Dental caries, and the prevention of dental caries : a series of papers reprinted from the Journal of the British Dental Association / by Henry Sewill.

Dental Caries and its Prevention

SEWILL

DENTAL CARIES,

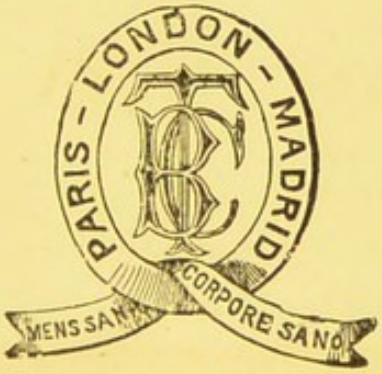
AND

THE PREVENTION OF DENTAL CARIES.

A Series of Papers reprinted from
THE JOURNAL OF THE BRITISH DENTAL ASSOCIATION.

BY
HENRY SEWILL, M.R.C.S. & L.D.S. ENG.

SECOND EDITION.



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PREFACE TO THE SECOND EDITION.

THE continued demand for this little book has induced me to prepare a second edition. With the exception of a few additions, alterations and corrections, the text remains unchanged. No new facts have been established since the first edition was published; and nothing has been urged to cast doubt upon the truth of the views I have endeavoured to make clear.

H. S.

40, WIMPOLE STREET,
LONDON, W.,
October, 1887.

DENTAL CARIES.

At the April (1884) meeting of the Odontological Society, in opening the discussion on Mr. Arthur Underwood's paper, I attempted to bring the whole subject of the etiology and pathology of caries within the limits of my speech. After proceeding for some length and having barely entered upon the first topics, I became aware that I had already taken up a large portion of the time of the meeting without yet fully reaching the proper subject of debate, namely, the influence of micro-organisms in the production of the disease.

It was obviously useless to proceed. I had opened the subject in a manner too elaborate to allow me to complete my argument, and I therefore at once cut short my remarks. It was suggested that I should amplify my speech, and publish it in the Journal. I have willingly adopted the suggestion. I think it will be useful to bring together very briefly the main facts on the subject of the etiology and pathology of caries, and the most cogent proofs upon which these facts are based. This has not been hitherto done. Although many authors have contributed to the elucidation of different points, there are only two works which deal exhaustively with the whole subject—Wedl's 'Pathology of the Teeth,' and

Messrs. Tomes' well-known Manual. The former of these is a most masterly work. Written by one of the most distinguished professors of general pathology, it is free from the narrowness so difficult for any specialist to avoid, however philosophical his mental attitude. It is exhaustive, and leaves no fundamental fact of any kind, no observation of any respectable investigator, unexamined. To dispute the clearly demonstrated deductions which it formulates is impossible. Messrs. Tomes' work is too well known to need description; that too is largely based, like Wedl's, on original research. These authors (as well as all really competent writers on the subject) are in essential points in full agreement; and although neither required the corroboration of the other, being each alone strong enough to prove his case, their agreement makes assurance doubly sure that their views are incontrovertible.

I shall, in these papers, endeavour shortly to state what we really know about caries, and to expose the fallacies of recent authors. My contention is that there remains no room for valid difference of opinion as to the nature of caries. Approached from any side, viewed in the light of physiological fact, or judged by the result of observation and experiment, there is only one possible solution of the problem. To establish this opinion full examination of opposing facts and arguments seems the proper method; and it is this which I have adopted.

Caries is a process of disintegration, commencing invariably at the surface of the tooth, proceeding inwards, and due entirely to external agents; enamel and dentine are passive under this process of disintegration, and manifest neither pathological action nor vital reaction of any kind. By pathological action I mean—(1) morbid changes in the tissues induced or produced by the influence

of the vascular and nervous system; and (2) morbid changes in the tissues, in which changes vascular and nervous influence may, perhaps, have no share, but which are not produced by external agents. By vital reaction I mean any change in the tissues not solely induced and produced by external agents.

The substantial truth of this definition of caries may be established on anatomical grounds. What concisely are the anatomical characters of enamel and dentine? Enamel is a densely hard, almost homogeneous mass, ranking in hardness with quartz, mainly composed of phosphate of lime, and containing a mere trace, not more than three to five per cent., of organic matter barely discoverable on solution of the tissue in acid. Dr. Bödecker, of New York, has recently stated that he has demonstrated this organic matter regularly distributed throughout enamel, having stained the tissue with chloride of gold. If this observation be correct (and it is very questionable) it will have no great importance, seeing that the organic substance would be in a condition of extreme tenuity. Enamel has no vascular or nerve connections. Dentine consists of a homogeneous calcareous matrix, in which no trace of cellular or other structure can be detected, ranking in hardness between enamel and the densest bone, permeated by minute tubes not more than $\frac{1}{4500}$ inch in diameter. The tubes radiating from the pulp cavity are occupied by fibrils which, proceeding from the pulp, endow the tissue with sensibility. The fibrils being so minute, their actual structure cannot be demonstrated; but there can be no doubt they are protoplasmic, and form the sole protoplasmic constituent of dentine.

The organic basis of dentine, about 28 per cent. of the mass, is contained mainly in the inner walls of the tubes surrounding the fibrils. It can be demonstrated by dis-

solving the earthy constituents in acid; and remains as a tough felt, even after boiling in strong muriatic acid. No cellular or protoplasmic elements are discoverable in this fibrous structure.

Dentine is in relation with the vascular pulp which it encloses, but a circumstance which is seldom mentioned should be remembered, that the vessels are not in intimate contact with the dentine—a layer of odontoblast cells, the membrana eboris, intervening. The dental pulp consists of vessels and nerve-fibres with numerous cells and delicate connective-tissue, and it contains no absorbents, or at least none have been demonstrated in its structure.

A consideration of the anatomy of enamel and dentine—unique in the body—would, it might be supposed, be alone enough to show that they are incapable of anything like pathological action, that such action in enamel is inconceivable, and in dentine hardly possible. Wedl is the only authority with whom I am acquainted who emphasizes this fact, and who rightly, in my opinion, dismisses many false speculations with the simple statement that on anatomical grounds they are impossible. I do not know whether we all ought to disregard disputers who seem to believe in various occult forces and phenomena, irreconcilable with fundamental truths of anatomy and physiology. I think a great authority like Wedl may well disregard them, but I fancy it may be useful to expose them in the columns of a periodical publication.

Pathology must have its basis on anatomy and physiology; and one might think a truth need not be stated so obvious as that no apparent pathological phenomenon incompatible with indisputable facts of these sciences can be accepted. In presence of our exact knowledge of the structure of the teeth, some recent writers and speakers have glibly talked of

inflammation of enamel and inflammation of dentine; of retrograde metamorphosis of the dental tissues, and of the influence of 'vital force' and of 'diminished vitality' of the hard dental tissues as predisposing causes of caries. Anyone acquainted with the meaning of these terms must feel disposed to pass by such utterances with the ridicule which their gross and palpable absurdity richly deserves. But, astonishing as it may seem, some of these views have been accepted and repeatedly expressed by a metropolitan teacher of dental surgery, and the hypothesis of retrograde metamorphosis has been seriously adopted. It seems, therefore, worth while to attempt an elaborate refutation of these fanciful views, if only for the sake of the student, who, at least, seems in danger of being confused by such teachings, put forth with what to him must appear authority. It is, indeed, for the student that I mainly compose these papers. It is not likely there can be any considerable number of duly educated scientific men who will support fantastic theories, solely based upon vague impressions and incompatible with physiological fact. Writers who, without first overthrowing established fundamental truths and forming a new basis for their hypotheses, construct elaborate theories, or put forth dogmatic statements, irreconcilable with those truths, seems to me beyond the reach of argument. Their style of reasoning reminds one of the mental attitude of certain individuals described by George Eliot. She speaks of some people who may with difficulty be made to agree that, as a rule, two sides of a triangle are together greater than the third side, but will qualify the reluctant admission by the reservation that one must, however, be careful, for there is such a thing as carrying mathematical reasoning too far. A man who can speak of inflammation of enamel and dentine, or of retrograde metamorphosis of

those tissues, must indeed, in a like fashion, believe that anatomical fact may be carried too far, and that it is better to rely sometimes, not upon facts, but rather upon the phantasms of a vivid imagination.

Later on I shall come to deal fully with the arguments which have been put forth against the view, but shall now proceed with the proof of the truth of the above definition of caries. Admitting that the direct exciting cause is to be found in external agents, we must inquire what agents exist in the mouth capable of giving rise to the first phenomena of the disease. The answer is easy, and is capable of verification by anyone. Acid, capable of slowly dissolving enamel and starting disintegration of dentine, is commonly present in the mouth. Caries can be started by merely packing a pellet of wool, pressing upon the gum, between two teeth. After the lapse of a few days the acid secretion caused by the irritating foreign body will have commenced to dissolve and erode the adjacent enamel. We all, unfortunately, perform this experiment in providing our patient with artificial substitutes supported by frames surrounding natural teeth. We all know what is the effect inevitably, unless the most scrupulous care be taken, by frequently cleansing the frame and teeth, to prevent lodgment of particles, and formation of acid decomposition products on the surface of the enamel. The destructibility of the dental tissues, and the production of the initial stage of caries by the agency of acids commonly generated in the mouth, have been verified by too many authorities to be open to contradiction.

The further progress of the disease will be best made clear, while at the same time my argument is carried forward, if I now proceed briefly to summarize the researches of modern investigators worthy to be accepted as authorities. Those researches are alone worthy of notice

which are fully described and conducted upon scientific methods, including histological examination of the affected tissues by the microscope. Except for curiosity, and to note the gradual evolution of full knowledge, there is little use nowadays in discussing the crude guessings of ancient and early writers. Regarded in the light of modern science, the opinions of the early dental pathologists are not more absurd than those of workers in other departments. It was first necessary, before exact knowledge of disease could be obtained, that exact knowledge of the structure of the tissues in health should be gained; and this was impossible before the introduction of the microscope, and the perfection of methods of histological investigation. It was natural for old writers, ignorant of the real structure of enamel and dentine, to look upon these tissues as more highly organized than we now know them to be; to speak of their 'vitality,' to suppose that they underwent constant nutritive changes, to identify the phenomena which disease made visible in the tissues with similar effects in more highly organized parts, and to classify these appearances as atrophy, gangrene, and inflammation—conditions to which they, perhaps, bore a superficial resemblance.

I must, before quoting authorities, premise that I shall subsequently enter more fully into the question of the predisposing causes of caries—so often alluded to by authors; and that I shall also in due course examine more thoroughly the statement that caries, although, perhaps, excited by external agencies, yet does not consist of mere unresisted disintegration of enamel and dentine, but is really a disease attended by vital reaction in the affected tissues, by inflammation, or at least some true pathological phenomena. All the authorities I shall quote start from the foundation of anatomical facts which I have given; and it cannot be

too often insisted upon that these facts, if not alone enough to prove the truth of the view of caries which I am attempting to expound, are, at least, enough alone to prove the absurdity of other hypotheses to which I have referred, but which, nevertheless, I shall, before I finish, fully examine and refute. Much of this epitome and much of what I shall have to say throughout, must, I fear, necessarily consist of reiteration of what I have previously written on the subject, and may sound to some readers like a thrice-told tale; but I wish my argument, if concise, to be complete; and, I repeat, I write for the student, rather than for those who are already masters of the subject.

Sir John and Mr. C. S. Tomes' research demonstrates that caries is an effect of external causes, in which so-called 'vital' forces play no part; that it is to a great extent due to the solvent action of acids which have been generated by fermentation going on in the mouth, the buccal mucus probably having no small share in the matter; and when once the disintegrating process is established at some congenitally defective point, the accumulation of food and secretions in the cavity intensifies the mischief by furnishing fresh supplies of acid. The presence of globular masses of calcareous salts, which are sometimes found in the dentinal tubes near a carious cavity, and which are relied upon by some writers as evidence of pathological action, they regard as probably depositions from solutions of salts, and not the results of such action; and they maintain that even if it were conclusively shown that dentinal fibrils became obliterated by calcification, this would not be absolute proof of vital action; for they point out that albumen, even when out of the body, is able to form with calcareous salts combinations having a definite structure. They describe a peculiar appearance at one stage of decay, in which the

dentine in cross section looks as though built up of multitudes of tobacco-pipe stems. The zone of altered dentine surrounding the cavity where caries is advancing, they demonstrate is present in caries of dead teeth, and they show that this appearance, which was once thought to indicate pathological action, is due merely to passive decomposition of the tissues. Inasmuch as no characteristic appearances can be found to distinguish caries occurring in living from that in dead teeth refixed in the mouth as artificial substitutes, the hypothesis of vital action in any way modifying the disease must be abandoned *in toto*, and 'dental caries cannot, strictly speaking, be said to have any "pathology."'

Messrs. Tomes regard as the main predisposing cause of caries structural imperfections in the tissues (such as I shall fully describe later); and they recognise that the physical signs visible at the onset of the disease vary mainly in consequence of the disintegration commencing sometimes on an unbroken surface, sometimes on a surface the seat of congenital defect, as, for example, on a patch of porous enamel, or in a fissure. The other great predisposing cause is vitiation of the secretions of the mouth.

The appearances visible to microscopical examination during the progress of the disease, Messrs. Tomes regard as governed by the physical characters of the tissues. For instance, in the enamel the central portion of the fibres is the first to undergo decomposition. In the dentine they consider that, as a rule, the connecting material (the matrix) is the first, the walls of the tubes (the fibrous element of the tissue) the last to undergo disintegration.

Professor Wedl's observations agree substantially with the views of Messrs. Tomes. He classes caries under the heading 'Anomalies of the Secretions,' considering the

disease to be due to the effect of morbid secretions. Caries, he says, has its origin chiefly in abnormal secretions of the gums, of the oral mucous membrane, and salivary glands. He looks upon the secretion of the gums as most active, this secretion coming in immediate contact with and forming a viscid covering on the teeth. Commencing at favourable points on the exterior of the tooth, the disease spreads in the direction of the pulp-cavity. In consequence of decomposition of the secretions, acids are formed, which extract the calcareous salts from the hard tissues, and give rise to disintegration of the affected portions of the latter. These tissues are passive under this disintegrating process, and show no vital reaction, and no change whatever of an inflammatory character. He draws a distinction between dental caries and caries of bone. The latter is an inflammatory process which originates in the soft parts of the bone and erodes its hard tissues. Dental caries, on the other hand, commences in the hard tissues and spreads to the vascular pulp. The destructive process is promoted essentially by the accumulation of secretions and particles of food, and opportunity is afforded for the proliferation of leptothrix buccalis in the dead and softened dentine. Wedl fully recognises the part played by inherent structural defects of the tissues as a predisposing cause of caries. The other main predisposing causes are found in those diseases of the general system, during the existence of which the mixed saliva generally acquires an acid reaction.

With regard to the minute phenomena occurring in the tissues, Wedl observes in the enamel, as an early change, the presence of pigment of varying shade, such as I myself suggest is probably deposited by bacteria. The persistence of the contours of the prisms in places—that is, the more rapid solution of their centres than peripheries by the acid—

he considers at present unexplained, but thinks it possibly due to proliferation of a mass of leptothrix into the decalcified enamel. In the dentine he recognises two stages in the progress of the disease, which, however, cannot always be nicely discriminated—a preparatory stage of decalcification and softening, and a stage of direct disintegration. During the latter stage the tubes become enlarged and varicose and filled with leptothrix, which also he believes may be found in all the ramifications of the carious cavity, although he has not discovered its presence in the earliest stages of the disease. Wedl elaborately proves by microscopical examination, which he illustrates by accurate drawings, that carious dentine of reinserted human teeth (*i.e*, as artificial substitutes) and of artificial teeth made from the ivory of the hippopotamus, present the same appearances as are shown in ordinary caries, in respect of pigmentation, granular condition of dentinal fibrils, and thickening and varicose enlargement of the latter. The phenomena ascribed to inflammation, or to any so-called vital, or, more properly, physiological action, cannot therefore possibly belong to any such pathological process.

Leber and Rottenstein's work, a clear and thoroughly scientific exposition, goes to prove the entire identity of caries of dead teeth and of teeth made of ivory, with caries of living teeth; and they deny that any recorded observations authorize the assumption of any vital reaction, or anything to be compared to an inflammatory process in the affected tissues of living teeth. The initial stage of caries is due to the solvent power of acid, and when, in consequence of this, the enamel and dentine have lost somewhat their power of resistance, or a breach of substance, however small, has been produced upon the surface of the dentine, the fungoid growth, leptothrix, penetrates into the interior

of the tissues, and by proliferation, particularly in the dentine, occasions more rapid progress of the softening and disintegration than would have been the case under the action of acid alone.

M. Magitot holds the opinion that caries is due to purely chemical action upon the enamel and dentine. He describes a large number of his experiments, proving that enamel and dentine are soluble in acids not more powerful than such as may be formed and deposited in the mouth. He believes that in extracted teeth, submitted experimentally to the action of such acids out of the mouth, he has produced nearly all the phenomena of caries.

Mr. Salter's investigations, which are hardly less thorough and complete, or less solidly based on incontrovertible anatomical fact, are in agreement with the authorities already cited. He defines caries as softening and disintegration of the tooth's surface, gradually penetrating towards the centre. It is dependent altogether on external influences which are chiefly chemical.

The most recent investigators who have, like those just quoted, examined experimentally and by observation the whole question of caries, are Messrs. Arthur Underwood and Milles.

The more important portion of these investigations consists of microscopical examinations of carious dentine stained with an aniline dye. These examinations have been carefully carried on for several years, and with one perfectly constant result—namely, that in every section observed under an eighth of an inch objective with transmitted light, micro-organisms have been found in the canals in greater or less number according to the degree of softening and disintegration of the tissue. The organisms consist of micrococci, rod-shaped and oval bacteria, and

short bacilli. The presence of these micro-organisms has proved so far to be invariable in natural caries, and the number of sections cut and examined is now so enormous, that the observers feel justified in assuming that the presence of organisms is indispensable to the process. They do not remain like leptothrix on the outskirts of the diseased tissue, but penetrate as far as the disease, and even seem to extend slightly beyond the limits of the tissue recognisable by the unaided senses as carious. Another direction in which these observers have obtained a valuable negative result, is that although a weak acid is able to dissolve out the lime salts from enamel and dentine, the result of such solution is not completely like caries, either in its mode of attack, its colour, consistency, naked-eye appearances, or microscopical appearances. This statement may be verified by experimentally decalcifying a healthy tooth in an acid bath. It will be found that the enamel is completely destroyed *first of all*, and reduced to a condition in which it can be removed with a paint brush, and that the dentine is afterwards rendered elastic. The negative evidence was further strengthened by submitting a number of teeth to the action of malic and butyric acids, and even of saliva, under aseptic conditions—*i.e.*, in boiled flasks capped under the carbolic spray, both the fluid and the teeth having been previously purified. Years' exposure in these fluids produced no caries. With regard to the pigmentation which takes place in the progress of the disease —the tissues being stained of a brownish hue—I have myself suggested that this is probably due to the presence of pigment-forming bacteria. With this view Mr. Underwood agrees, and he has already produced experimentally a brown pigment by developing organisms on potato.

Messrs. Underwood and Milles describe the microscopical

appearances of carious dentine as follows: The tubes are filled with micro-organisms, which can be seen very plainly when stained with methyl violet or any of the aniline dyes. They appear to penetrate the canals at first in single file, and then, accumulating in vast numbers, to encroach upon the matrix until the canals are so enlarged that two adjacent often amalgamate and form one irregular tube full of organisms. Organisms can be seen in the branches of the tubes; and the microscope discovers here and there a narrow line of bacteria or micrococci penetrating, like the advance guard of an army, beyond the sphere of visible decay, in tissue which to the naked eye shows no trace of commencing caries. Besides the disintegrated tissues and foreign particles, there is to be found by the microscope in most carious cavities abundance of the peculiar fungoid growth, leptothrix buccalis, to which I have more than once already referred, similar to that which is deposited upon the surfaces of the teeth in all mouths in which the most extreme care is not taken in frequently cleaning the teeth. Leptothrix assumes the appearance of minute threads projecting from the surface of the carious dentine in enormous numbers. It was thought by Messrs. Leber and Rottenstein, whose admirable observations have been just quoted, that the leptothrix took the principal part in promoting the advance of caries. Leptothrix is a fungus, and Messrs. Leber and Rottenstein in their essay of 1868 classed, probably by mistake, all micro-organisms under that common heading—the general knowledge of micro-organic life being at that period in its infancy.

Examination of carious teeth in various stages of decay demonstrates the fact that certain changes apparently take place prior to actual disintegration in that portion of dentine through which the disease is advancing, and which is

situated immediately contiguous to the already disorganised tissue. This altered dentine, which has already been briefly mentioned, has a translucent appearance, and forms either a regular zone, or exists in isolated patches around the walls of the cavity. The appearance, which is, however, visible under only a *low* magnifying power, was thought by Magitot to indicate vital re-action, perhaps a natural effort to arrest the disease by calcification of the dentinal fibrils. It is found, however, that a precisely similar translucent appearance is always produced in caries of dead teeth and during the gradual softening of dentine by acid, and to this softening, and not to consolidation, the appearance in caries is due ; and I believe that Magitot, whose first observations were made many years ago, no longer adheres to his early opinion.

Messrs. Underwood and Milles once more demonstrate the fact that caries of extracted teeth retained in the mouth as artificial substitutes, is absolutely identical with the disease in living teeth. Attempting to nearly reproduce in every respect the conditions existing in the mouth—as to temperature and presence of saliva and decomposition products usually found there—they have endeavoured to produce caries in extracted teeth by carefully conducted experiments, but state that they have hitherto failed to induce changes in the dentine similar to those occurring in the mouth. They ascribe the failure (which, however, of course does not affect the main issue for which I am contending) to the impossibility of reproducing an exact imitation of all the conditions present in a living mouth—such as flow of saliva, secretion of mucus, and the various physiological processes going on in relation to the teeth.

Dr. Miller, has followed Messrs. A. Underwood and Milles through all the steps of their research, and confirms

them in many essential points. Dr. Miller not only holds that decay of extracted teeth retained in the mouth is in all its phenomena identical with caries of living teeth, but he affirms that all the microscopical appearances characteristic of caries may be produced out of the mouth simply by subjecting teeth to decomposing agents, such as are constantly found in the human mouth.

Many other observers, including Mr. C. Tomes, have already confirmed the statement that micro-organisms are invariably present in carious dentine, and have recognised all the principal appearances described by the last cited observers.

Mr. Charles Tomes considers that the constant presence of bacteria in carious dentine at a depth below the surface is now fairly established, and this seems to show that they are not merely preying upon already disintegrated tissue. In this respect there is a marked advance beyond the standpoint of Leber and Rottenstein, who described the leptothrix only, an organism which, however abundant on the surface, does not dip in far. The matter, Mr. C. Tomes goes on to say, seems to stand thus : micro-organisms play a part in the process of caries, and in their absence it is probable that it could not go on, or that, if it did go on, the result would be different ; but the exact share which micro-organisms take is as yet not made out. The microscopic appearances described by the older writers were, he thinks, perhaps due to their having seen masses of organisms without recognising them as such, but others, such as the well-known tobacco-pipe appearance (mentioned further back), though eminently characteristic of caries, remain quite unexplained. While therefore, he continues, a substantial advance has been made in the understanding of the matter, there still remains an extensive field for investigation in the exact interpretation

even of appearances which are familiar to all observers. But interpret these appearances as we may, we cannot ascribe them to pathological action; and, as regards the 'vital theory,' adds Mr. Tomes, its supporters, to be logical, ought to affirm that the process of caries proper ceases with the inflammation and death of the pulp, and that what takes place afterwards is something different, its apparent continuity notwithstanding; but none of them, to his knowledge, have ventured to face this difficulty.

On *à priori* grounds, in the light of recent discoveries, it might have been safely reasoned that micro-organisms must be present in carious cavities. The decomposing *débris* contained within such a cavity must necessarily undergo a process of putrefaction or fermentation. These processes are due to proliferation of certain organic forms always present in the dust floating in the atmosphere. Admission of dust-charged atmosphere to dead organic matter gives rise to putrefaction and fermentation.

All these later discoveries lead to a tolerably sure explanation of the stages of the progress of caries, and these are as follows: the enamel is first perforated by acid, or when a fissure or flaw exists this is gradually enlarged by the same agency. The dentine being reached, the organisms find their pabulum in the fibrils of this tissue, in which they proliferate, and destroying them, penetrate along the tubes. Acid is generated by the organisms, and this assists in dissolving the lime salts of the matrix. In this manner the more organic tissue, the dentine, is eaten away more rapidly, while the enamel, partly dissolved and undermined, either remains as a shell, or breaks away for lack of support.

To sum up so far, we have first the fact that the anatomical characters of enamel and dentine are such as preclude the possibility of these tissues either initiating or carrying on

any pathological process, or that at least it is impossible to put into terms of plain language any explanation of the *modus operandi* of pathological change in structures of such a character. Secondly, we have the facts established by the investigators I have cited, namely, that the appearances in carious dental tissues are all interpretable, and solely and exclusively interpretable, as changes due to disintegration, unaccompanied by vital reaction of any kind; and thirdly, we have the fact demonstrated beyond dispute, *that caries of dead teeth retained in the mouth is identical in all its objective phenomena with the disease in living teeth.* This last fact alone is surely enough by itself to establish the truth of the definition of caries with which I started, and I must contend that in this fact alone we have ample knowledge of the essential nature of the malady. In addition to anatomical considerations and to evidence based upon the researches which I have attempted to epitomise, there is a considerable number of facts which further testify to the truth of the view I am endeavouring to expound. Of these facts I shall have to speak later, when I examine the other hypotheses which have been from time to time propounded; and when I shall have shown how untenable any of these hypotheses really are, it will be admitted, I think, that I have not spoken too confidently of our full and certain knowledge of the significance of all the really important phenomena of caries.

Before proceeding to the next link in the chain of my reasoning, a few more words must be added as to the sources whence are derived the acids—the active agents in initiating caries. These acids, principally malic, butyric, and acetic, are mainly the products of chemical change and fermentation, set up in fragments of organic matter—food, mucus, and epithelial scales—which are commonly present in the mouth, and lodged upon the teeth.

Acid may be derived from several other sources. It may be secreted by the mucous membrane. The normal secretion of the membrane is small in quantity and slightly acid. In health the acid is at once neutralized by the alkaline saliva, with which it mingles; but when the membrane is congested or inflamed the mucus increases in quantity, and becomes more strongly acid in character. Then, again, many forms of organisms themselves produce acid. Acid is eructated in many gastric disorders; and an acid, instead of alkaline, reaction is shown by saliva in several diseases.

The next topic in continuation of my argument is the predisposing causes of the disease. If the view of the nature of caries which I am attempting to demonstrate be correct, what can be the predisposing causes of the disease? These are, first, innate structural defects in the tissues, which render them more easily susceptible to the action of agents. Imperfections in structure may be owing to defect either in the quantity or in the quality of the tissues. Defects in quantity consist of pits and fissures in the enamel and dentine. These vary, in extent, between minute cracks perceptible only under the microscope and cavities plainly visible by the naked eye. They may penetrate the enamel alone, or may extend to a greater or less depth into the dentine also. Prof. Wedl especially notes that minute cracks, often only visible under a low magnifying power, are to be found here and there in enamel, even of the best quality.

Defects in the quality of the tissues may affect the whole body of the tooth, or may be confined to certain spots in the enamel and dentine. The durability of the dental tissues varies considerably; in one individual the teeth withstand the extremest hard usage combined with neglect, in another they show traces of disease within the earliest

years of childhood, and are destroyed sooner or later even in spite of active treatment. Enamel and dentine of such delicate teeth, examined microscopically, are found to present well-marked evidences of imperfect formation. The enamel, instead of being a densely hard, almost homogeneous mass, is comparatively soft, owing to imperfect calcification, and porous in consequence of incomplete coalescence of its formative elements. It retains a marked fibrous character. The fibres are imperfectly blended ; their tranverse striæ are clearly evident, and they are often penetrated at their centres by tubes or small cavities. At parts the fibrous character may be altogether lost, the tissue consisting of an imperfectly united granular mass.

The dentine, in addition to undue softness, exhibits throughout its structure, and especially immediately beneath the enamel, patches of defective tissue similar in character to the granular layer, which in well-formed teeth exists only at the point of juncture with the cement. In the spaces within this defective tissue—sometimes called interglobular spaces—the tubes end, or they may even run on and terminate in dilated extremities within the substance of the enamel.

It does not of course by any means always happen that all these structural defects exist together in one tooth. Their degree and character vary infinitely. In teeth of otherwise good organisation one or two pits or fissures, or small patches of defective tissue, are, as just mentioned, often found ; whilst in teeth of generally inferior structure there are often to be discovered portions of still lower formation.

It is easy to perceive how these structural defects furnish lodgment for acid-forming substances, and render the teeth more easily acted upon and destroyed. The fact that these

innate defects vary infinitely in extent and character in teeth of different individuals, must especially be borne in mind in following my argument.

With regard to the origination of innate structural defects of the dental tissues we know very little. We know that inherited syphilis, which seems to interfere with the due development of all the tissues derived from the epiblast, leads also, in some instances, to imperfect formation of the tooth tissues; and the characteristic short, narrow, peg-shaped, notched incisors (first identified by Mr. Jonathan Hutchinson), furnish, when present, unequivocal testimony to the presence of the hereditary taint. In my experience—which, I believe accords with that of the majority of observers—it is, however, comparatively rare to find this typical form of defect in syphilitic children. Out of the large number of cases of children undoubtedly subjects of syphilis which I encountered in hospital practice, not one per cent. displayed the dental sign. Many possessed teeth perfect in form and beautiful (for their whiteness) in colour, but I have seldom seen a syphilitic child with other than ill-made dental tissues—as evidenced by the early onset and rapid progress of caries. I have therefore concluded that hereditary syphilis usually causes dental deterioration, although it may not often leave an unmistakable mark modifying the external form of the organs. Honeycombed teeth—teeth with enamel full of small pits, and which are quite distinct, although sometimes mistaken for syphilitic teeth—are found in a large number of instances in which convulsions have occurred during infancy, and with this condition is sometimes associated lamellar cataract. This points to some interference with the development of the same series of embryonic structures, and it has been sought—without complete proof as yet—to ascribe these cases to the

administration of mercury, and consequent production of inflammation in the neighbourhood of the developing teeth.

Beyond these more or less solid facts the whole subject of the origination of dental deterioration is still, it must be admitted, in the region of hypothesis; yet we know not only that there exist a vast number of individuals with ill-made teeth, but that it is comparatively rare among highly civilised races to find an individual with really well-made dental tissues. No doubt dental deterioration has accompanied civilisation. Probably other factors (into the discussion of which I must not digress) have had their share in producing the result; but it may be largely accounted for by the comparative disuse of the dental organs to which civilised and luxurious habits lead. A high standard of physical comfort implies comparative disuse of teeth and jaws. The advance of the art of cooking, together with flour refining and soft bread making, renders all food so comparatively soft that it calls for little chewing. Disuse of the organs of mastication leads in time to their wasting; characteristics of parents are transmitted as hereditary characters to their progeny, until at length a deterioration commenced in one individual becomes a family defect, and, finally, a generation is produced in which the whole apparatus of mastication—teeth, muscles and maxillæ—is inferior. Evidence of the influence of heredity on dental development appears daily before every practitioner. Children's jaws and teeth commonly resemble those of one or other parent. Abnormalities such as 'underhung jaw,' the absence of a particular tooth, or the malplacement of one of the set, frequently run through a whole family. The occurrence of visible patches of defective enamel or the onset of caries in corresponding teeth are not less frequently encountered family traits.

This slight digression leads me to the next predisposing

cause of caries, namely, crowding and irregularity of the teeth—due to smallness and malformation of the maxillæ. Whilst it is the exception to find an individual of a civilised race with dental tissues perfect in structure, it is, as I have just suggested, equally uncommon to find one with perfectly formed jaws. In a well-formed jaw every tooth is subjected to the constant beneficial friction of the tongue, and of food during mastication; whilst all the dental surfaces are in health constantly washed by alkaline saliva. These conditions render impossible the prolonged lodgment of decomposing acid-forming products about the teeth, and so do away with one potent factor in the causation of caries. But it is the exception to meet with a case in which at least some crowding of teeth does not exist—most often, nowadays, there is, at least, no room for the wisdom teeth, and one or other of the set more or less overlaps its neighbours. Between such a slight case all degrees of crowding and irregularity are to be observed up to the extreme instances of small V-shaped maxillæ where the teeth are found leaning at all angles, wedged together in what looks like one confused mass. It is easy to understand how such conditions of crowding and irregularity make certain the accumulation of decomposing foreign particles in the unnaturally narrow interstices between the teeth, and in the nooks and crannies formed by the irregularity. And the fact upon which I wish specially to lay stress is that the extent and character of irregularity and crowding of the teeth vary infinitely in different individuals.

The third predisposing cause of caries which I have to describe is made up of all such diseases as are accompanied by vitiation of the oral secretions; or which tend to the formation or deposit of acid, and the accumulation of products of decomposition within the mouth. This is, perhaps,

the most important of the predisposing causes of caries; and, indeed—as I have already stated—Wedl, in his great work on the pathology of the teeth, classes caries under 'Anomalies of the Secretions,' holding that it is not really a disease of the dental tissues, but rather disintegration of the tissues, due to the physical effects of morbid secretions. It is certainly evident that with a free flow of healthy saliva, and a due secretion of normal mucus, there would be much less caries; but we must not overlook the effect of decomposing remains of food, and the opportunity which is afforded by structural imperfection of the tissues and irregularities of the teeth for the prolonged lodgment of *débris*, and its fermentation and putrefaction in contact with, or even within the substance of, the enamel. There is hardly a single derangement of health which is not attended by some vitiation of the secretions of the mouth; and the physician finds, as a rule, no surer indicator of a lowered standard of health than a foul tongue. From the occasional foul tongue and clammy mouth accompanying a transient attack of dyspepsia, from which probably even every robust individual suffers now and again, every degree of derangement of the secretions of the mouth is met with in disease, up to the severe condition associated with the zymotic fevers, such as smallpox or typhoid. During these diseases the secretion of saliva is scanty, often almost suppressed, and the teeth remain, perhaps for weeks, coated with sordes,—accumulations of epithelial scales, viscid mucus and other foul secretions, crowded with bacteria and overgrown with leptothrix. The condition of ill-health accompanying pregnancy in some women furnishes another instance in point. In most of these cases the symptoms are mainly due to disorder of the digestive organs; the appetite is morbid and capricious; vomiting and eructation of food frequent; the tongue is

foul, and the gums very frequently in a condition of chronic congestion or sub-acute inflammation attended with erosion of epithelium and secretion of muco-pus or tenacious mucus around the necks of the teeth. Then in gouty attacks the saliva has often an acid instead of alkaline reaction. In scrofulous subjects there is commonly a characteristic condition marked by chronic congestion and swelling of the gums, with secretion of viscid mucus. Some phases of syphilis and of phthisis, diabetes, chlorosis, and chronic alcoholism, are marked by congestion and inflammation of the gums, by stomatitis in some form, or by distinct and easily recognisable morbid changes in the secretions of the mouth. It is needless, for my purpose, to multiply instances or to examine further or more minutely these affections. The main point to be observed is that like the other predisposing causes of caries, this last varies infinitely in different individuals ; but unlike the other causes I have mentioned, which are constant and persistent, vitiation of the secretions is marked by the utmost variability and inconstancy ; for, as I have pointed out, it accompanies throughout life, in corresponding proportion, the smallest, not less certainly than the greatest departure from the standard of perfect health. If these facts be borne in mind in conjunction with what has been said upon the subject of the other predisposing causes of caries, there need be no difficulty in understanding how the origination of this affection is often coincident with the outbreak, and its advance simultaneous with the progress, of constitutional disease ; nor in understanding how caries when previously present and slowly progressing may, under these circumstances, be accelerated, and run a rapid course. It is instances of this kind which have led to the altogether erroneous assumption that the destructive process may be induced or hastened by morbid conditions

arising from within the teeth—an assumption about which I shall have more to say presently.

A consideration of the three predisposing causes enables us to understand clearly why caries is commonly associated with various diseases and cachexiæ, and why, to a greater or less extent, it shows itself, sooner or later, in every individual whose dental development is not perfect and whose general health is not invariably at the highest level. Given (1) structural imperfection of the tissues varying infinitely in different individuals; (2) crowding and irregularity of the teeth, equally varying in degree; and (3) vitiation of the oral secretions, not only varying in amount, but extremely irregular in occurrence and duration through the lives of different individuals, and we account fully for the facts without needing to invent any questionable hypothesis. The mutual relation of these causes is of course often too subtle and too intricate to be traced, even if we had the patient under constant observation; but these causes are demonstrated facts; they suffice to account completely for the effects ascribed to them, and these effects cannot be accounted for on any other theory not based upon pure supposition and not irreconcilable with the anatomy of the dental tissues.

Many points in my subject I have so far purposely passed over in a cursory manner, leaving them for discussion in the next section of these papers—the examination of the views of various representative writers who have put forth opinions at variance with those I have expressed. But first a few words on the subject of authority in matters of scientific opinion.

Near the outset of these papers I remarked that those authorities alone are worthy of notice whose researches are fully described and conducted upon scientific methods; but it seems worth while now to point out the larger grounds

upon which the value of utterances put forth with a show of authority by writers or investigators may be mainly gauged by the student; at least he can be taught to recognise salient features which may enable him to reject that which is utterly worthless. Now, the only ground upon which any statement in science can be accepted is the assurance that if we carried out ourselves the train of reasoning and research described by an author we should irresistibly be drawn to the conclusion which he professes to demonstrate. It is, therefore, necessary, first of all, that we agree to the truth of the premises from which a start is made. For instance, if a mathematician gives us the result of a new complex problem, which he has for the first time solved, we may with some safety accept it if we can assent to his postulates and can follow him through all the earlier steps of his reasoning, until he takes his new departure; but if he bases his argument on such a manifest absurdity, for example, as that parallel lines sufficiently prolonged may at length meet, we need not trouble ourselves further, for we must see at once that however elaborate the theory based upon such an hypothesis, it must be unworthy of attention. So in our case; if a writer set out with some statement, such for instance as that enamel is capable of undergoing inflammation, we may safely pass him by in full assurance that his observations will be worthless, since he starts from premises manifestly absurd. Of course, even the simplest physiological statement is founded upon highly complex data, not like a mathematical problem, upon self-evident truths whose negation is unthinkable. None the less is it often possible to perceive at a glance the absurdity of a quasi-physiological assertion; and so no one duly acquainted with the anatomy of the tissue, and clearly conceiving the nature of the pathological process, could hesitate for one instant in rejecting as absurd

such a term as inflammation of enamel. Yet this expression, which I have, therefore, purposely cited, has been by a recent author employed as though it were an unquestionable matter of fact needing no justification. And again, as every scientific exposition must have not only its basis on previously demonstrated fact, but must be built up entirely of similar material, no new term or statement can be received until it is fully explained and verified. The strength of a chain of reasoning is equal only to that of its weakest link, and if any indispensable link from first to last throughout exhibit a flaw under the strain of logical examination, the whole must be abandoned.

There are two fallacies from which even able scientists are not always able to keep clear, and these are especially the bane of an inferior class of writers, in whose works the failing is often so conspicuous that it cannot escape the notice even of the inexperienced. The first of these faults is hasty generalization from insufficient facts. This ought to be a maxim in scientific investigation: take care of the facts, the theories will take care of themselves—that is to say, accumulate facts, test and establish them, and when there are enough, deduction will almost evolve itself spontaneously. The second fault is the fallacy in arguing *post hoc ergo propter hoc*—the error of associating effects with causes merely because they have relation in sequence, when their connection cannot be fully demonstrated, and when such connection is not supported by, or is even incompatible with, incontrovertible facts of physiology. And it must not be forgotten that every new observation in science is at once eagerly scanned by ardent scientific workers; new researches properly described are soon gone over step by step by numerous experimenters, and the statements, if true, are never long without full corroboration. An investigator,

therefore, whose statements stand alone for any length of time, may be, at least, regarded as untrustworthy, or rejected if not bearing the severest scrutiny. Be the personal qualifications of an authority from whom it emanates never so high, no unproved statement is to be taken on trust. No scientific explorer worthy of the name thinks of resenting such healthy scepticism—the only safe mental position in regard to scientific research. On the contrary, probing examination is sought for and welcomed. Every one engaged in the elucidation of biological phenomena of any kind knows full well the constant danger of fallacy which accompanies each stage of the inquiry, and nothing can be more serviceable to the worker than, on the one hand, that error should be early discovered, and on the other hand, that new facts withstanding scrutiny should be speedily established in an unassailable position.

Unfortunately, however, it is by no means in every instance an easy task for the novice to discover and expose an author's fallacies by the application of the simpler rules of logic; and the difficulty of the task is often increased in proportion as a view of the error in its nakedness is obscured by an enveloping cloud of verbiage. As a famous writer puts it: 'A very long discussion is one of the most effectual veils of fallacy. Sophistry, like poison, is at once detected when presented in a concentrated form; but a fallacy which when stated barely, in a few sentences, would not deceive a child, may deceive half the world if diluted in a quarto volume.' And again, with equal wisdom has it been said: 'People talk about evidence as if it could be really weighed in scales by a blind justice. No man can judge what is good evidence on any particular subject unless he knows that subject well.' For these reasons the student, and more particularly the junior student, must, after all, be

very much at the mercy of his teacher, and hence is it the more obligatory upon every expounder of science, and especially upon such as set themselves up as instructors of students, that their facts be incontrovertible, their reasoning unassailable, and their language clear and unequivocal. And this mention of language leads me to another consideration which must not be passed over, in the case of many writers on dental subjects in late years—it is the quality of their writings from a literary point of view. When one scans for the first time a new author—I speak particularly of a scientific author—the best rough test of his value is the literary test; and this test is the one by which the student may most easily, at any rate, often at once detect the incompetent and unmask the pretender. Briefly the case stands simply thus: if one finds an author whose style is obscure; whose sentences at first glance seem to have no very clear meaning; at a second more careful examination are found ambiguous; and when construed according to grammatical rules are seen to be nonsense, we may safely throw him aside as utterly unworthy of attention.

It is the first duty of an expositor of science to master the mechanical art of making his meaning clear; he is not called upon for eloquence or ornate verbiage, but without lucidity he is worse than worthless. Let the student therefore unhesitatingly discard the author whose important statements are obscure from disregard of the common rules of syntax. There was once a German emperor who assumed a title implying his superiority to the vulgar conventionalities of grammar; but no air of authority on the part of a pseudo-scientific writer can disguise the well-established fact that a man's diction on any subject is the reflex of his thought, and if his words are confused it is only because his conception of the subject is equally involved and obscure.

Judged by this last test alone, not to speak of the others just named, most of the writers on dental pathology, whose statements are opposed to the authorities upon whom I have relied, are, in plain truth, really unworthy of notice.

Here may be the most suitable place to remark that I have relied upon no authority whose works will not stand all the tests mentioned; besides which, the student can verify for himself the fundamental facts upon which is based the explanation of caries which I am attempting to expound; and he is, in truth, asked to take nothing on trust, since such facts as he cannot investigate for himself rest upon the testimony, not of one, but of numerous independent unimpeachable authorities. Every student can examine for himself the anatomy of enamel and dentine, and, when he has grasped the full meaning of the term, can recognise the impossibility of enamel initiating or carrying on a real pathological process. Every student can demonstrate for himself the solubility of the dental tissues in acid no stronger than is produced in the mouth. Every student can recognise the continuity of caries before and after death of the pulp, and can see that in salient features caries of dead teeth and living teeth is identical. Everyone can see for himself that dentine, from its anatomical characters, is incapable of undergoing interstitial changes similar to those that affect non-vascular tissues of different structure under inflammation; and everyone can see that caries is unaccompanied by vascular action, such as must be present were it an inflammatory process. About the phenomena of inflammation I shall have more to say presently. Suffice it for the moment that there are facts connected with these phenomena, and many others besides those I have just mentioned, which the student can easily verify for himself; and if he cannot demonstrate from beginning to end the

truth of the explanation of caries which I am setting forth, he may at any rate furnish himself, by personal observation, with a sufficiency of solid fact to exclude by *reductio ad absurdum* every other opposing theory that has been promulgated.

Opposing theories may be divided into three categories. First, those that would make out caries to be a true disease, carried on by pathological action initiated within the tissues; second, those admitting the disintegration to be due to external agents, but insisting that these agents are inert without the prior occurrence of morbid changes in the tissues, lessening their power of resistance, and predisposing them to attack; and thirdly, those theories which admit that caries is entirely due to external agents, but maintain that the tissues—or at least dentine—are not passive under the process of disintegration; and assert that the process is accompanied by inflammatory phenomena, or some kind of vital reaction. I am, however, not aware of any recent author bold enough to support the first of these theories in its entirety. This view is confined almost exclusively to obsolete works composed before the anatomy of the teeth had been clearly made out, or their physiology properly understood. The anatomy of the tissues I have already briefly described; and we must not shut our eyes to the light which has been also shed on the subject (in regard to possibility of nutritive changes and pathological action in the tissues) by the full knowledge which we now possess of the histogenesis of enamel and dentine—the changes which take place in the tooth germ, by which its elements are converted into these tissues. Accurate knowledge of this subject was entirely wanting to earlier investigators, many of whose mistakes are thus accounted for.

We now know that enamel is developed from a pulp com-

posed entirely of epithelial cells, and that it retains its epithelial character throughout the process of calcification. This process begins at the surface of the dentine—of which surface calcification has previously commenced—and progresses outwards. The calcareous matter is deposited through the medium of vascular papillæ, which, arising from the contiguous surface of the dental sac, penetrate to a slight depth the external epithelium. Once formed, the enamel is cut off absolutely from all vascular connection. To believe in the possibility of nutritive changes inducing morbid states in this tissue, we must first imagine some means by which this almost homogeneous calcareous mass could assimilate molecules of nutritive material; and we must next imagine the conveyance of nutritive and effete material by the vessels of the pulp, through the odontoblast layer, *viâ* dentinal tubes and fibrils to and from its destination, as far as the surface of the enamel. An imagination capable of conceiving all this would have, of course, no difficulty in framing a theory of caries to match. And let it be always borne in mind that it is in enamel that caries commences, unless there be some very rare cases in which a flaw allows destructive agents at once to penetrate to the dentine.

Similar considerations apply with almost equal force to dentine. The dentinal pulp is derived from the rudimentary mucous tissue. After a time the epithelial cells, of which it is largely composed, become specialised and arranged at the periphery to form the odontoblast layer or membrana eboris. By calcification of these cells dentine is formed; the uncalcified centre of each cell constituting the fibril. The remains of the dental papilla consisting of fine connective tissue, bloodvessels and nerves, with numerous cells, occupies permanently the central cavity as the pulp or nerve of the tooth. The outermost cells of the

pulp—as already mentioned—form a layer on the surface of the dentine, the wall of the pulp cavity, and send off processes which are continuous with the fibrils. The vessels, therefore, do not ramify immediately in contact with the dentine.

Looking at these facts, and at all the anatomical characters of the tissue, it is scarcely conceivable that dentine, more than enamel, could be the seat of nutritive or morbid changes, and one cannot wonder at some writers contemptuously dismissing speculations involving negation of these irrefutable facts. But I have undertaken to examine some of these speculations, and I must proceed with my task. First, however, I must qualify my statement that no complete theory of caries, such as would be placed in my first category, has been put forth in recent years, for I think the work of the late Mr. Bridgman would fall under that classification, and it may be well, perhaps, before going on, to give a few words to it. This essay was written some twenty years ago, and was a most ingenious and consistently wrought out elaborate speculation, well deserving the prize which was awarded it by the Odontological Society. Pure speculation it was, however, from beginning to end, and I doubt if Mr. Bridgman would himself any longer uphold it in the face of our later knowledge. Briefly stated, the hypothesis was to the effect that the dental tissues, being in different electrical conditions, were capable—in conjunction with the fluids of the mouth, under some circumstances—of carrying on a process of spontaneous electrolysis, resulting in disintegration (caries) of the enamel and dentine. The foundation and every essential step of the reasoning was, I repeat, purely hypothetical, and deserved notice—or, at least, deserves notice now—only on account of its ingenuity.

Of those who have adopted the second class of theory—

those who attempt to prove that predisposing causes of caries are to be found, not in external agents, but in intrinsic morbid changes in the tissues, I shall choose for examination two writers who, from their professional position, must be considered representative men—men capable of saying the most that can be said in support of such views. There exists altogether a vast mass of literature on the subject, most of which it is difficult to characterise in moderate terms, and the best of which can hardly be described fairly as other than mere superficial impressions, unfounded on fact, and unsupported by scientific observation. With regard to compilation of useless voluminous literature containing the record of only such shallow impressions, some remarks were lately made by Mr. C. Tomes, which, it is to be hoped, may be taken to heart by the offenders in question. Speaking in a discussion on caries, which I had opened at the Odontological Society last year, Mr. Tomes remarked that the discussion arose, indirectly, as a kind of protest against the subject being befogged by a great number of generalisations based, not upon scientific facts capable of being recorded, and capable of being accurately described, but based upon general impressions. 'It seems to me,' Mr. Tomes continued, 'that any person who contributes to the literature of any subject his general impressions, and does not put down any recorded facts from which other persons can form their own impressions, has not only contributed nothing of value to the subject, but he has committed a species of literary scientific crime, because he has given other people the trouble of wading through a quantity of material, and when they have done that they will find that they will be not only none the wiser, but will probably be somewhat befogged.' However, I now give the first sample of the best that can be said in support of the

particular theory now in question. In a style well suited to a description of the mysterious forces to which he attributes the origination of caries, the author thus expresses himself:

'That the conditions prevailing in the progress of dental caries are dissimilar to those which take place in the inflammation of most soft structures, such as hyperæmia, effusion, cell-migration, softening, fatty degeneration, etc., etc., we can readily admit, without denying to the process a pathological character.

'The teeth of every person must in the mouth, like every other portion of the body—the hair, nails and skin included—be continually exposed to conditions, which, but for a prevailing something, would ere long subject them to those changes which all highly complex nitrogenous bodies undergo when removed from its sphere of influence. It is this prevailing something, ceasing or changing with the loss of life in an individual, that immediately permits the existence of those affinities, or no longer opposes their action, which tend to break up into simpler forms the more complex chemical compounds of which an animal or vegetable body consists, and to which consequently we apply the vague term "vital force," which, if actually a force, is probably not more distinct from the chemical than the latter is from the electrical, or any of the so-called forces; indeed, it may be only one manifestation of the chemical force. While this exists or remains unchanged, the teeth are, so to speak, protected from conditions, viz., moisture, warmth, action of acids, and bodies themselves undergoing change and decomposition, which would otherwise certainly allow the exercise which those affinities are ever exerting to break up complex organic compounds.

'We are prepared to grant that such affinities may when strong overcome that resistance, as well as have their full action when it is weakened, changed, or withdrawn, but this would in our eyes constitute it a pathological process.'

This remarkable exposition is further elucidated by the following equally striking paragraph:

'That the microscopical appearances as exhibited in the decay that occur in teeth employed as artificial substitutes should closely resemble the same in teeth naturally attached in the mouth is rather what we should look for, and, to our mind, is no evidence that the process is not a pathological one, both having been brought into the same or nearly similar conditions by pathological processes; the former by probably the death of the whole individual of whom the tooth formed a part, the latter by the death of the portion of the tooth attacked, the conditions of the subsequent decomposition being almost precisely the same. If we are bound to assert that the loss of vitality in the dentine of a living tooth is a result of inflammatory action, then we must admit

that our evidence of its existence are very small ; recent research would rather tend to show that it might be so, and, in the case of affected cementum, there is every ground for believing it to be so ; but the loss of vitality in a part may occur independently of inflammatory action, and yet, we presume, deserve the appellation of pathological.'

From a literary point of view, this is certainly a most astonishing passage. It is a wonderful specimen of tortuous involution, and, containing a rare collection of errors in syntax, it might well be set for correction as a test of the grammatical knowledge of students at a preliminary examination, and it at least deserves preservation among the 'Curiosities of Literature,' as a sample from a work made up mainly of similar stuff, which in these days of 'preliminary examinations' (not to speak of School Boards and compulsory education) could be seriously put forth for the instruction of students in the concrete facts of a subject in natural science. I recollect a certain author's style being described as worthy to be termed lucid by virtue of the ease with which it might be seen through, but the opacity of the above is baffling, and it is possible to catch no more than a glimpse of any solid statement within the enveloping cloud of words. The meaning, however, may be guessed—which, I repeat, is more than can be said of most of the 'impressions' recorded on the subject of dental pathology. And here is another utterance of the same author which may help us in our guess work from falling far from the mark :

'And then, with regard to the evidence of this acid state of the saliva, he had himself, many years ago, made a very large number of observations upon this point, expecting to be able to connect an acid condition of the saliva with, at all events, very active or acute cases of caries. But he obtained no such result. In cases of pregnancy and those convalescent from the exanthemata, the conclusion was the same, and yet in such cases it often occurred that teeth which, until these conditions supervened, had been excellent, then underwent very rapid decay. This had led him to doubt very much a simply acid cause for caries, and had brought him to believe that the greater liability to

disease in such cases arose from a lowering in the power of resistance of the teeth to agencies external to them—that is to say, to the ordinary laws of chemical affinity.'

It is difficult to render into plain language statements whose principal terms really pass comprehension, but I think we may not unfairly paraphrase these utterances thus: The dental tissues are *probably* pervaded by vital force. This force is a distinct entity which, diffused through the substance of the tissues, maintains their vitality and prevents their spontaneous decomposition. From diminution of this force, or from abatement of some other inherent potentiality (the 'prevailing something'), equally occult and indefinable, enamel and dentine frequently suffer impairment of vitality, and pass into a condition which renders them susceptible to external morbid influences.

I shall at once show that in view of facts which his practice every day demonstrates, and which are so constantly thrust before his eyes that one cannot believe in the possibility of his ignoring them, it is simply astounding that any dental surgeon can be found to talk of vital force—admitting the existence of such an entity—or of impaired vitality of the tissues—admitting the possibility of this—as elements in the causation of caries. But I may first remark that modern science goes to prove pretty conclusively that 'the known forces of inorganic matter operating in the special collocations of organic bodies account for the phenomena of life without leaving a residuum, and therefore the theory of vital force becomes unnecessary.' Be this, however, as it may, physiology deals only with recog nisable phenomena, not with noumena, not with phantoms; and concerns itself no more with vital force than with witchcraft, or the evil eye. The existence of vital force, witchcraft, and the evil eye is no doubt equally demonstrable to a certain order of intellect; and upon such an

intellect no disproof of their existence, however logical and conclusive, would have, in the presence of a fixed belief, the slightest effect. Vital force, and such like imaginary entities, had their place in men's thoughts once upon a time. They formed a species of metaphysical ghost; and ghosts of this kind are no longer visible to those who stand in the illuminated atmosphere of modern thought. It is, however, not necessary for me to lay this ghost of vital force—if it still haunts the imagination of some individual—for, as I have said, if it were no ghost, but indeed a tangible reality, its existence could, with equal ease, be shown to be entirely unconnected with dental caries. If it is not necessary to introduce the hypothesis of vital force into physiology, still less is it necessary to introduce it into pathology. The facts of pathology are concrete, and manifest themselves either objectively or subjectively—or in both ways—by inflammation, by tissue changes, by atrophy or degeneration, and by disordered function, or by other well-known phenomena of disease. That the hard dental tissues may, like structures more highly organised, undergo morbid changes, rendering them less able to resist disease, is a shallow impression; but I repeat I shall at once show that even if this were not so, an overwhelming mass of facts exists to prove that such changes have no part in the production of caries. But such changes in enamel and dentine are obviously impossible. We know very well that structures which, unlike enamel and dentine, are constantly undergoing nutritive changes, require for their maintenance in health and for power to perform their function, a proper supply of nutritive material by the blood, as well as due stimulation through a healthy nervous system. And it is from failure of necessary physiological activity of this kind, and by this means alone, that parts can be brought into a

state predisposing them to disease, or actual pathological change can be induced in them. We have only first to think of the anatomy of a tooth as a whole, and of enamel and dentine in particular; and more especially to think of the structure of enamel—the starting point of caries; and then to realise the nature of the physiological factors necessary in the production of malnutrition or impaired vitality —we have only to bring these things vividly before our minds to perceive the absurdity of a belief in the possibility of such morbid conditions in dentine, and *à fortiori* in enamel. To some of these points I shall recur presently.

Considerations such as I have thus briefly suggested would, one might have thought, have sufficed to prevent the reception—on any duly educated intellect—of the shallow impressions under discussion; and these considerations not sufficing, the glaring facts I have now to mention ought surely to have prevented such baseless speculations. The 'impressionist' does not condescend to explain how he places in line with 'highly complex nitrogenous bodies' enamel, which contains at least 95 per cent. of calcareous matter, and dentine, which contains 72 per cent. This is a detail too trivial to detain an 'impressionist' of this class. Probably he does not quite mean that these tissues are highly complex nitrogenous bodies; he only means that, *mutatis mutandis*, they behave in the same way under similar circumstances. But, of course, this is not at all the case. We know very well that if a piece of skin, fat, or muscle, or a portion of any highly organized tissue, a complex nitrogenous structure, be severed from its connection with the living body, it will speedily undergo decomposition; on the other hand, we know there is no limit to the number of years an extracted tooth exposed to the atmosphere may endure, without appreciable change in the enamel and den-

tine. We are not told to suppose that in the case of the tooth a modicum of the 'prevailing something' accompanies the amputated organ, and prevents 'the more complex chemical compounds of which it consists from breaking up into simpler forms;' but if we accept the 'prevailing something,' there is no reason why we should not ourselves add this simple item to the theory, and so make it complete.

We know, further, we may take a tooth which has been extracted for months or years; we may cut off the crown, and affix it, as an artificial substitute, by a pivot or peg, permanently to the root of a broken-down incisor, thus placing this dead crown in the fluids of the mouth, surrounded by all the circumstances favourable to its decomposition, and yet in this situation it will be neither more nor less liable to decay than its living neighbours—teeth with living pulps and living periosteum; and, indeed, so placed it will often outlast some of its neighbours if these are of innate structural inferiority. We know, moreover, that should such a dead pivot tooth decay it will decay precisely in those situations where it would be most liable to attack were it a living organ—namely, in places favourable to the lodgment of decomposing particles, and on surfaces where a solution of continuity allows access to exposed dentine; and we know that if the surfaces filed in preparing the crown for pivoting be finely polished, so as not to allow the ready adherence of débris, these surfaces will, like those of a living tooth under similar circumstances, be much less liable to decay. We know, furthermore, that when such a dead tooth does decay in the mouth it is affected by true caries identical in every respect with the disease in living teeth. And again, we know that dentine derives what low vitality it possesses from the pulp, yet caries is neither accelerated nor retarded in its progress nor altered in character by death

of the pulp. We know, yet again, that teeth affected by chronic wasting of the alveoli are not especially liable to caries, and, on the contrary, a singular absence of this disease may be noticed in the worst cases of premature shedding of the teeth. In such cases loose teeth very often remain for long periods almost completely denuded of periosteum, supported by the surrounding gum, between which and the root a probe may be passed around almost to the apex. Surely this is a condition of impaired 'vitality' in the most vital portion of the hard tissues—the cement—and in the whole tooth, and yet caries, I repeat, is not a common accompaniment of this malady. Am I not justified in saying that it is astounding how, with all these facts staring him in the face during his daily work, a dental practitioner and a teacher of students can go out of his way to invent the wild incoherent speculations which I have cited. It is indeed difficult to criticise with patience a writer like this who, not recognising his utter incompetence —literary as well as scientific—for his self-imposed task, painfully sets to work at an attempt to instruct students in what, after all, is among the most simple of pathological problems, and succeeds only in involving it in a fog of verbiage, in which nothing can be perceived but the shadow of a metaphysical bogie, good for nothing but to scare a simple reader.

The extracts quoted above are, I repeat, a specimen of the best that has been said on behalf of the theory of predisposition to caries through prior intrinsic alteration in the tissues, and the next sample which I shall examine is also from an author whose personal qualifications and professional position add all the weight to his utterances that can be added by these attributes. This author has, at any rate, the courage to deal in something more solid than vague

generalisations based upon fancy, the unsubstantial coinage of the brain, and in concrete terms boldly states it as his opinion that the main predisposing cause of caries is retrograde metamorphosis of the tissues. Here are his words :

> 'The teeth require as much constant nutrition as the muscles, the bones, or any other organs or tissues of the system. Teeth decay, primarily, because the nutrition of their organic structures being withdrawn, retrograde metamorphosis ensues ; and secondarily, because the agencies born of diverted, improper, or inadequate nutrition are capable of producing their chemical solution. Caries is simply solution, or disorganisation, of tooth constituents by agents which are always external, but which would be quite inert under other constitutional conditions. The vitality of well-organised tooth structure in a healthy body is quite sufficient to counteract the effects of any active external agents which might be temporarily present ; but when nutrition is insufficient or diverted, the resisting power of its vitality inadequate, and destructive agents present, the teeth will yield at their weakest points, and caries will result.'

It is, I say, a satisfaction to have to deal with a statement in which there is at least one solid phrase to take hold of, but it must be observed that the statement, as a whole, does not rise above the usual level of the average recorder of impressions, and like most of such writings will not stand the literary test. The whole paragraph is vague, and the second clause of the second and last sentences—the most important sentences of the paragraph—are nonsense. However, the gist of it all is unmistakable, and we are told with sufficient clearness that the prime factor in the causation of caries is retrograde metamorphosis of the teeth. Retrograde metamorphosis is a legitimate term of unequivocal meaning in pathological phraseology, and signifies a morbid process by which a tissue gradually becomes, by chemico-physiological action, converted into another of inferior organisation, or less adapted for the performance of the original function ot the part. The term is synonymous with degeneration. The most common varieties of degeneration are the fatty,

the calcareous, the pigmentary, and the fibroid, in which, either by actual metamorphosis of the tissue elements or by infiltration, the proper elements of the tissue are more or less displaced by the new material. True degeneration is usually associated closely with imperfect nutrition and atrophy.

Degeneration occurs in non-vascular structures, and we are all familiar with the external appearance of the arcus senilis, a true fatty degeneration of the cornea. In bone and cartilage—structures which most resemble the dental tissues—pathological changes take place closely associated with inflammation, and leading to alteration in the tissues commonly spoken of as degeneration, but which, perhaps, ought not strictly to be classed under that heading. An example in bone is afforded by the disease called mollities. In this affection, the pathology of which is demonstrable, the bone is gradually softened, the cancelli slowly enlarge as the earthy constituents are carried away through the vascular system, until at last the whole of the calcareous matter being absorbed, nothing remains except the periosteum enclosing a mass of fatty matter. The whole process is brought about through the medium of the vascular system, without the agency of which such changes are demonstrably impossible. Cartilage is destitute of nerves and devoid of bloodvessels, but undergoes constant nutrition by imbibition from neighbouring vessels through its vascular covering, the perichondrium. Cartilage is a cellular structure consisting of nucleated cells supported by a matrix, and it is by means of these cells that nutritive material is distributed through the tissue. The essential feature in all diseases of cartilage of a degenerative kind is increased cell development with disintegration of the matrix. The tissue changes can be clearly followed and demonstrated. The cells become

enlarged and filled with nucleated corpuscles, and then bursting, set free their contents within the altered matrix, which at the same time has been undergoing a process of softening and disintegration. At later stages of the disease the presence of abundance of fat globules gives to the condition the character of fatty degeneration; in other instances, fibrous tissue taking the place of the true cartilage elements destroyed, a species of fibroid degeneration is produced.

Examination of the process of retrograde metamorphosis or degeneration, in vascular and non-vascular tissues, fully demonstrates the fact—to decide which, however, anatomical considerations alone might suffice—that such a process is impossible, except in tissues freely supplied with blood, or made up of cellular elements undergoing nutrition by imbibition, and capable of interstitial change of the kind I have described as occurring in cartilage. The author of the impression under examination does not inform us into what form of tissue he considers enamel and dentine may degenerate—whether we are to look for fat, or fibre, or pigment, or any other constituent of structures known to the human economy in the place of the calcareous molecules of enamel and the dentinal matrix; he frames no theory as to how the changes are wrought, and does not tell us whether we are to believe that the inert earthy basis of enamel and dentine, devoid of cellular elements and of vessels, undergoes intrinsic change and actual chemical metamorphosis into new tissue; or whether we are to believe that molecules of fat, fibrous matter, or pigment, as the case may be, are transmitted through the vessels of the pulp through the dentinal tubes and fibrils to their destination as far as the exterior of the enamel; and he does not tell us through what agency, in this case, the original tissue elements are

carried away to make room for the inferior formation. And lastly, the recorder of this feeble impression does not do what he ought to have done at first, namely, produce a specimen of the dental tissues the seat of retrograde metamorphosis. A single specimen of dental tissue having been found in a condition possibly to be mistaken for retrograde metamorphosis, there would be a legitimate ground for some statement of the case and some record of the apparent fact. But the impressionist does not wait for proof, and cares nothing for physiological considerations; he is content to register his unsupported opinion that retrograde metamorphosis is a cause or the cause of dental decay. If this were so, nothing would be easier than to produce specimens of the tissues showing marked change. For, I repeat, such changes are not subtle and ill-defined, but gross and unmistakable; and out of the thousands of teeth extracted from year to year, hundreds would be found displaying the signs of degeneration. In the absence of any reasonable theory upon which the possibility of retrograde metamorphosis of the teeth can be made clear, and in the absence of a specimen of the dental tissues in a condition of degeneration, I can only characterise the term as ludicrously absurd, and I may repeat now what I said at the Odontological Society on this subject, that if it is possible to produce a specimen of the dental tissues in a condition even capable of being mistaken for retrograde metamorphosis, I shall humbly apologise for stigmatising the term in this connection as ludicrously absurd; whilst if it is possible to produce a single specimen showing unquestionable signs of retrograde metamorphosis, I shall not only apologise, but will humbly resolve and promise never again to obtrude a criticism on a subject of dental science, for I shall admit that the subject contains mysteries too deep to be fathomed

by my poor intellect. But till this is done, till proof is furnished of its existence, I shall continue to speak of retrograde metamorphosis of the dental tissues, under whatever name it be described, as mere sham scientific jargon totally devoid of meaning and incapable of rational interpretation.

Whilst revising these sheets for the press there comes to me a paper by Dr. Miller, in which there is a passage giving support to the theory just discussed. This passage is not so clearly expressed as it might be, and it appears that Dr. Miller, who usually writes lucidly, cannot, any more than other authors, help becoming obscure in proportion as he leaves the region of fact and approaches the realm of fancy. Dr. Miller writes :

'A most powerful influence (in the causation of caries), which we do not well understand, is exerted by the nutritive processes in the teeth themselves. I am assured by men who have grown old in the practice of dentistry, that mouths which have long been under their observation, and which practically have been completely free from caries for years, at once, on account of some sudden change of health, show a general breaking down or crumbling of the teeth, *en masse*, in the space of a few weeks. It has also been my experience that patients who have been dismissed by their dentists in America, with the assurance that according to previous experience their dentures would require no treatment for one or two years, have come to me a few weeks later with teeth looking as though they had not been under the hands of a dentist for years.

'At any rate, we have here a cause which lies without the domain of both bacteria and acids (either ferment or otherwise). The lime-salts of the teeth are supposed to form, with the organic matter of the tooth, a definite chemical compound, and it is probably due to this fact that simple salts of lime are so much more readily soluble in weak acids than pulverised tooth-bone, or that the tartar upon the teeth is so much more easily soluble than the teeth themselves; so that when anyone rinses his mouth with vinegar, and afterwards find lime in the vinegar, we know that the lime, in by far the greater part, if, indeed, we may not say altogether, came from the tartar. Now, though there is no positive evidence for the supposition, it is certainly not altogether improbable that, as a consequence of certain derangements in the nutritive functions of the teeth resulting from a change of health, etc., etc., a dissolution of the affinity between the lime-salts and the organic

matter may take place, thus setting free the easily soluble lime-salts, which are then carried away in solution or washed out mechanically.

'This is a supposition only, which I bring forward because facts in this case are absolutely wanting. If it should, perchance, contain a trace of truth, then adult and pulpless teeth should be less subject to these *sudden* attacks of caries than young teeth with living pulps.'

It is not necessary to examine the questionable chemistry of this statement, or to analyse minutely all its clauses. The essence of the whole is a repetition of the unsupported 'supposition'—to use Dr. Miller's word—that in certain cases the dental tissues, owing to malnutrition, pass into a morbid condition, leading to their rapid disintegration by external agencies. Neither is it necessary for me to recapitulate the arguments I have in previous pages used to combat the cardinal error here involved, and to show again that enamel is incapable of nutritive changes. Without physiological mechanism, physiological and pathological action are alike impossible. Before we can accept such an hypothesis as that before us, we must frame some conception of the means by which the supposed morbid changes can be brought about; and before we can believe as a fact in the occurrence of the morbid changes we must see specimens of the tissues in the altered state. Will Dr. Miller explain how nutritive processes in enamel and dentine can be carried on, and has he found a specimen of the dental tissues undergoing the degenerative changes to which he alludes? The cases of acute caries he mentions, moreover, are fully accounted for by the action of the predisposing causes which I have fully discussed; and when rapid onset and progress of caries occur, we may safely ascribe the phenomena, firstly, to presence of inherent structural defects in the tissues; and secondly, to solution of weak enamel by acid derived from decomposing food *débris*, and vitiated secretions. The element of time must not be forgotten in these cases. Is it

not easily conceivable that, in the course of months or years, patches of ill-made porous enamel, such as described, become further weakened by gradual solution and allow readier access of acid-forming products, even into the substance of the tissue? Given such extensive areas of weakened tissue, allowing infiltration of fluid and undergoing slow solution, and we can understand that a change in health involving vitiation of the secretions of the mouth and formation of acid should often be accompanied by rapid breaking down of the previously weakened enamel. Enamel inherently defective often covers dentine of equally bad formation; and the enamel of ill-made teeth once penetrated by caries, there need be no astonishment at the rapid destruction of the soft imperfectly calcified dentine which often follows. Dr. Miller's utterances on this subject may be appropriately criticised in the following words of his own, which occur a few pages earlier in the very paper from which my quotation is taken:

'Melassez and Vignal very justly say of Baumgarten, who claims priority over Koch in the discovery of the tubercle bacillus: '*Il ne suffit pas de* trouver, *il faut* prouver'—and I do not hesitate to say with reference to some of the discussions which for years have been carried on concerning the cause of dental caries: *Il ne suffit pas de deviner, il faut trouver* et *prouver*. It is not enough to guess the cause, or guess at it; we must *find* the cause, and, having found it, *prove* that it is the cause sought for.'

It is a little curious that Dr. Miller should so soon forget his own appropriate vigorous denunciation of unscientific generalisation, and for once be led to the commission of the very fault he so justly condemns.

Of the supporters of the theory that caries, although caused by external agents, is accompanied by vital reaction —inflammatory phenomena—in the dentine, Neumann is probably the most worthy of attention. He is quoted by Wedl, which is a guarantee that he is a representative writer

of those viewing caries from his special stand-point. His observations were published in a German periodical now many years ago, and I, knowing him only from Wedl's work, cannot quote him at first hand. So far as I am aware, no author the least worthy of attention has in late years supported Neumann's opinions. He has stood alone, and has received no corroboration from later authorities, whose new facts, on the contrary, all go to negative his hypotheses. Neumann has not reiterated his opinions in late years; and probably may have abandoned them as incompatible with the facts demonstrated by more recent observers. From Wedl's work I make this summary of Neumann's observations. By means of the imbibition of carmine he demonstrated—as he thought—in the thickened dentinal fibres of carious dentine uncoloured and brightly coloured segments alternating with each other with considerable uniformity. He suggested that the coloured portions were probably nucleiform bodies. He believed in consolidation of the fibrils as a phenomenon of the disease due to vital reaction. He considered there was a marked correspondence between the changes in dental caries and the phenomena of caries of bone, the difference being due merely to the anatomical peculiarities of the dentine. He believed he found in one instance calcification of dentinal fibres, and ascribed this to deposit of calcareous salts at the expiration of an inflammatory process—a phenomenon which is also to be observed in bone.

There exists, as we have seen, very much more than enough evidence to refute these views, and having really adduced already sufficient, it seems almost a work of supererogation to search for further proof in an examination of the minute phenomena of inflammation; but I must carry out the full intention with which I commenced.

Before discussing inflammation it will be well to decide what we mean broadly by the term.

We may take the definition of Dr. Burdon Sanderson, one of the latest and greatest authorities. Dr. Sanderson's definition is this : 'Inflammation is the aggregate of those results which manifest themselves in an injured part as the immediate consequences of the injurious action to which it has been exposed.' Dentine being perfectly passive under every form of injury—unless it be true that it inflames during caries—must, I repeat, be considered incapable of inflammation ; a term which we may now note includes morbid action of every kind due to injury. We know that dentine violently broken or lacerated does not inflame ; we know a broken exposed surface of dentine on application of an irritant does not inflame ; and we know that we may drill a hole into healthy dentine or expose a surface of healthy dentine after excavating a carious cavity, and forcibly wedge on to that bare surface a foreign metallic mass, a filling, and leave it there, and the dentine will not manifest inflammatory action of any kind. Yet by those who maintain that dentine inflames during caries, we are asked to believe that in this disease the irritation of weak acid, barely powerful enough to slowly dissolve out the earthy constituents of the tissue, is able to induce a morbid process which the more severe injuries I have instanced are quite powerless to produce. Similar remarks apply with greater force to enamel.

The phenomena of inflammation are essentially the same *mutatis mutandis* in whatever tissue they appear, and there is no suggestion that the phenomena would differ in the case of the teeth except as modified by anatomical peculiarities of the part. To judge what phenomena to expect we must examine the pathology of inflammation in

tissues most nearly homologous. In bone, inflammation leading on to caries is manifested by increased vascularity; then follow enlargement of the Haversian canals and disappearance of the canaliculi, whilst the cancelli are enlarged and filled with inflammatory exudation, and the bone is thus softened. When the inflammation is on the surface the periosteum is the seat of the greatest vascular activity; when deeper, the penetrating vessels and the endosteum. The inflammation may terminate in abscess and necrosis as well as in caries. Caries is the stage which follows the softening stage of inflammation. Suppuration takes place, the exudations break down into pus and dissolve the connection between the solid particles of bone and their fibrous stroma, and the bony particles are carried off from the surface mingled with the purulent discharges.

Ulceration of cartilage commences by inflammation of the external vascular connections of the tissue—as in synovial membrane—or spreads from within, from the surface of subjacent bone to the exterior. Vascular loops and granulations spread over the surface and gradually erode the tissue; and the process of disintegration is assisted by nterstitial changes in the tissue elements—the cells—similar to those which occur in degeneration, and which I have already described.

In the cornea the pathology of inflammation and ulceration is not very dissimilar to that of cartilage. Intrinsic tissue changes and the presence of inflammatory corpuscles —leucocytes—in the corneal structure have recently been demonstrated as the very outset of inflammation, and it is yet a very moot point whether these corpuscles are always migratory, finding their way from the vessels surrounding the cornea, or whether they may not sometimes be produced by germination from corneal corpuscles—cellular elements

of the corneal tissue. Inflammation of the cornea of every form is always speedily accompanied by visible vascular disturbance. Vessels around the margin gradually increase in size and invade more and more of the corneal tissue, passing beneath the epithelium, until perhaps the whole surface is visibly injected. This may terminate in formation of pus—abscess—in sloughing or in ulceration. The only points which we need note are that in all these structures—bone, cartilage, and cornea—inflammation is characterized by two indispensable features, namely, exudation (or at least the presence of inflammatory corpuscles) and vascular activity, without which any process of true caries or ulceration is impossible. Here we must be reminded that enamel and dentine are anatomically quite unique, and are not closely comparable to any other tissues. In their physical and chemical characters they most closely resemble dense bone; but even the densest bone has a free vascular supply, while these dental tissues have none. Between them and other non-vascular structures, such as cartilage and the cornea, there is the vast difference that the latter are largely composed of cells, are constantly undergoing nutritive changes, are capable of carrying on morbid processes and of undergoing intrinsic degenerative changes.

Dentine is devoid of vessels; the nearest, those of the pulp, are separated from actual contact by the odontoblast layer. Dental caries does not commence in proximity to the vessels, but at the surface—the point furthest removed from vascular influence, and no vascular activity of any kind accompanies the disease. There are no tissue elements, if we except the fibrils, which are capable of interstitial change like the cells of cartilage and the cornea. We know that caries goes on unchanged when the pulp, and therefore the fibrils, are dead. If inflammation corpuscles are present

in carious dentine they must arrive there by traversing intervening dentine by way of the tubes from the vessels of the pulp, a supposition which is manifestly absurd. Unnecessary as they are to add to the overwhelming mass of evidence adducible to prove that dentine is absolutely passive under the disintegrating process of caries, all these further considerations at least serve to show the shallow superficiality of the writings, the impressions, which I have felt called upon to criticise, and to justify me in characterising them in the strong terms I have occasionally employed. The errors of the few observers who, like Neumann, have *bonâ fide* adopted the theory I have just discussed, have arisen from their ignoring the wide paramount considerations which render its adoption impossible, and from mistaking the real significance of apparent changes in the tissues. The masses of organisms, for instance, in the dilated tubes of carious dentine readily taking up colour might no doubt be easily mistaken for inflammation corpuscles, particularly by observers writing at a period before the existence of micro-organisms was suspected in such a situation, and before their real nature had been ascertained.

Lest there should arise the least confusion, it will be well, before finally leaving the subject of inflammation, to explain the exact relation which the inflammatory episodes accompanying the later stages of caries really hold to the disease. These episodes are inflammation of the pulp, periosteum, and cement.

With rare exceptions, caries, unless checked by art, having once attacked a tooth, progresses with greater or less rapidity until the tooth is destroyed. In its progress there comes a time when the pulp chamber is laid open, and the pulp exposed to the atmosphere and other sources of irritation. It is not necessary for my present purpose to enter

into a lengthy examination of the pathology of the pulp. The pulp has not, like enamel and dentine, a unique pathology—one totally differing in its phenomena from that of every other tissue; and the pathological changes which take place in an exposed pulp are due to inflammation, and are in their nature essentially the same as similar inflammatory processes in other vascular tissues. It matters not whether an iris, a synovial membrane, a lung lobule, or a dental pulp be the seat of the morbid action, these processes are the same, modified only by the anatomical peculiarities of the part. It is probable that inflammation always speedily supervenes upon opening of the pulp cavity by decay, and that it often precedes the actual exposure of the pulp. We know that a joint or other analogous cavity, wounded, will speedily become inflamed, if not kept aseptic by art. The condition of an exposed pulp might be, perhaps, broadly compared to that of the tissues exposed in such a wound or in a compound fracture. Septic matter may slowly percolate to the pulp through an intervening layer of dentine; and we know that organisms may proliferate along the dentinal fibrils through dentine which to the naked eye appears healthy. A single drop of septic matter, a single bacterium inoculating the pulp, would certainly originate inflammation, and these facts explain the occurrence of inflammation even before the decay has quite reached the surface of the pulp. Dentine of inferior structure, containing much organic matter, and numerous imperfectly calcified spaces, would, of course, allow more easy passage for septic material. In ill-made teeth the pulp cavity often extends in the crown—sometimes by a narrow process only—to a point abnormally near the surface. In such teeth also numerous interglobular spaces are commonly found in the dentine, and thus tissue readily permeable by fluids

extends almost from immediately beneath the enamel to the pulp cavity. This accounts for the readiness with which inflammation often supervenes in the pulps of badly made six-year-old molars at an early stage of decay, or after excavation of the cavity, and insertion of a filling in childhood. There is, however, an 'impression' on record that in these instances inflammation really starts in the dentine. Exposure to changes of temperature through partial removal of the tissues, its normal covering, no doubt suffices in some other cases to excite inflammation of the pulp. The vascular connections of the pulp and dental periosteum are so intimate that inflammation, as one might expect, always extends in the later stages from pulp to the root-membrane and cement. Cement is identical structurally with osseous tissue, and its periosteum with the ordinary covering of bone, and the phenomena of inflammation which these tissues display are essentially the same as those manifested in other osseous structures. The only point in all this really pertinent to my subject is, that inflammation of pulp and periosteum and cement is not only in no way active either in the causation or progress of caries, but is itself the result, the sequel, of the disease.

Before proceeding to notice the last author upon whose work I feel called upon to briefly comment, I must here interpolate a remark on the subject of the spontaneous arrest of caries, a subject alluded to a little further back, when it was stated that in rare cases the disease might come to an end without treatment. Such cases are of the following description :—The decay occasionally commences on the grinding surface of a tooth the external portion of which alone is of inherently defective structure. The occurrence is most common in honey-combed teeth. The decay spreads over the whole of this surface, which gradually

breaks down until the denser, better formed dentine beneath the defective enamel is laid bare. The surface so exposed is often but little sensitive, and being more or less used in mastication, constantly swept by the tongue and washed by saliva, becomes in time worn smooth and highly polished, and frequently endures for many years in that condition without any renewal of the disease. This simple explanation is quite enough to account for these cases, the probable nature of which is confirmed by the great success of the operation of filing the teeth, which is often practised for the arrest of incipient decay, in imitation of the natural process. In incipient caries of the lateral aspects of the front teeth the treatment may be in many instances confined to cutting away the diseased tissues, polishing them, and leaving them of such a form that they may not allow the adhesion of foreign particles, may be readily cleansed, and constantly subject to friction during mastication. As usual in instances where the nature of a phenomenon may not at first sight be explicable, a writer was ready to come forward with his 'impression,' and record a far-fetched groundless explanation that the spontaneous arrest of decay was due to vital reaction in the dentine, to consolidation of the tissue through renewed calcification. The explanation which I have given is sufficient; but if it were not enough, that would be no more justification for the acceptance of the other hypothesis, the impossibility of which I have in previous pages fully demonstrated.

The last author that I shall notice is really quite beneath serious criticism. I notice him only for the purpose of putting in a protest against the acceptance of such extravagances by certain authors and teachers, whose most necessary accomplishment ought to be power to distinguish in science the sham from the false, and whose first duty is

to apply the severest tests to all new utterances in science before accepting them. A teacher who endorses worthless impressions and sets the seal of his approval to the useless work of a pseudo-scientific writer, is certainly guilty of a fault much greater than that of the original offender—he effectually bewilders the student and compels the critic to wade through and examine, for the sake of the student, a mass of matter totally undeserving on any other grounds the least notice.

This writer seems to base his ludicrous speculations on the fact—to which I have on a previous page referred—that Dr. Bödeker has succeeded in staining enamel with chloride of gold. Upon this fact new vague theories are built up. We are apparently asked to believe that enamel is permeated by a 'network of protoplasm;' that it undergoes constant nutrition, and is capable of displaying many of the phenomena of inflammation during the progress of caries. Enough evidence has been already adduced to show the baselessness of these speculations; and indeed the anatomical facts alone suffice. On this subject Mr. T. Charters-White, M.R.C.S., L.D.S., one of the most accomplished dental histologists of the day, writes to me as follows:

'My opinion, derived from careful observation, is that the amount of active protoplasm between the enamel fibres in an adult tooth is nil. The amount of organic material in adult enamel being only three per cent., it follows that whatever protoplasm be there is so infinitesimally small that it may be practically disregarded as an agent. I have stained very thin sections of recently extracted teeth, and stained them with chloride of gold, but while the protoplasmical contents of the dentinal tubuli were coloured, not

a trace of stain could I detect between the enamel fibres. I used the one-eighth of an inch objective, too. I send herewith a photo-micrograph of enamel, magnified about fifty-six diameters, and you will readily see that if protoplasm existed between these fibres it must have so little potentiality that it could do very little in pathological action—except theoretically.'

In fairness, lest it might be supposed that a mere isolated accidental failure had been picked out for criticism, I give the following ample quotation from the author under discussion :

'In dissolving the lime-salts on the surface of the enamel (or in any depression or irregular surface of a tooth, where food lodges until decomposition occurs), *the living matter in that tissue* becomes exposed, and at once, under the effect of the acid, becomes more or less irritated. This irritation extends into the substance of the enamel beyond the point at which absolute destruction of the tissue has taken place (which fact may readily be determined by diligent examination of specimens of carious enamel carefully prepared). Under this irritation, constantly applied, *inflammation soon follows;* this causes more or less of a *swelling of the living matter*, which affects the dislodgment of the lime-salts—*a melting down of the glue-giving basis substance*, and a bringing to view, under a power of from 1,000 to 1,500 diameter, *the medullary or embryonal elements of the enamel. As the caries reaches the dentine, the same inflammatory reaction with the swelling of the living matter, the enlargement of the canaliculi, dislodgment of the lime-salts, and melting down of the glue-giving basis substance*, becomes more intense, just in proportion as there is more organic and living matter in the dentine than in the enamel. These lime-salts are not necessarily dissolved and taken away, but may, and I have no doubt, as Prof. Mayr's experiments show, do remain mixed with this disorganised tooth-substance.

'Upon examination of a specimen of acute caries, cross-section, at a considerable distance from the disintegrated granular mass (always to be found upon the surface of caries), may be seen enlarged canaliculi, some double, some treble, some four, six, eight—yes, even fifty times as large as normal. In many instances, near the surface, as many as six or eight may be seen to have joined together, the lime-salts between and around the canaliculi having been dislodged, *the glue-giving basis substance* melted down, and forming *partly nodulated protoplasmic*

bodies in which the living matter is brought to view, in the shape of nuclei, with occasional threads running from one protoplasmic body to another. It is this living matter, so brought to view, which in my judgment has been mistaken by some observers for organisms. Many such enlarged canaliculi remain in a tooth after the supposed carious portion has been removed and the tooth filled. In such cases the acid irritant formerly in the cavity is removed, the inflammatory condition subsides, and the lime-salts become re-deposited. In other words, these deep-seated lesions heal, and become as solid and to all appearances as healthy tooth-bone as ever. Could this occur if these enlarged canaliculi were filled, as some observers claim, with organisms of decomposition?'

The following grotesque questions terminate the paper from which this excerpt is taken:

'1st.—Why is it that the teeth of all persons do not decay the same?

'2nd.—Why is it that in ninety-nine cases out of a hundred the lower front teeth (on which may be found the greatest number of organisms of any in the mouth) do not decay, while all others in the mouth do?

'3rd.—Why is it that teeth with the greatest amount of lime-salts, consequently the smallest amount of organic substance (that upon which, it is claimed, the organisms subsist) do not decay sooner and more rapidly than the reverse?

'4th.—Why is it that a pulp canal which has held a dead and putrifying pulp for many years, upon being opened is found to be as solid and free from decay as it was before the pulp died?'

If I have made myself at all clear in my previous remarks, it would now be an insult to the understanding of the youngest student of the least capacity to seriously criticise and point out the errors of this writing. It will stand not one test of criticism, whether as literature or science, and all the statements or phrases which I have italicised, including inflammation of enamel—a phrase glibly used—are so much nonsense. Several terms employed—such as 'swelling of the living matter,' 'melting down of the glue-giving basis substance'—are quite unknown to science, and, in the absence of explanation of their meaning, look like a ludicrous burlesque of technical phraseology. In truth, the whole thing at first sight seems an elaborate joke, and one

would feel inclined to treat it in a corresponding fashion, were it not, as I say, that such writings have been accepted in sober earnest by those whose professional position commands attention. The author I have just quoted has been actually cited as an authority in a work addressed to students.* One must protest against this kind of thing, although one can understand that in this instance a fellow-feeling may have made the writer wondrous kind, since he himself prints passage after passage (some of which I have quoted), the meaning of which can be barely guessed by an expert, so persistently and consistently ignored are all the indispensable rules of language, logic, and grammar. It is necessary, however, that a protest in unequivocal terms should be put forth against the serious discussion of such solemn nonsense; and more particularly desirable that a forcible protest should be registered on behalf of the Odontological Society, where some of these caricatures of science have been with serious intent from time to time paraded. The Odontological Society has a reputation to maintain, a reputation based on the *bonâ fide* scientific labours of its members, of whom Mr. Arthur Underwood (whose work gave origin to these papers) is, although the latest, not the least distinguished. The reputation of the Society has been hitherto kept up both by the value of the scientific achievements of members and the corresponding tone of the discussions. Nor have wild writing and utterances, such as I have stigmatised, been at all common at any time at the Society. There has been a much larger production of this kind of literary dross in America.

* This author is also quoted in a new edition of another better-known manual of dental surgery, issued since the first publication of these papers. That writings so absurd, such a travesty of science, can be seriously cited in a first-rate work is astonishing.

Let me not be misunderstood here. No one appreciates more fully than I the sterling qualities of the dental profession in America; and no one more fully recognises the great obligation we are always under to them for real advance in the science and art of dentistry—I speak only of a small minority. This small minority out of the healthy mass has, however, been affected sporadically by the *cacoëthes scribendi et loquendi;* and, if only by their noisy ravings, have compelled attention. The performances of these individuals remind one of a famous author's description of a character who, having swallowed several crude systems of philosophy, and having so induced a condition of moral dyspepsia, mistook the supervening flatulence for a veritable divine inflatus. Papers have been composed and read which, except for the show of technical polysyllables and of incoherent phrases indiscriminately snatched from the store of current physiological jargon, have contained nothing remotely resembling science. These papers have been seriously received at dental societies and discussed in a corresponding spirit. Floods of incomprehensible rigmarole have thus been poured forth, which could only be compared to the ravings of a party of physiological bedlamites—a tale told by an idiot, full of sound and fury signifying nothing. It may be imagined that this is a coloured and over-drawn description; but I can appeal to the columns of this journal, where many specimens have appeared—only, however, to receive the criticism they deserved—and I can appeal to many of those whose editorial duty it has been to examine the writings in question for proof that my language, far from being exaggerated, is well within the mark. And I say it is time a protest were made against reception of such worthless writings, or of the utterances of scientific pretenders at the Odontological Society; for if this protest

be not forcibly made, and if solemn discussions of nonsense be allowed to pass without comment, there will be a danger that the silence may be construed to imply the members' inability to discriminate; the Society may be thus brought into ridicule and contempt, and injury may be inflicted upon those good causes for the promotion of which the Society exists—causes which all have earnestly at heart, whether it be or be not in our power to contribute by our labours to their advancement.

Taking into consideration all the demonstrated facts—anatomical, physiological, and pathological—which bear upon dental caries, it may be fully claimed that the definition of this affection, with which I set out, is incontrovertibly established. The etiology is almost fully made out; the exciting causes are equally plain, and but little remains to be cleared up as to the exact sequence and significance of events in the tissue elements during disintegration. We know very little about the origination of one of the main predisposing causes, inherent structural inferiority of the tissues; but when this and some few other points—as, for instance, the exact part which micro-organisms play in the disease—are cleared up, our knowledge of caries will be complete. None of the questions remaining for solution are of supreme importance in helping to an understanding of the essential character of caries. These questions will, however, doubtless in time be solved; and some of them, especially those throwing light on the etiology, will surely help in the future to promote advances in dental prophylaxis, such as are now being so rapidly effected in other branches of preventive medicine.

THE PREVENTION OF DENTAL CARIES.*

Etiology being based mainly upon anatomy, physiology, and pathology, there can be no true science of preventive medicine without complete knowledge of disease. Preventive medicine is only now slowly being placed on a solid foundation of scientific fact. But even without a truly scientific system of preventive medicine, the art of preventing disease is capable empirically of vast development, and in this manner has already achieved great results. Let me illustrate my meaning. Through our knowledge of the processes of fermentation and putrefaction, and of the action of micro-organisms, we are beginning only now slowly to demonstrate fully the etiology of zymotic diseases, the proper means of preventing which have long been fully proved and recognised. Of the actual *materies morbi* of typhus, typhoid, diphtheria, small-pox, cholera, and such diseases we know very little; yet we know we possess in the art of sanitation the most powerful means of preventing them. Sanitary art has so far advanced that it may be truly said the diseases named ought no longer to exist in civilized states; and that when the nations shall have the resolution and the power to put their sanitary institutions on a level with

* Read at the Annual Meeting of the British Dental Association at Edinburgh, August, 1884.

science, these scourges of humanity will be as obsolete as the plague. Similar remarks apply to syphilis.

When we turn to some other classes of disease, we find that for them no prophylaxis exists because of our almost complete ignorance of their etiology. This remark applies to many surgical diseases, and especially to every class of new growths—every species of innocent and malignant tumour. Common and fatal as the latter are, it is quite beyond our power at present to prevent them. It is known that in individuals predisposed by heredity or other circumstances, an injury, such as a blow on the breast, or the irritation of pipe-smoking on the lip, may apparently determine an outbreak of malignant growth; but when this is said, the extent of our knowledge is reached.

Considerations such as these remarks suggest may be applied to dental caries. The true essential character of dental caries has been only within late years fully demonstrated; we have had to wait for full light to be shed upon the anatomical, physiological and pathological questions involved; and this light has only recently become full enough to illuminate all that was previously dark in these subjects. Nevertheless, the importance of personal hygiene, local and general, has been long recognised as preventive of dental decay. Now, however, that the nature of this affection is fully made out, it seems to me that the time has arrived when we may profitably discuss the subject of dental preventive medicine from the standpoint of our exact knowledge. The object of my paper is to start such a discussion.

Summing up the knowledge which we possess as to the etiology and pathology of caries in the papers which I have recently contributed to the Journal of this Association, I found that there exists much more than enough of fact—

anatomical, physiological, pathological, and experimental—to establish incontrovertibly the following truths with regard to caries. I must be excused if I state these matters shortly and dogmatically. I have given their proof in the papers mentioned, and the time now at my disposal will not allow me to enter into them.

Caries is a process of disintegration, commencing invariably at the surface, proceeding inwards, and due entirely to external agents; enamel and dentine are perfectly passive under this process, and manifest neither pathological action nor vital reaction of any kind.

The predisposing causes of caries are (1), innate structural defects in the teeth which render them more susceptible to the action of agents; (2), all such diseases as are accompanied by vitiation of the oral secretions, or which tend to the formation or deposit of acid, and the accumulation of products of decomposition within the mouth, and (3), crowding and irregularity of the teeth due to smallness and malformation of the maxillæ. The direct agents in initiating caries are acids—principally malic, butyric and acetic—the products of chemical change and fermentation set up in fragments of organic matter (food, mucus, and epithelial scales) which are commonly present in the mouth and lodged about the teeth. These acids are often assisted in their action by acid mucus secreted by unhealthy gums, acid (instead of alkaline) saliva in some diseases, and acid eructated from the stomach in others.

For any useful discussion of my present subject it is necessary to state at the outset the fundamental facts upon which reliance is placed; and although it is not essential now to enter fully into the subject of the development and structure of all the dental tissues, it is desirable that I mention some facts especially with regard to enamel.

Enamel is the starting place of caries, and with this tissue we are mainly concerned in studying the etiology of the disease. Enamel is a densely hard, almost homogeneous calcareous mass, containing not more than from one to five per cent. of organic matter. Examined microscopically, enamel is seen to consist of solid prisms lying side by side, intimately united and without any intermediate substance. Enamel is superimposed as a cap upon the subjacent dentine, to which it is closely united.

The enamel organ—that portion of a developing tooth from which enamel is formed—consists of epithelial cells, and it retains its epithelial nature throughout the process of calcification, the process commencing at the surface of the dentine, in which previously the hardening process has already well advanced, and proceeding outwards. The completed tissue results from direct calcification of the internal layer of cells. Numerous vascular papillæ arising from the contiguous tissue of the dental sac penetrate to a slight depth the external layer of cells and convey nutriment to the developing tissue. On completion of the enamel these external cells with the vascular loops undergo atrophy and disappear.

In estimating the possibility of pathological phenomena in enamel we must not lose sight of the anatomical characters of dentine, the tissue which intervenes between enamel and the nearest vascular supply, and through which any influences arising from within the tooth must find their way to affect enamel.

Dentine consists of a homogeneous calcareous matrix with a basis of fibrous tissue, about 28 per cent. of the mass. It is permeated by minute tubes, not more than $\frac{1}{1500}$ inch in diameter, which, radiating from the pulp cavity, are occupied by fibrils which endow the tissue with sensibility. The

minuteness of the fibrils prevents actual demonstration of their structure, but it is sufficiently proved that they are protoplasmic and form the sole protoplasmic element in dentine.

The bearing of these anatomical and physiological considerations upon my subject depends upon the obvious fact that enamel is totally devoid of any physiological apparatus whereby either vital or pathological changes could be brought about in it, and that whatever changes enamel undergoes, are induced by external agencies. Not only is enamel not capable of intrinsic changes, but it is not in relation with any mechanism capable of influencing it from within. To believe in the possibility of nutritive changes in enamel we must first conceive some means by which this calcareous mass, devoid of cellular elements, could assimilate nutritive material when conveyed to it; and we must next imagine the conveyance of new and effete material to and from the vessels of the pulp through the odontoblast layer of the pulp by way of the dentinal fibrils to its destination within the substance of the enamel.

The dates at which the calcification of the teeth commences must be also borne in mind. By the end of the twelfth week of intra-uterine life a cap of calcified material may be demonstrated on the pulps of all the temporary teeth. By the sixth month the first permanent molar has advanced to a similar stage of development. By the first month after birth the permanent incisors and canines are advanced to the same degree, and at the third year and twelfth year respectively, calcification has commenced in the second molars and wisdom teeth. It is usual to assume that ill-made teeth result from deficient supply of the lime salts which go to build up the tissues. We do not really know whether this is ever actually the case or whether the

defect arises always or sometimes from other causes affecting the tooth germ, whereby it is prevented from undergoing the process of conversion; but be this as it may, we can perceive that it must be useless to attempt directly, or indirectly, by medical treatment, to influence the formative process, to promote the production of enamel of good quality, after the lapse of the periods named in the different classes of teeth.

The one dark spot in our knowledge of the remote causation of caries, is found in this first pre-disposing cause which I have named—inherent structural weakness of the tissues. That the dental tissues vary infinitely in strength in different individuals, we demonstrate daily in our practice. We meet with well-made teeth—exceptionally, it must be confessed—the sound enamel of which tries the steel of our best tempered chisels; and on the other hand, we all encounter—and much more frequently—badly formed teeth, the soundest enamel of which is throughout so chalk-like in its softness, as scarcely to afford a secure foundation even for a plastic stopping. Between these extremes all varieties are to be found; and teeth of general defective character often present patches of surface, the enamel of which consists merely of an imperfectly united mass of granules. Similar defective spots are to be found here and there in teeth of better formation. Sometimes enamel fibres at parts are penetrated at their centres by minute tubes, thus making the tissue at such places porous. Defects in quantity are seen not less frequently than these defects in quality, and they are equally variable in extent. Honeycombed teeth have enamel full of small pits and fissures, and there is scarcely a set of teeth to be found in which, here and there, in one or more teeth, an isolated crack or cleft—a solution in the continuity of the tissue—does not exist.

Our knowledge, I repeat, of the origination of mal-

development of the dental tissues is very incomplete. We know that vast numbers—some say the great majority—of individuals in highly civilized states have ill-made teeth; and we know that the quality of the dental tissues varies very much in the different peoples. To instance a few nations in this regard, and to speak somewhat from my own experience, I should place the Scotch as a people in the first rank of dental development, after them I would place the North Germans; thirdly, the English; then the French; and lastly, the people of the United States. Among the last, I of course only refer to that part of the population native by sufficiently long descent; those upon whom—through their ancestors—the climate and other forces of evolution have set their mark. There is evidence, although not conclusive, that in all races of advanced civilisation there has been, in later generations, a notable further deterioration in dental development.

How far can we account for these circumstances, and trace the causes to their true origin? We have very few solid facts. One at the outset may be recorded, namely, that in accordance with the well established law of evolution, comparative disuse of the organs of mastication has led in civilised races to decrease in size and strength of the whole apparatus of mastication, jaws, muscles and teeth. This fact has been quite recently demonstrated by Professor Flower. He shows, as a result of examination and measurement of many thousands of skulls, that there is a gradual diminution in the size of the teeth, from the anthropoid apes through the lower races of man to the European. He has constructed a perfect dental index, and in this index the average size of the teeth of the gorilla being represented by 50·8, the Tasmanian by 47·5, and other savage races holding intermediate positions, the European stands at 40·5.

It has been plausibly argued that dental deterioration—and especially progressive deterioration—may be accounted for by the overwhelming demand upon the vital powers by the growth of the brain and its increased exercise in modern life. The brain and the jaws are alike fed from the common carotid artery, and, it is urged, the demand for blood by the growing and working brain leads to imperfect supply of the masticatory organs. This argument gains support from the fact that the people of the United States, with certainly the worst teeth, present a type of humanity one of whose most striking characteristics is enormous activity of brain and nervous system, with expenditure of vital energy through these channels. But I believe we shall find this hypothesis will not work, and it remains hypothesis, difficult of negation, impossible of proof.

Dental deterioration has been ascribed entirely to improper and imperfect feeding of children, to insufficiency in the food of lime salts, the proper pabulum of the developing teeth. If this were true, the whole osseous system—which is built up of the same chemical constituents as the teeth—must in every case be equally ill-constructed; it would be impossible to find a single individual with a solid skeleton, and such cases as so commonly present themselves, a massive bony system in conjunction with faulty dental tissues, would be unknown.

Perhaps with one exception, to which I shall at once refer, there is no modern people displaying general progressive physical degeneration likely to be accompanied by ill-developed teeth, and certainly the general physical development of most European nations has continually advanced from mediæval times. But it must be recollected that there has been at the same time among the populations a constant augmentation in the numbers of the physically imperfect, of

those unfitted to live under less favourable circumstances. Vast numbers of the phthisical, the scrofulous, the ricketty, and the congenitally syphilitic, who would formerly have perished in childhood, are now by science preserved alive, and these imperfect types are all associated with faulty dental development. Least of all among the nations can the people of these islands be suspected of general physical degeneration. On the one hand, no doubt, injury has been caused by the aggregation of great masses of the population in towns, the sanitation of which is imperfect, and in the employment of great numbers of men, women, and children in crowded, ill-ventilated factories. On the other hand, never before were the great bulk of the British so well sheltered, so well clad, so well provided with food and the smaller luxuries of life; never before was the public health so well cared for, and never before was there more freedom from diseases which leave their mark on the constitution of their victim and lead to degeneracy in his descendants. I express it as my opinion that the higher, the wealthier classes, display on the whole the worst dental development, but these classes surely do not show any signs of general decay. Although luxurious they are not enervated; they no longer take alcohol to excess, and their physique is maintained by their love of sport and athleticism: a dominant passion not confined to youth or sex, but affecting one and all.

In discussing questions of evolution we must be on our guard against hastily drawn generalisations, and must recollect that vast periods of time are needed to work permanent changes. Admitting that deterioration of the apparatus of mastication and of the dental tissues has advanced in the present and in recent generations, we are not, therefore, to believe that this decadence is to continue. The proper

performance of the function of mastication is necessary to perfect health, and no man can be in complete harmony with his environment, who is incapable of carrying on this necessary process. So long as mastication is an advantage to the race in making the man a better animal, it is, therefore, absurd to suppose that the forces of evolution will go in the end to produce a toothless man.

Dental caries is no more a necessary accompaniment of civilisation than are those other preventible diseases which I named at the outset. Science is giving to mankind the power more and more to mould his physical future, and we need not fear that this power will be wanting in the case of the teeth when once the problems are illuminated by the full light of exact knowledge. Looking into the far future, one can perceive that the time may come when there shall be no more dental caries.

And the nations are not interfering improperly with natural forces nor retrograding in physical development. To this, however, I believe, there is one exception. The French, having adopted almost universally the practice of restricting population by artificial means, are preventing the action of those evolutionary forces which, given fair play, ensure in the end the survival of the physically fittest as surely now in the nineteenth century as in the ages of primitive man. An effectual system of checking artificially the natural increase of population must act in several ways to the detriment of the nation's physical standard. It prevents in great measure that rapid destruction of the weaker in the battle of life which takes place where there is a redundant population ; it enables even poor parents to rear sickly offspring who would probably perish for lack of sufficient care and necessaries of life were the number of children greater ; and sickly offspring are most commonly found among the

firstborn, who are those most liable to inherit some diseases, especially syphilis.

The French are carrying on what is really a gigantic system of artificial selection, and by ensuring the survival of the least fit progeny are producing a stunted inferior race. France among European nations is the only one whose population does not increase. The arrest is due to known causes which are attracting the attention of her serious statesmen as well as that of foreign sociologists.

The physical deterioration of the French seems almost proved; but there is not evidence to show that their teeth are bad in proportion, and we must not forget a possible fallacy in this reasoning as regards the teeth. The French use their jaws less than any other nation; they are the best cooks in the world, and the whole population, without exception, lives upon the softest food, including bread of the most delicate manufacture. It is puzzling to observe that the teeth of the French are better than those of the Americans, who cannot be accused justly of adopting extensively those vices of civilisation practised by the French.

These observations and contradictory facts are intended to show, and I trust they may suffice for the purpose, that when we are asked categorically to name the cause of dental deterioration we must confess our ignorance; must state that we need more light, and must explain that probably the causation is not single and simple, but extremely complex. We do not know yet what are all the factors which can possibly go to produce the results which we note; and without this knowledge we cannot draw a sound deduction.

When we turn from the general consideration of this subject to the narrower question of the association of defective dental tissues with particular diatheses, we find ourselves on firmer ground, and we may take a solid fact to

start with—a fact, the importance of which I think has not been sufficiently dwelt upon, that hereditary syphilis, which seems to interfere with the development of all the structures derived from the epiblast, leads also in many cases, and I believe in most cases, to imperfect formation of the tooth tissues. The amount of injury to the developing teeth depends, I am convinced, upon the stage of the disease in the affected parent. It is, of course, most common for the father to be the subject of the disease. A woman pregnant by a man suffering from secondary syphilis of recent date, will usually either abort in the earlier months or bring forth a dead child about the full term. As the virulence of the disease in the parent abates under treatment or by lapse of time, the mother begins to bring forth living children; these may perish early or may survive in accordance with the extent to which their constitutions are modified by the syphilitic poison. It is a fact, I believe, that only secondary syphilis is transmissible, and thus so soon as that stage in the parent is passed, children, although often unhealthy, will not display signs of specific hereditary taint. In my experience the typical syphilitic tooth described by Mr. Hutchinson (and which must not be confounded with the merely honeycombed tooth), occurs only in children displaying the worst form of hereditary syphilis; but it by no means forms an invariable accompaniment to this condition. Indeed, I have been struck with the rareness of the malformation in children with unequivocal signs of syphilis. I reckon roughly I have not seen it in more than 1 per cent. of cases. I should be glad to know if similar experience is common.

But what I have clearly observed is that in many children of syphilitic parents, and even in cases where no specific symptoms existed, the teeth, although often good in form

and colour, have frequently been of imperfect formation, the enamel soft and easily broken down. I have noticed, also, that the children's teeth were, as a rule, markedly inferior to those of the parents; and I am strongly of opinion that children begotten of syphilitic progenitors may often display inherently defective dental tissues as the sole sign of the attenuated taint by which they have been affected. It is not for the dentist—perhaps not for the surgeon—to suggest to a father that his child is syphilitic; but the fact is often known to one or both parents, particularly when the first-born, as often occurs, are chronic invalids from the disease, and the later born children healthy. By one means or other, I have been able to verify my observations in a large number of cases; and the point upon which I wish to lay stress is, that seeing how commonly syphilis is diffused through all classes of the population, and considering that children of the third and fourth generations would inherit the dental characteristics of their parents, hereditary syphilis may be reasonably looked upon as one of the causes of imperfect development of the dental tissues.

An intimate connection between dental mal-development and any other diathesis, besides the syphilitic, cannot be fully demonstrated, yet there are some of these constitutional conditions with which badly made teeth seem more or less associated. In this matter I am recording the results of my own observation alone. My experience goes to show that imperfect dental tissues are found in the majority of cases of scrofula. Phthisis is now known by no means to indicate invariably the tuberculous diathesis. Keeping the distinction in view, I would say that ill-made teeth are not at all a constant accompaniment of any form of phthisis, nor are they especially noticeable in a majority of cases in patients of undoubted tubercular tendency. There is one

class of patients, however, the subject of phthisis of undoubted tubercular origin, which almost invariably presents faulty dental tissues. This is the type most frequently found in females, in which there is often, with a fragile form, considerable facial beauty, in which the eyes are large and expressive, the complexion fair with the blue veins visible beneath the skin. This is the not uncommon type, which, without further description, will be recognised, and in which I have invariably found the teeth, although well-shaped and often uncommonly white and beautiful to look at, covered with the softest and most defective enamel. With rickets I have generally found inherently defective teeth. I can in no way associate faulty tooth development either with the rheumatic or gouty constitution. Indeed, with the latter both teeth and jaws are often of unusually massive and solid character—a fact which was noted years ago by that acute observer, the late Dr. Laycock, of Edinburgh.

In view of our limited knowledge of the causation of dental degeneracy, what general measures can be suggested likely to lead to improvement and to aid in the prevention of dental caries? We, with other reformers, can direct attention to the need of national sanitation in the broadest sense. Syphilis, that fruitful source of moral and physical misery, is an affair mainly of police, and will—with many other diseases—surely be extinguished in this nation, when the people are sufficiently educated in science, and able to estimate at its true worth the sentimental false humanitarianism which now dominates a loud and active portion of the community. Scrofula and rickets are mainly poor children's diseases, and are the direct products of filth, darkness, and starvation. Of the etiology of tuberculosis little is known, although this disease seems in some measure

the outcome of climatic influences—of cold and damp. The observance of the well-known general laws of health, we may be sure, will tend to produce good teeth in the race, if not in the individual, and among these laws we must insert one inculcating the use of articles of diet that shall give due exercise to the muscles of mastication and prevent them, and the bones to which they are attached, from wasting.

The question of the marriage of syphilitic patients is one the dentist has not, happily, to decide, but we may remind the sanitarian that bad teeth will probably be included among the physical evils from which syphilitic offspring must suffer. The wealthier classes do not, as a rule, marry recklessly after syphilis, but it is doubtful if even enlightened people are sufficiently deterred by the presence of any other constitutional taint transmissible by heredity.

Turning from these general considerations to examination of particular instances, it may next be asked can we, by any treatment, influence beneficially the developing teeth of the fœtus in utero, through a mother the subject of either of the diatheses or cachexiæ which have been named. The answer must be that little reliance can in most cases be placed upon treatment specifically directed to this end, and we must depend mainly upon measures for the amelioration of the mother's general health, and the eradication of any definite morbid constitutional taint. It is unnecessary I should discuss the treatment of these conditions; this is quite beyond the province of the dentist, although it is the duty of the dental physiologist and pathologist to explain the laws which should govern treatment directed to the developing teeth. To show that defective teeth might occasionally result from deficient supply of the necessary pabulum through the mother, Dr. Thorowgood has brought

forward several cogent facts. He has pointed out that a hen with a broken bone lays, during the time the fracture is uniting, soft eggs; the earthy salts which should go to harden the shell being needed for the repair of the fracture. In a pregnant woman the union of a fracture is slow; the inference being that the lime salts are all required for the formation of the bones of the fœtus, and this inference is borne out by the fact that osteophytes and bony thickenings sometimes present in the early months of gestation, become absorbed as pregnancy advances and bone begins to form in the fœtus. In presence of these facts a rule may be established that in every case of cachexia during pregnancy, and particularly where there is a tendency to atrophy and bony wasting, there should be administered mineral nutrition, both through the medium of a suitable dietary and of the admirable therapeutical preparations of lime salts now to be found in the pharmacopœia. It is difficult to believe that any treatment could influence some cases in which a strong hereditary influence proceeding from the father gives a bias to the process of dental development. A mother with good dental tissues will often bear children with defective teeth, having the closest resemblance in form and structure to those of the father, who may be perfectly healthy, and in whom this may be the sole physical defect.

When the child is born, syphilis and specific diseases will receive their appropriate treatment, whilst the well-known rules of hygiene are enforced for the improvement of the general health. As I have so often insisted, we are ignorant of the actual physiological or pathological causation of defective enamel; but as uncertainty exists, we had better err on the right side—especially as the error would be harmless—and enforce the rule that in every case in which ill-made tooth tissues are likely to appear an attempt

by diet and therapeutics should be made to supply to the developing tissues the mineral constituents of which they may stand in need. This rule should, of course, especially be enforced in those diseases, such as rickets, in which the whole skeleton is ill constructed. All along must not be forgotten the dates at which the classes of teeth are calcified, and that after the formation of enamel any attempts to prevent caries by improving the quality of that tissue must be futile. The exteriors of the crowns of all the temporary teeth—from caries of which children suffer so much—are fully formed at birth, these teeth can therefore be influenced only through the mother. By this time the first permanent molars and the permanent incisors and canines are so far advanced in development that it is open to doubt whether our further treatment can have any effect upon the enamel of these teeth.

There is, however, no difficulty in administering lime to an infant from birth. A child should, as we know, have no food except its mother's milk, if this is of proper quality, up to at least the sixth month. Practically in the majority of cases this source is supplemented by cow's milk; which, of course, with well-managed children, is diluted and prepared in the usual way to resemble human milk. It happens that lime water (liquor calcis) is found to be an admirable aid to the digestion of milk in infancy, and this can either be added to the supplementary food, or can be given separately, as suggested by Dr. Thorowgood, in the form of the saccharated solution of lime of the pharmacopœia.

So far I have confined myself closely to consideration of one predisposing cause of caries—inherent defect in the structure of enamel; and this has involved the question whether organic deterioration of the dental tissues has been on the increase in recent years. Such an increase must, of

course, be accompanied by a proportionately greater prevalence of dental caries; but we must recollect that, although structural defect in the enamel is a prime factor in the causation of caries, there is another factor as potent—the second predisposing cause which I named—vitiation of the secretions of the mouth. It is certain that enamel of sufficient durability under favouring conditions will be destroyed if the agents necessary for its destruction be present in the mouth. We know as a fact that the diseases of our times, unlike those of earlier days, are largely those which give rise to the formation of such agents in the mouth, for many or most of them are either diseases of the digestive organs, or maladies accompanied by disturbance of the functions of digestion, and it may well be, therefore, that there is more tooth decay now than formerly, and at the same time by no means a relative increase in organic dental deterioration.

In this way we may account for the prevalence of caries among certain portions of the community whose physique and general health in other respects are not to a corresponding degree inferior, and whose dental tissues are not of the worst structure. For example, the constant presence of dyspeptic troubles in some classes of factory operatives is enough to account for the rapid tooth decay from which they suffer. These people earn good wages, and they are well housed and clothed; but their occupation is sedentary; their food, although abundant, ill-cooked, and often too solid and coarse; they drink too much bad beer and too much coarse spirit, and the result is chronic dyspepsia. This disease is so common in some communities that it is regarded as a matter of course, and endured as one of those evils from which there is no escape.

When speaking of the association of ill-made dental tissues

with certain diatheses, I was not unmindful that some of these diatheses have also as a common constant accompaniment vitiation of the secretions of the mouth. Thus one type of scrofulous subject—that with coarse features, muddy complexion, and long thick upper lip, has usually a chronic condition of congested oral mucous membrane with secretion of viscid mucus and saliva. Then, for another instance, rickets is often preceded and attended by a virulent form of acid dyspepsia.

The broad facts now before us are, first, that without acid, without vitiation of the secretions of the mouth, caries is impossible; and secondly, that acid capable of dissolving enamel—and of more rapidly dissolving enamel in proportion as the tissue is soft and ill-made—is always being formed in greater or less quantity in every mouth in which absolute cleanliness does not exist. In the term absolute cleanliness, I include the absence of vitiated secretions such as accompany, in greater or less degree, every derangement of local or general health. Although the third predisposing cause, to which I shall come presently, takes an important part in determining the localisation of caries in some cases, this second cause should, with the first, which has been already discussed, account fully for the association of caries as an accompaniment or sequel to constitutional disease. As factors these causes vary infinitely in quantity in different individuals. It is not possible to find two individuals with teeth exactly alike in structure, and with organic defects (from which it is rare indeed to find any set of teeth quite free) occupying exactly similar situations. Add to this that vitiation of the oral secretions as a factor in the causation of caries is of constantly changing potency in the daily life of each individual, and we fully account for the extreme variability in different cases of the time of onset, the point

of attack, and the rate of progress of the disease, as well as for its frequent intermissions.

In combating this second cause of caries, the patient's general health must first be considered, recollecting that probably every lapse from a perfect standard will be accompanied by a proportionate vitiation of the secretions of the mouth. The question of general health, whether the ailment be a passing indigestion or the dyscrasia of a confirmed diathesis or an attack of acute disease, is for the physician, not the dentist. We have only to call attention to the indirect influence of systemic disease upon the teeth and to devise treatment to overcome its evil effects locally. But even with perfect health and perfect dental tissues, caries may appear if organic *débris* be allowed to remain and decompose in contact with the teeth. The first thing, therefore, in every case, is to insure mechanical cleanliness by the use of tooth-brush and tooth-pick. A tooth-pick properly employed I reckon important. I advise the use of those of quill or wood only, metal may scratch and break the enamel. I direct that it be used at night to clear away remains of food from between the teeth, before they receive their final brushing. Where the teeth are crowded and are of delicate structure, the use of floss silk passed between the teeth and rubbed to and fro supersedes the tooth-pick. Until lately I have been prescribing as a routine for healthy mouths a tooth powder containing two drachms of Castile soap, half an ounce of powdered orris, two drachms of borax, and two ounces of precipitated chalk. To this has been added ten drops of oil of cloves and a few of oil of lavender, or oil of geranium, or attar of roses. It is, however, very rare indeed to meet an individual in whom perfect health is constant, and in whom one or other of the predisposing causes of caries does not exist. I have, therefore,

latterly begun to urge my patients to use habitually a tooth-powder composed of the solid ingredients named, but containing, besides perfume, carbolic acid. I begin with a mixture containing 1 in 40 of carbolic acid—that is, twelve drops to the ounce of powder—and I sometimes prescribe as much as 1 in 20. This, however, is too strong to be borne where the gums are tender. Besides carbolic acid, I add eucalyptus oil from ten to twenty drops, and oil of cloves five to ten drops, to the ounce. These, as well as being antiseptic, have the effect of largely concealing the flavour of the carbolic acid; and I find this mixture is not disliked by sensible patients. I direct it to be used freely; to be taken up on a wet tooth-brush, and the mouth to be freely rinsed with water at intervals during the brushing. The use of a powder like this seems at once to remove *débris* of food, to prevent putrefaction, and to neutralise acid deposited and formed about the teeth. Care should be taken that the soap-powder is fresh and of good quality, or it may taste disagreeably. When properly prepared this powder has no soapy flavour, although it produces a slight soapy lather. This lather some few patients cannot endure; and for these a powder may be made containing, instead of soap, a few drachms of powdered myrrh. Boric acid is a very pleasant and useful adjunct to tooth-powders, and may be used in any quantity from a drachm to the ounce. With fastidious patients it is well to add perfumes which they prefer; and with orris-root, powdered myrrh, and essential oil perfumes—such as attar of rose, oil of lavender, cloves, and geranium, it is possible to impart a very agreeable bouquet of perfume to the powder, and effectually hide the odour of carbolic acid when that is used. A dentifrice ought, of course, to be thoroughly triturated and reduced to impalpable powder.

In fevers and other diseases in which the patient is either too feeble or too listless to clean his teeth for himself, this should be done by an attendant. In this matter the medical profession and nurses are much in need of instruction. They seldom think of the teeth. In such cases, and in all those where great vitiation of the secretions is present, as in the dyscrasia of pregnancy, extra means should be adopted to prevent putrefaction and fermentation in the deposits which form upon the teeth.* Pasteur has shown that perchloride of mercury is by far the most potent drug we possess for this purpose; and a solution of a strength of 1 in 5,000 is antiseptically equivalent to a two per cent. carbolic acid solution, which latter, although not more powerful than needed to prevent fermentation, is too strong to be used as a mouth-wash.† I find a not unpleasant mixture of perchloride can be made if one grain be dissolved in one ounce of eau de cologne or tincture of lemons. A grain of chloride of ammonium must be added to each ounce to prevent the perchloride from decomposing. Of this mixture a teaspoonful is to be mixed with a wineglassful of water, and the mouth to be thoroughly rinsed with it several times a day. The mixture approximates a strength of 1 in 5,000. Some patients complain of a lasting disagreeable metallic taste following use of the perchloride wash. To overcome this, and also to guard against the swallowing of even a minute quantity of this very poisonous drug, I direct that the mouth

* Thorough cleansing of the teeth in this way will often suffice to prevent toothache, which in some conditions—particularly in pregnancy—is due to irritation of carious cavities by acid.

† These facts have been confirmed by Koch and numerous observers. My prescription is based upon the researches of MM. Arloing, Cornevin, and Thomas, reported in the *Lancet* of 1883. Dr. Miller, of Berlin, has, while I write, published the results of his experiments with organic *débris* such as is present in the mouth, and he shows—as might be expected—that the perchloride is not less potent in arresting fermentation in this than in other similar substances.

be well rinsed with warm water, or warm water to which has been added a little spirit of wine or eau de cologne, immediately after use. Mouth-washes may be prepared with any of the antiseptics in common use—carbolic acid, chloride of zinc, or permanganate of potash. Elegant, pleasant, and efficacious lotions may be composed of boric acid and tincture of myrrh, with lavender water, and eau de cologne, or tincture of lemons; and all lotions are much more likely to fulfil the desired end when used with a tooth-brush, friction being necessary to cleanse the teeth from shreds of food and adherent mucus. For this reason a powder, when it can be used, is to be preferred to a lotion, and in every case the importance of systematic sweeping and cleansing of the teeth and interstices by means of a thin flexible wooden or quill toothpick cannot be too much insisted upon.

I find no difficulty in persuading patients to use these preparations—powder and lotions—and few complain that they are disagreeable; and I have described these mixtures not for the instruction of this audience, but rather to excite a discussion and obtain an answer to the question, What is to be done with the large minority of patients who are not sensible, and who, although sorely needing them, will not use any preparations which are not highly agreeable to the palate?

Fanciful women and children frequently return to me after using these preparations for a time at my prescription, and state that they have gone back to the use of some one of the advertised dentifrices, which, whatever their virtues or defects, are invariably concocted to please the class of patients which I name. It is not right of course to encourage the use of secret preparations, and as tooth powders of this class are always stated to whiten the teeth

(an effect which must as a rule be injurious to the teeth) I strongly oppose the use of all such nostrums.

Before leaving this subject, I must point out that without due exercise in mastication, teeth cannot easily be kept clean ; and where the function is impeded by the presence of tender teeth, these must be brought into a healthy state or extracted.

The third main predisposing cause of caries is crowding and irregularity of the teeth. This acts in several ways. It favours retention of particles of food and of other organic *débris* for long periods in contact with the teeth, these particles becoming fixed in the nooks and crannies formed by the irregularities, or becoming wedged in the unnaturally narrow interstices. Crowding and irregularity also greatly prevent the constant beneficial rubbing and polishing of which, in a well-formed jaw, every tooth during mastication gets a full share, and they prevent the ready access of the alkaline saliva to those very surfaces where acid-forming substances find their location. This cause of caries, like the others named, presents, in different individuals, an ever varying quantity in its influence in promoting the disease. From the common cases in which there may exist slight crowding or accidental malplacement of one or two teeth in a well-formed jaw, all degrees are met with up to the worst form—the small V-shaped maxilla, where the teeth are wedged together, leaning at all angles in what looks like one confused mass. Crowding and irregularity of the teeth are mostly caused by smallness and malformation of the maxillæ —a condition which is no doubt largely associated with the physical type presented by highly-civilised man. I have already entered sufficiently into the causes of deterioration of the apparatus of mastication, but I have not mentioned one which, in my opinion, is not without importance, namely,

sexual selection. The type of female beauty for many ages has included a small delicate jaw—the heavy jaw, or anything approaching prognathism, being universally deemed a disfigurement. Darwin has demonstrated the potency of sexual selection as an evolutionary force, and I cannot doubt its influence in the production of the modern form of maxilla.

What can be done to promote the growth of better jaw-bones, with ample room for all the teeth? I am afraid we cannot hope that, for the sake of the teeth of posterity, men will be advised to pick out big-jawed wives, but we can at least seriously impress hygienists with the fact that the human jaw, for its due development, needs adequate use, and that no dietary—however otherwise suitable its constituents—can be perfect which is composed of uniformly bland and soft substances calling for little or no mastication.

In the meanwhile we have to deal with the crowded jaws which daily present themselves. The most universally beneficial measure to prevent caries in these cases, especially where the teeth are of imperfect formation, is the extraction of the six-year-old molars as soon as the second molars are well in place. The first permanent molars are, with rare exceptions, the worst made teeth of the set, and in the cases where room is urgently needed, it is seldom indeed that their permanent preservation is possible. I am firmly convinced that the removal of these teeth at the epoch named is more beneficial than that of any others in causing an equal and sufficient spreading apart of all the teeth and preventing the evils of crowding. Exceptional cases in which these teeth are well made and sound, whilst others of the set are defective, must be dealt with in accordance with their special circumstances, and instances do often present themselves in which the circumstances point

to the removal of others of the set, most frequently bicuspids, as the best course to be pursued.

The operation of filing and polishing the contiguous surfaces of incisors and canines as a most effectual means of cutting short the progress of incipient caries in crowded jaws, is too well recognised to need comment here. I would merely note that this operation does not, according to my observation, answer where chronic inflammation and sponginess of gums prevails, as in the scrofulous subjects of whom I spoke.

The regulating of teeth by means of mechanical apparatus may be of service in overcoming the cause of caries under discussion, but in the use of apparatus we must be on our guard lest we excite the very mischief which we desire to prevent. Regulating plates should not be allowed to chafe the teeth; above all, they should never be allowed to remain in place for more than a few hours at a time; they should be removed at frequent intervals and thoroughly purified; whilst with equal frequency the teeth should be well brushed with an antiseptic tooth powder, and thoroughly cleansed from the coating of food *débris* which is invariably retained upon their surface by the plates.

THE END.

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