoms being a disagreeable taste in the mouth, fœtor of the breath and sometimes the expectoration of 'matter'; as to whether




I II III

Fig. 17. Diagrams illustrating the course of septic dental arthritis. I. Showing normal relationship of parts; *a*, alveolar bone; *b*, peri-odontal ligament; *c*, free gingival margin. II. Chronic gingivitis and commencing arthritis; *a*, alveolar bone; *b*, peri-odontal ligament commencing to be involved in the inflammation affecting (*c*) the thickened gingival margin; *d*, deposits of calculus; *e*, spaces containing débris, organisms, and pus—'pockets'. III. Chronic arthritis in an advanced stage. *a*, *b*, *c*, as before; *d*, increased deposit of calculus; *e*, large accumulations of pus; *f*, osteoid tissue. More than half the joint is disorganized.

these things are noticed particularly or not depends entirely upon the character of the patient.

When the teeth are loose the patient finds it difficult to masticate and so may seek treatment.

If untreated the disease progresses until the dental joints are quite disorganized and the teeth drop out. Even then in some cases it continues as a chronic osteitis of the jaw-bone, and the alveolar processes are entirely destroyed.

**Secondary Effects.** These are those of septic infection and

toxic absorption, and in chronic well-established cases always severe and not infrequently very serious; that is to say, the effects are the same as those of a chronic suppurative arthritis in any other part of the body. This aspect is considered in detail under 'oral sepsis' and its effects (see p. 202); it will suffice to state here that such effects are usually shown as secondary infections of lower portions of the alimentary tract, muscular and joint pains, chronic sapræmia or toxæmia, septic and 'pernicious' anæmias; that such is the case is only to be expected when the *extent* of the original disease is understood. In cases which have progressed to the stage of osteoporosis and all the dental joints are becoming disorganized, it is well within the mark to say that at least one-eighth of a square inch of tissue around each tooth is involved in the suppurative process, and if the whole of the alveolar processes are infected, then we have an area of at least *four square inches* of infected suppurating tissue situated at the commencement of the alimentary tract.

**Treatment.** The disease has since its recognition always been regarded as more or less intractable and difficult to treat, and as a result a large number of 'remedies' and methods have at various times been put forward.

There are two points upon which there is general agreement, firstly that all deposits of calculus must be thoroughly removed, and secondly that the patient's general condition must be treated. Whether it be a 'predisposing' condition such as diabetes, nephritis, syphilis, or plumbism, or whether it be a general toxæmic condition initiated by the septic arthritis, it is necessary that the patient's tissue resistance should be raised.

No local treatment should be commenced until the patient's urine has been examined at least, and the presence or not of any other concomitant disease ascertained by the proper examination of other systems.

The nature of the treatment varies firstly with the extent and stage of the disease. The factor which must determine whether local conservative treatment should be undertaken or not is the extent to which the alveolar bone has been destroyed. If there has been much destruction, and the tooth is very loose and a considerable portion of the root of the tooth is exposed to view, conservative treatment will be of little or no use (since the lost tissue can never be reformed), and the immediate extraction of the teeth and curetting the sockets should be undertaken. It must be noted, however, that teeth may be loose and yet the osteoporosis not far advanced, the exact condition may best be ascertained by means of skiagrams; should these be favourable then local treatment may be undertaken with a good prognosis.

The methods which have been in vogue in the past have been those of removing the calculus and subsequently packing and dressing the pockets with strong antiseptic drugs and acids, such as copper sulphate, quinine sulphate, aromatic sulphuric acid, or more recently lactic acid or bismuth paste. These methods have the disadvantage of keeping up the mechanical irritation of the tissues perpetuating the 'pockets' and the chronic venous congestion of the gingival folds; they are, moreover, intensely disagreeable and often very painful to the patient, they are certainly slow and tedious (and therefore costly).

The author's method of treatment is as follows: the patient is received into a hospital, where proper aseptic methods are possible, is prepared for an anæsthetic, and the mouth sterilized as far as possible by the continuous use of a mercuric chloride (1 in 2,500) mouth-wash for three hours prior to the operation. Chloroform anæsthesia is used and maintained by the Junker apparatus, this is absolutely necessary for the thoroughness and success of the operation.

The calculus is rapidly and effectively removed—laceration

of the tissues does not matter in the least, and the hæmorrhage is controlled by an assistant. Then by means of special curettes, curved scissors, &c., the whole of the infected tissue is removed[1] (see Fig. 18), applications of tincture of iodine are made frequently, and massage is undertaken as soon afterwards as possible. The patient is put upon a mixed diet of fibrous and acid carbohydrates, proteids and extractives being reduced to a minimum. By this method excellent results are obtainable in a remarkably short space of time. On the fourth day after operation it is difficult to realize that a condition of 'pyorrhœa alveolaris' has been present. The advantages of this operation are: (i) that the morbid tissues are removed at once; (ii) that free and efficient drainage is established, the subsequent *collection* of pus being impossible; (iii) that it occupies a comparatively short time, and occasions no pain to the patient.

The method has the further merit of being the only one suitable for those cases in which a surgeon requires a septic mouth to be treated prior to a major operation on some other portion or organ of the alimentary tract in order to avoid secondary infection. It cannot, however, be very satisfactorily carried out, when the patient is not in a fit condition for chloroform.[2] Local anæsthesia may be used, but this usually means a longer time for the operation, and some pain for the patient.

The passive hyperæmic methods associated with the name of Beer have been adapted for such cases as these. A special appliance is necessary in the shape of a spring splint which grips the jaws along the roots of the teeth, or of a circular-shaped suction cup which encloses the teeth and gums. This is applied and the air removed by means of a rubber

[1] This may be termed 'gingivectomy'.

[2] Ether is unsuitable—the great increase in the mucous, salivary secretion, and the hæmorrhage obscure the field of operation.

ball with a valve. Sufficient cases, however, have not yet been treated by this method to be able to give any definite idea of its value.

*Vaccine treatment.* This method has now been carried out in a large number of cases chiefly though as an accessory to local and general treatment. The method is somewhat tedious because of the fact that the infection is always a very mixed one, and it may be necessary first of all to ascertain which are the causal organisms. This is done by making cultures from the purulent discharge, and estimating the

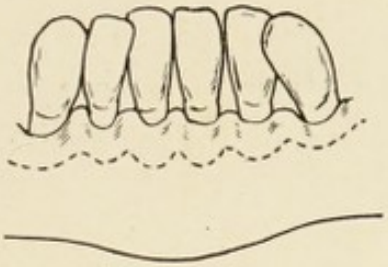


FIG. 18. The dotted line shows the line of incision in gingivectomy. All the tissue above the line is removed entirely.

opsonic indices of the patient to those organisms present which are known to be pathogenic.

Usually those organisms to which the index is either low or very high are selected for making the vaccines.

Five to fifteen inoculations of 50 to 250 millions are made at intervals of two or three weeks. Opsonic indices are taken during this period and the vaccines modified accordingly. The opinion generally accepted at the present time is that the vaccine therapy may be and often is a most valuable aid to local surgical treatment, but that by itself it is not sufficient. It is undoubtedly of most value in controlling and eliminating those secondary disorders due to septic absorption from the oral tissues, more particularly is this the case with regard to 'rheumatic' joint and muscular pains, loss of weight, headaches, pallor, and debility; all these have been repeatedly observed to clear up and the

patient to regain weight after suitable vaccine treatment. In addition to local treatment a course of arsenic or arsenic and iron is often very valuable in relieving the marked anæmia which so frequently accompanies these cases. Finally, whatever line of treatment is adopted it must be carried out boldly and energetically, and the seriousness of the condition pointed out to the patient in order to obtain his co-operation; for unless this is obtained, and the patient systematically carries out rules for oral hygiene, massage, and occasional applications of tincture of iodine, in addition to a proper dietary, there is always a risk that the condition may recur, and the more is this so in patients suffering from one of the chronic systemic diseases which tend to reduce the resistance of the peri-odontal ligaments.

# CHAPTER VI

## CARIES OF THE TEETH

### ÆTIOLOGY—PATHOLOGY—DIAGNOSIS OF THE VARIOUS STAGES

#### ÆTIOLOGY

CARIES of the teeth is essentially a disease of civilization, its numerical incidence may be said to be directly proportional to the degree of civilization to which any race has attained, but it is only within comparatively recent times that the disease has become so alarmingly prevalent. Among 'native' races leading natural lives the occurrence of dental caries is comparatively very rare, although spasmodic cases are found in the skulls at least of all races even the least civilized.

In this connexion it is interesting to notice the incidence of caries at different times in Great Britain.[1]

| | | | | |
|---|---|---|---|---|
| British skulls of the *Stone Age* show carious teeth in | | | | 2·9 per cent. |
| ,, ,, ,, *Bronze Age* | ,, | ,, | ,, | 21·8 per cent. |
| Romano-British skulls | ,, | ,, | ,, | 32·0 per cent. |
| Anglo-Saxon skulls i.e. decreased civilization | ,, | ,, | ,, | 15·0 per cent. |
| School children in England at the present time[2] | ,, | ,, | ,, | 86–98 per cent. |

Among 'native' races, too, at the present time the incidence of caries is directly dependent upon their mode of life.

In those natives who still lead the natural life of their

[1] Colyer, *Dental Surgery and Pathology*, p. 358.

[2] *British Dental Association Statistics*: Birmingham school children, Richards; Leith school children, Robertson; Kettering school children, Layton.

forefathers caries continues to be extremely rare, but in those who have been drawn into the vortex of civilization the prevalence of caries is as great as in European civilized people. Again, when caries does become prevalent in natives it does so in a rapid and extensive manner, in this respect being similar to the course of other ' introduced ' diseases such as exanthemata, phthisis, typhoid, &c.

This is well instanced in the case of the Maoris, physically one of the finest of native races.

A generation or so ago the incidence of caries in this race was lower than in any other native race for which figures are available, being only 1·09 per cent.[1] At the present time the average percentage of two schools of Maori children living under civilized conditions I found to be 95 per cent., and not only so but the extent of the disease in each mouth was far greater than in the mouths of European children of the same districts. It may be said then that dental caries affects about 95 per cent. of the present generation of civilized people, hence it is obviously the most prevalent of all diseases, moreover the extent of carious teeth in each mouth affected is no less alarming.

Ottofy, after an examination of 14,544 teeth of American school children, found that 30 per cent. were carious, and Cunningham, reporting on the work of the Cambridge dental 'clinic', gives statistics which show that 52·3 per cent. of the teeth of the children examined (1,403) were carious.

Females are slightly more susceptible than males.

Caries is chiefly a disease of childhood and adolescence, it appears very early in life, sometimes as soon as the deciduous teeth are cut, and the incidence steadily rises up to the age of 14 or 15. Should however the teeth remain immune to caries for a period of 8 to 10 years after

[1] The average of Mummery's and the Author's figures. See *Trans. Odonto. Soc.*, vol. ii, O. S. ; and Pickerill, *The Prevention of Dental Caries and Oral Sepsis*, London, 1911.

eruption, there is a probability that they will remain healthy for many years, or if not the occurrence of decay will be but spasmodic and gradual.

**Pathology.** Caries results from the acid fermentation of carbohydrates by micro-organisms.

The mouth, as is well known, is a septic cavity swarming with a very large variety of bacteria (Fig. 19); a number

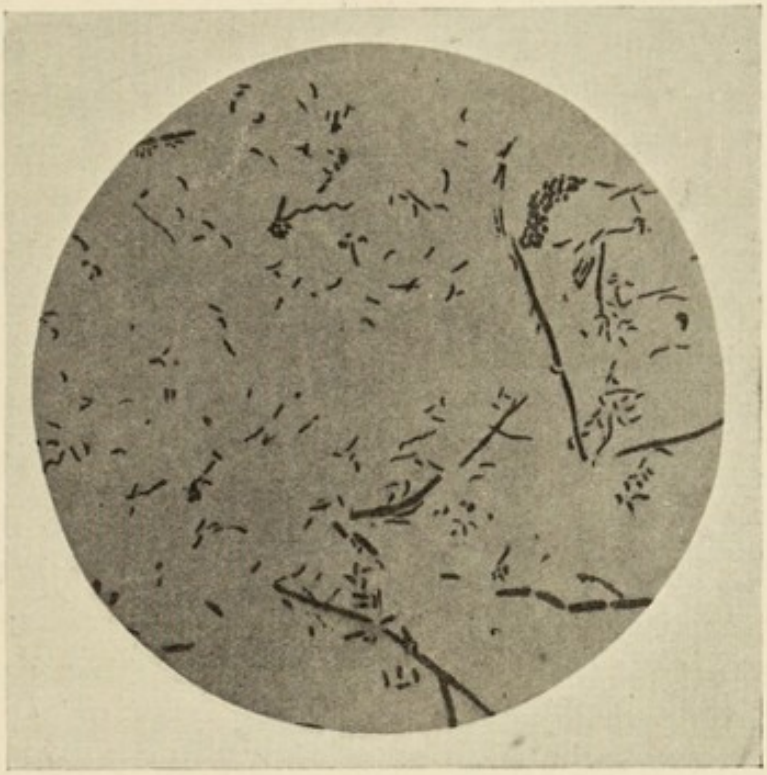


FIG. 19. Film of organisms from the mouth direct, to show the variety of forms normally present.

of these have the property of bringing about an acid fermentation of carbohydrate media.

The acid which is chiefly formed is lactic, occasionally this is further oxidized to butyric acid, traces of acetic and succinic acids are sometimes developed.

Such organisms are practically always present in all mouths: the following are the ones chiefly concerned[1]; Streptococcus (brevis), Staphylococcus (aureus and albus),

[1] Goadby, *Mycology of the Mouth.*

B. necrodentalis, Sarcinæ (lutea and aurantica). These are acid-forming and the following are proteolytic ; B. mesentericus (ruber, vulgatus, and fuscus), B. furvus, B. gingivæ pyogenes, B. fluorescens liquefasciens motilis, and B. subtilis.

Harm only results of course when the carbohydrates remain in proximity to the teeth and there undergo fermentation. There are two factors which influence the tendency for foodstuffs to lodge on or between the teeth ; (i) the disposition of the teeth, and (ii) the softness and 'stickiness' of the material. It is quite clear that, if the teeth be crowded and irregular, nooks and corners will be formed which it is impossible to keep clear of food detritus, and hence that more material will be available for acid formation in such cases. It is equally obvious, too, that certain materials like biscuits, chocolates, caramels, pastry, &c., are of themselves adhesive in nature and tend to ' stick ' to the teeth, and therefore also to the production of more acid.

The effect of mouth organisms upon proteids is to induce an alkaline decomposition. These facts may be experimentally verified in a simple manner as follows : masticate a small quantity of bread, and when it is insalivated (i.e. well infected with mouth organisms) expectorate it into a sterile test-tube and incubate it for twenty-four hours. Do a similar experiment with meat. Upon testing with litmus the bread-tube will be found to be strongly acid, and Ufflemann's reagent will show lactic acid to be present. The meat-tube will be strongly alkaline and very offensive.

In a similar manner caries may be produced artificially in teeth suspended in a mixture of carbohydrates and saliva for some weeks, provided that when the acid becomes too strong it be occasionally neutralized by the addition of an alkali. Microscopically such ' artificial ' caries in a tooth exactly resembles the ordinary pathological variety.

The equations expressing what takes place in the oral

fermentation of the various kinds of carbohydrates are as follows :—

Monosaccharide :

$$\underset{\text{(Glucose)}}{C_6H_{12}O_6} = \underset{\text{(Lactic acid)}}{2(C_3H_6O_3)}$$

Disaccharide :

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

$$C_6H_{12}O_6 = \underset{\text{(Lactic acid)}}{2(C_3H_6O_3)}$$

Polysaccharide :

$$\underset{\text{(Starch)}}{(C_6H_{10}O_5)^N} + H_2O = \underset{\text{(Dextrin)}}{C_6H_{10}O_5} + \underset{\text{(Maltose)}}{C_{12}H_{22}O_{11}}$$

$$C_{12}H_{22}O_{11} + H_2O = 2C_6H_{12}O_6$$

$$2(C_6H_{12}O_6) = \underset{\text{(Lactic acid)}}{4(C_3H_6O_3)}$$

$$2(C_3H_6O_3) = \underset{\text{(Butyric acid)}}{C_4H_8O_2} + CO_2 + 2H_2$$

The effect of this development of acid against the teeth is that the lime salts of the enamel are dissolved and a superficial depression or cavity results ; the latter owing to its constantly receiving increasing amounts of food débris, giving rise to an increased acid production, increases in depth, and soon the dentine is reached.

Here the process spreads in two directions : laterally along the junction between the enamel and the dentine, and centripetally along the dentinal tubules (see Figs. 20 and 21).

In the dentine the other organisms having a strong proteolytic action come into play, and after the tissue is decalcified by means of the acid-forming organisms the collagenous matrix is removed by the peptonizing bacteria.

The organisms penetrate along the dentinal tubules, and by their combined decalcifying and peptonizing powers

gradually enlarge individual tubes until several coalesce and form what are called liquefaction foci and the fusion of these latter causes a ' cavity ' to appear.

Thus a large cavity comes to be formed in the dentine which is frequently larger than the original cavity in the enamel, this is well shown in the accompanying sections of carious teeth (Fig. 20).

The result of this is that the enamel is undermined and weak, and under the stress of mastication gives way suddenly and a large cavity becomes apparent to the patient.

The next stage is marked by a hyperæmia of the pulp owing to the constant irritation by the acid and food débris, the organisms then reach the pulp and *myelitis*[1] results. If this be very acute and the dentine covers the pulp entirely, or if only a minute part of the pulp is exposed in the base of the cavity, it usually terminates in gangrene owing to strangulation of the vessels at the apical foramen. In the event, however, of the cavity being large and the area of pulp exposed likewise, there may be room for swelling to take place or the superficial capillaries may be ruptured, thus relieving the tension ; in this case the acute myelitis may subside. The attack, however, is bound to recur, or it may become chronic. Granulation tissue may develop on the surface and a ' polypus ' be formed which occasionally nearly fills the carious cavity ; the pulp is then very tolerant of mechanical irritation and may remain in such a condition for many months.

Gangrene of the pulp is, however, the more frequent termination ; it is then only a matter of a short time before the organisms make their way through the apical foramen and infect the peri-odontal ligament and surrounding bone of the jaw. An acute periodontitis is thus set up which may subside, but as a rule suppurates and an alveolar abscess results ; this *may* evacuate of its own accord via the

[1] Sometimes called ' pulpitis ' or ' papillitis '.

root canal, generally, however, the pus penetrates the outer alveolar plate and in the great majority of cases the condition becomes chronic—a chronic abscess sac at the apex of the tooth and a chronic sinus leading out into the labio-dental sulcus, continually or intermittently discharging pus, which of course is swallowed by the patient. If the condition remains untreated this may continue for years; meanwhile the combined action of the acid-forming and peptonizing organisms gradually destroys the whole of the crown of the tooth, and the roots are gradually extruded

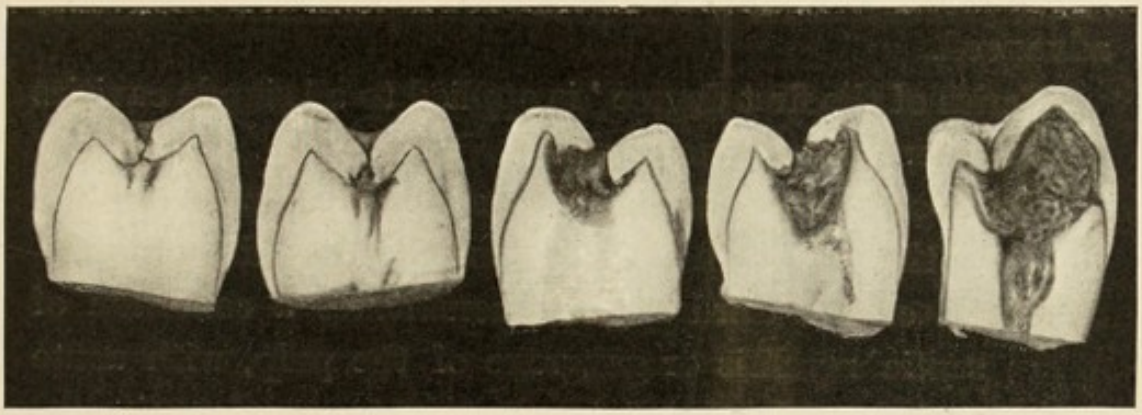


FIG. 20. Sections of teeth showing various stages of caries. They illustrate the important point that considerable destruction of tissue and gangrene of the pulp may occur with practically no visible cavity.

from the alveolus until eventually only a very small portion of root remains which becomes loose and is cast out as a foreign body.

An alveolar abscess does not always point in the labio-dental sulcus. It may appear in the palate from upper molars or lateral incisors, on the cheek from upper or lower molars, just below the inner angle of the orbit from upper canine teeth, or below the jaw from second or third lower molars.

*Caries presents different appearances* in patients of different ages; in children the progress is usually rapid and is known as acute caries or caries humida. The tooth substance is rapidly softened, is of light colour, and leathery in consistency.

In middle age the process is a slower one usually, and is termed chronic caries or caries sicca. It is characterized by shallow cavities, deep pigmentation and friability of the dentine.

It is frequently convenient to speak of any particular stage of the carious process above described, as being caries of the first, second, third, or fourth degree (Fig. 21). This is more especially useful in making notes of a case, since the seriousness of the condition present rests usually not on the number of carious teeth present, and not upon their mere appearance, but upon the stage to which the disease has advanced.

'Caries of the first degree' is applied to all simple cavities where there is no myelitis.

'Caries of the second degree' is used to designate extension to the pulp with actual or potential myelitis.

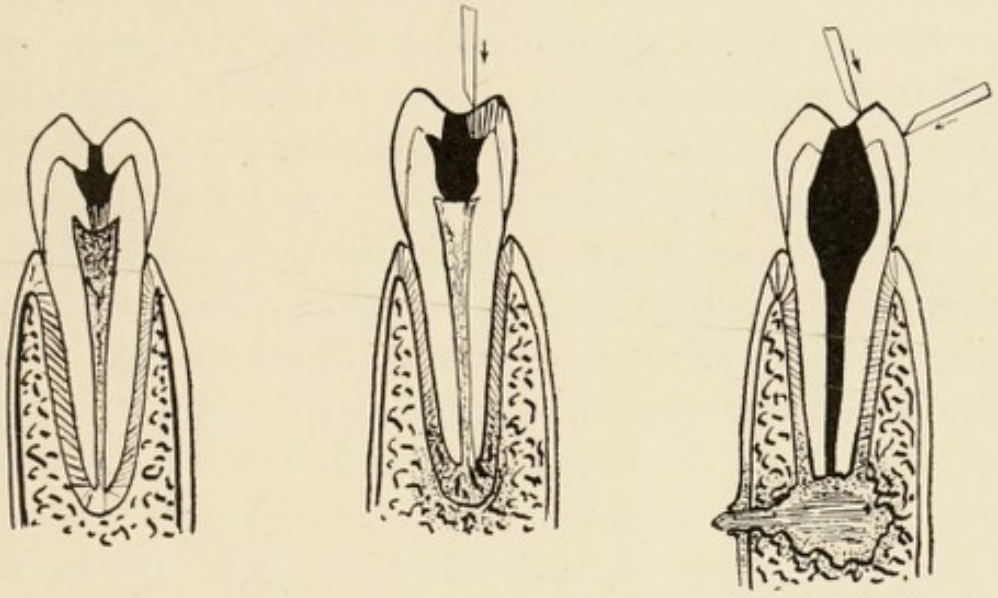
'Caries of the third degree' constitutes a condition of septic myelitis or gangrene of the pulp, with acute periodontitis present.

'Caries of the fourth degree' is applied to the condition when the crown of the tooth has been practically destroyed, an acute or chronic alveolar abscess being usually present.

**Signs, Symptoms, and Diagnosis.** The first sign of caries in a tooth may be said to be an opacity of the enamel due to solution of the interprismatic substance. In fissures of molar and premolar teeth, however, this is not the first visible sign, though doubtless it occurs; a dark brown line appears, caused by the infiltration of the remains of Nasmyth's membrane by chromogenic bacteria.

There are usually no symptoms at this stage, though in molars and premolars when the brown line has become well marked very sweet substances like chocolate may penetrate to the dentinal fibrils and cause pain. (When this occurs *immediate* treatment by 'filling' is necessary.)

The next sign is the presence of roughness and loss of tissue in the enamel, and the patient may begin to notice that food tends to collect in that situation ; there is as a rule no pain. When the dentine is reached the tooth begins to become discoloured and the enamel opaque around the area of the cavity—due to the 'lateral spread' at the amelo-dentinal junction. This should be recognized since it is



1 2 3

FIG. 21. Diagrams illustrating caries of various degrees. 1, Early second stage ; myelitis present ; no 'exposure' ; 2, Third stage : peri-odontitis and commencing gangrene of the pulp. The tooth is elevated in its socket ; 3, Early fourth stage ; large central cavity in tooth filled with débris and organisms ; absorption of bone, formation of abscess cavity with pus discharging through sinus into mouth. (2 and 3 also illustrate the use of the enamel chisel.)

frequently the only visible sign of a cavity, the orifice of the latter being small and tucked away between two teeth and thus, although serious in extent and causing pain, either local or referred, may escape observation.

The actual discoloration is due largely to the action of the mesenteric group of bacilli which are chromogenic, i.e. they are not themselves coloured but colour the medium upon which they are developing, in this case the *dentine*.

But because a tooth is discoloured it is not of necessity carious or septic.

Healthy teeth are frequently *discoloured on the surface*, due to staining or deposits of stained calculus. Such staining may be due to tobacco, silver, iron, copper, or nickel salts.

A green stain in the cervical region is usually due to a pathological condition of Nasmyth's membrane.

In the next stage of caries when the cavity has *suddenly* appeared owing to stress of mastication, the condition is obvious to the patient and to the observer. In many people caries reaches this stage without any pain whatever.

As soon as MYELITIS supervenes characteristic signs and symptoms appear. The patient experiences sharp lancinating pains, induced chiefly by the presence of food in the cavity or by hot, cold, or sweet substances in the mouth. *Biting on the tooth causes no pain.* The pain is relieved by ' sucking ' the tooth or by holding cold water in the mouth because of course these devices relieve the congestion of the blood-vessels. On examination a bright red spot may be seen in the base of a deep cavity ; if this cannot be seen a pledget of cotton-wool should be gently pressed into the cavity, if this gives rise to pain the diagnosis of myelitis with ' exposure ' is confirmed.

If the condition remains untreated the attacks of pain usually increase in frequency and severity, becoming most intense just before strangulation and necrosis of the pulp occurs. The pain may not be entirely localised, but may wander and give rise to more or less severe neuralgia of the other branches of the fifth nerve ; a circle of hyperæmia is not infrequently seen externally on the cheek at this time, but of course there is no cellulitis or swelling.

With the death of the pulp, symptoms subside for a varying length of time until, as is inevitable, periodontitis occurs. The first symptom of *periodontitis* is that the tooth becomes *tender to bite on*, and then slightly raised in the socket.

The pain is of a dull continuous nature, and heat and cold have not the same marked effect as in myelitis.

Firm steady *pressure on the tooth* in this stage relieves the pain because the blood is driven out of the peri-odontal vessels. Percussion of the tooth (with a metal instrument) gives a painful response, whilst cotton-wool or other foreign material in the carious cavity causes no pain. There is usually some accompanying hyperæmia and tenderness of the muco-periosteum over the affected tooth. In later stages pressure upon the tooth instead of relieving increases the pain, because exudation from the blood-vessels has occurred and no external pressure can make this return but only increases the pressure upon the nerve endings.

If suppuration takes place the tooth becomes very loose in its socket, pus may well up between the gum and the tooth, there is considerable swelling of the muco-periosteum, hyperæmia of the buccal tissues with more or less cellulitis. The pain is intense, throbbing and continuous in character, and does not abate until the internal pressure in the abscess cavity is relieved by perforation into the labio-dental sulcus, or by diffusion into surrounding tissues or organs. The tooth is excessively tender and any pressure aggravates the pain. The condition is usually accompanied by a rise in temperature, loss of appetite, foul tongue, and constipation.

*Chronic dental myelitis* if associated with exposure usually leads to proliferation of the pulp tissue, forming a 'polypus' which more or less completely fills the carious cavity. This frequently causes *loss of function* of that side of the jaws—shown by the presence of a film of calculus and detritus covering the masticating surfaces of the teeth on that side. Chronic myelitis not associated with exposure of the pulp, but due to some form of slow continuous irritation, frequently leads to fibroid or calcareous degeneration taking place. The former is usually painless, and the

whole of the normal pulp tissue may be entirely replaced by a dense fibrous network.

On the other hand calcareous degeneration or the development of 'pulp nodules or stones' may give rise to considerable pain, neuralgic in type and difficult sometimes to locate; it is more frequently found in patients of a gouty or 'rheumatic' diathesis (Fig. 22).

Since the effective treatment, even palliative, of pain in the teeth (odontalgia) is absolutely dependent upon a correct diagnosis, it is necessary in all cases to *determine exactly to what stage the lesion has advanced* before attempting to put any form of treatment into practice.

The following table may be found useful from this point of view :—

DIAGNOSIS OF ODONTALGIA

| *Myelitis.* | *Periodontitis.* | *Alveolar Abscess.* |
|---|---|---|
| Pain spasmodic, lancinating, and induced by stimulation. | Pain dull and continuous. | Pain excessively acute and continuous. |
| Pain relieved by deflation and cold. | Pain not much affected by cold and not at all by 'sucking'. | Condition too painful to apply either of these. |
| Pressure on tooth causes no pain. | Pressure on tooth first relieves pain and later increases it. | Pressure on tooth causes intense pain. |
| Pressure in cavity causes sharp pain. | Causes no pain. | Causes no pain if the tooth be held perfectly firm and still. |
| Small red point of exposed pulp may be seen. | Pulp either suppurating, gangrenous, or decomposed. | Pulp cavity filled with decomposing débris and organisms. |
| Percussion causes no pain. | Tender to percussion. | Percussion cannot be tolerated. |
| No hyperæmia, swelling, or tenderness of muco-periosteum. | Muco-periosteum tender and reddened. | All the typical signs of an abscess. Heat, redness, pain, swelling, fluctuation, and loss of function. |

Pain may be referred from one tooth to another, usually to one situated more anteriorly in the same jaw or to the corresponding tooth in the *opposing* jaw, but never to a tooth

on the opposite side of the mouth. The possibility of this must always be borne in mind, or influenced by the insistence of the patient a wrong diagnosis will be made and treatment will fail.

It is not pretended that these three conditions by any means exhaust all the causes of odontalgia, but they are

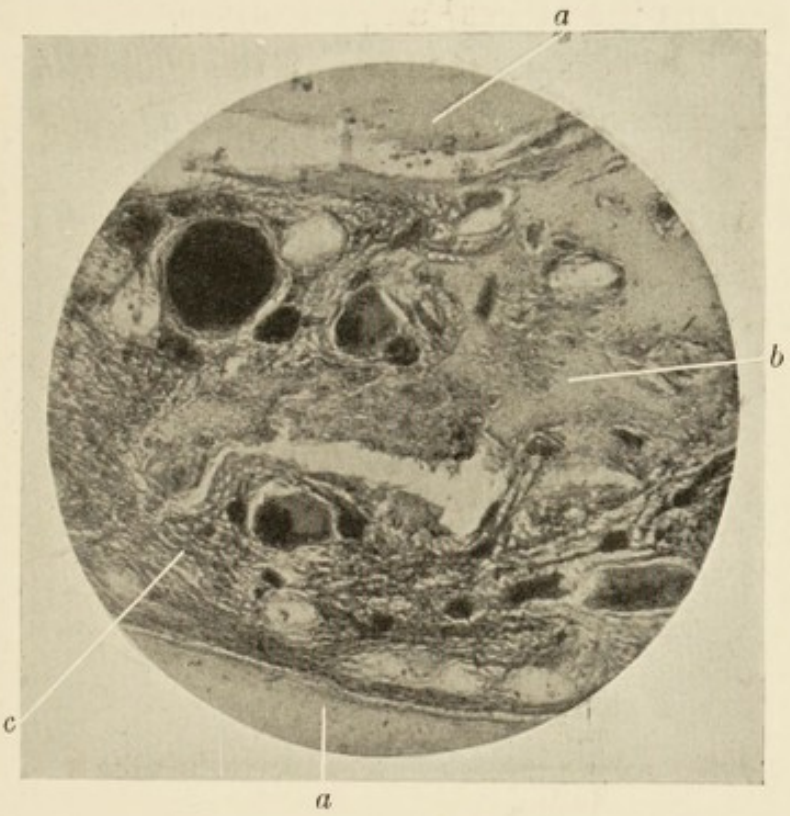



FIG. 22. Calcareous and fibroid degeneration of the dental pulp. *a*, Normal dentinal walls of pulp cavity; *b*, Calcareous degeneration; *c*, Fibroid degeneration. Notice the complete absence of normal cellular tissue, including odontoblasts.

the most common causes, and therefore all that can be discussed here. Other local causes are, calcareous degeneration of the pulp, erosion, attrition, abrasion, thermal and electrical shocks due to the presence of large fillings, foreign bodies around the necks of teeth, viz. calculus, ligatures, 'ledges' of filling material or crowns; also diseased conditions of the jaws or adjoining regions, such as tumours,

innocent and malignant, necrosis, syphilis, stomatitis of various kinds, more especially tubercular. Pressure on any part of the fifth nerve may cause intense odontalgia. The author recalls that a case in which pain in the left upper teeth, which were all quite sound, was the only symptom of a glioma pressing upon the left Gasserian ganglion. Pain is, too, occasionally referred to the teeth from remote regions such as the uterus[1] and intestines.

In patients of gouty diathesis and the subjects of malaria, very slight causes tend to bring on an attack of odontalgia.

[1] The author was recently consulted in a case in which four sound molars had been successively extracted for odontalgia without relief. Dysmenorrhœa was found to be present in a marked degree, and upon treatment of this condition the odontalgia cleared up.

# CHAPTER VII

## THE PREVENTIVE TREATMENT OF DENTAL CARIES[1]

Considered from the point of view of public health and from a purely medical aspect, undoubtedly the preventive treatment of caries is most important. By its means alone can we rationally hope to lessen the very serious prevalence of the disease, and from a medical point of view eliminate the starting-point of a great many serious systemic disorders ; in other words, lessen the occurrence of oral sepsis and all its widespread secondary disorders. This is a matter for the earnest consideration of the general medical practitioner, just as much as or more than it is for the dentist. The subject may conveniently be discussed under two headings.

1. Physiological or natural prophylaxis.
2. Artificial prophylaxis.

### Physiological Prophylaxis—Natural Immunity

It is quite obvious that there exist physiological means of preventing the occurrence of caries, since not only are natural races almost immune, but occasionally in civilized communities one sees perfectly healthy natural dentures. Bearing in mind, too, the fact that, as we have previously pointed out, caries of the teeth is due to the lactic acid fermentation of carbohydrate detritus, it is a remarkable fact that amongst the races least afflicted with caries are

[1] For a fuller account of this important subject see *The Prevention of Dental Caries and Oral Sepsis*, by the author, London, 1911.

the great rice and millet eating races of East and Southern Asia—exactly the diet which might be expected to give rise to prolific caries.

There must be some degree of immunity, too, in every individual albeit very feeble; were it not so, teeth would not last for a twelvemonth, and probably by now, by reason of the forces of evolution, teeth in the human race would have become mere embryonic vestiges.

What, then, is the cause of such immunity? And how has it been so greatly diminished in the great majority of individuals in modern races? The formation of an acid is the initial stage in the occurrence of caries; given, therefore, the simultaneous presence of a sufficiency of alkaline salts, it is quite clear that the acid would be at once neutralized and no free acid would be available to attack the lime salts of the enamel.

Now the saliva is an alkaline fluid which should normally bathe the buccal tissues to the extent of two or three pints daily; it cannot therefore be without effect, and this *a priori* one would assume to be a beneficial one.

Again, although there is a difference in the resisting power of various teeth, enabling them to be classed either as 'malacotic' (whitish soft teeth) or 'sclerotic' (hard yellowish teeth), the author has elsewhere[1] brought forward evidence to show that probably all teeth are originally malacotic in type, and should normally undergo a hardening process after eruption and become eventually sclerotic in type.

Since the enamel is beyond the pale of the nutritional and ordinary protective agencies of the body, immunity must be due to external agency, and the only possible agency is the saliva. It has been customary to regard the function of saliva as being that of facilitating the swallowing of food and helping in the digestion of starch in the stomach. These, however, are in the author's opinion to be regarded

[1] *The Prevention of Dental Caries and Oral Sepsis.*

as of very subsidiary importance, briefly for the following reasons.

(i) The salivary glands (in an animal) may be excised without any effect being produced on the general nutrition or the digestion of starches. This has been done in a dog by Schäfer, who noticed only that the animal drank more water. In order to eliminate this latter mode of compensation, and since dog's saliva frequently contains no ptyalin, the author excised the salivary glands of a rabbit; this produced only a temporary stomatitis, no increase in the undigested starch in the fæces, and no difficulty whatever in swallowing was noticed even with dry food.

(ii) The percentage of the total digestion of starch by ptyalin must be extremely small since its action is destroyed as soon as free acid is present in the stomach. (Cannon has shown that the digestion of starch in the stomach goes on for longer than was formerly thought, but even this only accounts for a small proportion of the whole, and, as has been shown, in animals at least, the pancreas is capable of digesting the whole amount.)

The salivary glands must have an important function or they would disappear; it must be, therefore, in the protection of the oral cavity that the salivary secretion is of importance to the organism; moreover, its composition and methods of secretion are perfectly adapted to this end. It is necessary to remember that the secretion of saliva is a reflex act and is normally excited by stimulation of the gustatory nerves; thus food in the mouth calls forth a flow of saliva automatically, but the composition of such secretion varies enormously with the character of the food.

The principal constituents of saliva are :—

1. Water.
2. Alkaline salts of sodium and potassium.
3. Ptyalin.

4. Potassium sulphocyanate.
5. Phosphates.
6. Mucin.
7. Chlorides.

Every one of these is extremely valuable from a protective point of view, and the more there is of them the greater *must be* the protection afforded. Now in saying 'the more there is of them' percentage composition is not meant; such indeed by itself may indicate very little—what is necessary to know is the *amount per minute present in the mouth*, since saliva does not remain in the mouth but is constantly being swallowed. This has been determined in detail by the author and the full results are given elsewhere.[1]

Electrical stimulation of the secretory nerves is not very useful, since it is an extremely rare form of stimulus and is untranslatable into the normal gustatory stimuli, ordinary dietetic stimulants have therefore been used in the following estimations.

**Water (or total amount) and alkalinity.** The following table shows that different substances induce very different secretions of saliva, that acid fruits produce most in amount and greatest alkalinity, that a soft demulcent substance like bread and butter actually depresses the secretion and alkalinity, and that neutral substances like biscuit and meat only cause a very feeble increase.

| *Substance masticated as a stimulant.* | *Flow in c.c. per minute.* | Alkalinity per min. *expressed as equal to c.c.* $\frac{N}{50}$ *NaOH.* |
|---|---|---|
| Resting saliva | 1·65 | 1·73 |
| Bread and butter | 1·6 | 1·26 |
| Meat | 2·6 | 2·18 |
| Biscuit | 2·8 | 2·7 |
| Apple | 12·0 | 13·24 |
| Orange | 11·0 | 15·0 |

[1] Op. cit.

It was observed also that tea acted as a salivary depressant and alcohol (especially acid wines) as powerful stimulants. Such increased secretion lasts too for some time afterwards, at least for fifteen minutes and probably for much longer. (For instance during an acid dietary the resting saliva increases in amount and alkalinity, and moreover the secretion in response to biscuit stimulation is also markedly increased.)

The beneficial effects of an increased amount, rate of flow and alkalinity, of saliva in preventing the lodgement of carbohydrates, washing them away when lodged, or neutralizing acid if formed, must be quite self-evident; and it is equally obvious that in order to obtain such beneficial secretion acid substances are the most useful. Sweet, salt, bitter and aromatic substances were tested similarly, but in no case did the increase in secretion approach that induced by acids. The most suitable acids are the 'natural acids' or salts such as tartaric acid, tartrate of potash, citric acid, &c.; mineral acids such as hydrochloric and sulphuric have a distinctly feeble effect and their 'astringent' effect and 'dry taste' is well known.

**Ptyalin.** It has not been customary to consider ptyalin as a protective agent, yet there can be no doubt that its most important rôle is as such. We have already indicated its very small value as a digester of starch for absorption, yet when it is remembered that caries of the teeth is largely caused by the lodgement of solid insoluble starch particles in the mouth, and that the property of ptyalin is to render these soluble and fluid and hence *unlodgeable*, it becomes at once apparent how important ptyalin must be in maintaining a healthy and hygienic state in the mouth; NOTHING ELSE IN FACT COULD PERFORM SUCH A FUNCTION, it is exactly the substance which is required for the purpose.

Considered in this light its presence becomes both logical and highly important.

The amounts in which it may be present have been determined by the author for a similar series of stimulations by means of Metts tubes. The results are given in 'ferment units' per minute (i.e. the square of the length of the column of starch digested).

| *Stimulant.* | *Ferment Units.* |
|---|---|
| Resting saliva | 14·8 |
| Biscuit | 18·4 |
| Bread and Butter | 36·2 |
| Biscuit soaked in Tartaric Acid 1·5 per cent. | 147·2 |
| Apple | 152·4 |
| Orange | 179·0 |

Again it is clear that acid substances call forth the greatest amount of ferment per minute, and that substances like biscuit and bread and butter are extremely feeble excitants of ptyalin secretion. This is undoubtedly a very important matter, for where the ferment action is most needed, there it is least present; in this we have one of the most potent causes of the prevalence of caries—of the breakdown of natural immunity—since such non-stimulating foodstuffs form a very large proportion of modern dietaries.

A small proportion of the pytalin is possibly destroyed by the acid substances, but not very much, since as we have seen, these latter substances also induce a saliva of high alkalinity whereby the acids are rapidly neutralized.

**Potassium Sulphocyanate.** This substance is present in saliva in varying proportions. Its effect is a feeble antiseptic one—or rather antifermentative. In a strength of ·01 per cent. *in vitro* it reduces the acid production by one-half, and a 1 per cent. solution prevented acid formation for seven days.

The percentage in human saliva varies from ·0075 per cent. to ·03 per cent., but for the same individual under the same conditions it is fairly constant. With an increased rate

of flow per minute the *percentage remains the same*, therefore the total amount present per minute is increased proportionately to the rate. Hence here also natural acid foodstuffs induce a greater amount per minute than anything else. Moreover, the amount in any sample of saliva seems to increase upon standing, and more so if incubated; this increment is probably derived from the decomposition of the proteid in the saliva. Therefore it would seem to be a special natural provision providing an antifermentative action during the night when salivary secretion is more or less at a standstill.

**Phosphates.** These follow an exactly similar law, not only the amount per c.c. but also the amount per minute is increased during the mastication of acid substances.

The presence of phosphates is desirable as being substances which readily combine with lactic acid, and which are, as the author believes, of value in 'hardening' the outer strata of the enamel. In any case acid foods tend to keep them in solution, and prevent their deposition as calculus around the necks of the teeth which invariably leads to sepsis.

**Mucin.** This may be beneficial or harmful in its effect—depending upon its physical condition. When it is completely dissolved in alkaline saliva it probably has no action at all and is negligible.

When it is in a semi-dissolved state it is very viscous, and is extremely harmful in that it tends to bind food débris to the teeth, thus promoting stagnation and acid formation. During the mastication, however, of acid substances, some of the mucin is precipitated and the saliva becomes more fluid and food particles can be more readily washed away, the precipitated mucin carries down alkaline salts with it and thus affords temporary protection both mechanical and chemical to the oral tissues. Afterwards the mucin is decomposed by the action of bacteria with the production of

alkaline end products, and these tend further to neutralize any acid formed by fermentation.

**Chlorides.** The effect of sodium and potassium chlorides in the saliva is doubtless to afford protection to the soft tissues and maintain the vitality of the mucous membrane, and also to act as natural auxiliaries in the stimulation of the gustatory nerves—Nature's method of increasing to some extent the sapidity of more or less insipid food, and of neutralizing the depressant effect of a highly alkaline saliva upon the gustatory nerves.[1] The maximum amount of chlorides per c.c. and per minute is produced by the stimulus of acid foods.

The effect of reducing the sensibility of the gustatory nerves by cocainizing the tongue in man is to lessen by one-half the salivary secretion, and the experimental removal of the taste buds in animals is followed by a perceptible diminution of weight in the salivary glands (submaxillary).[2]

**Acid dietary.** By adopting either a partial or complete acid dietary the author has shown that both the amount and alkalinity of the saliva per minute may be definitely increased, and not only so, but that the saliva resulting from the mastication of biscuit which previously gave a very poor response may, during a period of acid dietary, give a much increased response both in amount and alkalinity.

Thus in amount the saliva to biscuit stimulation was increased in a fortnight from 3·8 c.c. per minute to 5·8, and in alkalinity per c.c. from ·7 to ·83.

By a complete acid dietary is meant that at no time is anything eaten which has not an acid reaction, and all salivary depressants are carefully avoided. By a partial acid dietary is meant that at least substances having an acid reaction are taken at the commencement and at the end of all meals. It was found that the partial acid dietary gave

[1] See below.

[2] See the Author's *Prevention of Dental Caries.*

the best results, and moreover is far more practicable and pleasant.

**Salivary Depressants.** In the series of experiments already alluded to it was shown that certain articles of diet &c., actually depressed the salivary secretion below normal. Such depression is brought about by strong tea, butter, and *substances having an alkaline reaction.*

It was also shown that the drinking of pure water tended to reduce the flow of saliva in response to neutral substances eaten just afterwards. In view of the fact that a very large proportion of modern dietaries consists of 'tea and bread and butter' and of substances having a neutral or weak alkaline reaction, the failure of the resulting saliva to cope with the acid developed is only to be anticipated.

**Phagocytosis** takes place to some extent in saliva, but is very limited in amount since the medium is not isotonic with blood serum, and the author has not been able to show definitely that '*opsonins*' are present in the saliva of immune individuals, though there is some reason to suspect that this is so. At least it seems clear that more phagocytosis occurs in the saliva of a person immune to caries than in that of susceptible persons. It is possible, however, that this may be due to other causes than the presence of opsonins.

That such 'cleansing' of the mouth is brought about by the use of salivary stimulants after other carbohydrate food with a high potential for acid fermentation has been demonstrated as follows.

It was estimated by experiment[1] that the amount of acid developed in a mouth after eating chocolate and cake amounted in twenty-four hours to 5·0 and 4·0 acid units respectively (the unit being equal to 1 c.c. of $\frac{N}{50}$ $H_2SO_4$). Whereas if these substances were immediately followed by

[1] Op. cit.

apple and orange respectively, instead of there being an acid reaction an alkaline product resulted equal to 2·0 c.c. $\frac{N}{50}$NaOH in each case.

The mixture of such substances as opposed to a sequence has an effect in reducing the acid formation but it is not so marked, and in order to produce a similar effect the acid must be in excess.

We cannot, therefore, conclude otherwise than that the great physiological means of prophylaxis must reside in the saliva. That in the saliva we are provided with a natural mouthwash perfectly adapted to preserve the health of the oral cavity, and moreover that the amount of this fluid and its composition are largely under control and can be 'regulated' in a very exact manner by means of dietetic stimuli. The most beneficial of such stimulants we have seen to be 'natural' acids and acid salts, therefore the logical conclusion is that these should be utilized as much as possible and that all meals should be both begun and ended at least with such excitants of automatic protection.

That it is just in the amount of 'natural' organic acids which modern dietaries are particularly deficient has been shown by the author elsewhere.[1] That this is so might, too, be inferred quite correctly from the universal extent to which patent pills and aperient medicines are advertised at the present time. The natural physiological stimulus to the peristaltic action of the intestines provided by such natural acid substances is too frequently entirely absent, the result being that artificial substitutes become increasingly necessary in order to relieve the chronic constipation which has developed. Thus, by adopting such a partial acid dietary, not only are the tissues of the very commencement of the alimentary tract preserved in a healthy condition, but

[1] Op. cit.

also the function and tone of the lower part of the same tract will be maintained or recovered.

The alimentary tract, it is to be remembered, is a physiological and pathological entity from mouth to anus, and must be treated as such.

**Note on the Use of Sugar.** Sugar in any form is an important food, and especially so in children who are unable to take fats ; it affords a valuable and cheap source of energy and heat, and attention has recently been called to its use as a therapeutic agent in adults.

Sugar, on the other hand, even cane sugar, is fermented by mouth organisms and produces lactic acid, and therefore destroys the teeth and leads to oral sepsis, and this may outweigh any benefit derived from its internal absorption.

Is the use of sugar, therefore, to be abandoned ? By no means. Cane sugar, which is the form chiefly taken, is only slowly fermented by mouth organisms, but by reason of the fact that it stimulates a profuse secretion of mucin, and thus an extremely viscid saliva, it is bound down to the teeth for some considerable time, and hence in the end produces a fairly large amount of acid.

But if an organic acid such as lemon juice or acid tartrate of potassium be added first of all to the sugar, the mucin is not so profuse and a considerable quantity of it is precipitated by the acid, and thus the saliva is less viscid and more watery and the sugar gets swept away—it does not lodge and ferment.

This, then, is a simple method of giving sugar in a perfectly harmless manner, and one which should always be adopted in order to obtain the beneficial effect of sugar as a food without inducing a future septic condition of the mouth. Moreover, the addition of the acid probably prevents the hypersecretion of mucin and hyperæmia of the oral and gastric mucous membranes which are known to follow the ingestion of large amounts of sugar.

**Soured Milk.** Patients taking lactic acid bacilli, whether in powder or in milk, should always cleanse the mouth thoroughlyafterwards, or there will be an increased formation of lactic acid in the mouth and a corresponding increase in the tendency to caries. The best method of cleansing the teeth and mouth is for a piece of orange to be eaten immediately after taking the bacilli. This may be combined with the use of a tooth-brush, but the latter by itself would be quite inefficacious and probably would do more harm than good.

**Inoculation of the Mouth.** As a means of rapidly disposing of the carbohydrate débris in the mouth, the author has suggested [1] the inoculation of the buccal cavity with cultures of *Saccharomyces coagulatus*. This organism of course splits up dextrose (which is always an intermediate product of acid fermentation) into alcohol and carbon dioxide, and this it does when well established in a remarkably rapid manner, so that chiefly by reason of the $CO_2$ formation the food débris is broken up and dispersed before any acid can be developed.

For instance 'interdental' spaces filled with a mixture of biscuit and saliva remained full and apparently unchanged, although strongly acid, for *eight days*.

Exactly similar spaces filled with a mixture of biscuit, saliva, and *S. coagulatus* were clear in from *forty-five minutes to forty-eight hours*, depending upon the numbers of the organism present. The organism may be dried slowly and kept in this condition; activity is regained in suitable surroundings.

The dose I have at present used is 10 grains twice daily. The effect of giving yeast is also beneficial in other ways in that it increases the number of leucocytes in the blood, and since boils may be successfully treated in this manner (probably by raising the patient's opsonic index to the organisms), it is very probable that this particular organism, *S. coagulatus*,

[1] Op. cit.

will be found to have a similar beneficial effect in assisting to clear up suppurative conditions in the mouth associated with dental caries.

By such means as these, if properly and scientifically applied, caries may be prevented, and I have reason to believe arrested even, when it has not proceeded too far.

It will be sufficient in general cases to lay down general dietetic and hygienic rules for patients. But in special cases it is necessary to ascertain that the salivary glands are reacting normally to the stimulus and that the stimulus is not too great. For if the latter obtains it will have a depressant effect upon the glands, and then the acid of the fruit, &c., in the diet may be free to attack the lime salts of the tooth. Therefore, in such cases where a special course of treatment for the prevention of dental caries is being undertaken, the saliva should be analysed at the commencement and at frequent intervals to ascertain how it is responding, and in order to know when the maximum secretion of salts, water, and ferment is reached. This being found out the amount of salivary stimulants in the diet may be reduced a little. In special cases, too, the development of extensive caries may be due to the irregular disposition of the teeth, or to the presence of an abnormal number of acid-forming organisms; these factors should be ascertained and of course suitably treated.

# CHAPTER VIII

## THE PREVENTIVE TREATMENT OF DENTAL CARIES (CONTINUED)

### Artificial Prophylaxis

**The Use of the Tooth-brush.** It might be argued from the results of the last preceding experiments that, given a sequence of salivary stimulants and detergent articles of food, a tooth-brush would no longer be necessary. Logically this is correct, and practically also—if one could always be absolutely sure of the sequence—and it is always the end, the ideal to be aimed at ; but ideals are not reached in a jump, neither are customs and habits changed in a day, therefore it is necessary to take such habits as are already established and mould them in the right direction. Moreover, the prevalence of dental disease is much too serious a thing to rely entirely upon a single line of defence.

Quite apart from this aspect, however, a divergence of opinion has latterly arisen as to the benefit to be derived from the use of the tooth-brush ; it being inferred at least that the tooth-brush is not only useless for the prevention of caries but is positively harmful. Such suggestions contain some truth but much error.

Undoubtedly if a tooth-brush be used in a dirty and septic condition and the gums are inoculated by means of its bristles with extraneous germs, more harm than good will result, as also may occur by the too vigorous use of a clean brush in a markedly septic mouth. But because ill accrues from the wrongful use of a therapeutic agent its use cannot therefore be universally condemned.

Again, many patients who show marked susceptibility to caries claim frequently to have 'used a tooth-brush regularly'—patients to whom the above arguments would not apply, i.e. whose mouths have not as yet any suppurating foci, and who as a matter of personal cleanliness would take great care that the tooth-brush was at least 'scrupulously' if not 'surgically' clean.

It is not, however, so much the use or disuse of a tooth-brush as the use or *abuse* of a brush which is the important matter. That a brush may serve a useful purpose, may in fact be made to remove practically all the food detritus from the teeth, has been shown by the author.

But in order to accomplish this end it needs to be used *intelligently*. A person must know what it is he is endeavouring to accomplish in any act or he is not likely to achieve much success in the performance.

**Abuse of a Tooth-brush** usually arises from one or both of two causes, firstly from the size and shape of the brush or secondly from its manner of use. Both the size and shape of the 'ordinary' brush utterly preclude its proper use. It is necessary to have in mind when the teeth are being brushed that it is not a smooth continuous surface which has to be scrubbed, but a number of individual, separate, but contiguous rounded objects; further, that the surface of these is not absolutely smooth but is *imbricated* and is covered by a dense but thin membrane. It is necessary to remember that the food detritus has not soaked into the teeth but is merely lying on them and against them, hence the *material merely requires brushing off the teeth* thoroughly, the teeth *do not require to be scrubbed*. If a patient can be made to grasp these facts he will automatically adopt the right motion in handling the brush—to use a homely simile, a tooth-brush should be used after the manner of a clothes-brush rather than as a scrubbing-brush.

This point once gained, it will become apparent to the

user than an ordinary brush is quite incapable of being used in the mouth in the manner in which he automatically desires to use it. In fact the usual brush compells the user to scrub the teeth because he cannot well do otherwise, it is much too large to be used in any other way and covers as a rule nearly eight teeth at a time. This may ' save time ' but it does not save the teeth.

Fig. 23 shows the kind of brush which should be used. That is to say, it should be quite small, the bristles set not too close together, of unequal length, and of a fair stiffness. If in addition the handle be bent as shown in the illustration it facilitates its use considerably. All the surfaces of the teeth should be brushed in several directions, no one direction preponderating over others, and especially should the masticating surfaces of the molars and premolars be *brushed* thoroughly,—to brush the detritus out of the fissures and not to scrub it into them.

There can be no doubt that at night is the time when most benefit is derived from cleaning the teeth, in order to prevent fermentation occurring when the salivary secretion is at its minimum. Ideally, of course, the teeth should be brushed after every meal, and in this we might follow with advantage the practice of those native races whose food being softer and less sapid than others assiduously cleanse their mouths and teeth after every meal.

In cases of acute and rapid caries, in all prolonged fevers and in all abdominal operation cases, in fact in every case of decreased salivary secretion, this latter course of procedure must be absolutely insisted upon.

**Toothpicks and Floss Silk** are articles which are sometimes useful and necessary for the proper cleansing of the teeth, but their use should be limited to such cases as specially require their use.

The passing of floss silk between the teeth is quite impossible in a large number of mouths on account of the

closeness with which the teeth are set together, and this contiguity is not to be regarded as abnormal—at least in the mouth of any 'native' with a perfect denture it would not be at all possible to pass floss silk between the teeth.

Toothpicks may be useful when atrophy of the interdental papillae has occurred, thus leaving wide spaces between the teeth; quill or wooden toothpicks are preferable to metal ones, they cannot do any damage and can be discarded when used.

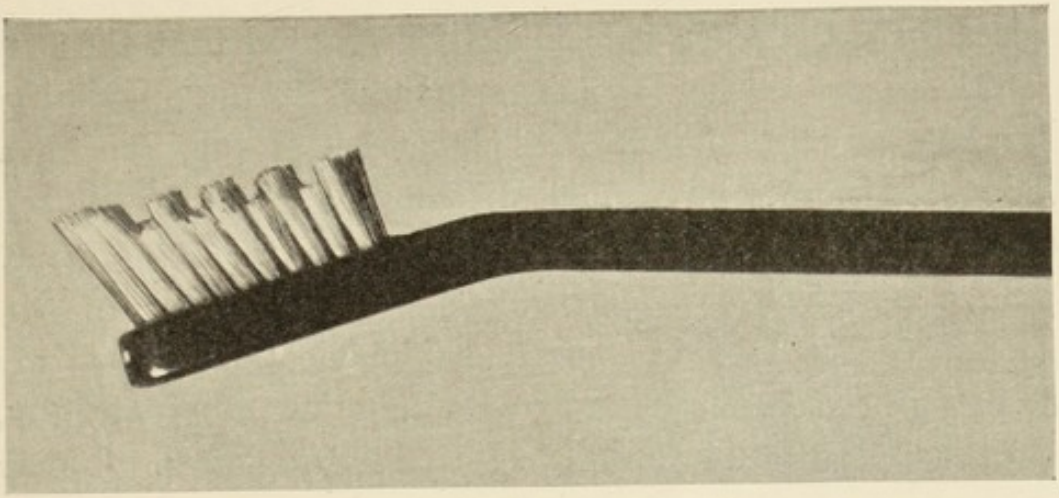


FIG. 23. The type of tooth-brush advocated. The illustration represents the actual size of the brush. Smaller brushes may be used with advantage—certainly nothing larger.

**The Use of Dentifrices.** The great majority of dentifrices either prescribed or proprietary at the present time are compounds of alkalies (flavoured and with a small admixture of some antiseptic), the rationale of their use being to neutralize the acids formed by fermentation. It is extremely questionable, however, whether they accomplish even this end, for the acid which is formed by the fermentation of carbohydrates lodging against the teeth combines at once whilst in the 'nascent' condition with the lime salts of the enamel: it cannot remain 'free' in the mouth, waiting (as it were) until it is neutralized by the infrequent use of an alkaline dentifrice.

If any acid is neutralized at all it is only that extremely

minute amount which has just previously been formed. The further production of acid is not prevented either; it is rather increased, for, as is well known, a much larger total amount of acid may be developed from a given amount of carbohydrate by occasionally neutralizing the acid formed, thus giving the organisms, as it were, a fresh start. On these grounds alone, then, there is extremely little to recommend the use of alkaline dentifrices.

But moreover, and above all, their use is quite unphysiological. By their use it is taken for granted that the oral secretions are neither sufficient in amount nor alkalinity, therefore a very clumsy artificial substitute is provided. The result is quite physiological; the salivary glands, if they were deficient in secretion before, certainly become more so now.

It of course is a universal physiological law that any organ or organism relieved of its function must atrophy, and this indubitably applies to the salivary glands. It may be shown quite readily that the use of alkaline dentifrices diminishes both the amount and the alkalinity of the saliva. The following table represents a comparison between the amount and alkalinity per minute of saliva before and five minutes after using such dentifrices.

| | *Amount per minute.* | *Alkalinity per minute.* |
|---|---|---|
| Before . . . . . | 1·65 | 1·73 |
| After A. . . . . | 1·0 | ·9 |
| B. . . . . | 1·3 | 1·3 |
| C. . . . . | 1·1 | 1·43 |

A. Dentifrice composed of a mixture of simple alkalies unflavoured.
B. Similar dentifrice but flavoured with oil of cloves and gaultheria.
C. Precipitated chalk alone.

These substances are, therefore, obviously salivary depressants and are on that account to be avoided.

There is, therefore, extremely little to be said in favour of alkaline dentifrices and a very great deal against them. They

must do more harm than good, for it is obviously much more beneficial to have a permanent and normal flow of saliva at maximum alkalinity than a spasmodic, transient, and infrequent high alkalinity followed by a prolonged period of subnormal salivary secretion.

In view of the fact that alkalies of various sorts, from decomposing urine[1] to burnt and powdered bones, have been persistently prescribed for dental caries for hundreds of years amongst civilized peoples, and the annual 'consumption' of proprietary dentifrices must at the present time be enormous, it is not at all improbable that such continual depression of the salivary glands has been in a very large measure directly responsible for the present prevalence of dental disease; it has been an empirical, symptomatic remedy continually applied to a chronic ailment; being such it could not be expected to accomplish much permanent improvement. It has utterly failed to reach the source of the trouble and has undermined the patient's powers of natural resistance. It is quite possible, too, that the daily swallowing of even small quantities of the chalk composing such dentrifices is a factor in the production of chronic constipation.

Compare the above results with those obtained after the use of an acid salt as a dentifrice.

| | *Amount per minute.* | *Alkalinity per minute.* |
|---|---|---|
| Before . . . . . | 1·65 | 1·73 |
| After . . . . . | 1·9 | 2·28 |

In this case acid potassium tartrate was used, and the contrast is most marked and obvious.

Instead of a depression in amount and alkalinity a considerable increase in both is noted.

Here, then, we have a means of physiologically increasing

[1] 'Quod quisque minxit, hoc solet sibi mane
Dentem, atque russam defricare gingivam.'—CATULLUS.

the normal salivary secretion, thus providing a permanent mouth-wash of increased alkalinity. Moreover, the activity of the organisms is not increased by the application of the acid salts, but rather decreased.

It might be urged that it would lead to a softening or erosion of the enamel if a solution of such a salt were used continuously—the author believes not,[1] but even if it did, slight mechanical erosion is infinitely preferable to widespread caries. The most rational procedure in cleansing the teeth is as follows :—

To brush the teeth thoroughly as previously directed, using water only.

To wash the mouth out thoroughly with an acid sweet solution such as the following :—

| | |
|---|---|
| Potass. Tart. ac. . . | gr. ij |
| Ac. Tartarici . . . | gr. j |
| Glusidi . . . . | gr. $\frac{1}{6}$ |
| Sp. vini rect. . . . | m. xx |
| Ol. limonis . . . | m. iij. |
| Aquam ad . . . | $\bar{3}$j. |

The brush also may be dipped in the same solution and the teeth lightly brushed, especially if there is a tendency for stains or calculus to collect upon them.

Afterwards the resulting copious flow of highly alkaline saliva should, by the muscular action of the cheeks and tongue, be forced through the interdental spaces.

During the use of such a mouth-wash—10 c.c. for one minute—the author found that a flow of 4 c.c. of saliva occurred, which diminished the acidity of the mouth-wash from 2·25 to ·65 $\left(\frac{N}{50}\ H_2SO_4\right)$, and that five minutes after use

[1] In the author's laboratory, teeth have been daily *scrubbed* with a saturated solution of acid potassium tartrate for six months without the slightest visible effect being produced.

the alkalinity per minute of the saliva was 3·7, i.e. more than twice 'normal'.

It is in such a manner that stagnation can be broken up and a proper oral circulation established.

**The use of Antiseptics in the Mouth.** It has been previously shown that the presence of certain organisms in the mouth is an essential factor in the causation of dental caries; it might therefore be thought that the continual use of an antiseptic or disinfectant would eliminate this factor and so render the occurrence of caries impossible. As a matter of fact this is not possible, for two reasons: firstly, the mouth is an exceedingly difficult cavity to disinfect, requiring the use of the strongest disinfectants for a comparatively long time; secondly, it is not possible under modern conditions for persons to daily and regularly devote such time to the partial sterilization of their mouths. The perfunctory use of a more or less mild antiseptic is not of the slightest value; even if a very few organisms are destroyed, the remainder very rapidly proliferate and make good the loss. The time during which an antiseptic mouth-wash is used does not amount to more than three minutes in the day at the most, and the effect of the antiseptic is very rapidly lost, that is to say, the organisms are in contact with the antiseptic for three minutes out of twenty-four hours—it would be futile to expect any beneficial result. Not improbably it has the reverse effect—that of producing a more vigorous and resisting strain of organisms due to the *slight and intermittent* opposition to their growth and to the survival of the fittest.

A great many workers have investigated this subject, and all those who have conducted their experiments along rational lines (i.e. by cultures from the mouth direct after the use of antiseptics, and not conducted entirely *in vitro*) have come to the conclusion that it is PRACTICALLY impossible to sterilize the mouth, and that the only drugs of value in reducing for

a short time the number of organisms present are perchloride of mercury and peroxide of hydrogen.[1] The various formic aldehyde preparations, although much vaunted, are comparatively valueless; the growth of organisms is probably retarded whilst the lozenges are being sucked, but three minutes after their use a copious growth can always be obtained from any part of the mouth or from the saliva.

It is therefore clear that for a chronic and comparatively slowly progressing lesion like caries of the teeth, the intermittent (albeit regular) use of antiseptics is of no value. Perchloride of mercury (1 in 2,500) certainly reduces the number of organisms present, but it is not a drug which could be prescribed for general use, and especially not for children, who are very prone to swallow a fair proportion of the fluid which is being used as a mouth-wash. Moreover, it is a very strong salivary depressant, and therefore its continuous use from this aspect would be harmful.

The exhibition of antiseptics in the mouth should be limited to *acute* conditions, and then they should be used frequently and in full strength, i.e. as strong as can be tolerated, with the object of rapidly reducing the number and virulence of the organisms.

Such conditions are *acute* stomatitis, gingivitis, septic dental arthritis.

Antiseptics may also be beneficially used thus both before and after, but chiefly before surgical operations within the mouth—including the extraction of teeth.

[1] For a *résumé* of this subject see the author's *Prevention of Dental Caries*.

# CHAPTER IX

## THE SURGICAL TREATMENT OF DENTAL DISEASE

Caries of the teeth is treated surgically in a number of ways, depending principally upon the stage to which the disease has progressed.

The following are the operations which may be practised:—

1. Excision.
2. Application of drugs and dressings.
3. Excision followed by ' filling ' or ' crowning '.
4. Extraction.

The second and fourth methods are the only ones which will be discussed in detail here.

### 1. Excision

This operation is now confined to the treatment of incipient caries when no loss of substance has occurred. The softened enamel is cut away and the surface remaining is highly polished.

### 2. The Application of Drugs and Dressings

This form of treatment is more or less palliative, the procedure is simple, and therefore being in accord with our present purpose will be described more in detail.

In the treatment of *shallow cavities*, especially those near the gum margin, which are usually extremely sensitive, and may be giving rise to pain or originating a neuralgia of other branches of the fifth nerve, the application of drugs is extremely useful. The most reliable agent is silver nitrate in a solution of 20 grs. to the ounce. It has to be remembered, however, that the cavity will be stained black, and therefore

will become unsightly if it be near the front of the mouth. The rationale of its use is that it destroys the sensitive terminations of the dentinal fibrils, and combines with the organic portions of the dentine to form an insoluble silver albuminate; it is a powerful antiseptic and destroys the organisms *in situ*. Thus the condition is arrested, and recurrence is prevented, or at least considerably retarded, by the albuminate, which is insoluble in lactic acid.

The method of its application is as follows :—The tooth to be treated should be isolated and packed off as far as possible by means of a strip of folded lint or gauze. The cavity is then dried with pledgets of cotton-wool, the silver nitrate is taken up on an extremely small and tightly rolled pledget

Fig. 24. Enamel chisel.

of wool and rubbed on the surface of the cavity, this as a rule causes very little pain. It should be allowed to remain for four or five minutes, when the cavity and the adjoining gum should be well swabbed with a strong solution of sodium chloride to neutralize any free silver nitrate. One such dressing is usually sufficient to stop the pain and relieve the symptoms, it may, however, be repeated with advantage in a few days' time.

Other drugs which may be used similarly are—zinc chloride, zinc oxychloride, chloride of gold and phosphoric acid. These are not so reliable but have the advantage of not staining the teeth. The zinc oxychloride is inserted in the cavity as a paste made by mixing zinc oxide with a solution of zinc chloride, it is somewhat irritant and may give rise to some initial pain.

**The Palliative Treatment of Deep Cavities.** As we have indicated before, it is absolutely essential to make a correct

diagnosis of the condition present and causing the pain before attempting even palliative treatment. If this be not done the result will not improbably be a considerable increase in the pain. (The student is therefore referred to the table giving the differential diagnosis of the more common causes of pain, given on p. 80.)

(*a*) *The Treatment of Dental Myelitis.* The object in the palliative treatment of this condition is to apply a dressing to the pulp which shall be obtundent, antiseptic and protective.

It is necessary if possible to apply the dressing to the pulp itself; it is of no use to place it in a cavity on the top of a

FIG. 25. Right and left spoon-shaped excavators.

layer of decomposing food detritus and septic dentine. The cavity must therefore be cleaned out first of all. This should be done firstly by syringing it with tepid water, then opening it up by means of a chisel (see Fig. 24), and lastly removing the layers of softened dentine with a medium-sized spoon-shaped excavator (see Fig. 25).[1]

The enamel chisel is to be used with a steady pushing motion in a direction at right angles to the surface of the tooth, i.e. in the long axis of the enamel prisms (see Fig. 21); access is thus obtained to the cavity, and all the weakened

[1] These two instruments every medical practitioner should possess. They should be provided in all surgical out-patient departments, and students should, as opportunities offer, familiarize themselves with their use, instead of sending all cases of 'toothache' along to the 'dental department' or perhaps ruthlessly extracting the tooth complained of.

and overhanging enamel is thus to be removed. Particular care must be taken to rest the second or third fingers of the hand in which the chisel is held on an adjoining tooth, otherwise the chisel may slip and do considerable damage to the buccal tissues. The excavator is then to be used in a direction at right angles to the chisel, that is with a scooping or circular motion and the softened dentine flaked out.

Great care must be taken that the excavator does not enter or touch the pulp.

There is nothing difficult or very technical in this procedure and a very little practice will make it quite simple. The cavity being opened up, the dressing is applied. In addition to the properties required in such a dressing as stated above, it must be inserted *without pressure* and should be capable of being retained in the cavity without losing its properties. A mixture of carbolized resin and zinc oxide is one which fulfils all these requirements excellently.

A small quantity of carbolized resin (see Appendix II, No. 2) is placed on a porcelain slab or a plate, and mixed (by means of a spatula or flexible knife) with zinc oxide until a thin paste is obtained, a few wisps of cotton wool are then incorporated in the mixture to give it stiffness, and the whole is then rolled up into a small ball.

The cavity is then dried and the dressing inserted (without pressure); if there is too much the whole must be removed and less inserted.

When the saliva comes into contact with the dressing the resin is precipitated, the spirit is washed away, and the dressing in a few hours becomes more or less hard.

The carbolic acid acts as an obtundent, is antiseptic, and the plug very efficiently protects the pulp from further irritation, the pain being relieved almost immediately.

The only danger is lest the dressing should be allowed to remain in too long and suppuration should occur under it. The patient should always be warned that it will be necessary

to obtain further treatment of a permanent nature within the next few days.

Carbolic acid alone on cotton wool should *never* be used to apply to an exposed pulp. It relieves the pain, certainly, but it also forms a dense coagulum on the surface of the pulp through which it is difficult afterwards to get another drug like arsenious acid or cocaine to penetrate, when it becomes necessary to undertake permanent treatment by extirpating the pulp.

This coagulating effect of carbolic acid is entirely prevented by mixing it with equal parts of thymol (or with resin as above).

Oil of cloves and creasote are popular remedies and certainly temporarily relieve the pain, but they are rapidly removed from the cavity and the pain returns.

If purely liquid drugs are used they should be sealed in the cavity by means of ' cement ' or gutta-percha, but since the manipulation of these materials requires some experience it is probable that the general practitioner will not find them very useful.

(*b*) *The Palliative Treatment of Periodontitis.* If it is certain that this condition is present, and if suppuration has not yet occurred, the procedure should be as follows. The cavity should be opened up as before, but very thoroughly in this case, taking care to open well into the pulp chamber, syringe the cavity out well with warm water, and if possible clear out the decomposing pulp from the root canals with a fine barbed bristle (this, however, is usually a somewhat difficult operation and may be left for the dental surgeon to deal with). *Nothing at all should be placed in the cavity,* and the patient must be instructed to keep it quite free from food debris.

The whole object of such palliative treatment is to afford a means of escape for the products of inflammation at the apex of the tooth.

In addition, counter irritants may be applied to the mucous membrane over the affected tooth; the most useful drugs being the tinctures of iodine and capsicum.[1]

These should be applied on a small piece of cotton wool wound tightly round a clean match stick and thus rubbed well into the mucous membrane on the inner and outer sides of the tooth. When the condition has subsided somewhat (or even at first if there is no acute pain) a dressing of formalin 10 per cent. solution may be *sealed* in the cavity. The formalin must not be allowed to escape into the mouth: because firstly, it is extremely irritant to the tissues; and secondly, if there is a means of escape into the mouth the liberated formic aldehyde gas will not pass through the apical foramen, and cannot destroy the organisms in the periodontal tissues. If the organisms remain, and at the same time the cavity is partially blocked, the condition will in all probability again become acute. Formalin may be most conveniently sealed in by means of oxyphosphate of zinc cement.[2]

When pus is formed, or is about to form, at the apex the above treatment will probably be inefficient. The tooth may be so tender that it is impossible to open up the cavity in the ordinary way.

Recourse must then be had to heat in the form of hot fomentations to relieve the pain.

*The fomentations must always be applied inside the mouth, and on no account to the outside of the face,* because the abscess then tends to point on the exterior of the face, and the cicatrix which afterwards forms binds the healed sinus down tightly to the bone.

The latter practice is popular but leads to unsightly,

[1] For formulæ see Appendix II, No. 4.

[2] Obtainable at all wholesale dental supply houses; it consists of a powder (zinc oxide) and a liquid (phosphoric acid). These are mixed together into a paste which becomes hard in a few minutes.

disfiguring, and permanently depressed scars. Hot antiseptic mouth-washes may be prescribed, or tightly rolled pieces of lint soaked in the hot antiseptic may be held in the mouth in the buccal sulcus against the affected tooth. The popular bread poultice is an effective means of conveying heat to the muco-periosteum and alveolus.

In using it the patient should be instructed to make the little poultice first with bread softened in cold water, folded in thin muslin, and afterwards to soak it in hot water, otherwise all the heat is abstracted from the bread in the process of making up the poultice.

Another useful form of fomentation is a decoction of poppy heads—useful because it keeps the patient warm, gives him something to do and something to think about, all of which are more valuable therapeutically than the minute amount of opium which will be absorbed by the mucous membrane of the mouth. The patient is to be instructed to obtain six poppy heads, to place them in a quart of water, and to watch this very carefully simmer down to a pint *exactly*. It is then to be strained, and the fluid portion heated again if it has become cold and used as a mouth-wash, i.e. portions of it as hot as possible are to be held in the mouth around the affected tooth, and this is to be repeated as often as possible. In addition to the fomentations, if much pain be present and pus seems to be on the point of forming, a deep incision in the labiodental sulcus will considerably and rapidly relieve the tension and pain.

(*c*) *The Treatment of Alveolar Abscess.* The treatment of an abscess arising from a septic tooth does not differ in any respect from the treatment of an abscess elsewhere in the body; there is only one principle—*open and drain immediately*. The method and situation of the opening may, however, vary in different cases.

When the abscess has not reached large dimensions the

dental surgeon nearly always prefers to obtain drainage through the apical foramen by enlargement of the latter, instead of making an incision into the soft tissues.

This, however, requires special knowledge and training in the use of the particular drills to be used for the purpose. When a distinct fluctuating swelling is present it may be most advantageously drained by an incision into the muco-periosteum, which should always be carried down to the bone. A small and very narrow piece of gauze should be inserted for a day or two to keep the incision open. In these cases immediate and intense relief is experienced by the patient as soon as the pus finds an exit.

If the abscess appears to be pointing towards the outside of the cheek when the patient is first seen, the reddened and hyperæmic area should be well painted with collodion flexile, and if necessary a small piece of gauze may be stuck on with the collodion to further strengthen the skin and prevent its perforation. An incision should then be made in the buccal sulcus opposite the 'pointing' on the exterior, and hot fomentations prescribed to be used *inside* the mouth.

When the abscess has tracked down below the level of the lower jaw, incisions within the mouth are of no avail. The abscess must be opened at its most dependent part; the opening should be as small as is consistent with the size of the abscess and be made in a line with the branches of the seventh nerve, the facial artery being avoided; a drain of gauze may be inserted through the fibres of the platysma to keep the wound open. In these latter cases only is it permissible to apply hot external fomentations.

In all cases of alveolar abscess the original source of the trouble—a septic tooth—must be treated as soon as possible; unfortunately this is too often forgotten, and what should be an acute condition and healed up in a week or ten days drags on, becomes chronic, and then is most difficult to treat successfully. If the tooth is much broken down, or is

useless from a masticatory or æsthetic point of view, it should be extracted at once.

It by no means follows, however, that because a tooth is the cause of an acute alveolar abscess, it cannot be made both healthy and useful in the future, provided the proper treatment (the sterilizing and filling) of the root canals be undertaken as soon as possible.

The question as to whether a tooth should be extracted or treated conservatively when it is associated with an acute alveolar abscess is one for which no definite or hard and fast rules can be laid down.

On the one hand a large number of deaths from alveolar abscess are on record, many of the fatal results being due to the retention of the infected tooth; whilst on the other it is equally certain that many teeth are quite needlessly sacrificed in the treatment of alveolar abscesses.

The extent, position, and acuteness of the abscess, the condition of the patient—temperature, pulse, age and tissue resistance—the usefulness or otherwise of the tooth, must all be taken into account in deciding on the treatment of the tooth—as also of course must the social position of the patient and accessibility of a dental surgeon for the treatment of the root canals.

In any case there is not the slightest excuse for the old idea of postponing active treatment ' until the swelling has subsided '.

The treatment of chronic alveolar abscess is scarcely likely to come under the notice of the general medical practitioner, because it does not give rise to acute pain, and its treatment is essentially very technical. The following are the chief methods available for the treatment of the condition :—

(i) Forcing strong antiseptics from the tooth through the sinus by means of a hypodermic syringe.

(ii) Injection of bismuth paste into the sinus.

(iii) Amputation of the apex of the tooth and excision of the fistula.

(iv) Extraction of the tooth, curettage of the socket and sinus followed by reimplantation. (The tooth being root-filled, sterilized, and secured by means of a splint.)

It is, however, essential that the presence of chronic alveolar abscesses should be recognized and effectively treated, since they are potent and frequent causes of oral sepsis and its many secondary systemic disorders.

### 3. The Restoration of Lost Dental Tissue by means of Fillings, Crowns, and Bridges

So much attention has been paid by dentists to the development of this branch of work that only teeth which are hopelessly loose and septic are now regarded as being unfit for some form of restoration.

For obvious reasons it would serve no useful purpose to discuss the subject in detail here.

The various methods, however, may be briefly indicated with advantage.

The operation of Filling consists of the following :—

(i) The excision of infected and weakened tissues.

(ii) Further extension of the cavity in order to obtain self-cleansing margins and retention for the 'filling'.

(iii) Sterilizing the cut surfaces.

(iv) Restoration of the lost tissues by means of some artificial substance such as porcelain, gold, amalgam (an alloy of silver and tin or copper with mercury), oxyphosphate of zinc, or gutta-percha.

There is a popular impression that filled teeth are bound to ' ache ' later on. Such an occurrence means usually that the caries has been imperfectly removed or the cavity not sterilized—if these operations have been properly carried out

there is no reason why a filled tooth should not last as long as a normal ' unfilled ' tooth.

When the natural crown of a tooth has been completely destroyed by caries it may be restored by the fixation of an artificial crown, which is made either of gold or platinum and porcelain. The crown is made to fit and grip the root either by means of a metal post passing up the root canal, or by a band or collar encircling the root, or by a combination of both methods.

In any case the crown is fixed to the root finally by means of oxyphosphate of zinc cement.

Because a tooth has been septic or has originated an alveolar abscess is no reason why it should not be crowned after all sepsis has been treated and eradicated ; but if absolute sterility cannot be obtained, or if the tooth or root does not become reasonably firm under treatment, the operation of crowning cannot be considered.

If a small space exists between two carious teeth (caused by previous extraction), the two teeth may be crowned and the space 'bridged' over by joining 'dummy' crowns to the two abutment crowns at either end. This constitutes what is known as a ' bridge '.

Both crowns and bridges require the greatest care in construction, and have in the past been much abused by certain classes of dental practitioners, owing either to their being fixed to septic teeth or to their originating an unhygienic or suppurative condition after fixation.

Bridges may be made either fixed or removable, and as a general rule it may be said that removable bridges are much more hygienic.

# CHAPTER X

## THE SURGICAL TREATMENT OF DENTAL DISEASE (CONTINUED)

### The Operation of Extraction

**Indications for the Operation.** It may be said that the extraction of a tooth should be only undertaken as a last resort or when the patient's general condition demands it.

It is not justifiable to extract a tooth simply because it is aching and the patient perhaps wishes under the stimulus of the pain to have it extracted.

If, however, teeth are the cause of oral sepsis and no other immediate treatment is possible, extraction becomes not only justifiable but absolutely necessary.

It is better that a patient should suffer loss of masticating power, and have a clean mouth, than that he should perhaps be able to masticate food to a certain degree, but his food be always infected with pus and organisms.

A similar question often arises in connexion with the treatment of deciduous teeth. The too early extraction of deciduous teeth undoubtedly leads to future deformity when the permanent teeth erupt ; on the other hand the retention of septic deciduous teeth leads to enlarged cervical glands and general toxic absorption. It becomes necessary to choose the lesser of two evils, and undoubtedly extraction of the deciduous teeth under such conditions is the less harmful to the child. It is one of those conditions where the general medical practitioner's opinion is sometimes of more value than the dentist's.

A deciduous tooth should not, however, be extracted if

possible before the age at which its permanent successor should erupt, and it should not be allowed to remain much after this period without due reason; it is, therefore, eminently necessary to remember the numbers

7. 8. 11. 10. 11.

which represent the ages at which the respective deciduous teeth should be lost physiologically.

**Selection of Forceps.** Before attempting to extract any tooth it is necessary to have an exact idea of the shape and number of the roots of the tooth to be extracted; for this reason attention is directed to Fig. 3, which should be committed to memory, and should be able to be visualized at any time. Having realized the shape of the neck of the tooth to be removed, the correct forceps are selected and sterilized by boiling.

Provided there is a full set of forceps to select from, as there should be at least in all hospitals, a forceps is chosen whose blades may be accurately adapted to the cervix of the tooth.

For all teeth except the molars there is a single groove on each blade, for upper molars the outer or buccal blade has two grooves corresponding to the two buccal roots, and the inner or palatine blade has one broad groove for the palatine root; there are, therefore, left and right forceps for these teeth.

The forceps for lower molars have two grooves on each blade, and come to more or less sharp points between the grooves to enable a grip to be obtained of the inner and outer surfaces of the anterior and posterior roots. (Fig. 35.)

The forceps for upper incisor and canine teeth are straight throughout their length (Fig. 26), those for upper premolar teeth are slightly curved in order to avoid the anterior teeth and to enable force to be applied in the direction of the long axis of the tooth. (Fig. 27.)

The forceps for lower incisor, canine, and premolar teeth have single grooves on the blades, the latter being placed at right angles to the handles. (Fig. 28.)

Where the number of forceps is limited these should consist of upper incisor forceps, upper premolar forceps, and lower premolar forceps. (Figs. 26, 27, 28.)

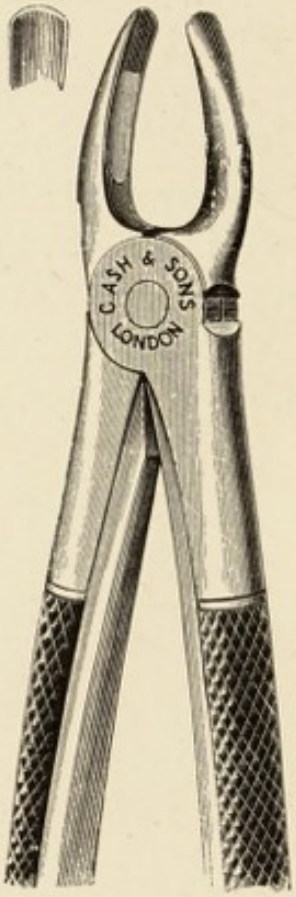



Fig. 26. Forceps for upper incisor and canine teeth.

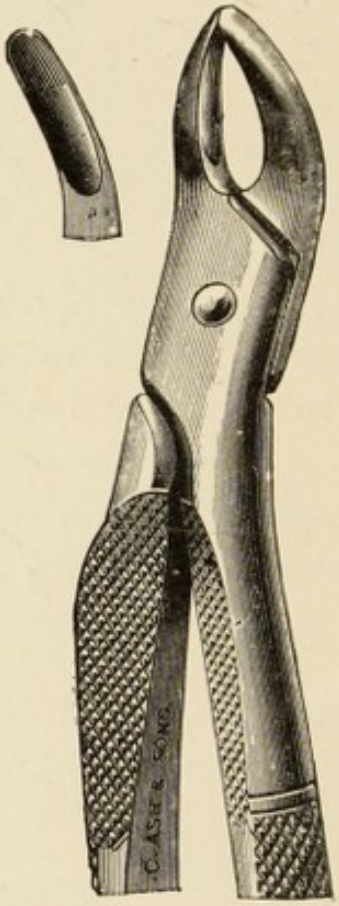


Fig. 27. Forceps for upper premolars and molar roots.

With these three pairs of forceps almost any tooth which it is necessary to extract can be extracted. It is becoming increasingly uncommon to have to use 'full' molar forceps, since these are adapted for use when the crown of the tooth has not been destroyed, and such teeth are as a rule treated conservatively; or if they are to be removed for a septic periodontitis or arthritis they are usually so loose that the premolar forceps are all that is necessary.

**Position of the Patient.** This is an exceedingly important matter, since upon its correct observance largely depends the success of the operation.

The following rules must always be observed :—

(i) The patient's body and head must be so supported that shrinking away from the forceps as force is applied becomes impossible.

(ii) In the extraction of upper teeth the patient's mouth must be above the operator's elbow line, and in the extraction of lower teeth the patient's mouth must be below the operator's elbow line.

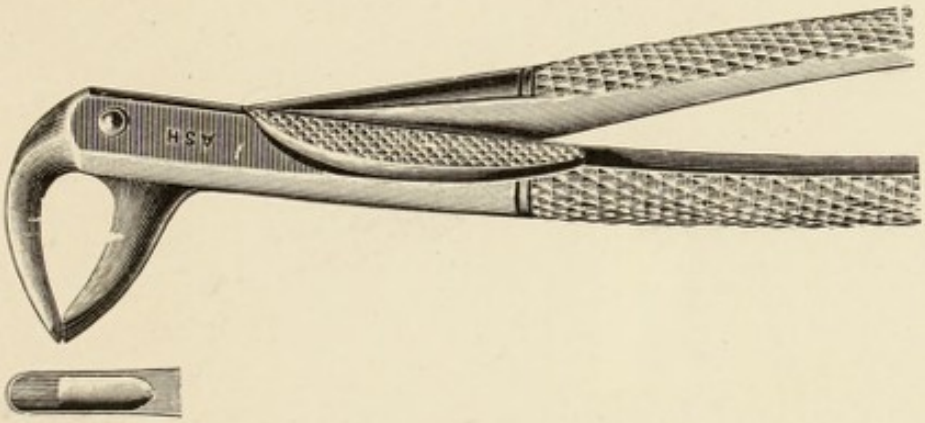


FIG. 28. Forceps which may be used for the extraction of all lower teeth.

With a modern dental operating chair such positions are obtained at once by manipulating a few levers, but the medical practitioner or the student in hospital has to utilize whatever chair may be available. A fairly solid and not too low an ' arm-chair ' makes a good workable substitute for a dental chair, if it be used judiciously according to the following plan.

In the extraction of *lower* teeth the patient tends to sink *down* into the chair. To prevent this the chair is placed in front of a window and so close to it that the patient's knees are touching the wall and cannot move forwards; that being so, the head cannot sink down in the chair (Fig. 29); if the patient's head can then be supported in the occipital region

so much the better, but this is not so important as the fixation of the knees.

If, owing to the tallness of the patient, the height of the chair, or the shortness of the operator, the patient's mouth is not then below the operator's elbow line, the operator must stand on a stool—improvised or otherwise. (Fig. 29.)

FIG. 29. Position of patient and operator in the extraction of lower right teeth. For the extraction of lower left teeth the operator stands on the patient's right and facing him.

In the extraction of upper teeth the patient's head tends to travel upwards and backwards. To control this the back of the chair should be placed *nearly* against a wall facing the light. The stool or cushion should then be placed in the chair and the patient seated thereon in order to bring the mouth above the operator's elbow line. The head should be tilted backwards and upwards, so that the vault of the

cranium is opposed firmly to the wall (or in the case of children to the back of the chair). (Fig. 30.)

By these simple means almost under any conditions (even if a tree trunk or a tent pole has to be utilized instead of

Fig. 30. Position of patient and operator in the extraction of all upper teeth. Note the patient is now sitting on the cushion and his feet are off the ground.

a wall) a patient's head may be maintained stationary during the extraction of any tooth.

**Position of the Operator.** For all *upper* teeth the operator stands to the right and in front of the patient.

For *lower* incisors and lower left premolars and molars the operator's position is the same.

For *lower right* premolars and molars the operator stands *behind* the patient.

The operator should keep his head well back from the patient, so that the light is not obstructed and a clear view is obtained of the tooth to be extracted.

**Disposition of the Fingers of the Left Hand.** Before putting the forceps inside the mouth, the 'field of opera-

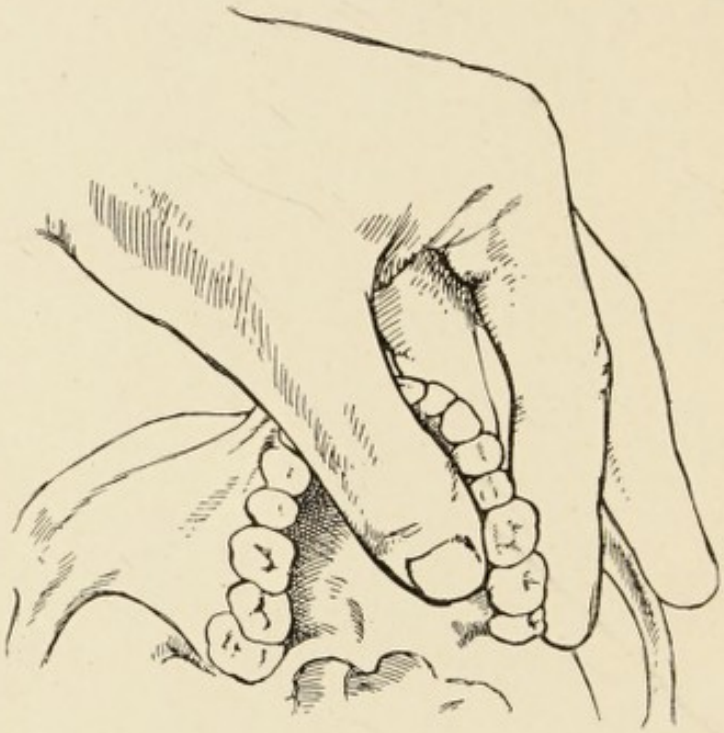


Fig. 31. Position of the thumb and fingers of the *left* hand in the extraction of upper teeth.

tion' has to be cleared, defined, and guarded by the fingers of the *left* hand.

The first finger and thumb should be placed on either side of the tooth in the upper jaw as is shown in Fig. 31.

For lower *left* teeth the first and second fingers are placed on either side of the tooth, and the thumb under the jaw to support it and make upward pressure (see Fig. 32).

For lower *right* teeth the first finger and thumb are placed within the mouth, and the remaining fingers support the jaw underneath, as is shown in Fig. 33.

The function of the fingers of the left hand is to keep the lips, cheeks, and tongue away so that a clear view can be

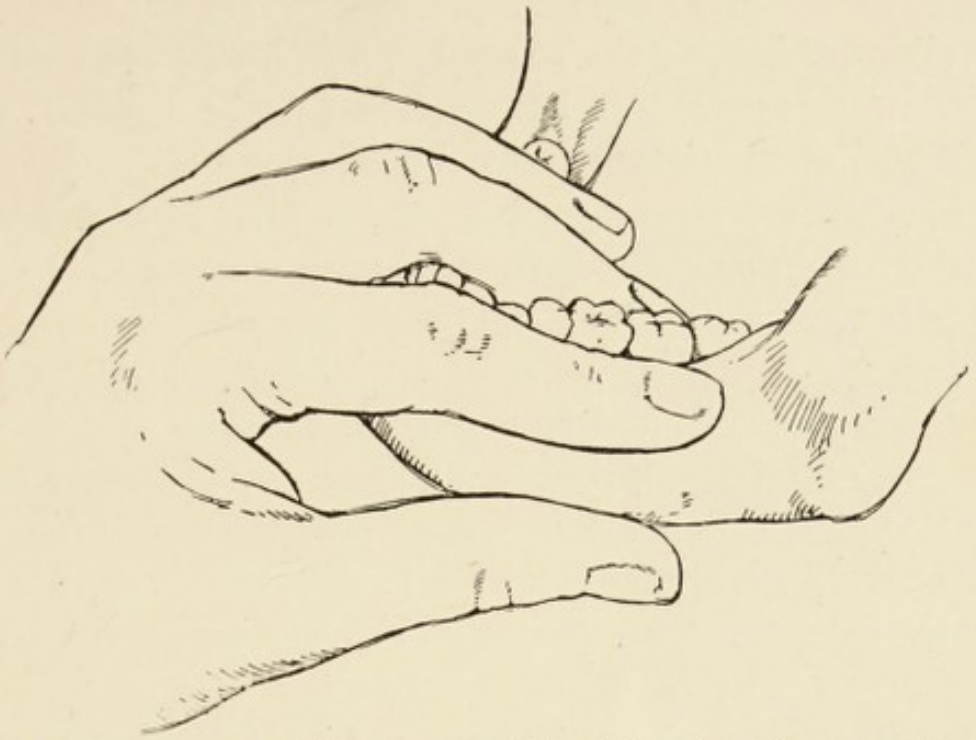


FIG. 32. Position of the fingers of the *left* hand in the extraction of lower *left* teeth.

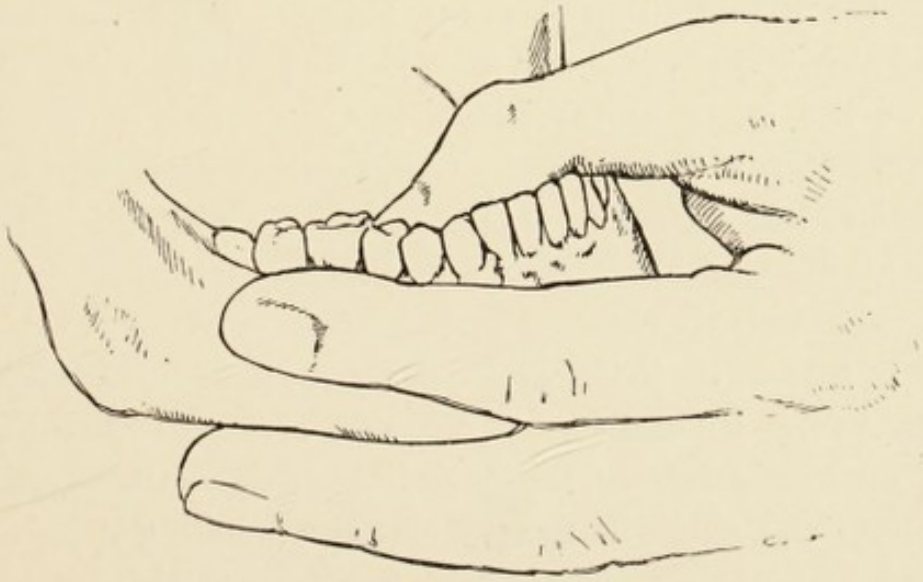


FIG. 33. Position of the fingers and thumb of the left hand in the extraction of lower *right* teeth. (The operator standing behind the patient.)

obtained to apply the forceps, and, secondly, to be prepared to prevent the tooth passing into the larynx or œsophagus

should it accidentally leave the blades of the forceps; it is, therefore, highly important that the fingers of the *left* hand should be correctly disposed.

**Method of holding the Forceps.** All forceps are held in a similar manner. The handles are grasped between the lower part of the thumb and the first three fingers, the ball of the thumb is placed between the handles as high up as possible; by this means undue squeezing or closing pressure on the handles is avoided ; the fourth finger is kept between the handles low down in order to open the blades when necessary. Figs. 34 and 35 explain graphically, and better than any verbal description, how forceps should be held. In the extraction of upper teeth *upward* pressure is made on the ends of the forceps handle resting in the palm of the hand.

In the extraction of lower teeth *downward* pressure is made with the ball of the thumb on the lower handle of the forceps.

**The Application of the Forceps and the Removal of the Tooth.** The fingers of the left hand being correctly disposed, the lingual or inner blade of the forceps is applied first to the tooth, followed by the outer blade. Strong upward or downward pressure is made in order to get a firm grip of the root of the tooth as far below the gum line as possible. The next step is to loosen the tooth ; all teeth except upper incisors are loosened in their sockets by firm inward and outward movements or rocking ; the upper incisors alone are rotated slightly through one-eighth of a circle. When the tooth is felt to be loose, *and not before*, it is to be steadily removed from its socket. In the case of molar teeth this is done, not by pulling either upwards or downwards, but rather by rolling the tooth outwards through about a quarter of a circle, and using the thumb or finger on the outer side of the tooth as a fulcrum.

The same method modified applies to all teeth except upper incisors and canines, which are removed in a downward direction.

Rolling the tooth outwards of necessity (and purposely) fractures the outer wall of the alveolus; this is of no consequence, but the bulged-out fragments should be immediately compressed with the thumb and finger of the left hand in order to lessen the surface available for possible infection, to lessen hæmorrhage, and to induce the formation of a

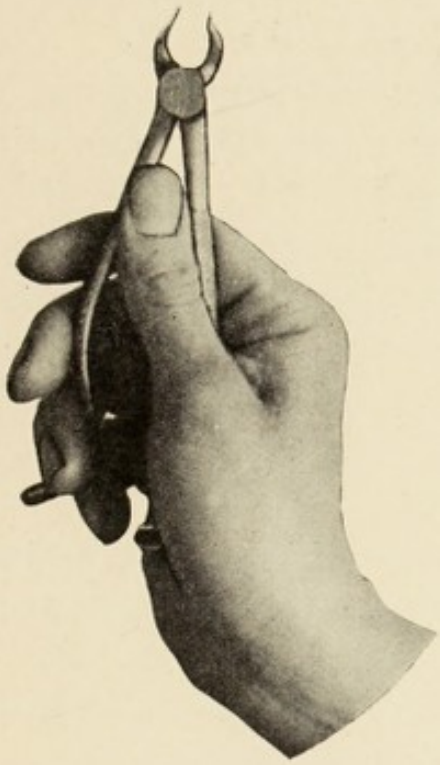


Fig. 34. Method of holding forceps for the extraction of upper teeth. (A left upper molar forceps is illustrated.)

Fig. 35. Method of holding forceps for the extraction of lower teeth.

shapely alveolar ridge for the subsequent fitting of artificial dentures.

There is one *exception* to the above rule; owing to the presence of the external oblique line of the lower jaw, lower third molars or wisdom teeth cannot be rolled outward, but must be taken rather in an inward direction.

The essential point in the extraction of all teeth is to make quite certain of having a firm grip of the root, and that it is loose before attempting to remove it.

It sometimes happens that in attempting to extract a tooth one root is difficult to seize firmly with the forceps. Under such conditions recourse may be had to the 'elevator'. These are made with left and right blades. The suitable one being chosen, it is passed down the empty socket and rotated in such a manner that the root remaining in the other socket is elevated from below; this method is very simple and efficacious and rarely fails.

In all but quite simple extractions the patient should be ordered a hot antiseptic mouth-wash (see Appendix), to be used frequently for at least two days.

**Sequelæ.** These may be hæmorrhage, pain, sepsis, necrosis, and there may be many other complications, more or less infrequent, which cannot be discussed here.

**Hæmorrhage.** The general practitioner is perhaps more often called upon to treat this condition than the dental surgeon, frequently at inconvenient hours, hence it will be well for him to be prepared at a moment's notice to be able to simply and satisfactorily arrest the bleeding from a tooth socket when required to do so.

**General Directions.** Unless the patient has lost much blood and is very pallid, he should be directed to sit up and not lie down. Fluids either very hot or very cold should be held in the mouth, and any nourishment taken should be at a similar temperature. If this does not arrest the bleeding the socket must be plugged. The simplest and most efficacious method of doing this the author has found is to make a cone-shaped plug by mixing carbolized resin, zinc oxide, and cotton-wool together in the manner already described for dressing a case of myelitis.

The socket is then temporarily cleared of blood with another plug of cotton-wool, and the carbolized resin plug rapidly and firmly inserted; it causes a little pain at first, but this soon subsides.

As a rule this is all that is necessary, and the bleeding

should cease, or almost so, in a few minutes. Should it not do so, however, it means that the plug is being forced up out of the socket by the pressure of blood below; in order to counteract this a thick pad of lint must be placed over the plug, and the lower jaw firmly compressed upon this by means of a four-tailed bandage; this, however, always causes considerable pain to the patient.

If carbolized resin is not available simply cotton-wool or lint may be used—preferably soaked in some antiseptic. 'Adrenalin' is of no value in such cases; ergot is sometimes recommended, but its use either internally or locally is quite unphysiological, and it may be worse than useless, since it has no special local action on the blood-vessels at the bleeding-point, but raises the blood-pressure and so may increase the hæmorrhage.

Ferric perchloride arrests the bleeding well, but forms a slough which, when it separates, is likely to lead to secondary hæmorrhage.

In hæmophilic cases the patient should be given full doses of calcium chloride (or lactate) for several days prior to the extraction. In severe cases of this nature continual digital pressure may be the only means of absolutely controlling the bleeding.

If the patient is collapsed from previous hæmorrhage, as soon as the socket is plugged the usual surgical measures for the condition (injection of saline, &c.) should be immediately undertaken.

The advantages of the carbolized resin plug are that it is simple, the plug is antiseptic and analgesic, it accurately adapts itself to the wall of the socket and therefore to the bleeding-point, it becomes hard *in situ*, and is not painful to the patient (if the four-tailed bandage can be dispensed with). The plug usually comes away of its own accord on the second or third day, but if it does not it should be removed very carefully.

**Pain** may be caused either by infection of the socket, injury to a nerve, or the too rapid healing of the surface of the wound.

In an infected socket there may or may not be pus; in the event of suppuration being present the wound should be syringed out frequently with a hot antiseptic solution and the patient directed to use poppy-head fomentations. With an infected and 'dry' socket the pain is usually very severe, and this condition most frequently arises after the injudicious use of a local anæsthetic.

Treatment should be directed towards promoting bleeding, either by warm fomentations or by scarifying the bone of the socket; unless bleeding can be induced and the socket filled with blood-clot necrosis will almost certainly follow; should this appear likely incisions should be made in the gums, and warm antiseptic mouth-washes used continuously; the patient should be kept in the warm, and a sharp saline purgative given.

A great deal of pain is not infrequently caused by the cellulitis of the jaw and face, which follows the extraction of many septic teeth. This can practically always be avoided by the use of strong disinfectant mouth-washes used with a tooth-brush *prior* to the operation.[1]

Injury to a nerve is most likely to happen in the extraction of a lower third molar whose roots are frequently grooved by, and sometimes perforated by, the inferior dental nerve. Cutaneous anæsthesia usually follows in the region of the mental foramen.

To relieve the pain in such cases nothing is better than a pledget of cotton-wool soaked in Glycerinum carbolici placed in the bottom of the socket.

[1] In my general hospital work I frequently have to operate on extremely septic mouths, but post-operative cellulitis practically never occurs because all my patients use a hot mouth-wash of perchloride of mercury, 1 in 2,500, for three hours before the operation.

In severe cases of laceration transverse incision of the nerve may be necessary.

Pain may be due also to the early healing of the surface when the socket has become rapidly closed by unhealthy looking granulation tissue. The perforation of this with a small scalpel at once reveals the presence of pus or decomposing blood-clot and relieves the pain.

To prevent its recurrence a crucial incision should be made and the four triangular pieces removed with a fine scissors. A small piece of gauze should be left in the wound to act as a drain and to keep it open.

# CHAPTER XI

## FRACTURES AND DISLOCATIONS OF THE JAWS

### Fractures

Fracture of the lower jaw is more common than that of the upper and generally results from direct violence, although one case has been recorded of fracture of the lower jaw occurring during a violent fit of coughing. Probably the most frequent cause of a fractured mandible is the forcible 'argument' of another man's fist, and on this account the treatment of such patients is likely to be unsatisfactory from several points of view. Other causes are kicks from a horse, crushing by the wheel of a vehicle, falls from a height, and gunshot wounds.

Previously, when the 'key' was the instrument used for the extraction of teeth, fractures of the jaw from the application of excessive force were not uncommon.

The alveolus and the maxillary tuberosity are occasionally severely fractured even now during the extraction of teeth, usually when a chronic osteitis has been present.

Nearly all fractures of the horizontal ramus are compound into the mouth, and comminuted fractures are common after severe injuries such as a kick from a horse or a gunshot wound.

**Fractures of the Horizontal Ramus** occur in the following situations and in the same order of frequency.

1. Between the canine and first premolar, owing to the depth of the canine socket weakening the bone at this spot.
2. At the mental foramen (for similar reasons).

3. At the symphysis, usually said to occur in the young (but in two cases treated recently by the author the fractures were in this situation in adult men of particularly strong physique).
4. In the region of the last molar.

Nearly all fractures of this ramus are slightly oblique, except at the symphysis, when they may be vertical or T-shaped, the cross of the T running along the apices of the teeth.

**Fractures of the Ascending Ramus.** These are less common and are usually simple; they result as a rule from direct injury or from a fall from a height upon the chin.

The usual situations of the fractures in their order of frequency are :—

1. At or just above the angle.
2. At the neck of the condyle.
3. Across the coronoid process (such cases are extremely rare).

### Signs and Symptoms

**Horizontal Ramus.** In simple fractures there is no displacement, since the slightest displacement makes the fracture compound, the sharp ends of the bone readily penetrating the muco-periosteum of the mouth. The patient can speak well and eat to some extent, but there is pain and a sense of mobility at the seat of fracture. Slight crepitus and visible mobility may be obtained; this should be tested for in a vertical direction, and not horizontally, for fear of lacerating the muco-periosteum.

In compound fractures there is always a definite history of severe injury, though the patient may be unable to give this on account of his inability to speak at all distinctly. The patient is unable to use or move the jaw and may be supporting it with one hand. There is a considerable amount of pain and swelling, due to extravasation at first and to sepsis later. All the usual signs of a fracture are present.

The *displacement* occurring in compound fractures in the region of the canine teeth is as follows :—

The smaller (or posterior) fragment is drawn upwards and outwards by the temporal and masseter muscles, and also forwards by the external pterygoid muscle.

The larger or anterior fragment is drawn downwards and backwards by the digastric, geniohyoid and geniohyoglossi muscles.

The result of this is that the ends of the bone overlap more or less, and when the patient attempts to close the jaws the incisor teeth do not meet the upper teeth and are obviously ' too far back ' in the mouth ; the lower premolar teeth also tend to articulate *outside* the corresponding upper teeth. (Fig. 36.)

There is one exception to the rule of the incisor teeth being ' obviously too far back '. In the case of patients with an underhung jaw or ' inferior protrusion ' the result of such a fracture may be to make the incisor teeth articulate in an apparently normal manner, as happened in one of the author's cases. Such a condition is very puzzling at first, and unless it is recognized may lead to the fracture being put up permanently in a wrong position.

In bilateral compound fractures the lateral fragments are both drawn upwards, the central fragment is retracted downwards and backwards and tends to be rotated somewhat upon itself.

In comminuted fractures in the region of the symphysis with, as is usual, loss of incisor teeth, both halves of the bone tend to be rotated inwards toward the middle line, so that the lower premolar teeth on *both* sides articulate too far *within* the corresponding upper teeth.

**Vertical Ramus.** The chief symptoms of fractures in this region are considerable swelling and pain, the latter most marked on attempting to use the jaw.

There is no very obvious increase of mobility, and owing

to the wide attachment of the masseter muscle there is little displacement. The only reliable sign is crepitus. In order to obtain this the condyle and coronoid process should be fixed with the fingers of one hand against the temporal bone, whilst the other hand manipulates the horizontal ramus; this, however, is not easy on account of the dense and brawny swelling usually present.

Fractures of the *neck of the condyle* are usually caused by a fall from a height upon the chin and may be bilateral, but

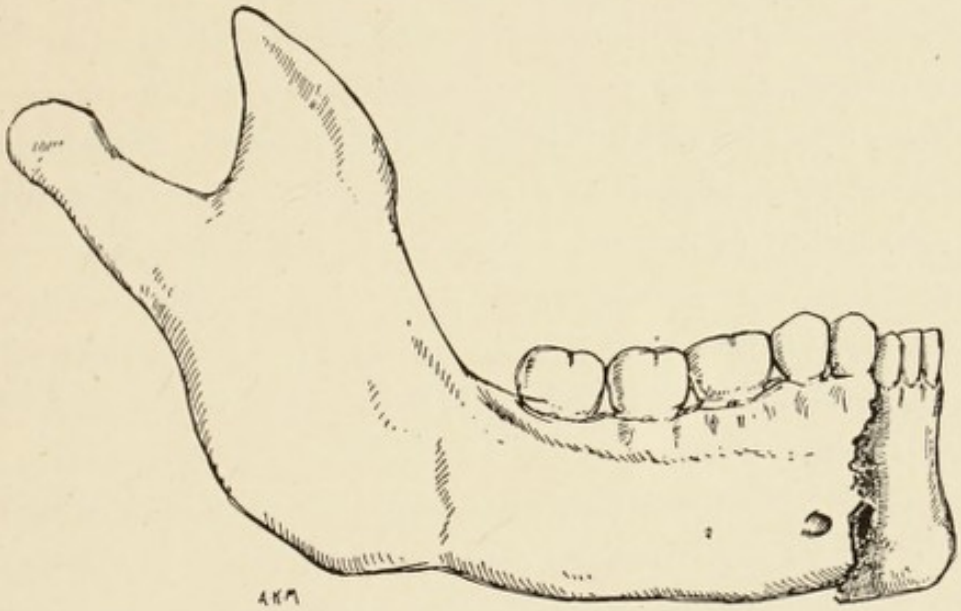


Fig. 36. Fracture of the lower jaw in most common situation, showing the usual type of displacement.

this is rare. There is considerable pain and difficulty in moving the jaw, *the chin is deflected towards the affected side*, and the patient may be able to detect crepitus; this is difficult for the observer to obtain, but pressure over the condyle is always painful.

In a fracture of *both condyles* the whole of the lower jaw is greatly retracted.

**Fractures of the Upper Jaw.** These are always due to the application of considerable violence. The most common forms of such fractures are :—

1. Horizontal fractures along the apices of the incisor teeth.

2. Horizontal fractures along the apices of premolar and molar teeth.
3. Medial fractures along the suture between the two maxillæ (and palatal bones).
4. In severe crushes the nasal or malar process may be fractured.

In horizontal fractures the displacement is always in the direction of the force applied, and this is nearly always inwards towards the centre of the mouth. There is no secondary displacement due to the action of muscles. The fragments may be quite loose, and in cases where the fracture has occurred during the extraction of a tooth the fragment has been brought away bodily with the tooth. This is liable to happen more especially at the maxillary tuberosity during the extraction of a third molar when the joint has become disorganized and a subsequent sclerosing osteitis resulted.

Excessive thinness of the bone round the lower part of the antrum may also conduce to this accident in the case of other molar teeth.

In medial fractures there is commonly very little displacement; the patient notices a sense of 'giving' in the centre of the palate when the upper teeth are pressed upon. Mobility and crepitus are obtained by grasping the premolar teeth on either side and manipulating the bones in an antro-posterior direction. Occasionally the muco-periosteum in the centre line is also ruptured and a visible space exists between the two bones.

Neither severe *hæmorrhage* nor *paralysis* of the inferior dental nerve are common accompaniments of fractures of the lower jaw, as might be expected to occur from laceration of the contents of the inferior dental canal. This is chiefly due to the fact that most fractures with marked displacement occur anterior to the mental foramen.

Neuralgia occasionally occurs subsequently owing to implication of nerves in the callus.

Since the majority of fractures of the jaws are compound, *suppuration* nearly always occurs. This as a rule subsides soon after the fragments are immobilized, but occasionally considerable *necrosis* of the ends of the bone has resulted, due to prolonged suppuration.

This is more likely to occur in the lower than in the upper jaw, since the latter is more vascular, although naturally it depends a great deal upon the degree of the original injury and the method of treatment, and also upon the resistance of the patient. Illustrating the latter point, Heath recorded that two patients were once admitted to hospital within a few hours of each other, in both of whom the superior maxillæ were fractured and freely movable. One patient made a good recovery, in the other *death* ensued; a post-mortem examination showed that there had been no injury to the base of the skull.

**Treatment.** In the treatment of fractures of the jaws the first object of course is to remedy any displacement and to immobilize the fragments.

As regards reducing the displacement; if this is at all marked, and if some form of interdental splint is to be applied necessitating the application of some force, which would be very painful, an anæsthetic is necessary; this is not the case, however, with simple fractures.

It is to be remembered that the lower jaw is a bone which the patient is constantly moving, unconsciously or otherwise, that particularly strong muscles are attached to it, but nevertheless it is a bone which can be kept quite still without the least interference with the general health of the patient. The presence of the teeth in the jaw-bones offers the means of obtaining a very secure control over the fragments such as is not possible with any other bone in the body. The teeth also serve as guides to the amount of displacement, and for the correct alignment of the fragments in a more exact manner than can be obtained in fractures of other bones.

In treating fractures of the jaws the object to be attained should be to restore full function to all the teeth by ensuring their correct or normal occlusion. If this can be done without undue pain or the use of any clumsy or unsightly apparatus it will be of distinct advantage to the patient, since with fractures of the jaws it is not necessary for a patient to stay either in bed or indoors, but he may, within reasonable limits, go about his ordinary vocation after the first week or ten days in uncomplicated cases.

## Treatment

In order to save repetition it will be convenient to describe the various methods and appliances which may be used, and to indicate for what variety of fracture each is most suitable. The methods are divisible into five classes.

1. External bandaging.
2. Interdental splints.
3. A combination of both.
4. Interdental and intermaxillary lacing.
5. 'Wiring' the fragments.

1. **External Bandaging.** This is one of the most ancient methods of treating fractures of the jaw, a form of a four-tailed bandage being mentioned by Celsus as a recognized method of treatment in the second century A.D., and it should now be considered as of historical interest chiefly.

Its only claim to persistence is simplicity, but this is in nearly all cases at the expense of efficiency.

Its disadvantages are :—

(*a*) The displacement in compound fractures of the horizontal ramus is not controlled, and may be exaggerated or perpetuated, since the chin is always drawn backward, and this is in the direction of the original displacement. It is therefore quite contra-indicated for this class of fracture.

(*b*) The same objection holds good to a certain extent for all other fractures of the jaws, but not so much for *fractures of the ascending ramus.* In these cases it is necessary to

immobilize the jaw with the lower and upper teeth in absolutely normal contact. This can be done by the four-tailed bandage only if it is applied with excessive tightness, and this the patient either cannot or will not tolerate, since it occasions intense pain, neuralgia and headaches; moreover, the patient, in endeavouring to eat, unconsciously or otherwise always stretches the bandage, and mobility results. The four-tailed bandage is always either too tight or too loose.

(*c*) For simple fractures in the incisor region it is unnecessary; there are other methods equally simple, more efficacious, and less unsightly.

2. **Interdental Splints.** An interdental splint is an apparatus which is made to embrace and fit the teeth of either one or both jaws. It is 'cemented' into place or held in position by interdental ligatures or external bandaging. Briefly the technique of making such a splint is as follows :— An impression is taken of the fractured jaw, and from this a plaster cast is made. The cast is then sawn through at the site of the fracture and fixed together again with the teeth in correct alignment. To such rearranged cast is then made a metal or vulcanite cap covering all the teeth, and this constitutes the splint (of which there are a number of varieties).

It is important to remember, however, that *no form of interdental splint must be used for fractures of the lower jaw posterior to the last molar*, because

(i) The lesser fragment is not controlled;

(ii) The jaws being separated malunion is bound to take place.

**The Hammond Splint** (Fig. 37) is perhaps the best known of this type. It consists of a single stout wire bent to fit round the lingual and buccal surfaces of the teeth, the ends being soldered together. It is ligatured in place by fine wires passing through the interdental spaces. It is

usually supposed to be simple to make and apply; this, however, is more apparent than real.

The most serious objection to its use is that it does not efficiently control the upward and downward displacements of the fragments, and where such is present its use is contra-

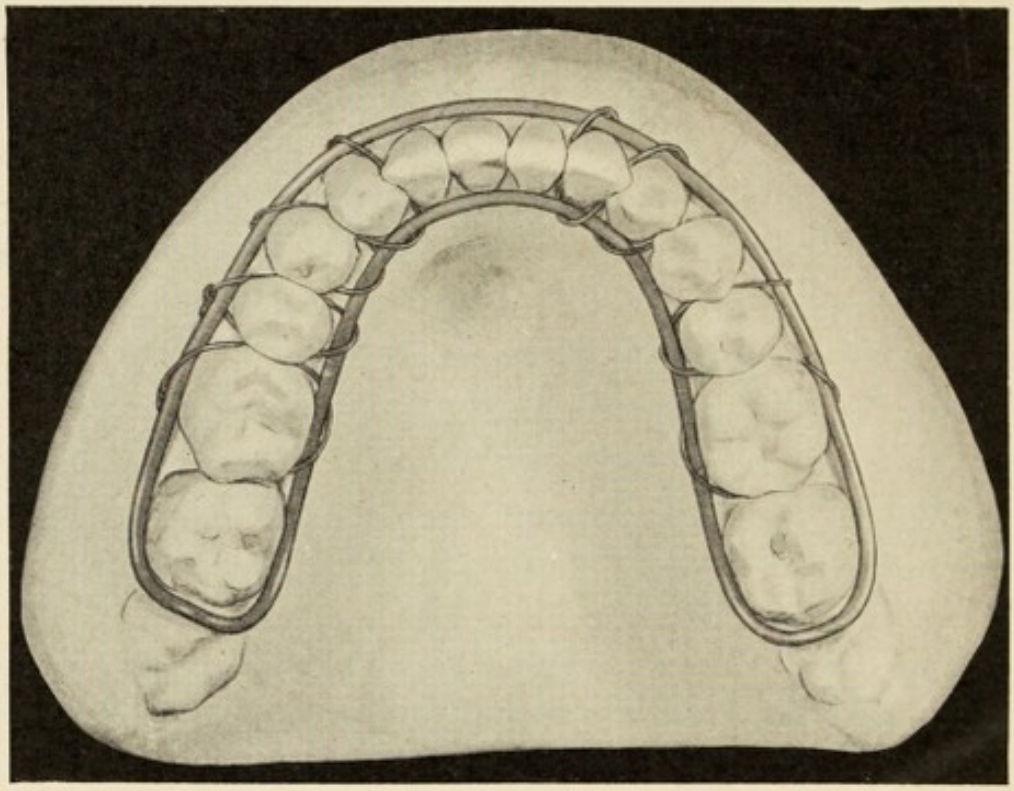


Fig. 37. The Hammond splint shown in position on a plaster cast.

indicated. It should be limited in use to simple fractures in the incisor region.

The author has seen at least one case where permanent deformity, i.e. 'open bite', resulted from its use. Moreover, the splint had become completely buried in the soft tissues on one side and had to be cut out.

**Moriarty or Metal Cap Splint.** This consists of a single cap of metal, usually german silver gilded, swaged to fit over the crowns of either all the teeth or of several teeth on both sides of the fracture. In its simple form it is very suitable for fractures in the incisor region of the lower jaw and for all fractures of the upper jaw. It is secured in position

in such cases by cement,[1] and this should be well covered immediately with mastic varnish to prevent the saliva acting upon it. For compound fractures of the lower jaw, however, owing to hæmorrhage and excess of saliva, sufficient hold is not obtained and the cement gives way. In order to overcome this the author uses the following *modification* (see Fig. 38). The splint is cut away opposite the premolar teeth on either side and two studs are affixed just above this.

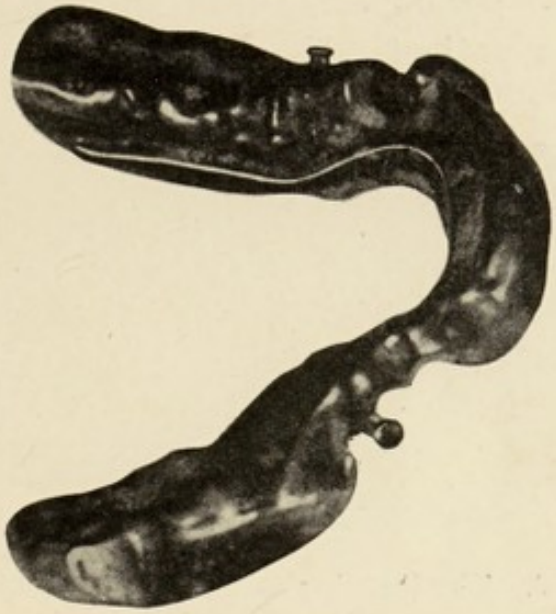


Fig. 38. Author's form of metal cap splint for compound fractures of the lower jaw.

Before inserting the splint wire ligatures are passed round the necks of the premolar teeth and the ends left long; the splint is then fixed, either with soft (hot) gutta-percha or 'cement', and the wire ligatures wound round the studs on either side. The splint is thus firmly anchored down. By this means both vertical and lateral displacement is corrected, the patient can use the jaw, and there is no external bandage or apparatus. No harm has been seen to follow the use of the wire ligatures.

**The Gunning Splint** (Fig. 39). This is a splint somewhat similar to the last, but it is 'double', i.e. it is practically two

[1] See footnote, p. 110.

splints, one for the upper jaw and one for the lower, joined together, having spaces for the admission of food. It is usually made of vulcanite. This apparatus controls all displacements well and may be used for the upper as well as the lower jaws; it is particularly useful when the jaws are edentulous and when there are very few teeth remaining, in which case a four-tailed bandage is necessary to keep the splint in place. In ordinary cases it is a little clumsy and keeps the mouth permanently open, and this is apt to become irksome to the patient. Nevertheless it is practically the

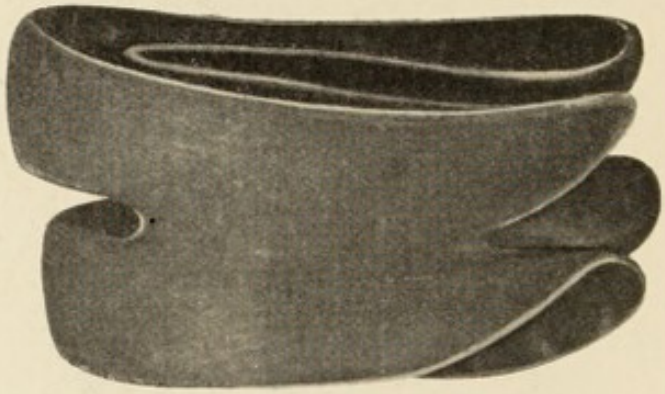


FIG. 39. The Gunning splint—made of vulcanite.

only variety of splint which is suitable for those rare cases where the whole of the alveolar process of the upper jaw containing the teeth is detached.[1]

*The Hern splint* is a modification of the Gunning, but it is a single splint with vertical projections containing gutta-percha for the reception of isolated upper teeth.

*The Ottolengui* is a similar splint to the Gunning, but made of metal and with no feeding spaces. The food passes round behind the last molar.

*The cradle splint* is similar; it may be described as a skeleton form of Gunning, being made of stout wire, and somewhat resembles a ' double ' Hammond.

[1] Two such cases are quoted by Heath, *Injuries and Diseases of the Jaws*, p. 47.

All these splints have the disadvantage of propping the jaw open considerably, thus rendering control of the lesser fragment more difficult, and if the fracture is very far back they are all absolutely contra-indicated on this account. They moreover absolutely immobilize the lower jaw, thus the advantage of passive movement is absent and union is apt to take longer.

3. **Combinations of External Bandaging and Interdental Splint.** *The Kingsley*[1] is the best type of this class (Fig.40). It consists of a vulcanite cap fitting over the lower teeth with

FIG. 40. The Kingsley splint. The impressions seen in the vulcanite are for the upper teeth.

two arms of very stout wire attached, which pass outside the mouth and run backward parallel to the lower border of the jaw. A bandage is passed over these arms and under the jaw; thus the fragments are 'clamped' as it were between the vulcanite cap above and the bandage beneath.

This is an excellent splint for controlling marked displacement, whilst the jaw can be moved and used during the time that union is taking place. Its disadvantage lies in the outside arms preventing the patient from lying on his side, and also gives him the appearance of being bridled (which I have had patients object to in a most forcible manner).

These objections may be overcome to some extent by

[1] Or Hayward.

having one central arm with a clamp under the jaw attached, instead of the two lateral arms. The author has had no experience of this modification, but it seems that this must result in a diminished ' grip ' or control of the fragments, and from the point of view of appearance is very little better.

4. **Interdental Lacing.** This is a simple and the oldest method of treating fractures of the jaws. It is mentioned

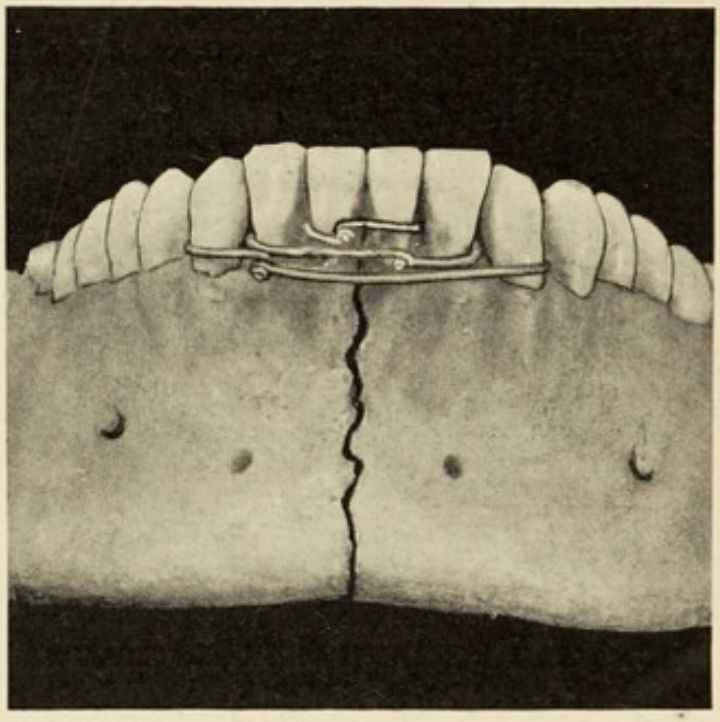


FIG. 41. Interdental lacing. The wire shown in the illustration is somewhat thicker than it is desirable to use.

by Hippocrates more than two thousand years ago. It consists in passing wire ligatures through the interdental spaces on either side of the fracture (Fig. 41). The first ligature includes the two teeth adjoining the fracture, the second embraces these two and the next two also, i.e. four teeth altogether, the third ligature is passed round six teeth, i.e. three on either side of the fracture. The longest ligature is tightened first and the shortest last.

The method is only suited to simple fractures in the incisor region of the lower jaw, or to simple fractures of

the upper jaw without displacement, especially to such as are in the median line.

The chief advantage of the method is its simplicity, requiring neither special skill nor apparatus, there is nothing unsightly, and the jaw can be used. It is said that the ligatures are apt to injure the teeth or gums, but with ordinary precautions as to cleanliness and antiseptic washes nothing of the sort need occur. Of course, if the teeth are loose, either from traumatism or disease, it is obvious that the method is inapplicable. It is a rather remarkable fact that those who object to this form of treatment on account of the 'irritation liable to be caused by the wires' all advocate the use of the Hammond splint, in which there is as much or more wire passed between the teeth. Moreover, the Hammond splint is far more difficult to keep clean than the simple interdental lacing.

*For fractures posterior to the last molar, and for all fractures of the ascending ramus, one of the following methods must be used.*

**Intermaxillary Lacing.** This consists essentially in absolutely immobilizing the lower jaw by lacing it up to the upper jaw by means of wire ligatures around the teeth. The following is the method used and described by the author (*B. M. J.* 1909, p. 882). The ligatures should be of silver or copper gilt wire (gauge 21 or 20 B. W. G.), and the premolar teeth are the most suitable to which to affix them. These teeth are not so far back as to be inaccessible, and the comparative narrowness of their necks makes them useful for retaining the ligatures, see Fig. 42. Two horizontal ligatures are first passed through the interdental spaces between the canine and first premolar, and the second premolar and first molar in both jaws, so as to include both premolar teeth. This should be done on both sides of the jaws. The horizontal ligatures on either side are then connected by vertical ligatures, all the wires being applied quite loosely.

The horizontal wires are next tightened, and then the lower teeth are made to articulate absolutely accurately with the upper teeth, and whilst in this position the vertical wires are tightened and the jaw thus immobilized. The method may be used as a temporary measure in all fractures of the mandible, in which cases ligatures of silk or silkworm gut may be placed round the anterior teeth, as shown in Fig. 42. This affords the patient much relief and is much

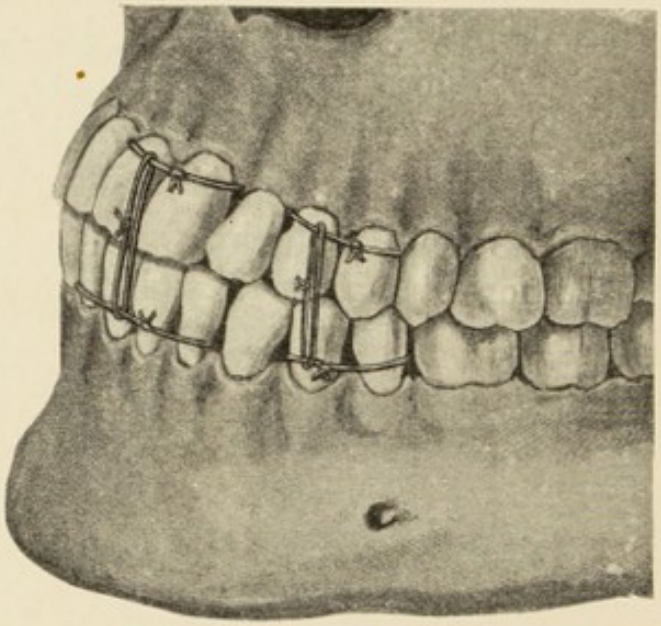


FIG. 42. Author's method of intermaxillary lacing, for all fractures of the lower jaw.

more reliable than a four-tailed bandage. The method may also be used as an adjunct to the open surgical methods of wiring the fragments, when fixation of the temporo-maxillary joint may be desirable for a time at least. Even if all teeth are present the patient does not suffer from inability to take sufficient nourishment. There is always sufficient space between the teeth and behind the last molars for liquid food to pass. Patients do not suffer very much from a diet of milk, porridge, arrowroot, soup, &c., for five or six weeks. In a similar way a patient can use an antiseptic mouth-wash, and use the tongue as a toothbrush on the lingual surfaces of the teeth.

The vertical wire ligatures may be left on for three weeks, after which in some cases they may be removed and passive movements allowed ; they may be replaced by silk ligatures, as being easier to apply. A vertical ligature should be continued until the sixth week, especially during the time or times the patient sleeps, in order to prevent yawning, as the latter does far more harm than even a slight amount of mastication.

The method is, of course, only applicable to those cases in which there is a sufficient number of sound teeth present, but such fractures usually seem to occur in strong vigorous patients with a good set of teeth.

Another method of intermaxillary lacing is by means of 'Angle's bands'. These bands are either made or procured and clamped to the teeth by means of a thread and nut. There are studs on the buccal surfaces round which ligatures are passed in order to bind the two jaws together.

5. **Wiring the Fragments.** This operation is of course applicable to all forms of fractured mandible, it is not suitable for the maxilla. It is absolutely essential in badly comminuted fractures, and in fractures of the coronoid process. It is not very suitable by itself for fractures above the angle of the jaw, or beyond the last molar tooth, because it does not sufficiently immobilize the jaw. It may, however, be very conveniently combined with intermaxillary lacing.

The disadvantage of the method, of course, for fractures of the ascending ramus, is that it necessitates a scar on the face.

For practically all fractures of the horizontal ramus the operation can be performed wholly from within the mouth. The difficulty, or otherwise, and success of the operation depends entirely upon the amount of swelling and sepsis present. Where this is marked and the method of treatment is deemed most suitable for the case the author prefers to immobilize the jaw by temporary intermaxillary ligatures

for some days or a week, whilst antiseptic mouth-washes are being used and the wound cleaning up. The method then employed is to drill through the bone between the roots of the teeth about ½ to ¾ of an inch away from the fracture on either side, with a suitable drill. (For fractures in the premolar or molar region a 'contra angle' bandpiece is used in the engine.) In order to mark the site of the perforation the drill is dipped in pure carbolic acid. Well gilded copper wire is then passed through the holes [1] and tightened by twisting the ends, which are cut off and covered with either gutta-percha or fine rubber tubing. It is then advisable wherever possible to supplement this single suture by *interdental* lacing in the neighbourhood of the fracture.

The wire suture should be removed (by being cut) at the end of six weeks: this may require a general anæsthetic, but it can usually be done under cocaine. This method has the advantage of being rapid, simple, and inexpensive.

**Ununited Fractures.** The fractures which show most tendency to non-union are horizontal ones either in the upper or lower jaw, which are at about the level of the apices of the teeth. These fractures are usually very septic, and not infrequently there has been extreme mobility of the fragment for some time before the patient is seen. No rule can be laid down as to whether an attempt should be made to retain such fragments or whether they should be removed at once: it is entirely a matter of degree of injury, vitality of the patient, and experience of the practitioner, though as a general rule it may be said that there is more hope of retaining such fragments in the upper than the lower jaw, on account of the greater vascularity of the former.

Cases of absolute non-union in the body or ramus of the

[1] It is sometimes stated that there is considerable difficulty in returning the wire through the holes, but with a few common-sense precautions this is really a very simple matter.

lower jaw are very rare; fibrous union practically always occurs, though this indeed may be so weak, and the ends of the bone so much separated, as to constitute a false joint. The causes of such union are usually either severe comminution leading to necrosis or mobility of the fragments.

The two following cases[1] illustrate this and the treatment:—

(*a*) The patient, a man aged 40, gave a history of having been treated for six weeks by means of a four-tailed bandage for a fracture at the angle of the lower jaw. A considerable amount of brawny swelling was still present and the patient complained of pain on endeavouring to eat; on examination, slight mobility and crepitus could be obtained.

An incision was made over the fracture, the ends of the fragments freshened and some gelatinous granulation tissue removed. The jaw was then immobilized by *intermaxillary lacing* for a month, when perfect union had taken place.

(*b*) A man aged 25, as a result of a kick from a horse, suffered a compound comminuted fracture of the lower jaw in the region of the symphysis with the loss of three incisor teeth. This was treated by interdental splints for nearly six weeks, when it was found that fibrous union only had occurred.

The line of fracture was opened up and a number of small pieces of necrosed bone removed and the fibrous tissue divided. The bone was then *wired* and this was *supplemented by interdental lacing*. Firm bony union had occurred in four weeks. In this case, of course, the articulation of the teeth was interfered with to some extent, since the lower jaw was made narrower than it had been previously, but this, of course, was unavoidable, and illustrates the point that in such cases too much attention may be paid to obtaining perfect articulation of the lower with the upper teeth at the expense of bony union.

[1] Recently treated by the author.

## Dislocation of the Jaw

Dislocation of the temporo-maxillary joint is brought about through force acting backwards and downwards on the horizontal ramus. This may be occasioned either by a violent blow or fall on the chin, or more usually by the sudden and powerful contraction of the digastric muscles as in yawning or opening the mouth too wide.

It is occasionally seen after a dental operation when the lower jaw has been depressed considerably during the extraction of lower teeth: in such cases the patient has usually been under an anæsthetic. Such accidents as this the anæsthetist may help considerably in preventing by keeping his fingers under the angle of the jaw, and making upward and forward pressure; especially should this be observed during the difficult extraction of lower teeth.

Dislocation of both joints is seen slightly more often than of one alone; the condition is commonest in women at middle age.

In almost all cases the condyle is displaced forwards over the eminentia articularis into the zygomatic fossa. The mechanism and pathology of this dislocation have been much discussed at various times, but the different theories advanced cannot be dealt with here. There is one elementary fact, however, which it is necessary to remember in considering this condition, and one which is not usually mentioned: it is that the temporo-maxillary articulation is *not a simple hinge*. That is to say, the condyle is not the central point on which the mandible turns. The geometrical point around which the mandible rotates is situated further back and lower down about the region of the posterior inferior border of the external auditory meatus. Expressed otherwise, when the chin moves downwards and backwards (as it always does when the mouth is opened widely) the condyle, instead of being a fixed point, moves forwards and downwards. (See Fig. 43.)

It is true that it is the external pterygoid muscles which are chiefly concerned in drawing the *jaw forward*, and, since these muscles are attached to the neck of the condyle and to the interarticular fibre cartilage, the student frequently infers that they are the only muscles which draw the *condyle forwards*, whereas it is the digastric muscles which bring the condyle furthest forwards; the forward movement of the condyle when the external pterygoids are acting is insignifi-

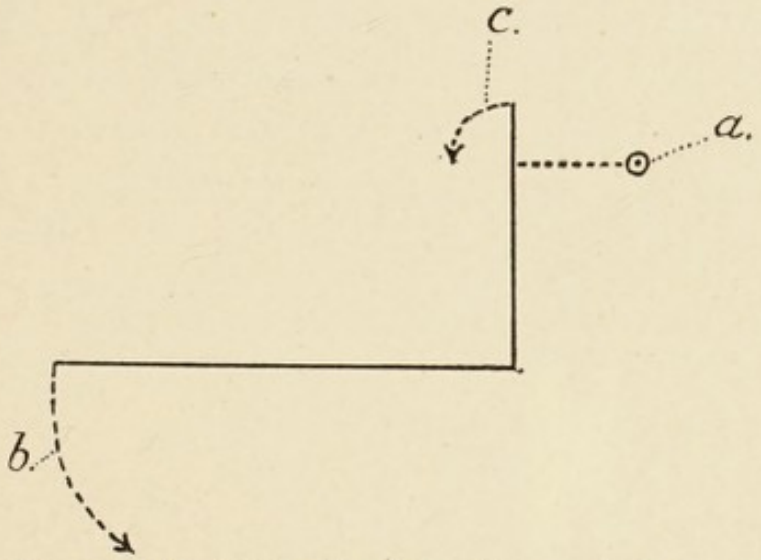



FIG. 43. Diagram illustrating the path of the condyle during depression of the anterior part of the mandible. *a*, Point about which the mandible hinges; *b*, Path of chin; *c*, Path of condyle.

cant compared to what it is when the digastric is fully contracted. The result of this is that if the action be continued sufficiently the condyle passes below the level of the eminentia articularis, and the masseter, temporal, and pterygoid muscles being taut, suddenly contract and draw it forwards and upwards into the zygomatic fossa.

The capsule is rarely lacerated and the interarticular fibro-cartilage follows the condyle over the eminentia articularis. (Fig. 44.)

All the ligaments are considerably stretched, the temporal muscle too is stretched so much that some of its fibres may be torn off the coronoid process.

**Symptoms and Signs.** In bilateral dislocation the mouth is open and the jaw fixed, mastication and distinct speech are impossible and the saliva may be dribbling away.

There is considerable pain in the region of the joints, and a hollow is noticed just in front of the ear and below the zygoma. Further forwards, just behind the malar bone, there is an abnormal fullness due to the presence of the coronoid process. In some cases there may be a swelling

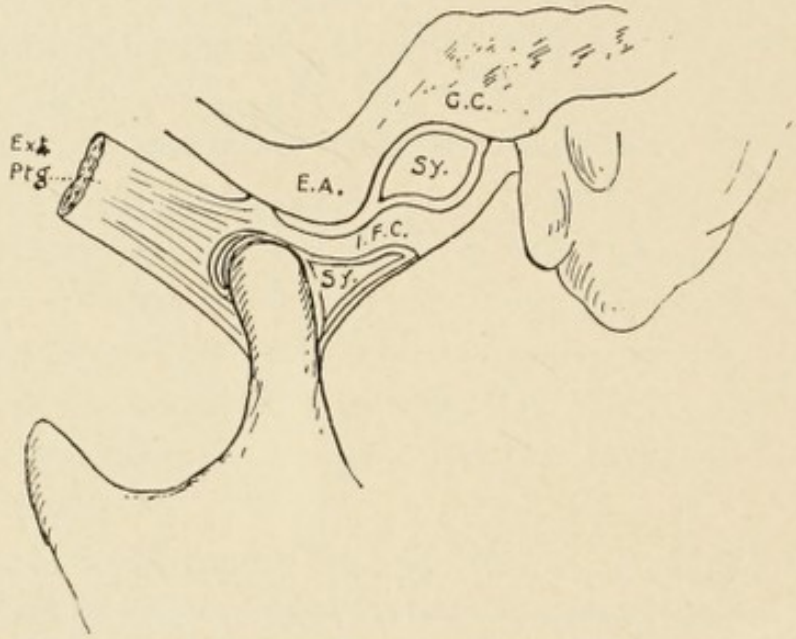



FIG. 44. Semi-diagrammatic representation of the position of the parts in dislocation of the lower jaw.

G.C. = Glenoid cavity, E.A. = Eminentia articularis, Ext. Ptg. = External Pterygoid muscle, I.F.C. = Interarticular fibro-cartilage, Sy. = Synovial membrane.

above the zygoma due to tonic contraction of a portion of the temporal muscle.

In unilateral dislocation the symptoms are similar but not so marked, and, owing to the fact that the patient retains the use of the jaw to a certain extent, the dislocation may pass unnoticed. (This is likely to happen after tooth extraction, especially if there has been previous swelling and pain on the same side, as not infrequently is the case).

The chin in these cases is rotated *away from the lesion*; this,

however, may not be very noticeable; it is in contradistinction, however, to fracture of the neck, when the chin is *inclined towards* the injury.

Dislocation backwards, with and without injury to the bony wall of the meatus, have been described, as also have cases of congenital dislocation with more or less malformation.

Recurrent dislocations are not uncommon in certain women in whom the ligaments are stretched and lax and the eminentia articularis probably not well developed.

The differential diagnosis between fracture of the neck of the condyle and dislocation is usually simple; in recent cases it may be expressed briefly in the following table :—

| *Dislocation.* | *Fracture of Neck.* |
|---|---|
| Jaw more or less fixed and open. | Jaw not fixed and is preferably closed. |
| Chin deflected away from injury. | Chin drawn slightly towards injury. |
| Hollow in front of ear and fullness below zygoma. | Diffuse swelling in front of ear. |
| Crepitus absent. | Crepitus present, but may be difficult to obtain. |

**Treatment.** Reduction is as a rule readily accomplished. The lower jaw is grasped firmly on both sides between the thumb and fingers, the thumb being on the occlusal surface of the lower molar teeth and the fingers under the jaw outside. The jaw is then firmly depressed by the thumbs, the chin slightly raised and pushed forwards by the fingers, and then, when it is felt that the condyle is clear of the eminentia, the jaw is pushed backwards.

This method always succeeds in recent cases; when, however, some considerable time has elapsed it may be necessary to place some object, such as corks or wedges of wood, between the molar teeth at the back, and to forcibly elevate the chin in front by means of a tourniquet over the head.

In difficult cases an anæsthetic may be necessary. Cases of recurrent dislocation have been successfully treated by cutting down on the joint, and suturing the interarticular

fibro-cartilage to the capsule at its attachment to the zygoma.

When strong adhesions have formed it may be necessary to divide these and then to maintain the condyle in the glenoid fossa for some time by securing the lower to the upper jaw, preferably by means of intermaxillary lacing. (See Fig. 42.)

**'Subluxation'**. This is a vague term for which there now seems to be no further use. Under it there seem to have been included several distinct conditions, at least if not by Sir Astley Cooper the originator, by other writers since. The term seems to have included osteo-arthritis of the joint, slipping of the interarticular fibro-cartilage, and laxness of the ligaments leading to or occasioned by recurrent partial or complete dislocation.

# CHAPTER XII

## CLOSURE OF THE JAWS

**Amasesis** (inability to masticate) may be due to a variety of causes. It may be due to the absence of teeth or the presence of defective teeth, or it may result from inability to use the jaw owing to fracture, dislocation, or 'closure of the jaws'. The latter condition may arise either suddenly or may come on insidiously; it is, therefore, proposed to speak of 'acute' and 'chronic' closure of the jaws, although the closure of the jaws is not a disease in itself but is really the most pronounced symptom of a number of different lesions. It is a convenient classification, however, since cases naturally fall into these two divisions, and on seeing or hearing of a case of inability to open the mouth, the first thing always to be asked about it is its method of onset and how long it has been present. The diagnosis, prognosis, and treatment hang very largely upon the answer to these questions.

**Acute Onset of Closure** may be due firstly to *tetanus*, in which case it is due to the stimulation of the nerves supplying the muscles closing the jaw and may be the only symptom, but it is rapidly followed by typical 'tetanic' contractions of the muscles of the neck and back. There may be a history of injury, but cases have been recorded in which it seems probable that the tetanus bacilli have found an entrance through an oral lesion already present.

Secondly, acute cases may arise on account of a *local inflammatory focus* in the region of the angle of the jaw; such as acute parotitis, mumps, lymphadenitis, tonsillitis,

or *impacted wisdom tooth*. The term 'impacted' is used to designate that condition when a wisdom tooth is only partially erupted, is lying more or less horizontally in the jaw, and its further eruption is prevented by reason of its crown impinging upon the distal surface of the second molar, as is shown in the Skiagram. (See Fig. 45.)

Of the above causes probably the last is the most common; certainly it gives rise to the most pronounced and rigid 'closure'. This result may be brought about in one or two ways, (i) by reflex irritation of the motor branch of the fifth nerve leading to a spasm of the masseter muscle, or more commonly (ii) by an infiltration of the muscles by inflammatory products, which may or may not cause a myositis.

The history of such cases is usually vague, the patient often cannot locate the site of the origin of the trouble, but knows there has been a soreness, a stiffness about the angle of the jaw, either inside the mouth or outside, accompanied perhaps by some swelling, and this has rapidly led to inability to open the mouth.

Consequently a very careful examination is necessary, and this is not always a simple matter. Parotitis and lymphadenitis are more readily palpated and diagnosed, but tonsillitis and impacted wisdom tooth may give rise to difficulty, firstly, owing to the patient's inability to open the mouth, and secondly, owing to the confusion of symptoms, since in the latter condition as well as the former one of the first and most constant things the patient complains of is pain and difficulty in swallowing.

In tonsillitis, however, the firmness of the closure is hardly likely to be so severe as in impaction. In the latter case it is frequently impossible for the patient to separate the jaws at all, and neither can they be separated by instrumental means unless the patient is given an anæsthetic.

It may be possible to diagnose the impaction from the history and by palpation and examination with a mirror

and probe; failing this, if the patient is not too ill, a skiagram may be taken which will give valuable indications as to treatment as well as diagnosis (Fig. 45). If this is impossible, an examination under an anæsthetic should be made, and at the same time preparations should be made to deal with whatever condition is found to be present. The mouth should be forced open first of all by wedges of boxwood

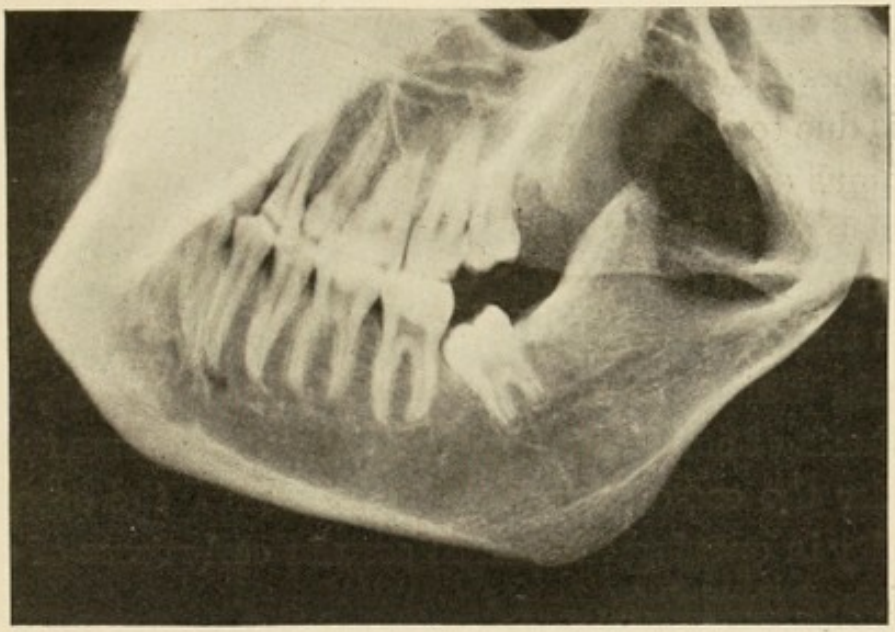


Fig. 45. Skiagram of an impacted wisdom tooth. Note the proximity of the still incomplete roots to the inferior dental canal. (Radiograph by Dr. Cameron, Radiologist to the Dunedin Hospital.)

about five inches long, and a *Mason's* gag then inserted. No other form of gag is suitable, because sufficient leverage cannot be obtained. Even the 'Mason' is insufficient on some occasions to effect an opening, and when this is so a 'thumb-screw' gag in the front of the mouth should be employed. If an impacted wisdom tooth is found, it should, of course, if possible be extracted, but this is sometimes extremely difficult or even impossible, in which case the second molar may be removed with equally good results as far as the relief of the immediate condition is concerned.

The wisdom tooth may erupt into a good and useful

position, or it may have to be extracted later. These cases are often accompanied by pyrexia, and perhaps rigors, there is more or less complete anorexia and considerable foetor, in fact the patient is not infrequently extremely and dangerously ill; nevertheless active treatment of the cause *must not be postponed* until symptoms have subsided, but must be undertaken immediately. Hot fomentations should be used in the mouth, both prior to and for some days after the operation.

Another cause of acute closure of the jaws is *pain*. The pain is due to acute inflammation in the region of the ascending ramus or to an acute arthritis of the temporo-maxillary joint. Here it is not the mechanical effect of the swelling which prevents the jaw being opened, but the patient more or less unconsciously keeps the jaw closed in order to avoid the pain which movement causes. In such cases, under anæsthesia of course, it requires no force to depress the jaw.

Under the same heading is to be classed the occasional difficulty in opening the mouth seen in early cases of osteo-arthritis of the joint.

**'Chronic' Cases of Closure of the Jaws** are due either to adhesions, cicatricial contractions, or to neoplasms.

Either of these may be situated in different places but may produce the same results.

The adhesions may involve (i) the structures of the joint; (ii) the tissues of the cheek, either inside the mouth or on the face.

The adhesions in the joint are subsequent to an acute arthritis which has occurred usually many years previously. In perhaps the majority of cases this is *post-scarlatinal*, it may have been associated with an otitis media, or may have resulted from traumatism, such as a fall upon the chin. Usually the adhesions are fibrous, but occasionally bony adhesion takes place or it may proceed to complete osseous ankylosis.

When cicatricial fibrous tissue is situated in the cheek it is due to previous loss of tissue occasioned by severe ulceration or gangrenous stomatitis, in which case all the tissues of the cheek may have been lost, or it may be due to thermal, chemical, or other traumatic destruction of the exterior of the cheek. In any case adhesive bands slowly form between the upper and lower jaw and eventually prevent movement of the latter.

A case has been recorded where laminæ of bone existed congenitally between the alveolar processes of the two maxillæ, united by cartilage in the centre.

Neoplasms causing closure of the jaws may be either innocent or malignant, usually the latter. The tumour may act either by mechanical obstruction to the movement of the jaw in the region of the angle, or by an exostosis of the zygoma, or by causing a reflex spasm of the muscles due to inclusion of the motor branch of the fifth nerve in the growth. In one case, increasing difficulty in moving the lower jaw was the first and for some time the only symptom of a sarcoma at the base of the skull. Such cases (when due to malignant disease) are usually accompanied by much neuralgic pain, which distinguishes them from those due to adhesions.

All cases due to adhesions or neoplasms come on slowly and insidiously, those caused by adhesions in the joint and cheeks being slowest, and those due to malignant growths being more rapid in their process.

In the case of adhesions and contractions it may take from five to twenty years for the jaw to become completely immobilized.

One case coming under the observation of the author was in a woman of fifty, in whom immobility was almost complete, and who gave a definite history of a slowly increasing difficulty in moving the jaw since she had scarlet fever at the age of eleven. The condition had been 'relieved'

occasionally by the extraction of teeth from the back of the mouth.

On the other hand, an equal degree of immobility was present in a boy of fifteen, in whom it also was due to scarlatinal arthritis some six or seven years previously.

In this class of case, where the movements of the jaw become limited early in life, development ceases owing to loss of function, and the lower jaw remains infantile in shape and size, giving a characteristic appearance to the patient (Fig. 46).

**Treatment of Chronic Cases.** In the treatment of cicatrices and adhesions there are two things which naturally first suggest themselves—division and mechanical stretching. Neither of these is effective, and experience has shown that either singly or combined they always fail to give a permanent result owing to the re-formation of fibrous tissue.

For adhesions in or near the joint there are two possible lines of treatment, (i) excision of the condyle, and (ii) Esmarch's operation. The former has given good results in some cases, though it may cause deviation of the chin towards the side of the lesion; the general opinion now, however, seems to be in favour of Esmarch's operation, as being simpler, more effective, and more constant in results. The operation consists in removing a wedge-shaped piece of bone from the angle of the jaw with the apex towards the alveolar border; a false joint is formed and the patient regains the use of the jaw in about three weeks. Division of the ramus from within the mouth has been occasionally performed, but the operation is more severe, there are more risks, and the results are not so good.

For adhesion near the anterior part of the mouth where the lower alveolar border is tightly bound up to the alveolus of the upper jaw the above operations are of no service. For such cases there are two methods of treatment.

(i) One or more broad ligatures are passed through the

adhesion and tied loosely in position. They are left *in situ* until healing has occurred along their course, when the fibrous tissue covering them in is divided, and thus the band of adhesion is, as it were, ' let out.'

(ii) In more severe cases the jaws are well freed from the adhesions by division close along the alveolar borders, and in order to prevent re-attachment ' shields ' are inserted to keep the raw surfaces apart and on which the new tissue

FIG. 46. Incomplete development of the mandible in a patient with slowly developing ankylosis of the temporo-maxillary joint. The black portion shows what should have been the normal development.

may mould itself. These shields are something like an inter-dental splint (see Figs. 38 and 39), except that there are prolongations upwards and downwards into the buccal sulcus at the points where the division of the adhesions has taken place. Such shields should be inserted as soon after the operation as possible.

It seems to the writer that in such cases as these latter a form of plastic operation for inserting more tissue into the cheek would be more rational and more likely to yield permanent results.

# CHAPTER XIII

## ORAL TUMOURS

In scarcely any other region of the body, or in so circumscribed an area, are so many different varieties of 'tumours' found as in the mouth. It is possible that a 'swelling' in the mouth or on the jaws may be due to any one of some twenty-four or so causes. To treat all these causes extensively would be entirely outside the scope of this book; it is therefore proposed to discuss such tumours—cysts and neoplasms—more in relation to their manner of origin, and to indicate to some extent their diagnosis and treatment.

The following classification is convenient clinically :—

I. Originating in the mucous membrane and submucous tissue.
- Papilloma.
- Epithelioma.
- Angioma.
- Endothelioma.
- Adenoma.
- 'Polypus'.
- Myeloma.
- Fibroma.

II. From periosteum.
- Chondroma.
- Osteoma.
- Sarcoma—Spindle-celled.

III. From centre of bone
- Endosteal Fibroma.
- Osteoma.
- Chondroma.
- Sarcoma—Myeloid and round-celled.
- Odontomata (including dental cyst and zahnleiste epithelioma).

IV. Diffuse overgrowths of muco-periosteum and alveolus.

It will be convenient to consider together '*Polypus*', *Fibroma*, and *Myeloid* (?) *Sarcoma*. Any of these may

be a swelling, ἐπὶ οὐλὶς (on the gum), and therefore be literally termed an 'epulis'; the latter, though, is such a vague term that it is best avoided. The three tumours, wherever they occur in the mouth, are practically always associated with some form of chronic irritation, and may be regarded as pseudo-inflammatory in nature. Moreover the source of the irritation is most frequently dental—either natural or 'artificial'. Whenever a carious cavity in a tooth extends down to the gum margin, a localised hypertrophy of the mucous membrane takes place, forming a polypus, which grows into and eventually fills up the cavity. Such polypi are of exceedingly common occurrence, and act as a form of natural protection sometimes to a large exposure of the pulp in the tooth.

They are composed of a thin but normal epithelial covering, with a centre of loose areolar and very vascular tissue springing from the submucous tissue or periosteum. As a rule they cause no pain, and terminate either by being removed by the dentist before filling the cavity, or by atrophy and absorption if the tooth is extracted. Similar polypi uncommonly occur in the buccal sulcus or on the buccal mucous membrane, due to ill-fitting dentures. If the polypus remains for some time, however, and the teeth are not treated, it becomes irritated and inflamed, connective tissue cells proliferate, and eventually fibrillation and fibrosis occur in the central part. The growth is now a firm compact mass, and the longer it stays in position the more dense it becomes and must be classed as a fibroma. Owing to superficial irritation and frequent ulceration, granulation tissue is constantly forming on the surface, and thus, whilst the formation of fibrous tissue is taking place internally, the tumour tends to increase at the circumference. Such fibromata may be either pedunculated or sessile on the buccal mucous membrane; in the sulcus they are usually pedunculated, but on the alveoli they tend more to be sessile, unless the base

is situated in a constricted interdental space or becomes 'pinched', as it were, between two teeth.

Instead, however, of more or less complete fibrosis taking place in such a tumour, myeloid cells may be formed amongst the condensed and fibrillated areolar tissue, and in this case a 'malignant epulis', 'myeloma' or 'myeloid sarcoma' is said to be present.

The malignancy of this growth, however, is extremely small, being merely 'local', and the character of the tumour is usually different from a myeloid sarcoma springing from the medullary cells of bone, and, having regard to its mode of origin, it is doubtful whether it should be classed as a sarcoma. The term 'myeloma' is probably a much better word. In fact the writer recently had under treatment such a case, which seems to throw some light upon the origin of the myeloid cells.

The patient, a girl aged 10, had a loose lower deciduous molar causing some discomfort. On examination it was found that the molar was surrounded, and the almost completely absorbed roots were tilted up posteriorly, by what appeared to be dark granulation tissue. The tooth was removed, and it was then seen that the swelling showed the typical mulberry-like purplish coloured surface of a myeloma. A short time afterwards this was removed, and it was found to shell off like a cap from the surface of the permanent tooth below. Microscopically it was a typical myeloid tumour of the jaw, but the site in which it developed is just the position which should normally be occupied by the 'absorbent organ' of the deciduous tooth (which is composed chiefly of myeloid cells or osteoblasts), and the inference can hardly be avoided that this was the source of the myeloid cells of the tumour; this is further supported by the fact that a large proportion of such myelomata are found in quite young patients.

Periosteal fibromata of slower growth and embryonic in

origin arise near the symphysis in the lower jaw or the pre-maxillary suture in the maxilla.

Any of the above forms of tumour may by growth displace teeth and cause an irregularity of the alveolar border, and especially the last named.

A keloid in the buccal sulcus has been observed by the author arising from the scar of an old chronic ulcer. It was removed twice and then disappeared.

**Papilloma** of the oral mucous membrane, excluding the tongue, is uncommon. Many cases described as papillomata should be classed as ichthyosis or leucoma, since in many no central 'core' of connective tissue and blood-vessels is present, the growth is wholly epithelial, and may show a tendency to ingrowth. True papillomata, when they occur, are usually on the palate.

**Angiomata** of the simple or cavernous variety are occasionally found in the mouth and on the lips, buccal mucous membrane, and 'gums'. They are never likely to obtain a large size by reason of the fact that they are easily injured, and the consequent hæmorrhage leads the patient to seek treatment early.

**Epithelioma** of the squamous-celled variety commonly arise from the oral mucous membrane; they run a typical course, and are identical both macroscopically and microscopically with such growths in any other part of the body. They usually commence as an ulceration, which is in the majority of cases induced by the irritation of a carious or septic tooth, or by the edge of a badly fitting denture. They may originate in a patch of leucoplakia in which the epithelium, instead of proliferating outwards, suddenly dips inwards. Smoking is said to be a cause, but apart from the irritation of a clay pipe on the lower lip there is no very conclusive evidence in support of it.

Another cause, of which the author has seen two instances, is the biting of healthy but sharp teeth into the opposing

jaw from which the teeth have been lost. It is extremely surprising how long some patients will tolerate such a condition, and it is not therefore surprising that when ulceration occurs it should become malignant.

All chronic ulcers in the mouth, owing to the unavoidable persistant irritation to which they are subject, require to be kept under constant and serious observation until they clear up, and if this does not occur within a reasonable time they may with advantage be excised.

**Endothelioma** is somewhat rare in the mouth. It may occur on the palate or muco-periosteum, springing from the blood or lymph vessels. In a case recently described by Mr. Fairbank[1] the growth was a bright-red vascular tumour encircling the upper left second premolar, and had previously given rise to a sudden attack of hæmorrhage.

**Adenoma** of the palate and buccal mucous membrane occurs occasionally. The growths may be multiple, they are always small and imbedded in a capsule from which they are readily removed. Sometimes the adenoma breaks down and ulcerates, and it may become malignant. An adenoma of the soft palate may cause an alteration in the tone of voice and difficulty in swallowing, and also dyspnœa or cough.

**Endosteal Fibroma** is not of common occurrence. It takes origin from either the lining of the antrum, the peri-odontal ligaments, or from the sheath of the mandibular nerve. The tumour is a typical fibroma, usually encapsuled, the fibres are densely packed, and areas of calcareous degeneration are often present. It is usually of very slow growth, and distends the bone and distorts the direction of the teeth markedly. In the upper jaw the antrum is frequently encroached upon, and thus the tumour may grow to a considerable size before giving rise to any symptoms. Heath records several cases which he believed to have originated through carious teeth imbedded in the surface

[1] *Proc. Roy. Soc. Med. Odont. Sec.*, December, 1910.

of the tumours. Formerly these growths attained a considerable size and caused much disfigurement, but at the present time patients seek treatment much earlier.

**Chondromata** of the jaws are rare; they may arise endosteally or from the periosteum of the bone, especially in the upper jaw, or from the muco-periosteum of the antrum. The tumours are very hard and, if simple, of slow growth, causing separation of the alveolar plates. Occasionally, however, they are sarcomatous, and then development is more rapid, but the malignancy is not great. It is recorded, for instance (by Heath), that one patient was operated upon ten times in eighteen years for recurrent growths of this nature in the lower jaw, and in another case six operations were performed at intervals of six months to two years for a similar reason (Virchow).

**Osteomata** of the cancellous and ivory type both occur in connexion with the jaws. The regions most frequently affected are :—(i) the angle of the jaw ; (ii) the region of the symphysis or premolars—internally ; (iii) invading the antrum ; (iv) the hard palate.

In the latter situation the ' tumour ' is frequently central and is caused by a marked thickening of the bone on each side of the suture; it simulates an ' ivory exostosis ' and is termed ' torus palatinus '. Osteomata invading the antrum may be very dense, or quite spongy, to palpation. At the angle and symphysis hard ivory osteomata also occur, and Heath [1] relates a case occurring in the latter situation which caused considerable separation of the ascending rami. Osteomata of the cancellous variety not infrequently show a tendency to become sarcomatous.

It seems not improbable that some central osteomata may spring from the peri-odontal ligament. The writer has recorded a case of a small osteoma in this situation firmly adherent to the apex of a tooth.

[1] Loc. cit., p. 327.

**Sarcoma.** The myeloid and round-celled forms may arise from the centre of the bone, the spindle-celled originates in the periosteum.

A myeloid sarcoma developing in the centre of the bone differs only slightly from the form which is more superficial, the stroma is usually more cellular, the myeloid cells more numerous, and the vascularity is perhaps greater. Round-celled sarcomata in this situation are in all respects similar to those in other localities. Both these tumours tend at first to distend and thin the bone markedly, but later the round-celled variety infiltrates and destroys it. In the lower jaw these are more frequently seen in the region of the premolars and first molars; they develop in an upward direction, causing looseness of the teeth and 'toothache'. Ossification is liable to take place; in the myeloid form this is usually sporadic, but in other forms it may be considerable and convert the growth into an osteoid sarcoma. Round-celled sarcoma has been observed arising from the peri-odontal ligament, and sarcoma of the tooth follicle is described by Bland Sutton.

Spindle-celled sarcoma arising in the periosteum shows a tendency to follow this tissue and to grow round and enclose the bone, afterwards invading and eroding it.

These growths, but especially the round and spindle-celled, may invade (or originate in) the antrum. Symptoms are frequently absent until the cavity is filled, then displace-ment of the teeth downwards and the orbital plate upwards may occur, and pressure on the nasal duct causes epiphora. Neuralgic pain is prominent owing to implication of the second division of the fifth nerve.

The malignancy of these varieties is usually, as elsewhere, i.e. (i) round-celled, (ii) spindle-celled, (iii) myeloid, the latter never forming secondary growths and only tending to recur if incompletely removed.

All forms in many cases are associated with a history of

previous traumatism ; the myeloid not infrequently with an unsuccessful attempt at extraction of a tooth, a portion of a root remaining buried in the jaw. One case seen by the writer was definitely connected by the patient with such an event eleven years previously, and on removal of the tumour the fragment of tooth was found imbedded in its deepest portion, and again it is suggestive that the myeloid cells (? osteoclasts) may be present in order to attempt absorption of the unrequired bony substance.

In a case of large round-celled sarcoma in a girl aged 11, who was operated on by the writer[1] two years ago, the first symptom was toothache in a right lower molar. This had been extracted although sound, and the swelling had been incised on several occasions without relief. The swelling had then become noticeable externally and had distended the bone in an ovoid fashion. A small piece of the growth was removed under nitrous oxide and examination showed it to be sarcomatous. The parents refused the major operation of excision of a considerable portion of the jaw, and so the growth was removed locally from within the mouth, all the bone around it was removed, and a considerable portion of the body, leaving quite a thin bridge in order to prevent deformity (to which the parents objected). Up to the present time there has been no recurrence.

## ODONTOMATA

It is absolutely essential in order to obtain a rational and workable conception of these tumours that the details of the normal development of the teeth should be quite familiar to the reader ; if this should not be so, he is referred to Chapter II of this book, and to Tomes' *Dental Anatomy* or Schäfer's *Essentials of Histology* for detailed description of the tooth germ.

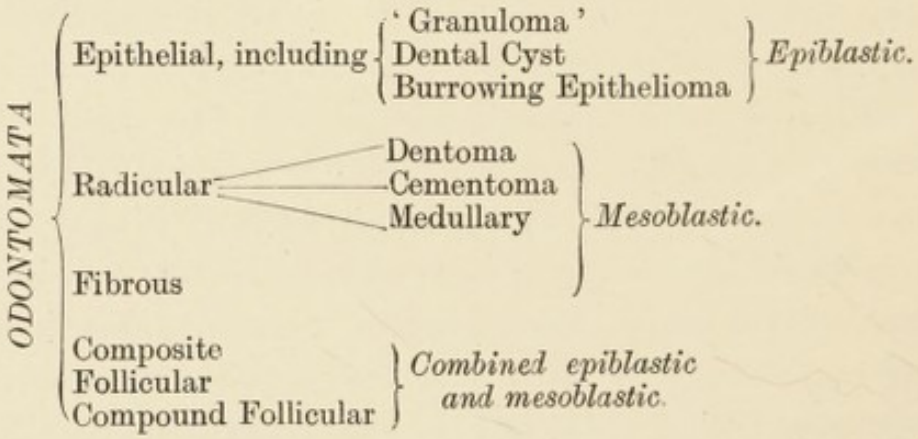
Odontomata are usually regarded as a rare kind of tumour

[1] *Proc. Roy. Soc. Med. Odont. Sec.*, July, 1909.

about which it is not necessary to know very much. This, as we shall show, is by no means the case, and largely arises through a somewhat inaccurate knowledge of what an odontoma really is. An odontoma may be defined as 'a tumour composed of dental tissues in varying proportions and different degrees of development arising from tooth germs or teeth still in the process of growth '.[1]

The tumour may arise from an aberration of any part of the tooth germ—from the enamel organ, dentinal papilla, or tooth sac; or the whole dental germ may be involved. Therefore an odontoma may be either an epiblastic or a mesoblastic neoplasm, or it may be compounded of both, it may be calcified or uncalcified (usually the latter), and it may be either innocent or malignant.

The following is a useful clinical classification :—

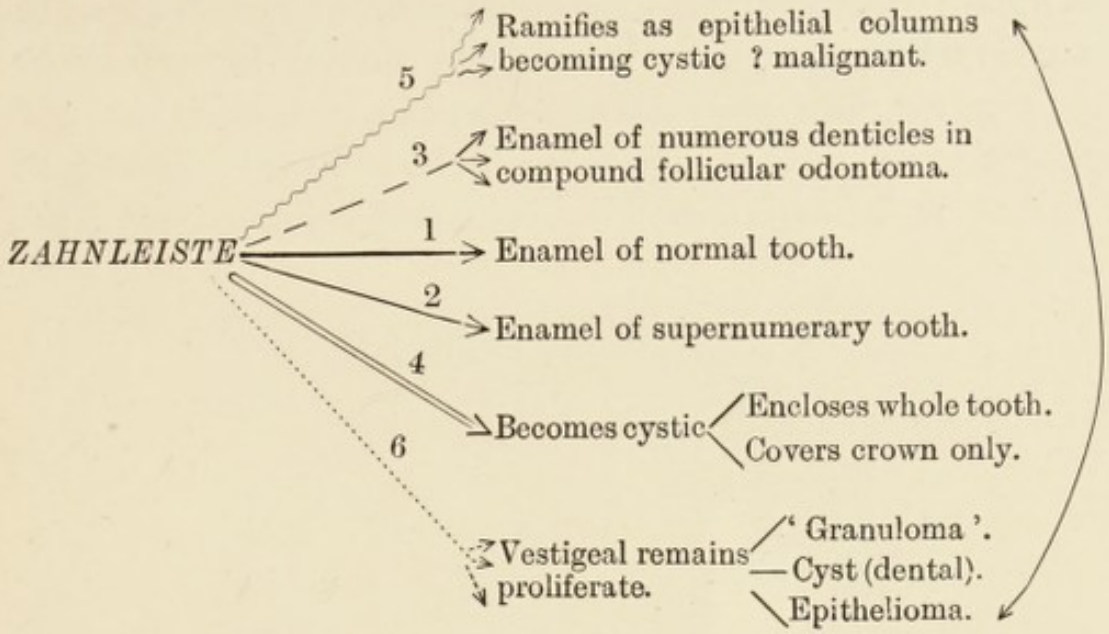



*Epithelial odontomata.* These varieties are without doubt the most important, both on account of frequency of occurrence and of seriousness of consequence.

As pointed out above they are all derived from the zahnleiste, and this structure has pronounced characteristics; it has (i) a tendency to persistence, (ii) a tendency to ingrowth, (iii) a tendency to become cystic. These three characteristics explain and correlate the various forms in a simple manner, the form depending largely upon which characteristic is predominant in each particular case.

[1] Bland Sutton.

The possible destinations of any individual group of epithelial cells in the zahnleiste may be expressed graphically in the following schema :—




It is only No. 5 in the above schema which is usually termed an epithelial odontoma. That is to say the zahnleiste, instead of forming the enamel of a normal tooth, divides, subdivides, and ramifies throughout the jaw.

The cells in the centre of the epithelial columns always tend to swell up and may become cystic. Fig. 47 is an example of such a tumour. The central light-coloured areas are the swollen epithelial cells, showing a slight tendency in places to become cystic and in another to colloid degeneration; the darker surrounding zones are the growing proliferating cells resembling exactly the cells of the zahnleiste before specialization. The stroma is that of a light connective tissue with denser fibrous bands in places, the vascularity is slight. The whole appearance of the epithelial growth has a distinct resemblance to the early formation of enamel organs. At a later stage, when possibly all the available stroma has been occupied or the particular epithelium has a more cystic tendency, nearly the whole of the cells break down and numerous cysts are formed

lined only by flattened epithelial cells, an example of which is well shown in Fig. 48. This constitutes the old 'multilocular cystic tumour of the jaw'.

**Symptoms.** Such growths doubtless always commence to develop in quite young individuals, though there may be no sign for some years. The growth is as a rule slow, and erodes and distends the bone gradually in various directions; probably the active growing epithelial columns erode the

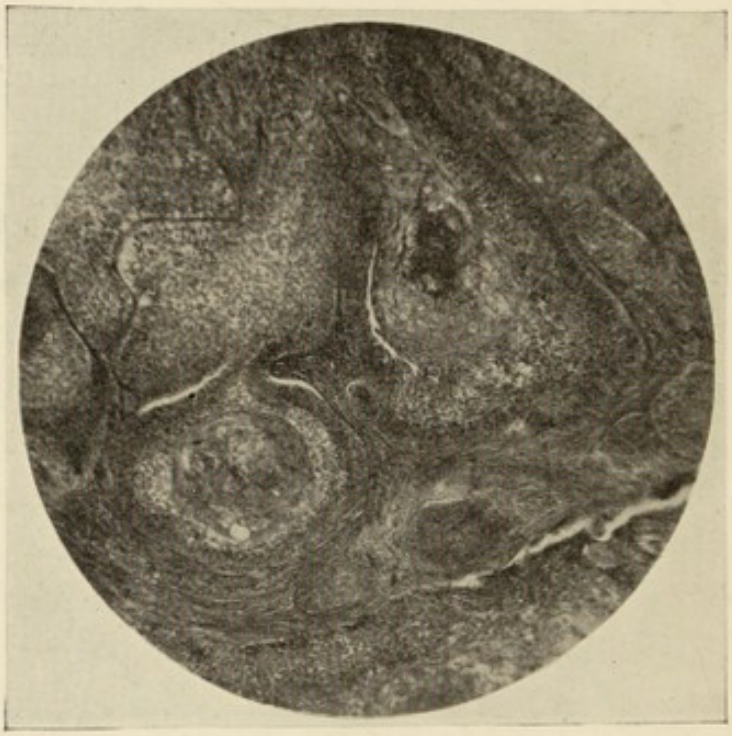


FIG. 47. Epithelial odontoma. Note the flask-like arrangement of the epithelial masses and the central swollen cells.

bone and when they become cystic distend it, so that eventually the bone comes to be multiloculated. There may be eggshell crackling and some slight fluctuation or 'bogginess'. There is not much pain, and in later stages some of the superficial cysts may rupture and discharge a small amount of glairy fluid.

**Compound Follicular Odontoma.** In the formation of this tumour also the zahnleiste breaks up, ramifies for a short period, but then stops and reverts to its normal function of

forming enamel. This results in the development of numerous denticles in one common follicle, which may or may not secrete a cystic fluid, the denticles however do not erupt. The number of denticles may vary from just a few to forty or fifty, and Bland Sutton describes such a tumour in a thar which contained over three hundred denticles.

Nearly all the cases of these tumours have been in young patients (æt. $8\frac{1}{2}$ to 25) and situated in the region of the

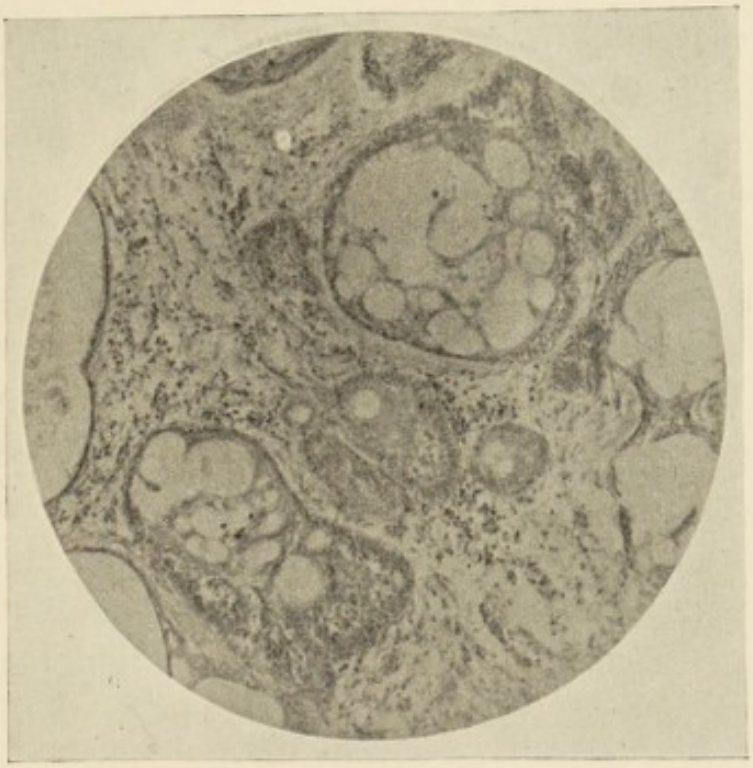


Fig. 48. Epithelial odontoma becoming cystic.

incisor teeth. A swelling is noticed which persists without symptoms, except that it may increase in size slightly. Sometimes the permanent tooth is missing (unerupted) or there may be a history of non-eruption of deciduous teeth.

**Follicular Odontoma.** The view held at the present time as regards the formation of these odontomata (which are cystic—the so-called 'dentigerous cysts') is that the fluid is formed in the stellate reticulum between the inner and outer layer of epithelial cells forming the enamel organ, and that

this fluid commences to be formed when the ameloblasts cease to be active in function. The cyst grows in size, distending the fibrous follicle, and usually entirely envelops the tooth, which thus comes to be in a cavity. If this is correct then we should not find Nasmyth's membrane [1] present upon the enamel of the tooth in the cyst—it should in fact be transferred to the lining wall; and this in fact is what has been found recently in a small number of cases.[2]

In Figure 49 is shown the wall of a follicular odontoma, and the epithelial lining is clearly seen becoming detached from the thick but delicate fibrocellular wall. (This, so far as the author can ascertain, is the only published photograph of the wall of such a cyst showing the epithelial lining; the latter usually disappears owing to suppuration or treatment before or after removal.) The tooth enclosed in this cyst was an upper canine, this being one of the commonest situations of these odontomata, and examination of its surface with acid showed the absence of Nasmyth's membrane. [Two cases of similar tumours have recently been described as 'odontoceles' [3] because the cysts were over the crown of an unerupted tooth only, and in one case outside the follicle. But since there can be no doubt that in both these cases also the cysts were derived from zahnleiste cells either within or without the follicle, there would seem to be no reason for special names. The one within the follicle is simply an incomplete or partial follicular odontoma, and the other, outside the follicle, a dental cyst [4] in a somewhat unusual situation.]

It will be now convenient to consider together **Granuloma**

[1] Nasmyth's membrane is composed chiefly, it will be remembered, of the *external* epithelium of the enamel organ.

[2] A case showing this was first noticed in 1894 by Pare and Maggs, and later two others by Hopewell-Smith. I have myself also observed the same absence of Nasmyth's membrane in two cases of follicular odontoma.

[3] Hopewell-Smith, *Proc. Roy. Soc. Med. Odont. Sec.*, May, 1910.

[4] See description later.

(*epithelial root tumour*), **Dental cyst** and **Epithelioma** (*burrowing zahnleiste*). It is not customary to include these under the heading of odontomata, but logically they belong to this class, since they are derived from the embryological remains of tooth germs, or in other words from zahnleiste 'rests'. They are formed from precisely the same cells as an 'epi-

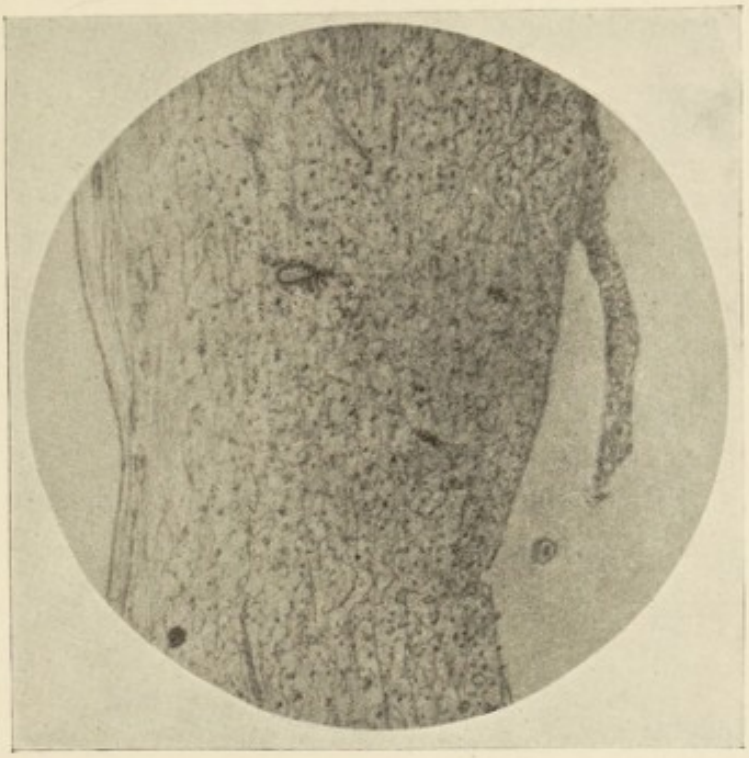


FIG. 49. Section through the wall of a follicular odontoma. (The stroma consists of a very delicate fibrous network.)

thelial' or a follicular odontoma. It has long been known that such 'rests' persist alongside the root of a tooth for many years, and they are known either as the *débris épithéliaux paradentaires* of Malassez or as the Epithelial sheath of Hertwig, and may be seen as small isolated groups of epithelial cells in the peri-odontal tissues. In general these cells either atrophy or remain quiescent during the whole of life, occasionally, however, under persistent and mild stimulation they 'revive' and proliferate.[1] The source of

[1] Sometimes they revert to their original function and form a little nodular mass of enamel on the side of the root of a tooth.

such stimulation is in nearly all cases a septic tooth—one in which the sepsis has not been so great as to cause acute suppuration, and so early loss of the tooth, but is simply sufficient to cause a chronic irritation, and this may be either at the apex or gum margin. The epithelial cells of any 'rests' in the neighbourhood of the apical foramen proliferate and form a small round solid tumour, a 'granu-

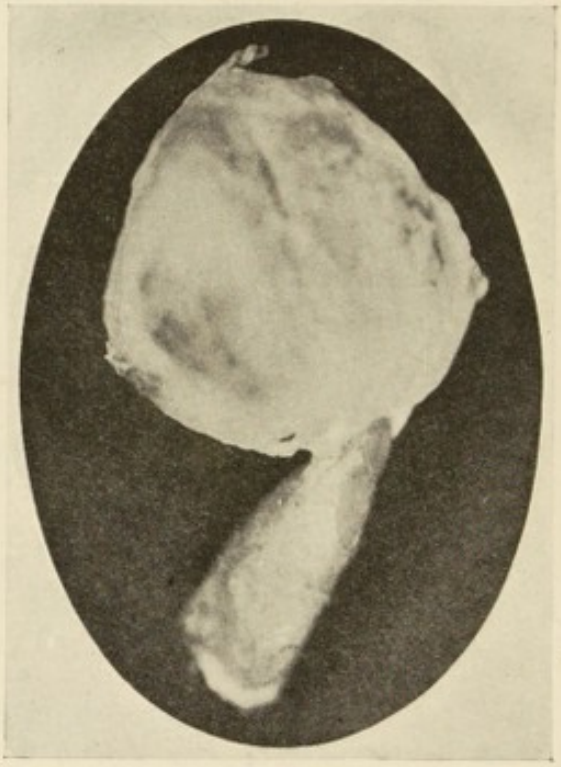


FIG. 50. Dental cyst attached to the root of a tooth.

loma'[1] or epithelial root tumour. The cells may be more or less scattered in a fibro-cellular stroma, or they may be tightly packed and arranged somewhat concentrically. These little tumours give rise to no symptoms, and are only discovered accidentally when a tooth is removed—if they happen to remain adherent to the root. Their significance and importance, however, lies in the fact that the cells having once started to proliferate they may either continue

[1] Oscar Römer, *Correspondenz-Blatt f. Zahnärzte*, 1899.

to do so or become cystic. In the latter case a '*dental cyst*' is formed which continues slowly to increase in size, expanding the bone, and eventually causing its complete atrophy. Fig. 50 shows an example of such a cyst. Owing to the slow growth the epithelial cells become backed as it were by a layer of cellular and fibrous tissue forming the wall of the

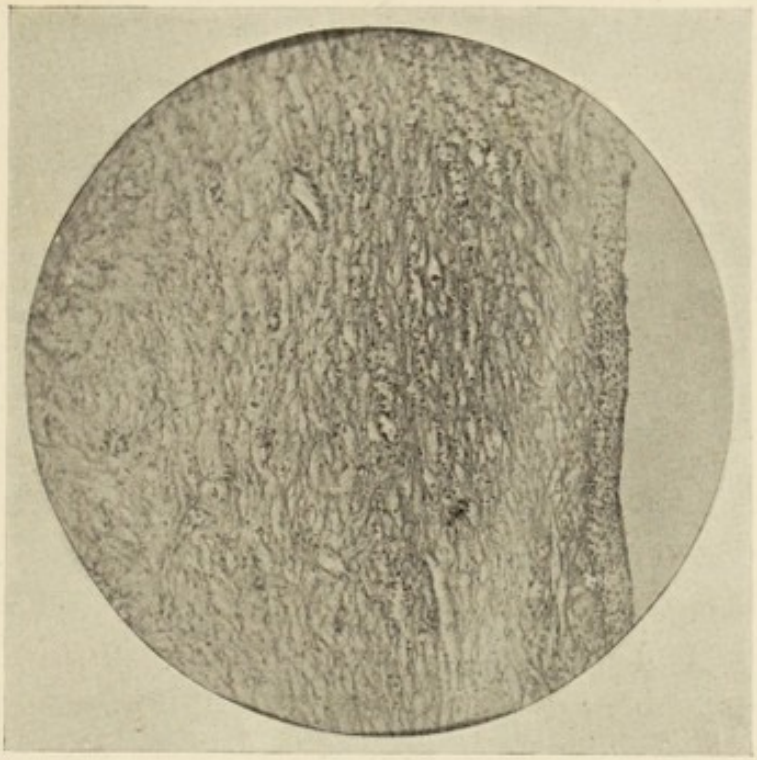


Fig. 51. Section through the wall of a dental cyst showing lining of epithelium (zahnleiste).

cyst. In Fig. 51 a section of the wall of a similar cyst is shown and the epithelial lining is clearly distinguishable. The epithelial lining in some cases may become ciliated. It has been suggested that 'dental cysts' may result from a chronic abscess sac becoming filled with serous fluid, but such 'cysts' are always very small and microscopically are seen to be quite inflammatory in origin.

Such cysts may arise at any time, though more frequently in early adult life; the symptoms are exactly similar to those of a follicular odontoma—the only difference being that in

this case the swelling usually commences somewhat later in life, and there is no history of an unerupted tooth. The teeth are present and usually septic—or indeed they may have been *extracted*, as in a case operated on by the writer in which a typical 'dental cyst' was present in a woman of 28 in the right upper premolar region; all the teeth had been extracted two years previously, but there was a distinct history of sepsis and broken-down teeth extending over several years. Another of the author's cases was bilateral in the same regions and no teeth were present, but the history was similar to the last case.

These cysts contain a yellowish glairy fluid with crystals of cholesterin suspended therein. By being opened they may become infected and suppurate. In the author's experience, however, this never amounts to very much; the opening either soon closes, and swelling of a non-inflammatory nature recurs, or if the opening is kept patent a communication between the oral and cyst epithelium is established, and a sinus without swelling results, which does not discharge pus but typical cystic fluid.

Of course if a part only of the cyst is removed that area suppurates, but it heals sooner or later, and the cystic discharge continues.

**Epithelioma** or 'Burrowing Zahnleiste'. If the epithelium of the zahnleiste 'rest' is stimulated to proliferate as stated above but the cells are scattered, or if they have more tendency to ingrowth than to become cystic, then they grow and proliferate in the form of epithelial columns which show only a slight tendency to become cystic, but whose chief characteristic is that all structures are invaded and eroded, especially the alveolar bone. 'Burrowing epitheliomata' are sometimes said to arise from the lining membrane of the antrum, but the majority of cases seem to occur in positions remote from the antrum.

The beginning of a zahnleiste epithelioma is shown in Fig. 52, where the epithelium of the resting zahnleiste is seen to be proliferating and growing out in columns and loops from the centre of a small tumour attached to the apex of a tooth.

In this case the tumour came away with the tooth, but it might have remained behind and a malignant growth would probably have resulted. It then might have been argued

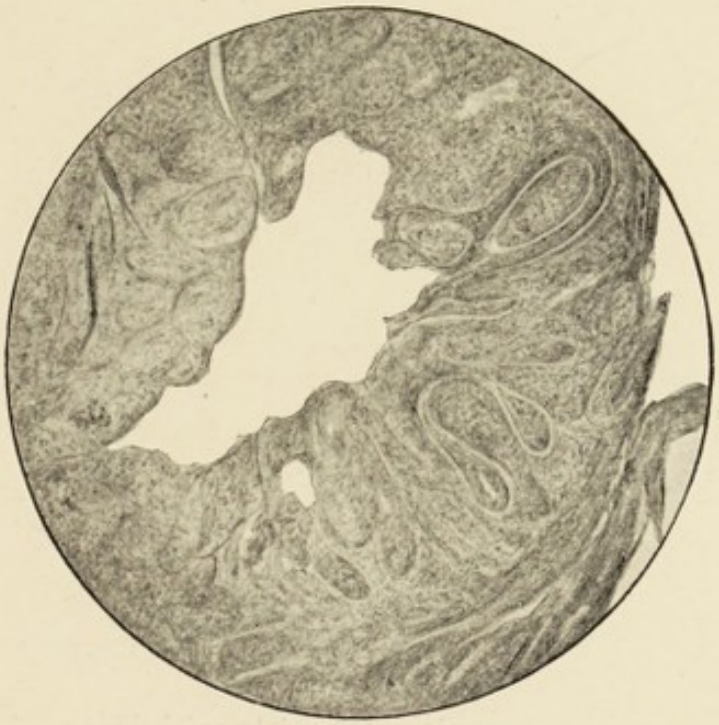


FIG. 52. Section through a small root tumour 'granuloma' showing proliferating epithelium—zahnleiste—invading the fibro-cellular tissue in loops and columns.

that the teeth had no causal connexion with the growth because none were present.

The structure, character, history, and course of these growths is essentially different from the usual epitheliomata of the surface origin. The cells resemble more the zahnleiste cells in size, shape, disposition, and direction of growth. They do not form typical cell nests: the central cells, instead of becoming arranged concentrically and becoming

keratinized, become enlarged, clear with well-defined margins and small nuclei, and show a tendency to break down and leave a clear or reticulated space (see Fig. 53), in these respects showing a noticeable resemblance to the stellate reticulum of the enamel organ.

The lesion *commences in the centre of the bone*, and not until it is well established are superficial signs observable.

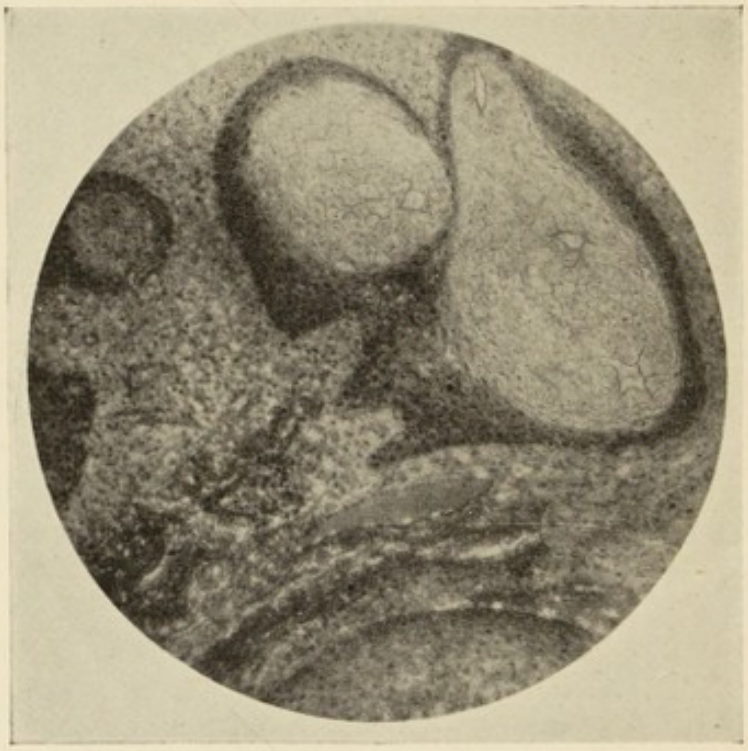


FIG. 53. Epithelial masses in a zahnleiste epithelioma. The tissue was taken from the centre of the maxilla in a woman aged 50.

Indeed the writer has seen several cases which had advanced to a very late stage before even the patient noticed or complained of anything. Yet in each case the whole of the bone in the neighbourhood was totally destroyed, and it was possible to pass a fine probe right through the jaw without meeting any solid resistance, and in one of them to pass it vertically from between the incisors into the nasal cavity. Such growths occur, as is usual, at 40 years of age or after, and there is always a history of long-continued sepsis of the peri-odontal ligaments.

There is *no superficial ulceration* in these cases primarily; the first thing the patient complains of is looseness of the teeth, and a dull aching pain; if these teeth are extracted the sockets do not heal, and then a somewhat suspicious ulceration may result; the growth becomes infected and suppurates. Glandular infection occurs late, but the growth once established spreads rapidly, and soon involves

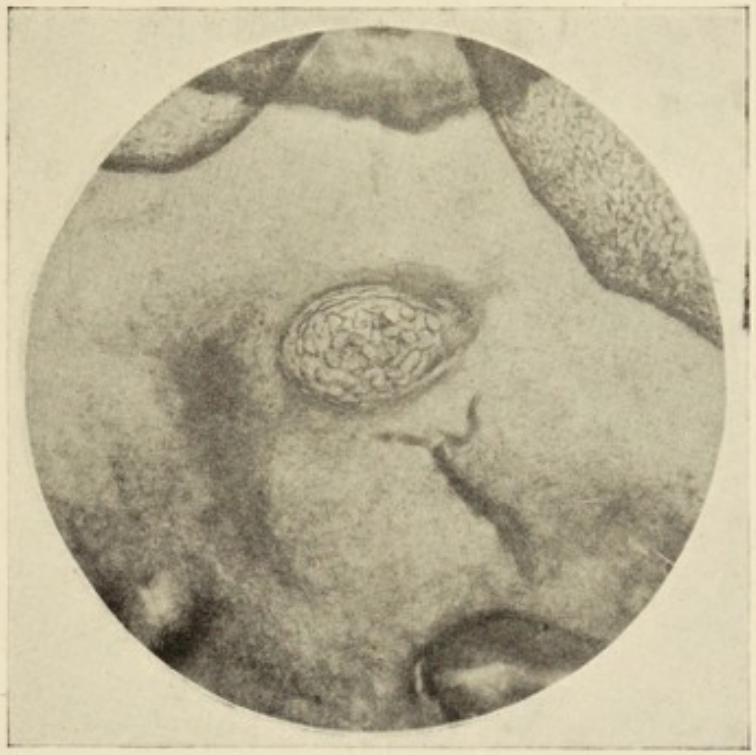


Fig. 54. Section from the jaw of a fœtal kitten, showing cells of zahnleiste cut transversely in the centre.

the antrum or the floor of the mouth and the tongue, and pain in this stage is often severe.

In all these odontomata just described the cellular elements are distinctive, and retain their resemblance to those in a normal tooth germ. Compare for instance the preceding illustrations of odontomata—particularly the epithelium—with Fig. 54, which is a section of the zahnleiste from a fœtal kitten showing similar 'cystic' cells.

**Composite Odontoma.** In this variety all the elements of

a tooth germ, epiblastic and mesoblastic, run wild as it were, and become inextricably mixed. In some cases the process of growth ceases sooner or later, and calcification sets in, in which case a mass of hard tissue results containing dentine, cement, enamel, and osseous material, extremely irregularly distributed, but not uncommonly the enamel is towards the centre of the mass (Fig. 55). The tumour may not give rise to any symptoms if calcification is complete before the growth has extended beyond the dimensions of a normal tooth, but when of larger size, ulceration of the surface may occur, and the mass may become virtually a foreign body and be enclosed in a suppurating cavity, under which circumstances a diagnosis of necrosis may easily be made. Spontaneous exfoliation has occurred. These tumours, of course, always occur in young adults, and the tooth or teeth represented by the aberrant tissues are missing. Symptoms are negative, except those in late cases due to inflammatory changes.

Such aberration of the whole tooth germ, however, may not calcify, and may continue to grow, in which case there is present an indefinitely proliferating epithelium, and indefinitely proliferating mesoblastic cells of the dentine papillæ and follicle; it constitutes, in fact, a mixed epithelioma and sarcoma. This form seems to have been first fully described by Eve, and is called by him 'Composite Embryoplastic Odontoma (Sarcoma)'.[1] The tumours are malignant, though they may run a rapid or a very slow course.

The following particulars from two cases quoted by Eve are illustrative of the conditions :—

(i) A very large central tumour involving the left half of the lower jaw was removed from a man aged 32. It had existed for eleven years. The mass of the tumour was

[1] *Brit. Med. Journal*, 1907, p. 152. But the term 'embryoplastic' seems not to be necessary, since all odontomata are formed from embryonic tissue which has not fulfilled its intended function.

a fibro-sarcoma, and imbedded in it were irregularly shaped masses and columns of epithelium.

(ii) A man æt. 67 was operated upon three times in two years for recurrent cystic tumour of the lower jaw. At the end of three years a large 'solid tumour' was occupying the site of the original cystic growth. This was removed, but the patient died three months afterwards with a recur-

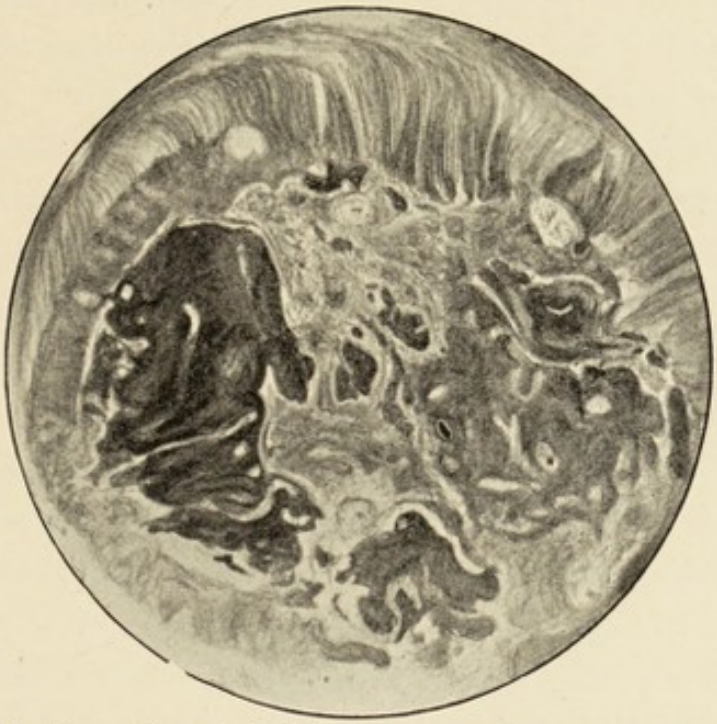


FIG. 55. Section of a calcified composite odontoma. There is a zone of dentine externally, and in the centre is a heterogeneous mass of bone, cement, and enamel.

rence in the skin of the cheek and metastases of round-celled sarcoma in the pelvis and right arm.'

**Fibrous Odontoma.** This might more logically than a 'dentigerous cyst' be called a follicular odontoma, for it is developed entirely from the follicle. It is, in fact, an excessive over-development of the tooth-sac at the expense of all the other elements of the tooth germ.

The fibro-cellular tissue of the sac commences to proliferate in a concentric manner at a very early age, and continues to increase concentrically at the periphery until a considerable

tumour is found. A tooth may be found in the centre, but usually all other dental structures than the follicle are almost completely obliterated, with the exception of a small mass of calcified cement substance in the centre. The whole of the growth is practically a fibroma, more fibrous at the centre, becoming fibro-cellular and cellular towards the periphery. The tumours are of considerable hardness, distending the bone at first, and causing displacement of teeth; later the bone is completely atrophied; they are usually well defined and encapsuled, but may not be so.

There should be a history of the non-eruption of a tooth, but this is not easy to obtain since the tumours are of very slow growth. Bland Sutton is of the opinion that rickets plays an important part in the formation of these tumours.

The recorded cases of this nature are not many, but it is not improbable that many cases regarded as endosteal fibromata are really fibrous odontomata. On one occasion the writer assisted Mr. Gilbert Barling in the treatment of a case of bilateral fibrous odontomata of the upper jaw in a young woman of 28, who stated that her brother (deceased) had always had similar swellings on both sides of his upper jaw. Both tumours were enucleated from within the mouth; the one on the left was the larger, measuring $2\frac{1}{4}$ in. $\times$ $1\frac{1}{4}$ in., and had almost completely obliterated the cavity of the antrum; the one on the right was smaller and not so definitely encapsuled. Microscopically they consisted of old and young fibrous tissue, with a small amount of central calcification which resembled cement (but might have been bone). The diagnosis of odontomata rested on the fact that they were bilateral and of very long standing—since early childhood; no connexion either with the periosteum or any peri-odontal ligaments; the family history, and their being in uncommon sites for ordinary fibromata. An equally interesting and important point is the sequel, for four years later a spindle-

celled sarcoma developed on one side close to the seat of the original tumour.

The late Mr. Christopher Heath [1] removed two tumours of this character from the lower jaw of a boy aged $7\frac{1}{2}$.

**Radicular Odontomata.** These are always calcified and arise after the crown of a tooth is complete. They consist of normal dentine or cement arranged in an irregular manner, they rarely attain any considerable size, and do not usually call for treatment. There is only one form, which the author has proposed to term '*medullary*',[2] which requires to be noticed here. This form is caused by an abnormal widening and thickening of the dentinal papilla, and the formation around it of a wall of dentine; thus the pulp cavity is abnormally large and 'ballooned'. This may give rise to a hard bony tumour of the jaw in the region of the root of the tooth, and if the latter becomes septic the tumour will be found to be hollow. The diagnosis, however, is made clear by the fact that by careful palpation it can be made out that the tooth-crown and the tumour move together, and are obviously rigidly connected.

**Diffuse Overgrowths of the Muco-periosteum and Bone** occur in the mouth and jaws, and only differ from similar innocent tumours in not being circumscribed. A number of cases of excessive and general hypertrophy of the oral mucous membrane in a pendulous and nodular form have been recorded. Microscopically these diffuse tumours are shown to be soft fibromata. In some cases, undoubtedly, they arise from chronic irritation and inflammation, and it has been suggested that the origin is always an infection; in one case it has been shown to be due to moluscum fibrosum.

[1] Quoted by Marshall, *Surgery of the Face, Mouth, and Jaws*, p. 670.

[2] 'Radicular aberrations,' *Proc. Roy. Soc. Med. Odont. Sec.*, July, 1909. (In the same communication is also described and illustrated a typical composite odontoma.)

Combined overgrowth of the alveolar bone and muco-periosteum is not infrequent in the region of the upper molar teeth. The outgrowth occurs chiefly on the inner or palatal aspect, and is usually bilateral, so that in severe cases the growths may approximate in the middle line and thus give rise to an appearance not unlike a cleft palate. A similar condition is occasionally seen in the front of the mouth in the upper incisor region. The outgrowth in this case is forwards and leads to much unsightliness, since the upper lip is pushed forwards and upwards, and a rounded mass of hyperæmic 'gum' is always visible.

Acromegaly and 'Leontiasis ossea' lead also to a diffuse overgrowth of the maxillary bones, but in this case the hypertrophy is limited to the bone, and is in the former case extremely regular in all directions. In the latter condition the growth is nodular but symmetrical and affects usually the external surfaces of the bones.

**Diagnosis of Oral Tumours.** It would not be possible here to give in detail the differential diagnosis for each possible tumour. An indication has already been given of a useful point in the site of origin, and a number of other points of principal importance are set out in the accompanying table on p. 185.

In addition to the sources there mentioned, acute abscess and necrosis have also to be kept in mind. Necrosis may simulate a malignant growth, and the latter an acute abscess. The history of the case is always important, particularly bearing on previous traumatism—recent trauma points to inflammatory change, distant to sarcoma. Occupation, too, is important in male adults.

Indications of malignant tumours are: rapidity of growth, neuralgic pain, displacement of teeth *usually accompanied by non-suppurative looseness*, the non-healing of the socket of an extracted tooth, and the presence of a tumour distending and eroding the bone, which is not

| *A 'Tumour' of the Jaw may be caused by—* | *Solid.* | *Fluid.* | *Superficial.* | *Central.* | *Expands bone.* | *Erodes bone.* | *Egg-shell crackling.* | *Fluctuation.* | *Inflammatory.* | *Tooth missing.* | *Tooth septic.* | *Secondary displacement of teeth.* | *Age of Patient.* | *Previous history or present appearance of tumour locality.* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fibroma (periosteal) . | + | | + | | | | | | secondary | | + | slight | any | Septic or broken down teeth frequently. |
| ,, (endosteal) . | + | | | + | + | | | | | | | + | ,, | |
| Myeloma . . . . . | + | | + | | | | | | secondary | | + | + | young | Mulberry-like — purple colour. |
| Angioma . . . . . | | + | + | | | | | ? | | | | | ,, | Frequent hæmorrhage. |
| Osteoma . . . . . | + | | + or + | | + | | | | | | | may be | early adult | No previous history. Chondroma very rare. |
| Chondroma . . . . . | + | | + or + | | | + | | | | | | ,, | ,, | |
| **Odontomata—** | | | | | | | | | | | | | | |
| Epithelial . . . . | ½ | ½ | | + | + or + | | | | | usually | | ,, | ,, | Tooth missing. 'X'-rays negative. |
| Radicular . . . . | + | | | + | + | | | | | | | slight | | Gradual swelling after eruption of teeth. No pain. |
| Composite . . . . | + | | | + | + or + | | | | | + | | ,, | | |
| Fibrous . . . . . | + | | | + | + | | | | secondary | + | | + | early adult | History of non-eruption of tooth. |
| Follicular . . . . | | + | | + | + | | + | later | secondary | + | | + | ,, | |
| Dental Cyst . . . . | | + | | + | + | | + | later | secondary | | + | | adult | Septic tooth—no acute pain. |
| Epithelioma (zahnleiste) | + | | | + | | + + | | | later | | | + | + 40 | Chronic peri-odontitis. |
| ,, (squamous) | + | | + | | | later | | | on surface | | | | + 40 | Non-healing ulcer. |
| Sarcoma (round, spindle-celled, and myeloid) | + | | | + | + | later | | | | | | + | young or old | Previous trauma—blows—incomplete extractions. |

completely solid and does not fluctuate nor give egg-shell crackling.

In squamous-celled epithelioma the submaxillary glands may be involved early, in epithelial odontoma and in zahnleiste epithelioma either not at all or very late.

A zahnleiste epithelioma may be diagnosed when a patient is forty or over, his teeth are loosening and painful without obvious cause, and when the bone is being eroded without much swelling and without superficial ulceration. Myelomata and fibromata (superficial) tends to separate the teeth, fibromata of the upper jaw involving the antrum give rise to considerable displacement downwards. Forward displacement of the upper teeth is usually due to malignant growth in the spheno-maxillary fossa or pterygo-maxillary fissure.

An *infected* dental cyst or follicular odontoma may be difficult to differentiate from an acute abscess; the history, however, of a persistent slowly-increasing swelling which was hard but is now soft, is against an abscess; moreover, some indication of expansion of bone can usually be obtained even in late cases, and in earlier cases parchment-like crackling is obtained upon pressure. Epithelial odontomata are partly solid and partly cystic, and usually distend the bone irregularly; they are uncommon.

The above four tumours constitute the fluid swellings.

The absolutely hard swellings are osteoma, fibroma (endosteal), chondroma, and calcified odontomata—spindle-celled sarcoma, too, is often quite hard to the touch: osteoma is more frequent in the upper jaw.

Differential diagnosis is of course important from the point of view of treatment; here we are dealing with a region in which extensive operation not only results in very serious loss of function, but also in considerable and obvious disfigurement. On the other hand it is a region in which it is highly important that a correct diagnosis should be made early because of the comparatively limited amount

of tissue that can possibly be removed, and on account of the rapidity with which a growth may pass beyond control.[1]

**Treatment.** Superficial fibromata and myelomata should be removed together with the subjacent periosteum and bone. Where they appear to spring from the peri-odontal ligament, the tooth and alveolus should also be removed to prevent recurrence.

Angiomata may be treated by galvano-cautery or electrolysis, but single or several ligatures are undoubtedly best if possible. Solid innocent tumours may be enucleated from within the mouth, and diffuse 'tumours' of the mucous membrane and bone must be freely excised. Follicular odontomata and dental cysts are to be opened up well, and the cyst wall curetted thoroughly; in fairly recent cases the lining will often peel out almost entire. In the author's opinion it is dangerous to attempt to treat these cysts with caustics, bearing in mind their epithelial origin and its tendency to proliferate and ingrowth; any remains of such a cyst may always be regarded as a possible source of epithelioma. The same applies to all odontomata containing uncalcified epithelial elements: they must be removed very thoroughly and as widely as possible.

Central myeloid sarcoma may be removed by taking out a rectangular piece of bone enclosing the tumour. Other forms of sarcomata and epitheliomata in other than quite early cases require usually the removal of that half of the mandible or the whole of the maxilla from which they spring.

In all cases where considerable portions of the jaws have been removed some form of obturator should be inserted as soon as possible, in order to prevent undue contractions taking place. Such an appliance is far more hygienic

[1] In this respect a trained dentist has many opportunities for the early recognition of oral diseases, and thus for the saving of life.

than a mass of gauze packing infiltrated with pus, saliva and decomposing food.

In the case of the lower jaw a stout gilded german silver wire may be inserted into the ends of the bones at the time of the operation in order to keep them apart, to preserve the position of the chin, and to prevent the dribbling away of saliva, which otherwise frequently occurs.

# CHAPTER XIV

## THE MANIFESTATION OF SYSTEMIC DISEASES IN THE MOUTH

### Effects upon the Oral Tissues

Many systemic diseases manifest oral symptoms during the acute stage and may leave permanent evidence of their effects upon the teeth ; especially does this latter apply to the exanthemata. These oral signs and symptoms are so common and, in many cases, constant, that it again suggests the advantage or necessity of a routine and *critical* examination of the mouth in all patients. Such conditions cannot be described in detail here ; they will be found scattered throughout medical and surgical literature, but indications will be given of the more important manifestations.

1. **Syphilis.** Signs of this disease may be observed in the mouth during all its stages.

The *Primary* infection may occur in the mouth, causing a chancre which may simulate at first an alveolar abscess, more especially if the infection is on the inner surface of the upper lip, since both this and an acute alveolar abscess in connexion with the upper incisor teeth cause swelling beneath the nose, more or less obliteration of the labio-dental sulcus, and the pain is somewhat similar. In a case of a chancre, however, the lip is more protruded, and the character of the swelling is not typical of an abscess, the teeth are not tender to percussion, and the submaxillary glands are enlarged more than they would be from an abscess. Attention is drawn to these points because of course a chancre

should be recognized *before* it is palpated. Ivanyi[1] states that out of 157 cases of extra-genital syphilis in 62 the lips were the seat of the original infection.

During the *secondary* stage, mucous tubercles—whitish elevated or ulcerated papillæ—appear on the mucous membrane of the cheeks and at the corners of the mouth or on the sides of the tongue, where they might be mistaken for a 'dental' ulcer. 'Snail-track' ulcers may be seen further back in the mouth, on the fauces and soft palate, and of course there is the characteristic sore throat.

During this stage there is considerable risk to the operator in performing any operation within the mouth (even the extraction of teeth); when this is absolutely necessary gloves should be worn if possible, and in all cases a gauze mask and spectacles should protect the lips, nose, and conjunctiva from exhalations and expectorations from the patient's mouth.

The *Tertiary* stage is characterized by the formation of gummata, and these, as is well known, frequently appear on the hard or soft palate, leading to necrosis and loss of tissue —the 'acquired' cleft palate. Such loss of tissue may be very slight or it may involve the whole of the palatal and alveolar processes of the maxillary bone.

As regards the treatment of syphilitic necrosis of the palate, many cases, if taken early and treated actively with mercurial mouth-washes, douches, and gargles, are arrested, and in some instances repair has taken place. More recently arrest of perforation and complete healing has been observed to follow the injection of salvarsan.[2] When the loss of tissue has been great the only means of treatment lies in the adaption of an obturator, Fig. 8. The peri-odontal ligaments and tissues are likely to be affected during this stage. Usually one or two contiguous

[1] *Pest. Med. Chir. Presse*, No. 24, 1910.

[2] *Brit. Med. Journ.*, February 18, 1911, p. 361.

teeth are 'selected'; they become painful at first, and then rapidly become loose. The tissue around them assumes *a soft dark-red velvety appearance*, sooner or later necrosis of the alveolar bone supervenes, and the teeth, with a sequestrum attached, come away.

The presence of loose teeth surrounded by such tissue as described is, the writer believes, quite sufficient to indicate a syphilitic history. Therefore, the ordinary local treatment in such cases is not sufficient by itself, but should be accom-

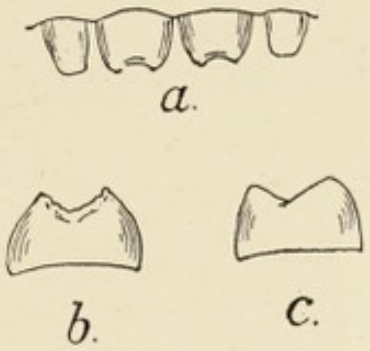



FIG. 56. Hypoplasia of the enamel of permanent teeth due to congenital syphilis. *a*, Upper incisors; *b*, First permanent molar; *c*, Normal unaffected molar.

panied by general systemic treatment, such as salvarsan or mercury, and potassium iodide.

**Congenital syphilis** gives rise in children to the presence of rhagades (*ῥαγάς*—a chink) around the angles of the mouth, and to the development of typically shaped incisors called 'Hutchinson's' teeth. This is due to the infection of the dental germ with the *Treponema pallidum* and to the consequent destruction or paralysis of the ameloblasts.[1] The upper incisors are narrower at their incisive edges than at the gum-line, and the central incisors show a crescentic notch at the cutting edge. The first permanent molars have dome-shaped crowns with ill-developed cusps (Fig. 56).

[1] *Syphilis in relation to Dentition*, by Dr. Joseph Cavallaro, Florence, Dental Cosmos., February, 1909.

Recently Cavallaro has placed a wider interpretation on what constitutes the dental stigmata of syphilis. He has shown that syphilitic children may have defectively shaped and developed teeth, which in many cases do not correspond to typical ' Hutchinson teeth '.

Although this may be so, the converse certainly will not hold, i. e. that all children who show such dental defects are of necessity congenital syphilitics. For instance, in New

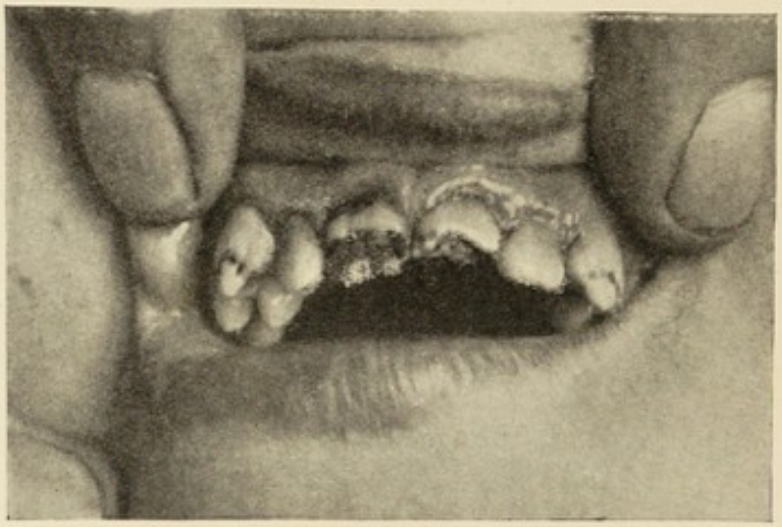


FIG. 57. Hypoplasia of enamel not due to syphilis. The first permanent molars were affected similarly.

Zealand congenital syphilis is comparatively rare, yet defective teeth such as are shown by Cavallaro are exceedingly common. The accompanying photograph (Fig. 57) illustrates a case of this kind. These teeth resemble those described by Cavallaro as syphilitic: yet they are not typical, it was absolutely impossible to obtain any history, but there was bulging of the forehead, a suspicion of rhagades and general under-development, so a Wassermann reaction was done with the patient's blood, and the result of this was negative; thus showing that the condition was not syphilitic.

Concurrently with the defects in the enamel producing Hutchinson's teeth, the dentine shows signs of irregular and intermittent calcification.

Dentition as a whole may be affected, thus the permanent teeth may either erupt very early or a varying number may be entirely suppressed: microdontism and diastemas (small teeth and spaces between the teeth) may be present. Fig. 58.

Dental stigmata, however, are usually associated with ocular, auricular, or facial lesions; they are rarely found alone; in Cavallaro's fifty-six cases this only occurred in three.

FIG. 58. Microdontism and diastemas in a congenital syphilitic. The patient never had any other teeth than these, either deciduous or permanent, and the lower teeth are exactly similar.

2. **Exanthemata.** In well-developed cases of Scarlet Fever there are always the well-recognized signs of hyperæmia, or inflammation of the fauces, and strawberry tongue; but in milder cases, which are often difficult to diagnose in the initial stages, it is well to remember that there may be a small punctiform eruption on the palate and mucous membrane of the cheeks when there is no rash visible elsewhere.

In fact, the oral symptoms are the most constant in the disease. There may be a general superficial stomatitis

or gingivitis, which may be simple, but may become so severe as to cause necrosis of the jawbone. This usually occurs in the region of the lower incisor teeth, which come away subsequently attached to the sequestrum. It has been suggested that this is caused or predisposed to by the use of the tongue depressor, when swabbing or spraying the throat, since this keeps the mouth forcibly open by pressing upon the lower incisor teeth. It may not always be possible to avoid such pressure, but its effect should always be borne in mind.

Scarlet fever with marked oral symptoms occurring in early childhood may leave a permanent effect upon the teeth, shown as either a sharp depressed ring running round the enamel, or as a pitted, honeycombed condition. Fig. 59.

In **Measles** the oral eruption is often visible before any other signs; this takes the form of small red patches with whitish centres, situated on the cheeks opposite the molar teeth; these are known as Koplik's spots; their presence is very constant, and is therefore valuable from a diagnostic point of view. In addition to such spots, however, there is practically always an acute gingivitis; the gum margins are red, swollen with a whitish scum, and this also occurs early, i.e. two or three days before the rash appears; it is, of course, not diagnostic, since it does not differ from any other acute gingivitis, but it is exceedingly suggestive where measles may be suspected, and if accompanied by a rise in temperature is sufficient to warrant isolation.

Measles affects the enamel organ in a similar manner to scarlet fever, leading to similar defects in the permanent teeth. Fig. 59.

It may be possible in such cases to form an opinion of the *age at which the illness occurred* from the position of the hypoplasic area in the enamel.

This is not a simple matter and depends firstly upon

the depth from the surface of the enamel, and secondly upon its superficial position being near the incisive or cervical margin. Roughly it may be said that 'healed' or covered hypoplasia in the incisive region (such as is seen in typical Hutchinson teeth) results from an illness prior to 12 months of age; open stomata or superficial hypoplasia in the same region is caused by diseases occurring during the second year, and similar defects in the cervical two-thirds result from illnesses between this and three and a half years of age.

*Small-pox* vesicles appear upon the soft palate, the buccal mucous membrane, and tongue early in the disease and

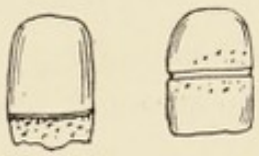


FIG. 59. Types of defective enamel due to the occurrence of exanthemata at various ages.

develop rapidly. This makes their observance of considerable value, since they can be recognized as vesicles at a time when the skin eruption is not sufficiently marked to be diagnostic. It is to be remembered, however, that similar but less numerous vesicles appear early in *chicken pox*.

During **Typhoid Fever** the mouth becomes quite characteristic, there is marked and almost complete loss of salivary secretion, the tongue becomes dry and brown, the mucous membrane dry, cracked, and fissured, the gums are swollen and glazed, and the teeth covered with dry yellow sordes.

Parotitis from infection via Stenson's duct is not unlikely to occur, and a number of cases of necrosis of the mandible have been reported; this latter may arise as part of the general infection, or from a local gingivitis or stomatitis becoming an acute periostitis.

3. **Rickets.** The effect of this disease upon the long bones, especially on those which have to bear stress and strain in

early childhood, is well known, but it is not so well recognized that a very similar effect may be produced in the mandible. That is to say, the horizontal ramus develops a downward 'bend' just in front of the angle; a frequent result of this is that when the permanent molars erupt they antagonize, but the incisor teeth do not, a condition termed mordex apertus, or open bite. Naturally in such cases the original under-development of the body tends to be aggravated by reason of the fact that the child can neither bite nor masticate its food.

The cause of such malformation may be either—

(i) Muscular action on comparatively 'soft' bone.
(ii) Lack of development of the ascending ramus.
(iii) Persistent mouth-breathing due to narrow nasal passages, and post-nasal growths.

Or indeed it may be, and usually seems to be, a combination of all three factors.

The eruption of the teeth is usually much delayed in rachitic children,[1] and hypoplasia of enamel of the pitted honeycombed variety is common, and the surface affected is usually large. A case is recorded (by Bokay) of severe and prolonged rickets in which at the age of twelve nine deciduous teeth were still present and no permanent teeth had erupted. But such delay as this is very uncommon.

**Addison's Disease.** Brown pigmented patches appear in the mouth as elsewhere, but these of course are not diagnostic, since similar patches are caused by other abdominal diseases, and by argyria. It is also said that a delicate black line may appear on the gums in this disease. The value of these patches is that if they are observed during the routine examination of the mouth they give a useful indication as to the direction of further inquiries.

**Diabetes.** There is as a rule marked salivary deficiency, which may be accompanied by stomatitis of varying intensity.

[1] In cretinism also dentition is much delayed.

The resistance of the tissues is lowered to bacterial agencies, and, the peri-odontal ligament being a locus minoris resistentiæ, a painless but rapidly destructive general arthritis is common. The pyorrhœa is marked, and the teeth are lost early.

**Scurvy.** Bleeding from the gum margins may be one of the first symptoms, but it may be absent; if not, the gums become congested, soft, and 'spongy', the peri-odontal ligaments are affected, the teeth become tender to bite upon, and in bad cases rapidly loosen and fall out. Submucous hæmorrhages in other parts of the mouth may occur.

**Purpura hæmorrhagica.** Here, too, the patient may first complain of bleeding into the mouth. This may be severe, filling the mouth with blood-clot, or it may be mild and resemble scurvy in being confined chiefly to the gums. The gums, however, do not swell, and the teeth do not become tender. In mild cases, when there is already present a chronic 'pyorrhœa', the condition may be mistaken for an acute suppurative gingivitis. The absence of acute pain and tenderness, and the presence of subcutaneous and submucous hæmorrhages in other parts of the body, should make the diagnosis simple. In these cases the breath has a characteristic and disagreeable heavy odour, which having once been experienced should always be recognized.

The possible effects of Gout and Rheumatism in causing a dental arthritis and degenerations of the pulp have been referred to previously.

'Gouty teeth' are sometimes spoken of, thereby being meant incisor teeth which bite edge to edge; this, however, is not due to any difference in development or structure, but simply to increased attrition, combined with, perhaps, the persistent use of acid beverages.

## Substances having an Injurious Effect upon the Mouth and Teeth

**Mercury.** It is unnecessary here to point out the effects of hydrargyrism, fortunately now quite rare. Certain people, however, have an idiosyncrasy for mercury, and small doses may cause a very painful gingivitis.

A case of severe necrosis of the mandible has been recently recorded in France, resulting from mercurial injections. It is important to remember that many teething or soothing powders contain mercury, and the constant giving of these to infants and young children frequently results in an ulcerative stomatitis.

It used to be held, too, that the dental stigmata of syphilis were due to the action of the mercurial treatment, and Bruck still holds that mercury is a frequent cause of leucoplakia.

**Lead.** The ' blue line ' seen in chronic plumbism is well known. The ' line ' is really a row of minute spots of precipitated lead sulphide stippled *in* the gums just below their free margins. It is necessary not to confound this stippled line with the bluish appearance caused by dark calculus on the teeth showing through the gum; also people who are exposed for a few hours to lead dust may develop a blue line *on* the gums, which of course is readily removable. The stippled blue pigmentation in chronic lead poisoning is not confined always to the gum margins, it may appear on the lips or cheeks, opposite the necks of the teeth. This is because the $H_2S$ from which the lead sulphide is formed, is developed chiefly from decomposing proteid material between and around the teeth; from this position, either as a gas or as ammonium sulphide, it invades the contiguous mucous membrane, which is either the gingival fold, or the lips or cheeks where they lie against the teeth.

A case of mild lead-poisoning with oral manifestations has been recorded from the lodgement of a lead shot in the

socket of an extracted tooth. In severe cases of lead poisoning an ulcerative stomatitis may develop.

**Charcoal.** People who habitually clean their teeth with tobacco ash or other form of charcoal, frequently tattoo their own gums by means of the bristles of the toothbrush and the powder. The appearance closely resembles the blue line of plumbism, and is not infrequently mistaken for it.

Workers in *brass*, *nickel*, *antimony*, and *bismuth* also may develop a dark line on the gums, which may vary in colour from a dark green to a bluish black. In the course of arsenic or bismuth poisoning, the mouth may become ulcerated.

**Iron.** In the form of ' tonics ' this is popularly supposed to injure the teeth. Dental textbooks as a rule deny this, and ascribe the injury to the disease for which the iron is given; with this, however, I do not agree.

Firstly, iron in nearly all its forms is a salivary depressant, and is therefore conducive to oral stagnation, and, therefore, disease. Secondly, the form in which iron is very frequently given is the perchloride, combined with a small quantity of hydrochloric acid; this may do much harm itself, since hydrochloric acid is a rapid solvent of enamel, and is not an active salivary stimulant; but what is more serious is that in dirty and septic mouths, if any perchloride remains, *nascent hydrochloric acid will be formed* by the action of sulphuretted hydrogen (which is practically always present in such conditions) according to the equation :—

$$2FeCl_3 + H_2S = 2FeCl_2 + 2HCl + S\downarrow .$$

When this has been neutralized by combination with the enamel (or saliva) the following type of reaction will occur in an alkaline medium :—

$$FeCl_2 + (NH_4)_2S = 2NH_4Cl + FeS.$$

Thus the sulphide of iron is deposited upon the teeth as a black stain so familiar after a ' course of iron '.

The hydrochloric acid formed in the above way is more dangerous than that originally contained in the mixture, because it is in small amount and *untastable*, and hence does not provoke any automatic protection whatever.

Therefore patients taking this form of iron should always be instructed :—

(i) To keep the mouth scrupulously clean.

(ii) To take the drug through a quill.

(iii) To take fruit or some other salivary stimulant immediately after the iron to neutralize its depressant effect.

**Phosphorus.** The fact that factory operatives in yellow phosphorus are liable to a disease called 'phossy jaw' is well known. This is a peculiar and distinctive form of necrosis of the jaw-bone which follows the entrance of phosphorus fumes through some oral lesion,[1] usually a carious tooth. It has been suggested that the necrosis is really tubercular, and that the phosphorus merely paves the way for infection by the tubercle bacillus, but there is no evidence of an increased amount of tuberculosis amongst workers in phosphorus.

The affection commences as a periodontitis, which becomes a localised periostitis of the jaw, suppuration occurs, and the tooth becomes loose, and if not extracted falls out, but the jaw condition continues. This of course is accompanied by a considerable amount of swelling and pain, and the sockets of the teeth which have been lost do not heal up. Necrosis of the bone occurs, and eventually a more or less large sequestrum comes away, which in some cases is covered with a peculiar deposit resembling pumice-stone.

Sir T. Oliver states that cases of necrosis of the jaw are not infrequently accompanied by the occurrence of fragilitas ossium in the long bones.

Since 1900 compulsory dental inspection and treatment of

[1] One case is quoted by Heath as having occurred in a child of six weeks old where the oral mucous membrane was apparently normal.

the factory operatives has been in force in England, and the effect of such prophylactic measures may be gathered from the following figures.

England: 1893–9 (6 years), 37 cases in 4,150 persons.

Belgium (no prophylaxis): 1896–1906 (10 years), 75 cases (official) and 160 (unofficial) in 4,611 persons.

England, since dental regulations: 1900–7 (7 years), 13 cases (six of these occurring in one factory where apparently the dental regulations had been inefficiently carried out).

Fortunately, however, in England and some other countries the condition will now become very rare owing to the use of yellow phosphorus having been made illegal.

# CHAPTER XV

## ORAL SEPSIS AND ITS EFFECTS

'None of these residents ever understood the meaning of oral sepsis.' 'They know of oral sepsis as an abstract statement, not as an actual potent factor, in the causation of every-day disease.'—Peter Daniel, *Lancet*, January 15, 1910.

THE general term oral sepsis includes any suppurating or septic lesions within the mouth, the chief of which are stomatitis, glossitis, gingivitis, suppurative dental myelitis, alveolar abscess, and dental arthritis—'pyorrhœa alveolaris'. Of these gingivitis, arthritis, and alveolar abscesses are the most frequent and potent causes of prolonged oral sepsis. In examining a mouth for oral sepsis a casual glance is of no avail and is often very misleading. The teeth may be quite black, and give a very dirty appearance to the mouth, and yet no oral sepsis be present; the discoloration being either due to accidental staining or to a condition of 'arrested caries'. On the other hand the teeth may look quite white and clean and yet a considerable amount of sepsis be present. There is some question as to whether simple carious cavities can be regarded as evidence of 'oral sepsis' since there is no inflammatory reaction to the bacterial invasion, but there is no doubt that the presence of many such cavities conduces to the vast increase of the number of organisms present in the mouth, and to the increased formation of the products of bacterial action upon the proteids of the *tooth substance*, and of food débris, and these must all be swallowed. Again, although the cavity may be a 'simple' one, yet the pulp may be infected and a localised suppuration may be taking place in it, although there is no visible communication

with the exterior. Fig. 60 is an example of such a condition, and since there is no exit for the products of this inflammation they must all be absorbed by the lymph and blood-streams. Oral sepsis in one or other of its forms is present in a very large proportion of patients; by far the greater number of all hospital patients at least exhibit it in a marked degree,

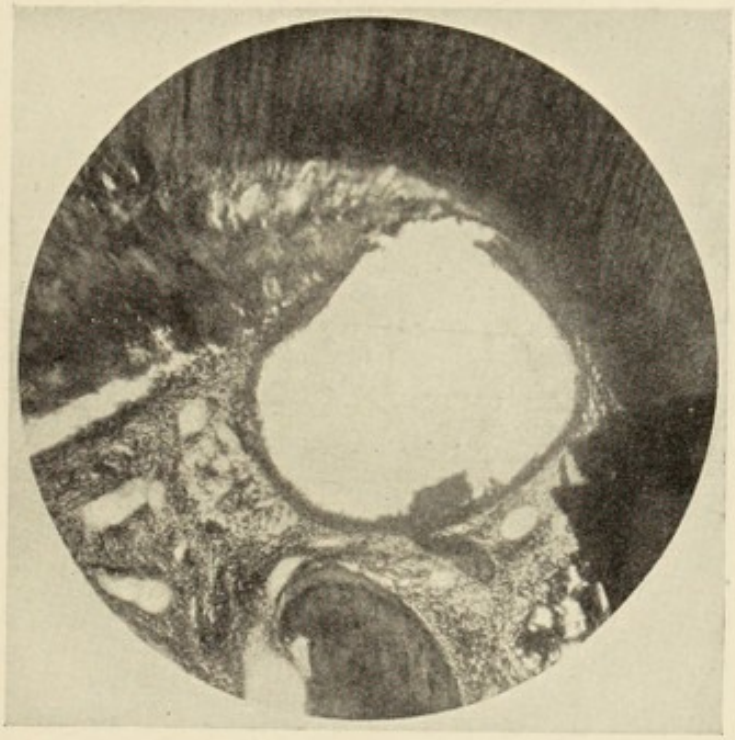


Fig. 60. Abscess cavity in dental pulp, there being no actual exposure of the pulp. Infection has travelled down the dentinal tubules.

and it is now exceptional to find a patient with a perfectly 'clean' mouth.

The secondary effects of oral sepsis upon other organs and systems may be produced in various manners as follows :—

i. Local extension to adjoining structures and organs.
ii. Absorption of toxins and organisms by the lymphatics.
iii. Infection of the mucous membrane at some point lower down the alimentary tract.
iv. Absorption of toxins and bacteria into the blood-stream.
v. Inhalation of organisms into air passages and lungs.

## I. Local Extension to Adjoining Structures and Organs

1. **Periostitis and Necrosis of the Jaw-bones** may frequently originate in small septic foci within the mouth, and the same applies to a *cellulitis* of the cheek. (Periostitis

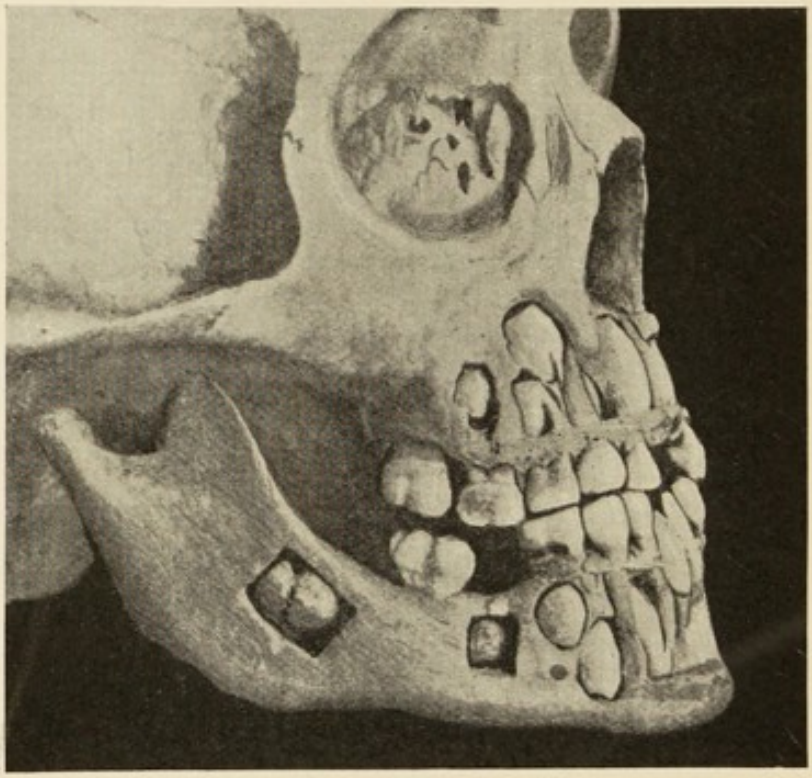


Fig. 61. Shrivelled-up, aborted, second lower premolar, due to septic infection from the deciduous tooth. (From a paper by Mr. J. G. Turner in the *British Dental Journal*, December 1, 1909, and reproduced by permission.)

and necrosis may arise, apart from sepsis, from an escape of arsenious acid for a dressing inserted in a carious cavity to devitalize the pulp of a tooth. A history of such treatment is, though, always forthcoming; moreover, the dentist who applied the dressing is most likely to see the case first.)

Sepsis in connexion with deciduous teeth frequently infects the subjacent permanent tooth germ, with the result that the latter may become necrotic and never erupt (Fig. 61). If the infection is not sufficiently severe to cause complete

necrosis of the permanent tooth, it causes a localised necrosis of ameloblasts, or an arrest of development, and this results in a pitted and honeycombed enamel. Prolonged suppuration in connexion with the permanent teeth always leads to loss of bone from the jaw, and in some cases this may be considerable, an instance of which is shown in Fig. 62.

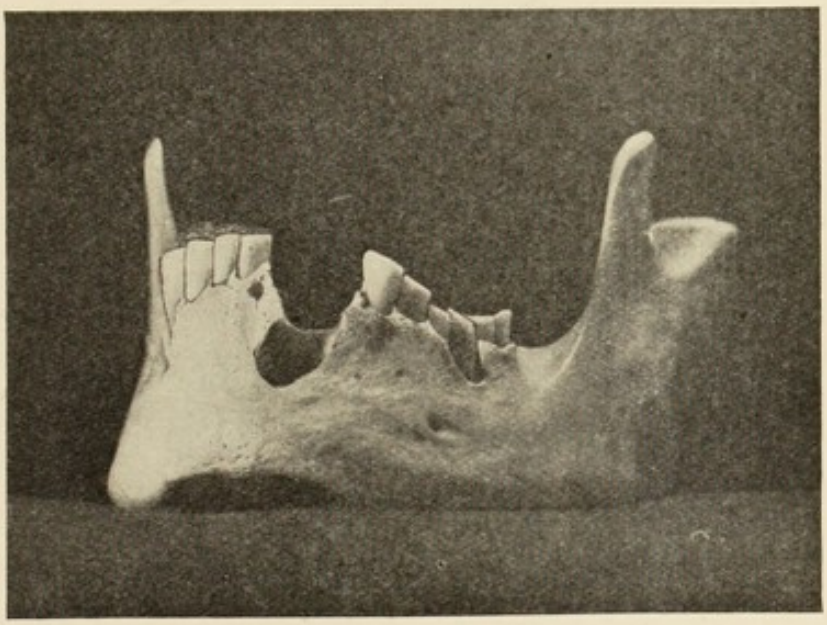


FIG. 62. Localised necrosis of jaw-bone due to septic dental infection.

2. **Angina Ludovici.** An intense cellulitis of the floor of the mouth and neck may result from a septic condition in connexion with the lower jaw. Suppuration around an 'impacted' (partially erupted) wisdom tooth or an alveolar abscess in connexion with the same tooth is frequently the original focus of the trouble.

The pus perforates the inner alveolar plate instead of the outer, because in this region it is thinner, and thus gets below the floor of the mouth or beneath the cervical fascia. Death may occur from septicæmia unless the condition is treated promptly. The cause of the condition should first be located and treated, and then an incision made and a drainage tube inserted in the most dependent part of the swelling. Hot fomentations should be applied externally,

and a hot antiseptic lotion used frequently in the mouth. Frequently much valuable time is lost and needless pain occasioned through making a small incision only and not at once removing the focus of infection (Fig. 63 shows such a case).

FIG. 63. Angina Ludovici, arising in connexion with a septic lower third molar.

3. **Empyema of the Antrum.** One of the most frequent examples of this mode of extension is infection of the maxillary antrum from a septic periodontitis, either on account of the roots of one of the premolar or molar teeth actually penetrating the floor of the antrum, or by means of the pus tracking through the thin intervening plate of bone. When this has occurred the best method of treating the empyema,

in the author's opinion, is through the socket of the tooth (which should of course be extracted). The socket is enlarged and free access is obtained to the antrum; it is syringed out with weak antiseptic solutions or a mild caustic if the case has become at all chronic. The orifice is kept closed by means of a solid vulcanite obturator which is smooth and polished; as soon as the discharge ceases the obturator can be reduced in length and thickness frequently so that the wound is made to granulate up from the sides and bottom. This seems to be a very rational mode of treatment, because the focus of infection (both bone and tooth) is removed and the antrum is drained from its most dependent portion. The obturator prevents any gross infection from the mouth, and if any other septic foci in the mouth are suitably treated and antiseptic mouth-washes ordered, there is no more risk of infection from the mouth than there would be from the nasal cavity, since the antrum is occasionally opened accidentally during dental and other operations on the jaws without any empyema resulting.[1] On the other hand the author has seen the worst results follow the use of tubes leading from the antrum into the mouth.

4. **Inflammatory Eye Conditions.** There is no doubt clinically that quite an appreciable percentage of cases of orbital cellulitis and periostitis owe their origin to a condition of oral sepsis, and only clear up in a satisfactory manner after the oral condition has been treated. Goulden[2] has recently reported several cases of irido-cyclitis, and one of panophthalmitis, in which the evidence is very strong in connecting septic conditions of the teeth with ocular disease.

[1] I have twice recently opened the antrum in this manner; on one occasion a small depressed portion of the floor of the antrum came away firmly adherent to and embraced by the roots of a first upper molar, and on the second occasion, whilst curetting a cyst, the instrument broke through the thin intervening bone. In neither case did the antrum become infected.

[2] *Proc. Roy. Soc. Med. Odont. Sec.*, February 1911.

And secondary to these conditions a papillitis or neuro-retinitis may arise. Cases of paralysis of ocular muscles arising similarly have been reported.

Infection may travel in these cases in several ways; it may be, and probably most frequently is, by ' local extension ' via either the perivascular lymphatics, veins, or muco-periosteum of the antrum. It is becoming increasingly common to find a ' latent ' empyema of the antrum in connexion with such ocular conditions, and the antrum is extremely likely to have received its infection from a septic oral cavity. Infection may travel via the general blood-stream, or it may be carried to the eye from the mouth by means of the fingers or handkerchief.

5. **Parotitis.** Inflammation of the parotid gland occasionally occurs during the course of some febrile disease. It is caused firstly through a decreased secretion of the gland owing to the pyrexia, and secondly to infection travelling up Stenson's duct from a septic mouth. Even if a mouth was not septic before, it rapidly tends to become so during any prolonged pyrexial condition, due to the lack of the normal oral secretions and to lack of normal function—the patient being upon a milk or ' slop ' diet.

In order to prevent this condition arising the patient's mouth should be kept clean by being washed out with weak antiseptics, such as Sanitas or peroxide of hydrogen (1 in 10), and a tooth-brush used regularly if possible. In addition, and most important, the salivary glands should be kept active by giving salivary stimulants; small quantities of fresh fruit should be given frequently if consonant with the general treatment, or the patient may suck some hard acid sweets (such as the old-fashioned ' acid drops ').

6. **Cavernous Sinus Thrombosis.** Not infrequently the focus from which infection spreads to the veins at the base of the skull is within the mouth and associated with septic peri-odontal tissues. The pus in these cases, instead

of perforating the external alveolar plates, passes upwards through the maxillary tuberosity to the pterygoid plexus of veins (which communicate freely with the cavernous sinus) or passes backwards and upwards from the mandible to the pharynx beneath the cervical fascia. An ascending thrombosis is then very likely to occur, and if serious infection of the cavernous sinus results a fatal termination may be anticipated. It is more than possible that such a source of infection is in many cases not recognized.[1]

## II. Absorption by the Lymphatics

It is very uncommon to see a case of marked oral sepsis without also finding evidence of lymphatic infection, as evidenced by the presence of either acute or chronic lymphadenitis in the submaxillary or upper cervical region.

The submaxillary glands are the ones most frequently affected, and next the cervical glands along the upper border of the sterno-mastoid. The parotid and submental glands are not enlarged so often from this cause. Lymphadenitis of this nature is most frequently seen in children as the result of alveolar abscesses, in connexion with the deciduous teeth, gingival ulceration, or stomatitis.

Since such conditions are very apt to be neglected in children, the lymphadenitis is correspondingly likely to become chronic, and as is well known chronically inflamed glands are prone to become the seat of tubercular infection. It is quite possible, indeed, that tubercle bacilli may infect the glands directly in this manner from the mouth, or of course the glands may become secondarily infected via the blood-stream from some other lower portion of the alimentary canal. But the experiments of Giliberti[2] support the

[1] Cases of this nature are recorded by Dwight and Germain, *Boston Medical and Surg. Journal*, 1902, p. 456 (34 cases). Warwick James, *Royal Dental Hospital Gazette*, December 1910. S. W. Milner, *Lancet*, November 12, 1910, p. 1416. Gordon Taylor, *Proc. Roy. Soc. Med. Odont. Sec.*, July 1911, p. 113.

[2] *La Pediatria*, December 1909.

former view. The cervical glands of 30 children dead from non-tuberculous diseases were excised, and guinea pigs (tuberculin negative) infected therewith. Of these 11 died of tuberculosis, 17 of other diseases, and 2 showed no sign of tubercle. From this it was concluded that the buccal mucous membrane may give passage to tubercle bacilli, and probably does so more frequently than is realized.

The association between oral sepsis and submaxillary or cervical adenitis is sufficiently common to suggest the rule that no form of treatment for the glands is complete without eliminating the possible source of original infection in the mouth (or it may be nasopharynx or tonsils).

### III. Infection of the Mucous Membrane at some Point lower down the Alimentary Tract

The first part of the alimentary tract being in an infected septic condition, and since the products of this infection must pass downwards through the whole length of the intestines, it might be inferred *a priori* quite logically that secondary infection would be extremely likely to occur, and that it would be situated at some such point where the resistance to the particular organisms was lowest. There is now abundant clinical evidence to prove that such indeed is the pathological origin of many gastro-intestinal lesions. It has been suggested that the buccal organisms would be destroyed by the gastric juice, but although this does take place it can only occur to a limited extent. The hydrochloric acid is not continuously secreted, and large numbers of organisms must be swallowed when no free hydrochloric acid is present in the stomach at all. Again, many of the organisms concerned in oral sepsis are spore forming, and even if they do come into contact with the gastric juice their spores escape. It is quite obvious that practically all intestinal organisms must at one time have come from or through the mouth. The infection of the intestines in tubercular,

typhoid, and anthrax ulcerations must have originally come through the mouth ; there may have been gross infection of food material, but on the other hand infection may have been slight and may have ' accumulated ' in the mouth, but owing to the higher powers of resistance of the buccal mucous membrane no local lesion has been caused.

Moreover, if the gastric juice sterilized all food material there would be no justification for lactic acid therapy. Hence it is obvious that organisms may pass from the mouth to the intestines without loss of function or virulence. Doubtless a certain degree of tolerance and immunity to the ordinary mouth bacteria is developed, but owing to an exacerbation of the oral sepsis, or to a temporarily lowered vitality on the part of the patient, such immunity sooner or later breaks down and the organisms produce a lesion at the most vulnerable point. The most common condition thus produced is *septic and chronic gastritis.* The following are two such cases reported by W. Hunter.[1]

A patient, a man aged 32, was admitted suffering from chronic indigestion and gastric trouble. His habits had been regular, his bodily physique good. There was no apparent reason why he of all men should be subject to gastric trouble. There was found the most intense septic inflammation and ulceration of the gums around a number of necrosed roots. After careful antiseptic dressing for several days to get rid of some of the septic infection, with immediate and obvious benefit to the patient, he ultimately consented to have the roots removed, and left the hospital in a week or two entirely free from all gastric trouble.'

The next case shows that infection may travel right through the small intestine and cause suppuration in the colon.

A very severe case of gastritis in a woman aged 33. She presented a dirty grey complexion and broken-down appearance. On examination she presented the most intense oral sepsis, ulceration of the gums, with large deposits of tartar covering the ulcers and extending up to and enclosing the

[1] *Lancet,* January 14, 1911.

teeth, many of these being quite loose in their sockets. She had swallowed this infection for years, and it had caused not only septic gastritis, but also septic colitis, for, as was duly noted, pus was present in the fæces.

Many similar cases of enteritis have been recorded, and since a catarrhal condition of the duodenum may spread up the bile duct, it is not improbable that such chronic infection may have a causal relationship to the formation of gall-stones. Undoubtedly, too, the present frequency of appendicitis is in a large measure dependent upon deficient mastication by defective and septic teeth.

The food is either swallowed in irregular masses, or to avoid this difficulty 'pap' food only is taken, in any case the food is infected seriously in the mouth, and peristalsis is deficient in the intestines; and, according to Sir Frederick Treves, the food often remains lying in the cæcum, where decomposition sets in. If the appendix is in the least injured all conditions for the disease are then present.

The mouth and buccal secretions are to the remainder of the alimentary tract as the first cog-wheels in an elaborate mechanism. Should the cogs of these primary wheels be broken or become coated with some corrosive material it is clear that the whole mechanism must sooner or later be disastrously affected.[1]

### IV. Absorption of Toxins and Bacteria into the Blood-Stream

Such absorption may occur either at the site of the lesion, and this is especially likely to occur if the bone is involved in an acute or chronic rarefying osteitis, or from the swallowed products of infection in the small intestine or stomach. Some doubt has been expressed as to whether ill results do follow the swallowing of toxins and organisms. It is evidently a question of dosage and im-

[1] Cf. Edkins' theory of hormones.

munity. The stomach and intestines are capable of dealing with a certain amount of toxin, but if the dose be considerable and long continued it is obvious that eventually a point must be reached when the toxins or organisms will be in excess of the antitoxins or antibodies. It is not a question of a 'few drops of pus' as a rule; as has already been pointed out, the suppurating area in the mouth may amount to several square inches, and the pus organisms and toxins are expressed from this more particularly during mastication; the toxins are therefore received into the intestine when absorption is going on; we know they are absorbable, the logical conclusion therefore must be that the toxins together with food material are absorbed. If such chronic suppurative lesions were present elsewhere in the body to a similar extent and for a like length of time, toxic results would be anticipated with assurance. Amyloid degeneration of the internal organs would be expected to occur, and although I am not aware that up to the present time lardaceous (or amyloid) disease has been definitely associated with oral sepsis, there is no reason to doubt that it must occur and only requires recognition. For such toxins as are absorbed from the upper part of the small intestine must pass through the liver and eventually be excreted by the kidneys, and it is impossible to conceive that the constant passage of an appreciable quantity of toxin through these two organs can be without effect upon them.

**Chronic Toxæmia.** This is the commonest and most frequent result of oral sepsis, it is likewise the one which is least connected by the patient with chronic oral disease. The symptoms are a dry earthy or sallow complexion, loss of appetite, furred tongue, constipation with occasional diarrhœa, headaches and debility—*an increasing inability to do the usual normal day's work*—together with loss of body weight in prolonged cases.

This group of symptoms is common, they constitute an

ailment which patients only too often imagine they are quite capable of treating themselves by means of 'pick-me-ups', pills, or alcohol. Such ailments are too often regarded as trivial in the outpatient department of a hospital, yet it is just such cases as these which will occupy a large part of the student's time after he has qualified. The first and foremost thing to be recognized is that they are *symptoms merely*, and next that the cause has to be discovered and eradicated, and that if this be not done, although one or other of the symptoms may be relieved, yet the disease remains and will in time give rise to other and more serious manifestations.

Not infrequently the chronic toxæmia of oral sepsis is present concomitantly with some other disease, and to what extent this toxæmia has paved the way for the second infection may not be certain, but it is absolutely certain that its presence does considerably retard recovery. Particularly is this the case with early phthisical patients.

Patients before they are sent for sanatorium treatment should have every trace of oral sepsis eradicated, or the 'pure' air which they are to breathe and the 'nourishing' food they are to eat will be contaminated and poisoned in a gross manner before either can be absorbed. The author sees a large number of sanatorium patients, and the improvement in such cases after the mouth has been treated is always very marked; the patient loses a good deal of his anæmia and begins to put on weight at once.[1]

A case of typhoid seen recently in an old man had drifted into the subacute and listless stage. It was then found that he had very marked oral sepsis; this, in spite of the

[1] I am convinced that many thousands of pounds are wasted annually on the sanatorium treatment of phthisical patients who cannot possibly benefit by such treatment on account of their physical inability to get either pure air or pure food. Patients before admittance to the sanatorium for this district have now to be passed by me, and I would suggest similar regulations elsewhere.

patient's conviction that he was going to die and that it was no use, was successfully treated. In a week's time the temperature had fallen and was steady, the patient bright, happy and very obviously less emaciated, and, moreover, enjoying his food. (The difference was so marked that I failed to recognize the patient in the ward owing to his altered appearance.)

**Arthritis.** It is now quite clear that there is a definite connexion between septic absorption from a local infection and some forms of arthritis; and suppuration within the mouth is frequently the source of the infection. The arthritis is usually of the 'rheumatic' variety, and may wander from joint to joint. It is occasionally acute and accompanied by pyrexia.[1] Eyre and Payne,[2] discussing the bacteriology of 'pyorrhœa alveolaris', state that in 24 out of 38 patients suffering from this disease so-called 'rheumatic' pains were present, 7 out of 24 had definite signs of osteo-arthritis, and that these joint-pains disappeared in nearly every instance after vaccines made from the oral infection had been injected. In other cases a similar result has followed the surgical treatment of the local oral condition.

(We have previously stated that 'pyorrhœa alveolaris' is really a septic arthritis of the joint between the teeth and the maxillæ. It would seem, therefore, to be in accord with our knowledge of organisms that having caused an arthritis in one locality they should, if absorbed into the general circulation, cause similar lesions in similar tissues in other localities, i.e. arthritis of other joints.)

**Anæmias, Septic and Pernicious.** Apart from the anæmia associated with a chronic toxæmia, oral sepsis frequently accompanies or is antecedent to a severer form of anæmia, which may be classed according to Dr. W. Hunter as either

[1] See case reported by C. W. Smith and A. G. Barnes, *British Med. Journ.*, September 18, 1909, p. 740.

[2] *Proc. Roy. Soc. Med. Odont. Sec.*, February 1910.

'septic' or pernicious. The following is such a case—regarded as one of septic anæmia.[1]

Patient aged 64. Red corpuscles 41 per cent., hæmoglobin 40 per cent., leucocytes 5,000. No history of loss of blood. I found he had only five teeth, markedly septic with two small necrosed roots buried in the gum of the right upper alveolus, the gum margin and alveolus being much thickened. The right cheek was somewhat swollen and puffy, with some pain in the left side and tenderness of the last molar.

I had the five teeth extracted with difficulty owing to chronic osteitis. Ten days later he had '75 per cent. of corpuscles and 75 per cent. of hæmoglobin', and two months afterwards 'he had 91 per cent. of corpuscles and 91 per cent. of hæmoglobin and was looking in good health'.

The following are two similar cases coming under the author's notice.

The first was a typical case: lemon-yellow complexion, muscular weakness, malaise, loss of appetite, &c., and marked oral sepsis. Red corpuscles 1,700,000. Hæmoglobin 41 per cent., Poikilocytosis, erythroblasts and megaloblasts present. The patient was seen early, the oral sepsis treated, and arsenic given in increasing doses, the red corpuscles rose to 3,800,000, and the hæmoglobin to 65 per cent. He made a gradual recovery.

The second case did not come under treatment until both the anæmia and the oral sepsis were in an extremely advanced condition. The only hope of rapidly eliminating the oral infection was by radical surgical treatment; but this was objected to, it being thought that the risk of hæmorrhage and of the anæsthetic would be too great. Swabbing with antiseptic lotions, therefore, was only adopted and arsenic given internally, but in spite of this the patient went rapidly down hill and died.

**Septicæmia.** As has already been indicated, a fatal septicæmia may and does result from acute cases of oral sepsis, or from chronic cases with acute exacerbations such as periostitis and necrosis. The septicæmia from gangrenous stomatitis is of course well known. Several cases of death

[1] For this and other similar cases see Dr. Hunter's paper, *Lancet*, February 14, 1911.

from this cause, originating in suppuration around the lower wisdom teeth, have come under my notice. The following is what might be termed a fulminating case of this kind.[1]

The patient, a middle-aged woman, presented herself complaining of pain in an upper incisor tooth. A gangrenous pulp and septic peri-odontitis were found to be present, and these were treated in the usual manner by evacuation via the root canal. The following day intense cellulitis of the face was present. A deep incision was made in the labio-dental sulcus and subsequently (later in the day) both central incisors were extracted. A small quantity of pus only was found, and this on examination proved to be ' an almost pure culture of streptococci ', on the next day ' large doses of antistreptococcic serum ' were administered, but in spite of this the patient developed a hyperpyrexia, became delirious, and died at midnight on the same day.

It is not improbable that since septicæmia may arise from oral lesions, a malignant endocarditis may also be caused in a similar manner by the organisms having entered the blood-stream infecting the valves of the heart.

At least, infection in such cases must come from some septic focus, and in one case post-mortem I obtained cultures of the same organisms from a septic mouth, the bronchi, alveoli of the lungs, and valves of the heart.

Such a case strongly suggests the next mode of infection.

### V. Inhalation of Organisms into the Air-Passages and Lungs

The most favourable opportunity for this to occur is during anæsthesia, especially if the breathing is at all spasmodic, or if the tongue is allowed to fall back and obstruct the air-way. On recovery from the obstruction the patient takes a deep inspiration through the mouth, not the nose, and infection in the shape of pus, organisms, septic saliva, or blood is drawn into the larynx. During chloroform anæs-thesia too the ciliated epithelium is not working normally,

[1] Recorded by M. E. Denniston, *N. Z. Dental Journal*, October 1909.

and the infection thus finds its way more readily down into the lungs.

The frequency with which cases of *septic pneumonia* follow operations on septic mouths is only too well known.

It is not improbable that a similar infection occurs during the deep inspiration of yawning or coughing, and now that pneumococci are frequently found in cases of oral sepsis, it is exceedingly suggestive that the infection of acute lobar pneumonia may arise in some cases from this source, especially when the vitality of the patient happens to be, through stress of work, at a low point.

**Recognition of Oral Sepsis.** The mouth of every patient should be systematically examined for evidences of sepsis. The lips, cheeks, and floor of the mouth should be examined for ulcers; the labio-dental sulci for evidence of chronic alveolar abscesses in the shape of either discharging or healed sinuses (a healed sinus is always likely to break down again, and there will probably be an abscess cavity in the centre of the bone all the time); the gums should be gently pressed upwards towards the crowns of the teeth, to express the pus of a chronic gingivitis and arthritis: teeth with suspiciously large cavities should be percussed for evidence of peri-odontitis.

The tongue and fauces of course must also be included.

Palpation of the submaxillary region may reveal enlarged glands, and thus give a history of past infection, although none may be present at the time; in a similar manner a history of the early loss of teeth, especially if they have 'dropped out', is presumptive evidence of prolonged oral sepsis.

Such an examination may be made quite rapidly, and the results amply justify its being made as a routine matter in all cases.

**Treatment of Oral Sepsis.** This must vary and be suitable to the exact condition present, and the means of treating many of the possible conditions have already been indicated;

they may be summed up as application of drugs, vaccine treatment, and surgical measures. But it cannot be too emphatically stated that the mere application of antiseptics, or even the chewing of sponges (as has been suggested for phthisical patients with marked oral sepsis), is utterly futile alone to eradicate or adequately treat cases of sepsis where the alveolar bone is involved, as it is in septic arthritis and alveolar abscess. In addition to appropriate surgical measures, prophylactic treatment should be instituted to prevent recurrence and to obtain a normal tone of the oral mucous membrane. Such measures should include: active mastication of coarser and more fibrous food material, an 'acid' dietary as far as possible, the use of salivary stimulants, and artificial prophylaxis by mouth-washes, toothbrush, massage with iodine, &c.

The secondary condition tends to clear up spontaneously after elimination of the focus of infection, but in order to aid recovery, saline aperients and tonics (either arsenic or iron and quinine) should be given. Fresh air and good food must be obtained, and graduated daily exercise undertaken in the open air.

# CHAPTER XVI

## THE RELATIONSHIP OF ORAL DISEASE TO VARIOUS LESIONS OF THE NERVOUS SYSTEM

### I. NEURALGIAS. II. REFLEX AFFECTIONS

### I. NEURALGIAS

The term neuralgia is applied to any severely painful sensation associated with the nerves in any part of their course. It is, unfortunately, frequently used as being synonymous with a definite disease of the nerve itself, and popularly of course 'neuralgia' is recognized as a definite and distinct affection or disease. It should be recognized, however, that in the great majority of cases '*neuralgia*' *is a symptom merely*, and not a disease.

Neuralgia may of course be due to lesions of the nerves themselves, such as a neuritis or degenerative or other changes, or, in its most severe form, tic-douloureux (or trigeminal neuralgia); there are no morbid changes discoverable to account for the pain. This condition is classed as Neuralgia quinti major by Head, and is regarded as 'a definite disease of the nervous system with a distinct course and character'. That is to say, it is considered as not due to any reflex peripheral irritation. In this condition the pain follows the course of one or other branches of the fifth nerve. It begins insidiously, often as pain in the jaws, or around one or more teeth; it may then be mistaken for odontalgia, but when this is treated according to the usual methods the pain is unrelieved and continues to increase in severity and frequency. Paroxysms occur periodically, and patients

frequently find it extremely painful to move the jaw or tongue, either in eating or speaking. Superficial hyperæmia, lachrymation, and sometimes salivation occur during these attacks, which may come on quite suddenly and without any apparent cause. A paroxysm requires to be seen in order to appreciate its severity, and then it will neither be forgotten nor mistaken in future for anything else.

The pain in neuralgia major is frequently so closely associated with the teeth and jaws that the patient may demand the extraction of perfectly sound teeth, or the removal of certain portions of the mucous membrane. To yield to such requests, however, is like following a 'Will-o'-the-wisp': the pain is relieved temporarily, but in 24 or 48 hours it recurs with all its intensity in some other region.

The only satisfactory method of treating such conditions in the past has been by excision of the Gasserian ganglion, but this is a serious and difficult operation with a high mortality.

More recently the injection of 90 per cent. alcohol by means of a hypodermic syringe with special needles into the main trunks of the nerve at the foramen rotundum and foramen ovale has been practised with success, particularly by Schlösser.[1] It has been shown that degeneration of the nerve fibres follows such an injection, so that the method practically amounts to a chemical resection of the nerve. The amount of penetration by the alcohol varies somewhat, and the pain may recur in a few months or in two or three years, when of course a further injection can be made. From personal experience of this method the author can say that if the nerves themselves are properly injected the pain, even in the worst cases, is most satisfactorily eliminated. The method, however, is difficult and requires considerable

[1] See *Lancet*, May 8, 1909, and *Brit. Med. Journ.*, 1910, p. 1404, for papers by Dr. Wilfred Harris on the subject.

practice. General anæsthesia may or may not be necessary, depending upon the type of patient.

*The technique of the operation is as follows*: A hypodermic syringe, 20 to 30 minims capacity, fitted with needles 2½ to 3 inches long and from ·8 to 1·2 mm. in diameter, is used. (A wire stylet is recommended, but this the author has not found necessary.) For the foramen rotundum the needle is inserted in the angle between the malar bone and the coronoid process of the mandible, and is passed slightly backwards and upwards at an angle of about 40° into the spheno-maxillary fossa; at a depth of 2 inches (about) the needle should strike the nerve at its exit from the foramen. For the foramen ovale, the needle is inserted 1 inch in front of the condyle of the mandible and about ½ inch below the zygoma, it is passed very slightly upwards and backwards and should engage in the foramen ovale at a depth of 1¾ inches. The patient, if not anæsthetized, experiences a characteristic sharp pain when the nerve is pierced. A few minims of 2 per cent. sterile beta-eucaine may be injected first, or the 90 per cent. alcohol may contain cocaine in the strength of 1 per cent.; of this latter 20 to 25 minims are injected into the nerve. Anæsthesia of the regions supplied by the nerve should result. The attendant risks are those of penetrating too deeply—into the optic nerve, carotid artery or cavernous sinus.

**Neuralgia minor** is frequently if not always symptomatic, or secondary to some disease of surrounding or distant parts. It, however, may closely simulate true tic-douloureux, especially in the early stages, and it may then be impossible from the symptoms to say which form is present.

It does not accord with our purpose to discuss the causes and treatment of all forms of neuralgia here in detail, but merely to point out their frequent association with oral diseases. 'Neuralgia' of this kind may be set up by any irritative condition of the terminations of the fifth nerve, and may be referred to other branches, and also to other nerves and more distant organs.

The most common oral conditions which may originate the neuralgia are chronic osteitis, dental arthritis, myelitis, or peri-odontitis; irritative lesions of the tongue and salivary glands are also to be included. The pain may be paroxysmal in character, and may wander and be referred to other

branches of the nerve, but it usually commences at the site of the lesion causing the condition. In true neuralgia minor the distribution of pain corresponds to the distribution of the affected nerves, whilst in another form, 'visceral referred pain,' the pain is referred, according to Head,[1] to definite peripheral areas which do *not* correspond with the peripheral distribution of the affected nerves. These areas

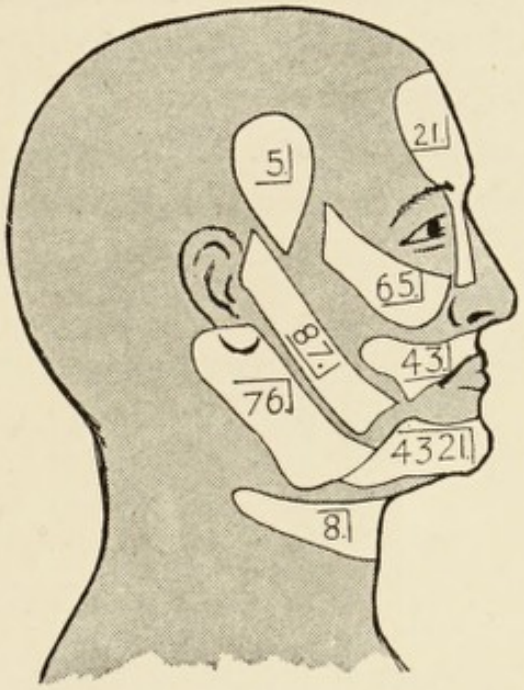



FIG. 64. Areas of superficial tenderness corresponding to lesions of the different teeth indicated. (For the meaning of the figures see Appendix I, p. 254.)

become hypersensitive during the attack of 'neuralgia'—pressure with the head of the pin becomes a painful sensation; they are fairly definite in outline, and correspond to definite points of stimulation within the mouth, and are, therefore, valuable from a diagnostic point of view when the causal lesion is obscure or difficult to locate. The accompanying diagram[2] (Fig. 64) shows the areas which correspond to stimuli arising in the various teeth. Neuralgia of the dental and oral branches of the fifth nerve may be caused by any

[1] *Trigeminal Neuralgia*, by Dr. Head; Allbutt's *System of Medicine*, vol. vi, p. 724.

[2] Modified from Allbutt's *System of Medicine*.

lesion which implicates the nerve in any part of its course, such as tumours (usually malignant), gummata, ossification of canals or foramina ; or owing to the proximity of the inferior dental nerve to the third lower molar ; ' impaction ' or sepsis of the latter usually causes pressure on the nerve-trunk. Pressure may also be made upon the Gasserian ganglion by intracranial tumours, and cerebral scleroses or degenerations may affect the deep centre of the fifth nerve.

In any case of neuralgia it is well to have some definite plan of examination in order that a diagnosis may be arrived at by a process of elimination, and no possible source overlooked ; the following scheme is suggested :—

Sources of Neuralgia.

*Peripheral.*
1. Caries of teeth—myelitis, periodontitis, arthritis (' pyorrhœa alveolaris ').
2. Inflammatory conditions of mucous membrane of mouth, nose, eye. Errors of refraction.
3. Periostitis, chronic osteitis (causing ascending neuritis).

*Trunk.*
4. Impacted or septic third molar.
5. Carcinoma or other tumour.
6. Gummata.
7. Ossification of canals—callus at base of skull.

*Central.*
8. Intracranial tumours.
9. Scleroses and degenerations.

The examination of the teeth in particular should be made very thoroughly by means of a mirror and probe. The reactions of the teeth to heat and cold and electricity should be ascertained in doubtful cases, and radiographs are useful in chronic bone conditions.

Very trivial peripheral lesions are sufficient in some individuals to set up a condition of neuralgia minor, and the more especially if the patient is suffering from anæmia or debility. Gout, plumbism, diabetes, and ' rheumatism ' also predispose to its occurrence ; with regard to hypersecretion Sir Victor Horsley is of the opinion that when this is definitely associated with the onset of pain, the condition is to be regarded as ' organic ' and not ' functional '.

A case illustrating the effect of callus or cicatrices in producing neuralgia is the following :—

A man, aged 56, presented himself complaining of severe pain in the region of the right lower molar teeth for which no adequate local cause could be found; moreover, the pain was atypical. The patient was noticed to have a squint, a paralysis of the external rectus, and a history was obtained of a fall from a train two years previously. The right side of the head was injured, and he was unconscious for one or two days (and on recovery he noticed that he had a squint). A diagnosis was therefore made of injury to the base of the skull, which divided or paralysed the sixth nerve with subsequent involvement of the Gasserian ganglion, or nerve trunk, in repair tissue.

Errors of refraction may cause neuralgic pain in one or other branch of the fifth nerve, and similar pain may be referred from a remote organ such as the uterus or stomach, as in the case of the girl with dysmenorrhœa and dental pain, recorded in the footnote on p. 82.

An interesting case of this nature has come under the observation of the author :—

The patient, a man aged 60, had severe paroxysmal neuralgia in the region of the mandibular nerve ; in type and position it was suggestive of pressure on the nerve by a wisdom tooth, but examination and radiograph failed to reveal any peripheral lesion whatever in the mouth. He was sent into hospital for observation and treatment, but on the second or third day he was unable to pass urine, and an examination showed a greatly enlarged prostate. This was enucleated, and the neuralgia of the fifth nerve at once completely disappeared.

These two cases are interesting in view of the fact that the sinus prostaticus in the male is the analogue of the uterus in the female.

Conversely diseased conditions of the mouth and teeth not uncommonly affect remote regions such as the ovaries and stomach, causing neuralgia, gastralgia, nausea or vomiting, or the brachial plexus may be implicated. The author

has seen three cases of brachial neuralgia definitely associated with dental lesions. In each case pain started in the lower premolars, and then, leaving the tooth, became transferred to the shoulder and arm, and only yielded to extirpation of the affected dental pulps. The pain in all these cases was confined to the region of the axillary and cutaneous branch of the radial nerves, and gave rise to considerable reluctance to move the arm.

**Treatment.** This should always be primarily directed towards removing the cause, and secondly to improving the 'tone' of the patient's nervous system by treating any anæmia or debility by change of air, rest, and administering iron or arsenic. Other systemic diseases or diatheses should be appropriately treated.

Palliative measures may be taken in acute cases; butyl-chloral hydrate, gelsemium, phenacetin, and acetanelid[1] may be found useful in modifying or relieving the paroxysms of pain. Injections of cocaine may also be used for the same purpose; but such drugs do not cure the condition, they merely relieve (and perhaps 'mask') the symptoms. When these measures fail, and when it is difficult or impossible to locate the originating lesion, treatment by the injection of alcohol into the nerves at the infra-orbital and supra-orbital foramina, or injection of the mandibular and maxillary nerves at their exits from the skull may almost certainly be employed with success.

## II. REFLEX AFFECTIONS

The diagnosis of 'reflex irritations' was often resorted to formerly to explain symptoms for which no other adequate cause could be found, but which with a more extended knowledge of the special pathology of the several regions concerned are now otherwise explained. Nevertheless there remain a number of affections of nerves, muscles, glands,

[1] See Appendix II for prescriptions.

and skin, the direct exciting cause of which may be traced to reflex irritations, originating in some lesion of the jaws, teeth, or mucous membrane of the mouth.

**Physiological and Anatomical Considerations.** It is necessary for the proper comprehension of the various reflex affections to be described that not only the physiology of reflex action in general, but also of the special physiological and anatomical relationships of the fifth nerve to other nerves, should be quite understood.

As an explanation of the possibility of such affections, as we are about to consider, arising reflexly from oral lesions, the student is reminded of two physiological experiments.

(1) *Reflex Stimulation.* It is found that if an animal be very lightly anæsthetized, certain cortical areas of the brain become sensitive to stimuli (which would not ordinarily be perceived) if numerous *other* sensory impulses are being received from the periphery at the same time.

(2) *Reflex Inhibition.* Acid placed on a frog's foot causes instant reflex retraction. If now a tight ligature be placed round the *arm* of the frog, the acid when applied to the *foot* either causes no reflex action at all, or a very much delayed one, i.e. the impulses from the arm have 'monopolized' the attention of the brain.

The sensory fibres of the fifth nerve arise from the cells in the Gasserian ganglion; they bifurcate, and one branch passes out of the skull to the skin, mucous membrane, and teeth, whilst the other passes backwards to the pons and arborizes around the sensory nucleus of the fifth nerve in the medulla; these fibres then bifurcate also, some being continued down the medulla and into the spinal cord as low as the second cervical nerve, and others ascending as high as the upper part of the corpora quadrigemina.

During the course of these fibres they are brought into anatomical relationship with the nuclei of many of the other cranial nerves.

The extremely low point to which the descending root reaches should be borne in mind, as this may serve to explain the connexion between diseased teeth and reflex affections of muscles of the neck.

Further, Gaskell[1] has shown that, apart from the first, second, and eighth nerves, all the cranial nerves are developmentally connected with the sensory root of the fifth nerve. The structure of the medulla oblongata shows that the sensory or (posterior root portions) of the ninth, tenth, eleventh, and twelfth nerves (excluding the visceral branches) are absent from the nerves themselves, but that they have become diverted to help to form the sensory part of the fifth and the Gasserian ganglion. With regard to the remaining nerves, i.e. third, fourth, fifth (motor), sixth, and seventh, it was shown that their sensory nuclei have degenerated although their anatomical outlines still remain, but are now functionally represented by the sensory root of the fifth.

In fact Gaskell demonstrated that the majority of the cranial nerves are exactly analogous to spinal nerves with an anterior or motor root, and a posterior or sensory root, but that whilst the motor roots have remained separate, the sensory roots have all become merged into the sensory division of the fifth nerve.

Under these conditions it would *a priori* be expected that stimulation of the terminal branches of the fifth nerve would give rise to very varied and numerous reflexes.

This in fact is found to be so, and for the sake of convenience, they may be classified as arising from the following :—

I. Stimulation of
- (1) Motor or volitional cerebral areas.
- (2) Motor nerves
  - (*a*) Tonic.
  - (*b*) Clonic.
- (3) Vasomotor nerves.
- (4) Secretory nerves.
- (5) Nerves of special sense.

II. Inhibition of
- (1) Motor nerves.
- (2) Trophic nerves.
- (3) Nerves of special sense.

[1] *Proc. Roy. Soc.*, vol. xliii, p. 382.

### Affections arising from Stimulation of the whole or a part of the Motor or Volitional Areas of the Brain

In these conditions certain cortical areas of the brain are probably in a state of abnormal excitability, and the stimulation of the fifth nerve merely acts as the spark which gives rise to the explosion of energy.

Such exhibition of energy in an abnormal form may be manifested as epilepsy, hysteria, chorea, or in some forms of insanity.

**Epilepsy.** A condition of epileptiform convulsions has been experimentally produced by Brown-Sequard by weak stimulation of the fifth nerve after section of a lateral column of the spinal cord in the cervical or dorsal regions.

So many cases of epilepsy having a demonstrably causal connexion with diseased teeth have been recorded, both by physicians and dental surgeons, that there can be no doubt that irritation of the dental branches of the fifth nerve (in common with other sources of peripheral irritation) is to be regarded as a frequent exciting cause both of *grand mal* and *petit mal* in those patients whose cerebral centres are in an unstable equilibrium.

A strikingly illustrative case is the following :—

A girl aged 11 had suffered from attacks of epilepsy for some time, and was being medically treated by Sir James Sawyer. On being asked to see her I found a condition of chronic myelitis without symptoms in a lower molar. This was treated and the epilepsy did not recur for twelve months. I then saw the patient again, and found that an exposure of the pulp had occurred in another lower molar; this was treated and the attacks again subsided, and as far as I know did not recur.

Tomes[1] records a case of epilepsy in a boy in which the fits occurred two or three times a day, and the ordinary medical remedies were used without avail for six weeks.

[1] *Dental Surgery*, p. 567.

The extraction of some (presumably) septic lower molars was at once followed by a cessation of the seizures. There had been no recurrence in eighteen months' time. Brubaker records a series of eighteen cases of epilepsy associated reflexly with oral conditions.

**Chorea.** Cases of chorea of apparently dental origin have been recorded, but in view of the fact that chorea is now largely regarded as resulting from an infection, it is probable that cases which have been so described have been a form of convulsive tic rather than true cases of acute or Sydenham's chorea. Nevertheless, many authorities are of the opinion that all possible sources of irritation of the fifth nerve should be eliminated in the treatment of true chorea.

A case where choreic movements were associated with diseased teeth is recorded by Pierce[1]:—

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

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

**Hysteria.** In persons with neuropathic tendencies it is quite possible that an attack of an acute oral condition may be sufficient to upset the normal correlation of control between mind and body, and thus induce symptoms of hysteria.

[1] *American System of Dentistry*, vol. iii, p. 498.

A number of cases have been recorded by reliable authorities, but the certain demonstration of cause and effect in these conditions is by no means easy or clear. The converse is more certain, i.e. that odontalgia is occasionally a manifestation of hysteria, and young girls especially, frequently are possessed with an ardent desire for perfectly sound teeth to be extracted.

**Insanity.** In view of the fact that stimuli arising from peripheral irritation of the fifth nerve are capable of affecting the motor and sensory areas of the brain by irradiation, as in epilepsy and neuralgia, and to the volitional centres as in hysteria, it would seem to be neither impossible nor improbable that such stimuli should be reflected in a similar manner to the higher centres of the brain controlling the conscious reasoning powers, and therefore give rise to effects and manifestations of mental energy, which are abnormal (causally) as to time, place, and the usual environments. Twelve cases of such a nature are recorded by Upson.

The majority of these were cases of dementia, and were all associated with either diseased, impacted, or faultily treated teeth. They were all treated by extraction of the affected teeth, after skiagrams had been taken by means of which various unsuspected lesions of the alveolar bone and peri-odontal ligaments were brought to light. Ten of the cases benefited considerably and were apparently cured by the treatment, in the remaining two no improvement followed. In none of the cases did the patient complain of local pain. It is not improbable, however, that such cases as these are due to the effect of long-continued toxic absorption from a septic mouth *combined* with the constant stream of unperceived afferent impulses reaching the brain via the second and third divisions of the fifth nerve.

All recent authorities are agreed that chronic toxic poisoning is the most important factor underlying insanity.

### *a.* Tonic Spasms

**Spasm of the Masseter Muscle.** This causes a more or less sudden inability to open the mouth, and is due to a reflex stimulation of the motor branch of the fifth nerve, which may arise from irritation of diseased teeth. The sensory nerve involved is usually the third division of the fifth, and the second or third molar is usually the seat of the lesion.

These are true cases of '*Trismus*'. Details of such cases are related by Salter,[1] Tomes,[2] and Ewart.[3]

**Inability to Close the Mouth** due to spasm of the muscles depressing the mandible, the exact opposite of true trismus, has been recorded by Tomes.

The patient was suffering from difficult eruption of a wisdom tooth, with much swelling and ulceration of the adjacent gums; whenever in closure of the mouth the upper wisdom tooth touched these inflamed tissues, the mouth was violently dragged open. This spasm was of so painful a nature that the patient went about with a cork between his teeth, so as to prevent any contact, and in this way he could obtain sleep.

**Lagophthalmos** (*inability to close the eyelids*). This condition may be brought about by spasm either of the levator palpebræ or of Muller's muscle. In the former case the stimulus affects the third nerve, and in the latter the sympathetic. Where the lagophthalmos is due to reflex irritation arising from diseased teeth, and if no other ocular spasms are present, the latter nerve and muscle are the ones probably involved, as in the case recorded by S. J. Hutchinson. In this case the condition had existed for more than a year. After other treatment had been tried an exposed pulp was found under an amalgam filling in a first upper molar tooth; this was treated by extraction of the tooth.

[1] *Guy's Hospital Reports*, 1867.
[2] *Dental Surgery*, p. 567.
[3] *Brit. Journal Dental Science*, January 11, 1900.

The spasm at once began to improve, and ceased within six months of the extraction.

Inability to close the eyelids may also be due to paresis of the orbicularis oculi, which, in the opinion of the late Mr. Henry Power, may be induced by reflex dental irritation, but Sir Job Collins and others strongly dissent from this on account of the 'impossibility of conceiving of a reflex paralysis'.

**Ocular Spasms** (*squints*). On theoretical considerations alone, seeing that the fifth nerve is the sensory root for the third, fourth, and sixth nerves, it might be expected that reflex spasms of the ocular muscles would be fairly common, at least as common as those arising from reflex stimulation of the seventh nerve. Yet this in actual fact seems not to be the case, at least very few cases of spasm of the muscles of the orbit are on record which can be assigned definitely to reflex irritation.

Concomitant strabismus is said to be 'caused by' difficult dentition in weakly children, but since Donders demonstrated that the actual cause in at least three-fourths of these cases is hypermetropia, reflex irritation has come to be looked upon as of secondary importance.

**Mydriasis** (*dilatation of the pupil*), due to stimulation of the sympathetic fibres supplying the radiating muscle of the iris, may be produced reflexly by stimulation from diseased buccal tissues.

The late Mr. Henry Power was of the opinion that it is to be regarded as a reflex paresis of the third nerve.

**Torticollis.** Wry-neck or spasm of the sterno-mastoid muscle may occasionally arise as a reflex manifestation of carious teeth, and may be either tonic or clonic in form.

A case of tonic spasm is recorded by Hancock,[1] and one of clonic spasm by Ormond.[2] In both of these cases the spasms ceased after the extraction of diseased teeth.

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

[2] *Trans. Odont. Soc.*, 1869.

**Cervical Opisthotonos.** Retraction of the head may, in the opinion of some authorities, be due to reflex irritation. Dr. Edmund Cautley[1] says that 'in a few cases' he has been 'unable to find any other explanation for simple retraction of the head', and Dr. R. Hutchinson[2] would seem to be of the opinion that when such cases do occur the immediate cause is cerebral congestion, which may be induced reflexly by stimulation of the dental branches of the fifth nerve.

### *b.* Clonic Spasms

**Histrionic Spasm** (*contraction of facial muscles*). In this condition the efferent reflex path is via the seventh nerve, and the whole of the facial muscles may be affected; when arising from dental reflex irritation one side only of the face is usually affected. If the orbicularis oculi is implicated the spasm may become tonic, and the eye can then only be opened with great difficulty; suitable treatment of carious teeth in the upper and lower jaws in one such case was followed by a complete cessation of the spasm. In another case[3] in which the spasms had lasted for two years no systemic or local causes (other than dental) could be found to account for the condition. 'The teeth were in a neglected condition, the right side of the mouth in this respect being much worse than the left. General treatment had been tried for nearly one year. Local treatment was then tried, all roots and teeth with septic pulps being removed and the others filled. The calculus was removed and the gums treated. The dental treatment lasted three weeks, during which time improvement occurred, the patient eventually making, as far as could be ascertained, a complete recovery.'

The orbicularis oculi may be the only muscle affected,

[1] *Brit. Med. Journ.*, 1905, p. 555.
[2] *Brit. Med. Journ.*, 1905, p. 556.
[3] Smale and Colyer, *Diseases and Injuries of the Teeth*, p. 531.

and instances are recorded in which this excessive blinking was clearly traceable to the teeth, since it ceased immediately after dental treatment. Power and F. J. Bennett[1] also record curious cases in which the blepharospasm ceased upon pressure being applied to or in the region of the lower wisdom tooth, yet was not cured in either case by simple local treatment in that situation. But in the case recorded by Power the blinking eventually ceased after section of the inferior dental nerve.

**Cough.** Instances of paroxysmal cough arising from reflex dental irritation in children are not uncommon during dentition, and cases are recorded by Sir Lauder Brunton and H. J. Mummery.

### Affections arising through Stimulation of Vaso-motor Nerves

**Cardiac Arrhythmia and Palpitation.** That the nervous mechanism controlling the heart's action is capable of being affected reflexly is well known, but it is not often recognized that sensory impulses set up by diseased teeth may be reflected along the cardiac nerves. Yet several cases of this nature have been recorded.

A case related by Sir Douglas Powell was that of a woman aged 36, who had experienced considerable pain and irregularity of the heart's action for two years. The teeth were extensively decayed, but there was no dyspepsia and no organic lesion of the heart. A course of Nauheim baths failed to relieve the condition, but eventually ' she consented to have her affected teeth removed under gas and ether, which she took very well, and from that time her cardiac symptoms have entirely ceased '. Sir Douglas Powell concludes that ' the cardiac symptoms seemed to be due chiefly to the reflected irritation of the decayed teeth '.

**Vaso Dilatation of the Ocular Vessels.** Undoubtedly this

[1] *Brit. Med. Journ.*, 1905, p. 557.

condition is often brought about by reflex dental irritation and may be only transient in effect, or it may serve to accentuate any latent or trivial lesion which may be present in the eye.

Thus **Hyperæmia of the Conjunctiva** and œdema of the lids may be caused in this way, and so serve either to aggravate or stimulate an original inflammation.

**Orbital Cellulitis.** The chief points in a case which was apparently caused reflexly by diseased teeth are as follows :—

The patient, aged 17, on three separate occasions suffered from attacks of 'orbital cellulitis'. The first two seemed to be associated with the presence of unerupted molars contained in their crypts, and the second attack occurred many years afterwards and synchronized with a non-suppurative periodontitis in an upper canine. Extraction of this tooth was followed by a complete cessation of orbital symptoms. Seeing that infection of the orbit by direct extension seemed to be negatived, and in the absence of any proof that the 'cellulitis' was infective in origin, it is probable that this case is to be regarded as an instance of reflex vasomotor disturbance of the orbital vessels causing intense engorgement of the tissues with blood and serum.

**Glaucoma.** The evidence that glaucoma may be causally connected with diseased teeth is divided. Experimentally Von Hippel and Grünhagen found that in animals, if the aorta were compressed and the fifth nerve then stimulated, a greatly increased tension of the eyeball resulted. Power and Reber are of the opinion that reflex dental irritation may be a factor in threatening glaucoma. On the other hand Priestley Smith, after examining the tension of the eyeball in a number of cases of odontalgia, is of the opinion that the two conditions are not related.

It must be remembered, too, that the precise pathology of glaucoma is not yet fully worked out, and hence it is one of that class of lesions to which a 'reflex' origin is apt to be assigned in the absence of any other obvious cause.

## Affections arising from Stimulation of Secretory Nerves

**Lachrymation.** A reflex flow of tears due to paroxysms of dental myelitis or peri-odontitis is observed so frequently as to need no comment.

**Salivation** in a similar manner is frequently brought about by reflex stimulation of the chorda tympani. This is usually quite transient, but the flow of saliva may remain excessive for a considerable time.

**Hyperidrosis.** A profuse secretion of the sweat glands may be incited reflexly by any severe pain. But the same thing may occur without excessive pain being present.

Two cases of hyperidrosis are recorded by Darguises[1] which were caused by 'mastication', though no mention is made of the condition of the teeth or buccal tissues.

A case has recently come under the notice of the author in which the patient, a healthy young man of 19, exhibited most profuse hyperidrosis of the frontal and malar regions whenever his teeth (upper incisors) were being treated, although he stoutly denied the slightest pain, and was not in the least 'nervous'.

**Coryza** may occasionally be traced to reflex irritation from diseased teeth. Rousseau-Decelle recounts a case of coryza on the left side (with other complications) which seemed to be dependent upon lesions of the two upper left incisors; on the teeth being treated the symptoms at once abated. Collett[2] relates a case of a patient who developed a coryza, accompanied by neuralgia, which had lasted for three or four weeks despite all local nasal and general treatment. An upper premolar was then found to be affected with peri-odontitis and was extracted; no pus was found. The neuralgia had disappeared next day, and the coryza also during the next three days.

[1] *Brit. Dent. Journ.*, 1909, p. 221.

[2] *Lancet*, January 1897.

### Inhibition of Motor Nerves

Some authorities are of the opinion that reflex inhibition of motor nerves does not occur, but the balance of modern physiological opinion seems to be in favour of such action taking place.

Dr. Head's explanation of 'spread' as due to lowered physical conditions, may be taken as explaining muscular paresis in remote parts sometimes found to be dependent on the teeth; there are other ways also in which reflex paresis is possible but which cannot here be discussed. Cases of this nature have been recorded by competent observers, so that this classification is adhered to. It is important, however, to recognize that referred pain or neuralgia may cause a patient to involuntarily simulate a paralysis in some cases, owing to the fact that movement is carefully avoided in order to obviate any increased pain.

**Facial Paralysis**. Many cases of Bell's palsy are on record which apparently have a causal relationship with diseased teeth; some of these would appear to be true reflex neuroses. Others are due to the implication of the seventh nerve in inflammatory products from the teeth. It is also well to remember that the most frequent cause of facial paralysis is exposure to cold, and this same thing often originates an attack of odontalgia, and the two conditions may thus merely synchronize.

**Ocular Paralyses, Ptosis, Paralytic Strabismus, Paralysis of Accommodation, Mydriasis, and Paralysis of the Orbicularis Oculi** have all been ascribed in a few cases as arising reflexly from diseased teeth.

Drs. Gutmann and Reber are of the opinion that such paretic conditions may often be connected with disease of the teeth and improve upon treatment of the dental lesions.

Mummery records the following case of ptosis.

A lady, aged 30, consulted me with ptosis of the left eye, accompanied by complete blanching of a lock of hair over the left temple; there was no toothache, but neuralgic pain in the left temporal region, from which she suffered previous to the blanching of the hair.

The second upper left molar I found tender on percussion, and finding it contained a partially dead pulp beneath a filling, I removed the tooth. The ptosis was relieved the next day, but the lock of hair remained permanently white.

With regard to paralysed accommodation Schmidt found a certain degree present in 73 out of 93 cases of dental disease examined, whilst Priestley Smith only found it present in 1 out of 16 cases of odontalgia. Norris and Oliver refer to paralysed accommodation as 'perhaps the most frequent ocular disturbance noted in connexion with dental irritation'.

**Paralysis of Arm.** Several cases of this nature have been recorded, the paresis in no case being complete but varying from 'a sense of lassitude in the arm' to 'inability to raise the arm'. The case recorded by Salter seems to have been the most severe. The patient suffered considerable pain in the arm accompanied by 'total inability to use the arm, to raise it, or to grasp with the hand'. A lower wisdom tooth which was carious and was erupting with difficulty was extracted, and almost immediately afterwards the arm symptoms 'vanished completely'.

Mummery states that in his own case, whenever he suffered from bad pain in the left lower molar he could only raise his left arm with difficulty, and 'experienced a sense of weight and fatigue in the arm almost amounting to pain'.

In the author's cases of brachial neuralgia due to myelitis of the lower premolars there was difficulty in raising the arm, but it was not easy to say how much of this was due to the pain; certainly in one case movement of the arm increased the pain, and for this reason it was very carefully held as still as possible.

## Affections arising from Interference with the Trophic Functions of Nerves

It is impossible to believe that actual inflammation, as it is now understood, could be caused *per se* by reflex dental irritation. Yet that such reflex irritation has some influence over the origin and course of inflammatory lesions in certain regions is a clinical fact.

The mode of such influence may be either, as Hilton said, that the reflex irritation from the teeth leads to interference with the nutritive function of the nerve supplying other regions, or it has to do with a congestion of the vessels which may be reflexly induced through the medium of the fifth nerve. More probably the effects are produced by a combination of the two causes, and in either case the tissues would be rendered less resistant to invasion by micro-organisms.

**White Hair.** Both Mummery[1] and Hilton relate cases in which the hair on the temple became more or less suddenly white during attacks of severe neuralgia originating in the teeth. In Mummery's case the bleaching of the hair was accompanied by ptosis. The latter condition disappeared on the extraction of an upper molar containing a partially necrosed pulp under a filling, though it is important to note that no ' toothache ' was present.

**Alopecia areata.** The frequent association of this condition with oral lesions has been worked out more especially by French authorities, and by Jacquet particularly.

Rousseau-Decelle[2] goes so far as to say that reflex irritation from the second and third divisions of the fifth nerve causes one-third of the total number of cases of alopecia. He further gives it as his opinion that the loss of hair is on the same side as the pain, and is more frequent on the left side, and that

[1] *Brit. Med. Journal*, 1905, p. 553.
[2] *Brit. Dent. Journal*, 1909, p. 879.

alopecia tends to follow lesions of the mucous membrane and alveolar bone rather than of the teeth.

Amongst others the following case is cited: ' In a patient presenting an area of baldness of the left side of the nape for over a year, the treatment of various teeth, including the extraction of the left lower third molar, produced no improvement till Monier discovered and removed a large fragment of the posterior root of the wisdom tooth, when the alopecia disappeared in a month '.

Another case is also related by the same author of alopecia of the moustache, which was cured in ten days by the treatment of the two upper left incisors. Underwood has also recorded a case of loss of hair caused reflexly from the teeth.

Alopecia of dental origin is said to be small in area and multiple. Extensive loss of hair is usually due to other causes.

**Ulceration of the Face, Mouth, or Cornea.** There can be no doubt, and nearly all authorities are agreed, that the irritation arising from diseased teeth tends to prolong such lesions even if it does not actually predispose the part to the initial attack of inflammation. A case observed by the author was that of ulceration of the face on the right side between the malar eminence and the nose, which resisted all treatment for a considerable time, and only yielded on devitalization of an exposed pulp in the right upper canine tooth, albeit it should be noted there was no dental pain.

**Conjunctivitis and Iritis.** Similar remarks apply to these lesions, and it is a matter of fairly frequent observation that the ophthalmic treatment is in some cases only successful after the elimination of other sources of peripheral irritation.

The author has certainly seen such cases of conjunctivitis. A case of severe iritis occurring in the practice of my colleague Prof. Lindo Ferguson is as follows :—

The operations ran a perfectly normal course in the left eye, and the right did well until after the second or third

needling. Some ten days after the needling, when all question of septic involvement could be put on one side, the child began to cut a tooth, and developed iritis, which caused a good deal of anxiety. The inflammation recurred with each subsequent tooth, the ciliary body became involved, and the globe became staphylomatous, and was finally removed.

**Furred Tongue.** This condition occasionally exists on one side, and is associated with some buccal or dental lesion on that side. This is sometimes quoted as an example of interference with the trophic function of the nerves supplying the tongue. But it is equally likely to be caused by deficient mastication on that side, in the same way that the teeth and gums on the same side are likely to be covered with epithelial and food débris.

**Otorrhœa.** That dental irritation is capable of exerting a neurotrophic influence in this condition would seem to be so from the cases recorded in which a persistent flow of pus from the ear only gives way to treatment after treatment of some oral lesion. Urban Pritchard also states that in his opinion 'dental irritation is a factor in these cases of suppurative otitis media, both in the causation itself and also in the keeping up of the inflammatory condition'.

### Affections arising from Stimulation or Inhibition of Nerves of Special Sense

**Amaurosis and Amblyopia.** Very many cases of such conditions have been placed on record from time to time as reflex neuroses arising from dental disease.

Some of them would now probably be recognized as being due to direct septic extension via the antrum, veins, or perivascular lymphatics, causing an acute optic neuritis. Nevertheless there are cases in which this seems to be excluded. Hancock records a case in which a patient had been blind for over a month; there had been no preliminary symptoms; the onset was quite sudden, and no structural

changes in the eye could be found. A diagnosis of reflex dental irritation was made and six teeth were extracted. Within a few hours light became visible, and during the week following the sight completely returned. The only other treatment adopted was the administration of aperients. It is recognized, too, in many instances, that 'cases of amaurosis and amblyopia often improve after the extraction of diseased teeth'.

**Deafness.** This, like amaurosis, may be occasionally caused reflexly.

Mummery[1] records a case of deafness on the left side from the delayed eruption of an upper wisdom tooth; very considerable deafness had existed for some months, which was much relieved immediately on the extraction of the tooth, hearing being fully restored the same day.

Catlin relates an instance of deafness which had lasted for four days, and in which hearing returned within an hour of the extraction of a right lower molar.

Such inhibition of hearing is probably accounted for by the proximity of the middle root nuclei to the auditory nerve-centre.

**Photophobia.** Increased sensibility of the retina to light seems in some cases to be induced through reflex dental irritation, instances of which are related by Tierlink, Hay, and De Witt. The latter records a case of lachrymation and photophobia of one eye of prolonged standing; the condition was intensified by dietetic errors, but yielded completely after the extraction of a carious tooth.

**Tinnitus aurium** is in a similar manner said to be occasionally set up reflexly from mouth lesions.

## Diagnosis of Reflex Affections

The lesion giving rise to such reflex affections as have been described may be either dental myelitis (due to acute or

[1] Loc. cit.

chronic exposure of the pulp or penetrating caries without loss of substance), calcareous degeneration of the pulp, peri-odontitis, gingivitis, arthritis, or the impaction of an erupting tooth.

It cannot be said definitely which of these lesions is most prone to give rise to reflex affections, but the author is of the opinion that chronic myelitis, with exposure of the pulp and impacted lower third molars, is more often than others the cause of the trouble. But more important than the class of lesion, in the author's opinion, is *the absence of local dental pain.* This fact is to be noticed again and again in records of such cases, and is of great value from a diagnostic point of view.

As a general rule it may be said that *if the patient is complaining of symptoms which may be reflex and a dental condition is discovered which should normally be causing pain, but is not, then that dental lesion is not improbably the source of the reflex affection.*

With regard to alveolar abscess. Although the presence of pus around a tooth suggests the probability of other affections being due to septic infection, yet it must not be forgotten that an abscess (especially in its early and acute stage) may give rise to powerful sensory impulses which may be reflected in any of the ways mentioned.

It is often possible to diagnose a reflex paralysis when only neuralgia is present—the pain preventing voluntary movement; this may be eliminated by eliciting involuntary movement in the usual manner.

A patient suffering from hysteria may present combinations of pain and reflex affections, but the combinations are as a rule very unusual or impossible.

In every case one has to guard against the fallacy of *post hoc ergo propter hoc* in seeking for a source of reflex affections.

# CHAPTER XVII

## LOCAL AND GENERAL ANÆSTHESIA

Local anæsthesia is obtained either by applications or injection. The only drug of any value as an application is *ethyl chloride in spray form.* Its use, however, is extremely limited: it can only be suitably used in the anterior part of the mouth, and there only for opening abscesses or the extraction of broken down and isolated teeth. In the lower jaw the flow of saliva usually prevents its freezing action, and in the posterior region of the upper jaw it is difficult if not impossible to apply accurately. Frequently, too, it is very questionable whether the pain of the freezing is not greater than the subsequent operation.

Ethyl chloride may, however, be used with considerable advantage in the extraction of deciduous teeth, especially if the child be refractory. In such a case one may aim at part local and part general anæsthesia.

A small strip of lint is placed in the mouth round the tooth to be removed, and held in position by the fingers of the left hand correctly placed for extraction. The ethyl chloride is then sprayed partly on the gum and partly on the lint. The child usually at this stage, if not before, commences to cry: after the second expiratory cry it will be noticed that the child becomes suddenly quiet.

This means that the child has taken two deep inspirations of air mixed with ethyl chloride, and is partly anæsthetized or is at least analgesic.

The extraction of the tooth or other operation must now be performed rapidly; the patient will complain of nothing,

and the parent watching will be quite surprised that after all the child 'never made a whimper' whilst the operation was being performed. Care should therefore be exercised always when using ethyl chloride for children to watch for its general anæsthetic effect, or it may be quite easy to give an over-dose whilst endeavouring to obtain a nicely frozen gum.

For *local anæsthesia by injection* cocaine or one of its numerous substitutes is used. Le Brocq has investigated the toxicity of these substances with the following results, taking cocaine as the unit:—

| | |
|---|---|
| The toxicity of Alypin | = 1·25 |
| ,, ,, ,, Cocaine | = 1·0 |
| ,, ,, ,, Nirvanine | = ·714 |
| ,, ,, ,, Stovaine | = ·625 |
| ,, ,, ,, Tropacocaine | = ·500 |
| ,, ,, ,, Novocain | = ·490 |
| ,, ,, ,, Beta-eucaine lactate | = ·414 |

As regards anæsthetic effect, he says that alypin, beta-eucaine lactate, novocain, and tropacocaine are about equal to cocaine, whilst stovaine is superior, and nirvanine inferior. Sloughing followed the use of stovaine, beta-eucaine lactate, and tropacocaine. Cocaine caused slight hyperæmia, whilst novocain was without any after-effect at all. Hence it would appear that novocain is the best substance to select; it has, too, the added advantage that it can be boiled and thus sterilized.[1] The author, however, still prefers cocaine as being more uniform in its results.

[1] Cocaine may be boiled without decomposition in distilled water, but not in an ordinary glass receptacle, it being the alkali from the glass or water which splits up the cocaine, and not the heat. A glass tube may be used in which to boil cocaine solutions, provided its alkalinity does not exceed 3 c.c. $\frac{N}{100}$ NaOH for every 50 c.c. capacity.

There are several most important principles to be observed in connexion with local anæsthesia by injection.

*Firstly*, to be quite certain of the substance and the amount of it in each syringeful used.

*Secondly*, to be absolutely sure of asepsis.

*Thirdly*, to use only dilute solutions.

In order to observe these precautions a solution should be made up *fresh* for each operation, and on no account should anything out of a bottle which has been previously opened ever be injected into a patient's tissues. It increases the value of the drug to add adrenalin to its solution in order to localize its action, thus preventing systemic effects and enabling a smaller quantity of cocaine or its substitutes to be used. The only safe way to use adrenalin is in 'ampoules' containing ·5 c.c. each, the glass ends of which can be hermetically and aseptically sealed again after use if necessary (either by holding the glass in a flame, or by sealing with well-heated sealing wax).

As regards *the syringe*. The usual hypodermic syringe is useless; it is both too small and too weak. The muco-periosteum of the jaws is exceedingly dense, and it requires quite a considerable pressure to force the anæsthetic into it, and satisfactory results are only to be obtained by using comparatively large amounts of weak solutions (rather than small amounts of strong solutions).

The most suitable form of syringe to use, if it is only used occasionally, is the all-metal syringe with a screw on nozzle, and detachable 'Schimmel' needles. Thus the whole can be sterilized by boiling before use and a needle discarded when blunt or rusted. With constant use the plunger of these syringes wears, and must be replaced.

'All-glass' syringes with a special finger-grip attached to the nozzle (without which the latter only flies off) may be used, but are in some danger of being broken either when being used or in boiling. Any syringe with a leather washer

is very difficult to keep water-tight and exceedingly difficult to sterilize—the only means of overcoming these objections is to keep the whole syringe permanently in an antiseptic bath, but even this in time causes the leather to deteriorate. The syringe, of whatever variety, should have a capacity of 30 minims.

The following is a convenient method of procedure :—

Fill the sterilized syringe with boiled water, eject it into a sterile minim measure; when cooled a little, drop into it one $\frac{1}{6}$-gr. pellet of cocaine hydrochloride and three drops of adrenalin chloride 1 in 1,000; this gives approximately a ·6 per cent. of cocaine with 10 per cent. of adrenalin. The gum is then to be sterilized by rubbing it well with tincture of iodine on a pledget of cotton-wool on the inner and outer sides of the tooth; about 15 minims of the cocaine solution are then injected on each side for molar teeth, less will suffice for incisors. If the tissues are well blanched it is not necessary to wait for more than a minute before operating; if no blanching occurs, operation should be deferred for five minutes. The result is that the operation is usually painless. No more than three such syringefuls ($\frac{1}{2}$ gr. cocaine) should be used for an adult patient on any one occasion.

No injection should ever be made into acutely inflamed or suppurating tissues—the pus is driven in deeper, the vessels are contracted, the organisms thus invade the tissues unchecked for a while, and necrosis not unusually supervenes.

**General Anæsthesia.** It is not proposed here to discuss the principles of general anæsthesia, but merely to indicate several points of importance to an anæsthetist for a dental operation. The four anæsthetics used are ether, chloroform, ethyl chloride, and nitrous oxide.

A dentist may justifiably, and usually does, administer what is known as 'short' nitrous oxide himself; this, however, only allows sufficient time for the extraction of two or three

teeth at the most, and the patient begins to recover as soon as the face-piece is removed.

If the operation is more extensive, and another anæsthetic or 'prolonged' nitrous oxide is required, a medical man should always administer it.

**Prolonged Nitrous Oxide.** There can be no doubt that, from the points of view of safety, rapidity of induction, free-

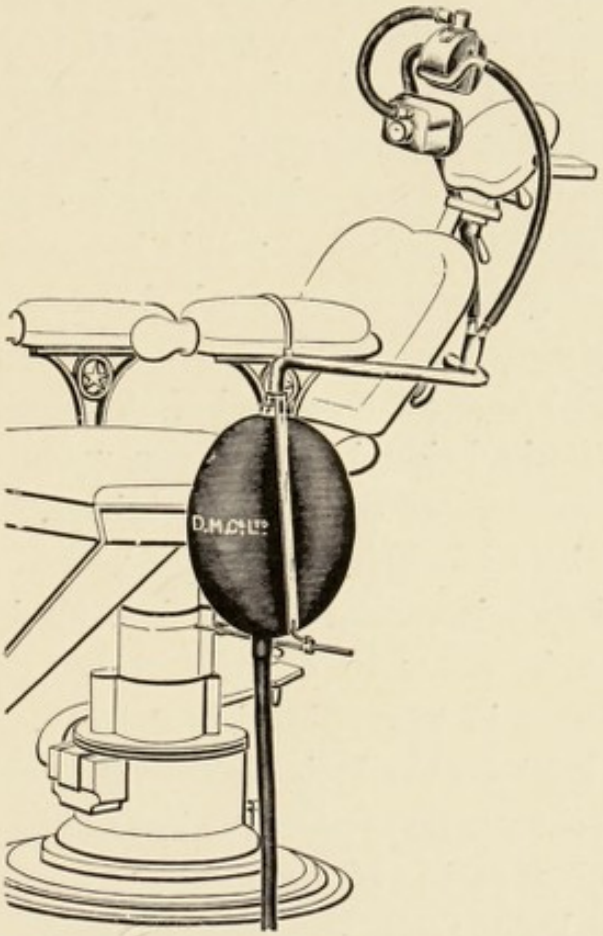


FIG. 65. Apparatus for the 'prolonged' administration of nitrous oxide by the combined nasal and oral method. (Trewby's apparatus.)

dom from after-effects, and convenience to the dentist, nitrous oxide administered by the prolonged or nasal method is much to be preferred. (Fig. 65 shows one form of the apparatus.) The more modern forms have a connexion between the nose-piece and mouth-piece, and some form of spring upon the gas bag to ensure a constant pressure; these are great improvements, and ensure a much more even anæsthesia,

and thereby eliminate the cyanosis and jactitation so frequently seen with the older apparatus.

**Method of Administration.** The anæsthetist should stand to the left of the patient, who should be seated in the dental chair inclining slightly backwards, but with the head and neck in a straight line with the thorax (i.e. the larynx and trachea must not be pushed backwards so as to obstruct the airway). The nose-piece is to be held in position with the thumb and first two fingers of the right hand passed over the top of the patient's head, and the mouth-piece should be held firmly pressed over the mouth with the left hand. The supply of gas should be regulated by a tap operated by the anæsthetist's foot; this is preferable to having an attendant to 'turn on the gas', since the supply of the anæsthetic is more completely (and after a time automatically or reflexly) under the control of the responsible person.

Anæsthesia being induced, the mouth-piece is removed, but the nose-piece of course retained. The fingers of the anæsthetist's left hand should be placed under the angle of the patient's jaw on the left side, forcing it upwards and forwards, in order to counteract the pressure applied by the operator in extracting lower teeth. One of the fingers may be kept on the facial artery, and the fifth finger of the right hand may be used to lift the right eyelid of the patient for the observation of reflexes.

Whilst lower teeth are being extracted the supply of gas can be lessened, because the tongue is nearly always thrust upwards and backwards to some extent by the operator, thus lessening the intake of air; as soon, however, as the last lower tooth is extracted, and the operator turns to the upper jaw, the supply of gas frequently needs to be increased to make up for the additional dilution with air via the mouth. During this part of the operation the anæsthetist may afford considerable assistance to the operator by making counter downward pressure on the head with his right fore-

arm and wrist. It should be arranged that the operator is to tell the anæsthetist when he is about to extract the last tooth in order that the nitrous oxide may be turned off, that which remains in the bag being sufficient to continue the anæsthesia.[1] By this method anæsthesia may readily be secured for the most lengthy dental operations, eight to ten minutes being frequently obtained, and there being no reason why the anæsthesia should not be continued for twice or three times as long.

The method has been used for major surgical operations where ether and chloroform were both contra-indicated.

**Chloroform.** Statistics are all against the use of chloroform for dental operations. Of the total deaths under chloroform those occurring during oral operations form the major part. The reasons for this are twofold: (i) the patient is frequently not properly prepared, (ii) in order to secure anæsthesia during the time when no anæsthetic is being administered it must be 'pushed' in the first instance further than it would be for any other operation.

These two factors, combined with a possible depression of the jaw by the operator, and thus obstruction of airway and a chronic toxæmic condition of the patient due to prolonged oral sepsis, combine to make the anæsthetic unsuitable and certainly not free from danger in dental operations.[2]

Conditions under which chloroform may justifiably be used are :—

(1) When the patient has been under observation, and

[1] This is an important consideration from the point of view of expense, especially in hospitals, since this is undoubtedly the most costly form of anæsthesia.

[2] I am well aware that some individual practitioners claim to have administered chloroform in many hundreds of dental cases without any mishap; and undoubtedly some practitioners are particularly skilful with this form of anæsthesia; but it is not for the skilled specialists that this is written—they probably will never read it—it is to the practitioner of average skill and ability, by whom the greater number of anæsthetics are administered, that one wishes to be of some service.

is properly prepared (as in hospitals—private or general).

(2) When a fully recumbent position of the patient can be secured as well as the convenience of the operator (i.e. an ordinary bed will not do).

(3) When a Junker apparatus can be used satisfactorily in order to avoid 'pushing' the anæsthesia in initial stages.

Chloroform under these conditions affords a very favourable anæsthesia for dental work: the muscles are relaxed, the salivary and mucous glands are not over-stimulated, and the capillaries are not dilated.

**Ether.** When nitrous oxide by the prolonged method is not available, and special precautions are not possible, ether should be selected. It is considerably 'safer', of course, than chloroform—that is, it may be 'pushed' further in the initial stages than chloroform can be—and it can be administered to a patient in the semi-recumbent position: a decided advantage to a dentist unused to operating on the recumbent patient. Its disadvantages are that it requires a special apparatus, it causes a profuse flow of mucous and saliva, and increases the hæmorrhage.

The odour of ether, too, in a dentist's rooms is very objectionable and difficult to ret rid of—the patient is a considerable time in recovering, and not infrequently vomits.

**Ethyl Chloride.** This anæsthetic gives an anæsthesia of slightly longer duration than 'short' nitrous oxide. It is advantageous where three or four teeth are to be extracted, particularly in children. Induction and recovery are rapid, and with proper preparation there should be no after-effects. Its use, though, is extremely limited; it is undoubtedly much less 'safe' than either ether or nitrous oxide, it is not suitable for long operations, and is unnecessary for very short ones.

It may be combined with nitrous oxide with advantage

for the extraction of deciduous teeth in children. The face-piece and bag as for ' short ' gas are used, and after three or four full inspirations of nitrous oxide, two to three c.c. of ethyl chloride are run into the bag (it is better to have a special aperture for this), and anæsthesia rapidly deepens. The child maintains a good colour, keeps quite still, and there appears to be an analgesic state lasting for 15 to 20 seconds after full anæsthesia, which allows perhaps for the extraction of another tooth. This method is of advantage nowadays, when children frequently have to visit the dentist early in life, and it is of extreme importance that during this age they should not be subjected to unnecessary pain or fright, and thus give rise to a permanent disinclination to have their teeth properly treated.

It has recently been proposed to administer ethyl chloride by the ' open method ' for dental cases, and the results are said to be a great improvement upon the closed method.

**Cleansing the Apparatus.** In hospital work it frequently happens that a number of cases have to be taken in quick succession ; when this is so the anæsthetist should see that the mask or mouth- and nose-pieces are properly cleansed between each administration. This may be done effectively by first holding the apparatus under a stream of cold water to remove any blood, and then by washing it in a 1 in 50 solution of lysol ; this should afterwards be removed with a dry clean towel.

# APPENDIX

## I

## METHOD OF EXAMINING THE MOUTH AND CHARTING THE TEETH IN THE EXAMINATION OF SCHOOL CHILDREN

The medical inspection of school children is now the routine work of many practitioners, and in the course of such work it is recognized that the mouth and teeth should be examined.

It is a matter for regret that in many cases such examination has been limited to a cursory glance, or such details given and recorded as do not at all accurately represent the extent of disease present in a mouth, nor give an indication as to the potentiality for future lesions either primary in the mouth or secondary elsewhere.

The following scheme of oral examination is therefore suggested :—

1. The **Mucous Membrane** of the lips and cheeks to be examined in a good light. To be recorded as normal, acute, sub-acute (or chronic) stomatitis, &c.

2. The **Lips** to be everted, and the **Cheeks** drawn back in order to examine the labio-dental sulcus for ulcerations, or *sinuses* in connexion with *chronic alveolar abscesses*. This is exceedingly important, since the constant swallowing of pus from such sources is apt to be overlooked, and gives rise to a chronic toxæmia which prevents the mental and physical development of the child.

To be recorded as number of sinuses or ulcers present.

3. The **Gum Margins** to be noted whether pale or normal, swollen and red, or hypertrophic. Pressure to be applied upwards and downwards towards the crowns of the teeth to determine the presence of pus pockets. To be recorded as normal, simple, or suppurative inflammation.

4. Each tooth should be examined seriatim and in detail. This is the only means whereby any value whatever may be derived from the inspection. It need not occupy much time, especially if, as is usually the case, some one is available to note down the details as they are dictated by the examiner. Of course a system of examination and charting is necessary, and the following is strongly suggested on account of simplicity and rapidity.

The following is the chart to be used :—

R. CONDITION OF MOUTH ON EXAMINATION L.

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | E | D | C | B | A | A | B | C | D | E | | | |
| | | | | | | | | | | | | | | | |
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| | | | | | | | | | | | | | | | |
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| | | | | | | | | | | | | | | | |
| | | | E | D | C | B | A | A | B | C | D | E | | | |
| | | | | | | | | | | | | | | | |

The upright dark line indicates the centre line of the mouth, the horizontal dark line represents the division between the upper and lower teeth.

The letters apply to deciduous teeth and the figures to permanent teeth. The teeth are numbered or lettered from the central incisor round to the last molar on either side in each jaw. Thus 1 represents a central incisor, and 2 a lateral incisor, $\underline{\text{D}}|$ represents the right upper first deciduous molar, and $\underline{5}|$ its permanent successor the right upper second premolar, $|\overline{6\ 7}$ represent the lower left first and second permanent molars, and the figure 8 indicates a wisdom tooth, &c. It is important to remember that it is the patient's right and left which is always spoken of, and *not the examiner's.*

The squares below the letters and figures are for noting the **extent** of caries in each tooth, i. e. whether a tooth is carious to the first, second, third, or fourth degree (see chapter VI, p. 76).

(It is essential to note this in order to gain an idea as to the amount of *sepsis* present in a mouth ; the mere inability to masticate is quite of secondary importance when the child is probably existing on a soft pretriturated diet.)

Letters or figures representing teeth which are not present are crossed out (a single stroke / can be used to indicate not present, and a × to mean extracted, but this is not so important).

The examination should always commence at a definite point, and be continued in a definite order, in order to save explanations with the clerical assistant. It is simplest perhaps to commence at the right upper central incisor, ' 1 ' or ' A ', to do the right upper teeth, then the right lower teeth, next the left upper teeth, and finally the left lower teeth. To give an example of how this works in practice. The above instructions having been given to the assistant, the examiner dictates (for a child of 7) :—

One, B-fourth, C-first, D-third, E-second, Six. This means permanent right upper central present and sound, deciduous lateral carious and septic, deciduous canine simple carious cavity, first deciduous molar pulp gangrenous, second deciduous molar myelitis present, first permanent molar sound. It would appear on the chart thus :—

| | | | E | D | C | B | A |
|---|---|---|---|---|---|---|---|
| | | | 2nd | 3rd | 1st | 4th | |
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
| | | | | | | | |

The charting of a mouth in this manner need only occupy a very short time—two or three minutes at most—and the information thus obtained can be, if desired, afterwards easily tabulated and elaborated by a clerical assistant, to whom the chart has been explained, and advice can from it be given as to the treatment required. Such records would be of exceeding interest and importance as bearing upon—

1. The number of carious teeth present in each mouth.
2. The degree of the disease.
3. The amount of sepsis.
4. The extent of masticating surface available.

To endeavour to obtain this information in any other manner

during the examination of each patient would entail too much time and labour, and yet it is just the information which it is necessary to have if the examination is to be of any service from a public health point of view.

As regards suggestion for treatment; it is to be remembered that even simple carious cavities of the 'first degree' are potential sources of future sepsis, and that the treatment of caries in its initial stages is simple and inexpensive, whereas in later stages it is more tedious, and therefore expensive, and also that the extraction of a tooth is only to be regarded as something better than no treatment at all.

## II

## FORMULÆ WHICH WILL BE FOUND USEFUL IN ORAL AND DENTAL CONDITIONS

### APPLICATIONS

1.

℞ Chloralis Hydratis, gr. 80.
Menthol, gr. 80.
Thymol, gr. 80.
Camphoræ, ʒ iv.

For use in painful *gingivitis* and *odontalgia*.

2.

℞ Acidi Carbolici, ʒ ii.
Resinæ, ʒ ii.
Spiritus Vini Rectificati, ʒ iii.

Sedative dressing in localised *odontalgia*. To be inserted in the cavity on cotton-wool mixed with an equal part of zinc oxide.

3.

℞ Olei Cinnamomi, ♏ 20.
Acidi Carbolici, gr. 40.
Olei Gaultheriæ, ʒ i.

For sterilizing dentine over a nearly exposed pulp. Also as a sedative dressing in *odontalgia*.

4.

℞ Tincturæ Iodi, ℥ iii.
Tincturæ Aconiti, ℥ ii.
Tincturæ Capsici, ℥ iii.

To be used as a *counter irritant*: 2–3 minims at each application on cotton-wool wound round the end of a match stick.

5.

℞ Thymol
Acidi Carbolici, āā ℥ ss.
Camphoræ, ℥ i.

For use as an antiseptic and analgesic in dental *myelitis*.

6.

℞ Tragacanthæ, ℈ i.
Ol. amygdalæ, ɑ) x.
Aquam, ad. ℈ ii.

To be applied as a protective in localised stomatitis.

## COLLUTORIA

7.

℞ Acidi Borici, gr. 10.
Glycerini, ℥ ii.
Aquam, ad. ℈ i.

Mild antiseptic mouth-wash for use after *extraction* or scaling, or in *acute stomatitis* in children.

8.

℞ Zinci Sulphatis, gr. 30.
Tincturæ Krameriæ, ɑ) 30.
Tincturæ Pyrethri, ℥ i.
Aquam Cinnamomi, ad ℈ i.

For use in sub-acute and chronic *gingivitis*, diluted with an equal quantity of warm water.

9.

℞ Aquæ Chlori, ℥ i.
Syrupi, ℥ i.
Aquam, ad ℈ i.

For use in ulcerative or *gangrenous stomatitis*.

10.

COLLUTORIUM HAMAMELIDIS.

℞ Liquoris Hamamelidis, ℥ ii.
Aquam, ad ℨ i.

Astringent mouth-wash for use in sub-acute and chronic *gingivitis.*

11.

℞ Hydrargyri Perchloridi, gr. ¼.
Ammonii Chloridi, gr. ¼.
Aquam Chloroformi, ad ℨ i.

For rapidly *sterilizing the mouth* before operations.

12.

℞ Tr. myrrhæ, ℨ ss.,
Sodii biboratis, ℥ ii.
Aquam, ad ℨ xii.

For use frequently as a mouth-wash in *acute stomatitis.*

13.

℞ Papaveris Capsulæ, vi.

To be placed in a quart of water and simmered down to one pint, strained and then used as a mouth-wash as hot as possible. This should be undertaken by the patient.

For use in *pain after extraction.*

14.

℞ Liquoris Potassæ, ℥ i.
Acidi Carbolici, ℥ i.
Glycerini, ℥ ii.
Aquam, ad ℨ i.

For use after extractions or in *suppurative conditions* of the mouth, ℥ i, in ℨ vi of hot water.

15.

℞ Acidi Tannici, gr. 8.
Sodii Biboratis, ℥ i.
Glycerini, ℥ ii.
Aquam, ad ℨ i.

Astringent mouth-wash. Useful in cases of '*spongy*' *gums.*

16.

℞ Zinci Sulphatis, gr. i.
Zinci Chloridi, gr. ii.
Morphinæ Acetatis, gr. ⅓.
Aquam, ad ℥ i.

To be used with an equal quantity of hot water as an *anodyne and antiseptic* mouth-wash.

17.

℞ Thymol, gr. ½.
Acidi Benzoici, gr. 15.
Olei Menthæ Piperitæ, ♏ 8.
Spiritus Vini Rectificati, ℥ i.

As a prophylactic in clean mouths. A few spots in a wineglassful of water sufficient to cause milkiness.

18.

℞ Cocainæ Hydrochloridi, gr. 4.
Acidi Salicylici, gr. ½.
Aquam, ad ℥ i.

For injection hypodermically in extracting teeth—10 to 30 minims for each tooth.

19.

℞ Beta-Eucainæ, gr. 8.
Adrenalin Chloridi, ♏ 20
Aquam, ad ℥ i.

For hypodermic injection in extracting teeth—10 to 30 minims for each tooth. It is better, however, in all cases to make up solutions with boiled water immediately before use, as suggested on page 247.

## LOTIONS

20.

℞ Acidi Carbolici, gr. 24.
Aquam, ad ℥ i.

21.

℞ Aluminis, gr. 16.
Aquam, ad ℥ i.

22.

℞ Lysol, ℥ iii. ss.
Acidi Carbolici, ℥ iii.
Sodii Bicarbonatis, gr. 36.
Aquam, ad ℨ viii.

For sterilizing pulp canals, &c., and also for delicate instruments such as hypodermic needles.

23.

℞ Beta-Naphthol, ℥ iv.
Alcohol Absolute, ℨ i.

For sterilizing cavities and pulp canals.

24.

℞ Hydrargyri Perchloridi, gr. ½.
Hydrogen Peroxide, ℨ i.

For injecting into the pockets in 'Riggs' disease—chronic *dental arthritis.*

25.

℞ Liquoris Potassii Permanganatis, ℥ ss.
Aquam, ad ℨ i.

For use as a deodorant in cases with marked 'fœtor ex ore'.

26.

℞ Sodii Sulphitis, ℥ i.
Aquam, ad ℨ i.

For use in '*apthous*' *stomatitis.*

## MISTURÆ

27.

℞ Butyl-Choralis Hydratis, gr. v.
Syrupi Tolutani, ℥ i.
Spiritus Chloroformi, ♏ x.
Aquam, ad ℨ i.

For use in '*trigeminal*' *neuralgia.*

28.

℞ Calcii Chloridi, gr. 5.
Aquam, ad ℨ i.

For use *prior to extraction* when much hæmorrhage is anticipated.

29.

℞ Liquoris Ferri Perchloridi, ℥ iii.
Potassii Chloratis, gr. 5.
Aquam Menthæ Piperitæ, ad ℥ i.

For use in *stomatitis* accompanied with *anæmia*.

30.

℞ Potassii Chloratis, gr. 10.
Syrupi Aurantii, ℥ 5.
Aquam, ad ℥ s.

For ulcerative stomatitis—Maximum dose of Potassium Chlorate for a child of six years : 5 grains.

## PASTÆ

31.

℞ Acidi Arseniosi, gr. 20.
Morphinæ Acetatis, gr. 10.
Cocainæ Hydrochloridi, gr. 10.
Creasoti, q.s.

For devitalizing the pulp.

32.

℞ Potassæ Causticæ.
Calcii Hydratis, āā ℥ i.
Glycerini, q.s.

To be used for removing hypertrophied gum in positions inaccessible to actual cautery.

33.

℞ Thymol, gr. 30.
Formaldehydi (40 per cent. solution), ℥ 30.
Zinci Oxidi, ℥ i.

For hardening devitalized pulps '*in situ*'.

34.

℞ Acetanelidi, gr. ii.
Quininæ Sulphatis, gr. iii.
Caffeinæ Citratis, gr. i. ss.

For use as an analgæsic in neuralgic conditions. Six powders to be ordered. One to be taken at once, and one every hour until relieved. In children or adolescents two or four powders only should be ordered with a proportionate dose.

35.

℞ Trochisci Potassii Chloratis (B.P.), gr. iii.

For ulcerative stomatitis.

## III

## MINIMUM LIST OF DENTAL INSTRUMENTS SUGGESTED FOR THE USE OF MEDICAL PRACTITIONERS

The references are to pages in the Catalogue issued by Messrs. Ash & Sons, Golden Square, London, but the instruments are obtainable from any of the usual supply houses.

1. Mirror (either special dental or a laryngoscope mirror will do).
2. Probe, Weston's No. 12 (Ash's Catalogue, G. 21).
3. Enamel chisel, Mitchell's No. 1 (Ash's Catalogue, G. 12).
4. Excavators, Darby Perry, Nos. 21 and 22 (Ash's Catalogue, G. 20).
5. Dressing forceps, 'College' (Ash's Catalogue, G. 76).

### Extracting Forceps

6. Upper incisor and canine forceps (Ash's Catalogue, D. 2, No. 1).
7. Upper premolar and molar root forceps (Ash's Catalogue, D. 23, No. 111, Thorne's pattern).
8. Lower hawk's-bill forceps (Ash's Catalogue, D. 22, No. 109).

### Additional List

1. 'Full' forceps for lower molars (Ash's Catalogue, D. 12, No. 73 S).
2. Full upper molar forceps, R. and L. (Ash's Catalogue, D. 18, Nos. 96 and 97).
3. Elevators for removing broken-down roots, R. and L. (Ash's Catalogue, D. 29, Hospital pattern with metal handles).
4. Thumb-screw gag (Ash's Catalogue, K. 46, called 'mouth opener').
5. Small irrigation syringe with fine curved nozzle (Ash's Catalogue, H. 37 or 'Harvard').

6. Hypodermic syringe (Parke Davis—all metal for dental work).

The above instruments may be obtained in an 'aseptic' metal case made for the purpose at my suggestion by Messrs. Ash. It is certain that such an 'outfit' will be found extremely useful by medical practitioners in country districts, and also by medical officers of expeditions, medical missionaries and others. All the above instruments should be included in the equipment of the out-patient department of every hospital.

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