THE SYSTEMIC FACTOR IN THE TREATMENT OF DENTAL CARIES.

Read in the Section on Dental and Oral Surgery at the Forty-fifth Annual Meeting of the American Medical Association, held at San Francisco, June 5-8, 1894.

BY G. S. DEAN, M.D.

SAN FRANCISCO, CAL.

It is not only because of its novelty that I have chosen my subject. I have chosen it, also, because it seems eminently a proper subject for presentation on an occasion like the present—an occasion whereon the practitioner of general medicine and the practitioner of dental medicine sit down together, to consider what they may do, jointly, for the welfare of mankind. The time was, in the not-distant past, when these two practitioners were sadly ignorant of one another's territory. But now the two have come together. They have familiarized themselves with one another's country. And now that they meet as allies in the cause of humanity, now that they are inquiring what they can do jointly for that cause to which their lives are devoted, it seems eminently proper to bring forward for discussion a subject which lies within the territory of both. It seems fitting on this occasion, to bring forward for attention the systemic side of the treatment of the commonest of dental diseases—the commonest in fact, of all the physical ills with which mankind is afflicted. That there is frequently (not always) a systemic factor in the etiology of dental caries, and that systemic treatment is often needed for the cure of this local affection, I shall endeavor to show. This being shown, it will appear that the disease has an interest, a practical interest, for the general as well as for the special practitioner. Hence it has seemed to me that, apart from other reasons, the subject which I have chosen has a peculiar appropriateness to the present occasion.

Let me introduce the subject by presenting a case. Let me do this, not merely because it is the proper way to introduce a new subject, not only because at the outset a single clear concrete presentation is worth a world of misty generalities; but let me do it also as a protest against the opposite course too often adopted and even defended by the presenters of new subjects, who, thoughtlessly, not having taken due note of the need of the human mind for concrete centers of knowledge, burden us with vague abstract or general statements without driving the initial nails on which these statements should be hung. Case reports are usually considered little more than cumberers of the ground; to be avoided or eliminated, or employed only as idle gossip to fill waste space or kill waste time. But this estimation can be due only to non-appreciation of the nature and the necessary conditions of human knowledge. A report of a case may, certainly, be uninteresting; for it necessarily contains only what the reporter has seen, and he has perhaps not seen the items which are relevant. But for the presentation of new truth vividly, convincingly, for the purpose of rendering truth graspable or assimilable, there is nothing which can take the place of contact with the facts. With the assurance, then, that this method of presentation is right, and that the contrary method is wrong, I proceed to my narrative:

A gentleman sought dental examination, occasionally, during some years, his teeth being always found free from caries. At length, on the proximal faces of two of his teeth, cavities appeared.

Examination of the saliva revealed nothing worthy of note; no known form of salivary abnormality was present. We are not yet able to make satisfactory clinical salivary analyses. Inquiry developed the fact that, while the patient was "well," there was perhaps a trifling gastric and quite certainly a slight intestinal indigestion. Search for the cause of the disordered function, oral and intestinal, proved futile. Fillings were inserted; and there was given such hygienic attention, general and local, as seemed possible in the absence of etiologic insight.

About a year later, the filled teeth were found in good condition, but three other inter-dental spaces, in different parts of the mouth, were bordered by decay. At this time the general gastro-intestinal error, though not great, was sufficiently pronounced to be noticeable; and it was accompanied by rather frequent attacks of diarrhea, serous in character, sometimes supervening on exertion, at other times appearing without perceptible exciting cause. The patient, I thought, looked more pale and dull than before. He said that, though "well," he "felt somewhat run down."

Let me pause to set forth explicitly the salient general features of the case as they then appeared to me—there being no cognizable local abnormalities. Though there may have been periods of mucous inflammation, yet on the whole it may be said that whatever evil existed was beyond the reach of the dissecting knife; in other words, there was no apparent structural error; the troubles were purely functional. The failure of function was twofold, general and local; there was systemic atony or asthenia, and there was disorder of the intestinal tract; the digestive disorder being, I may add for explicitness, extended as distinguished from localized, and simple or afeptic as distinguished from complex or parafeptic. The relation between the local and the systemic derangement was beyond my view. I desired on this point (as I had done the year before) an expression of opinion from gentlemen more capable than myself of forming a judgment, but, failing to obtain it, remained in darkness. I probably accepted tacitly the hypothesis usual in such cases, namely, that the general asthenia was due to the digestive error.

The etiology of the predisposition to dental caries was, then, beyond my view. The dental disease seemed incapable of actual remedy, capable only of retardation by persistent or repeated local attention. The progress of the destruction of the teeth was again checked by filling the cavities, and the patient was brought to an active resolve to care locally for his own teeth (in the sense of keeping them clean) the moving cause being a realization by him of the fact that the predisponent of the dental caries was undiscoverable and therefore irremediable, and that therefore, to prevent destruction of the teeth, the effects of the predisposition must be met by local measures.

In a few weeks he re-appeared, complaining of oral and pharyngeal inflammation. Examination showed his disease to be amygdalitis purulenta—"quinsy." It terminated in suppuration, its character being thus conclusively established. Believing that suppurative amygdalitis is usually due to aërial poisoning, I determined to investigate more minutely than I had previously done the man's aërial surroundings, with some hope that I might thus reach the cause of his dental caries. But my investigation showed nothing. It was brief and eminently unsatisfactory. I ascertained that he had been living in and frequenting the same buildings during somewhat more than the period of his dental decay; but I found that his quinsy had supervened on an absence from home of a month's duration, and had appeared three or four days after his return. Apparently, then, since he had inhabited the same localities so long without quinsy, this disease had been contracted during his absence.

The synchronism of his habitation of his present abode and the caries of his teeth seemed suspicious, but inquiry, made on his presentation of dental caries and now repeated, showed that the house was apparently healthful; that it had in fact, every apparent advantage, and seemed above suspicion. The synchronism was therefore set down as a chance coincidence, and the problem of the dental caries was no nearer solution than before.

A few months elapsed, and the patient appeared again, suffering a second time with quinsy. The tonsillar abscess being evacuated, his case came up for consideration.

I hold, as I have said, that purulent amygdalitis is caused by ærial poisoning. I published some years ago, and justified by evidence fulfilling every logical requirement, the following theses: 1, suppurative amygdalitis is not in any way related to or dependent on mucous or catarrhal inflammation; it is a disease *sui generis*; 2, the predisponent of purulent amygdalitis is neither "plethora," "debility," nor "a previous attack;" it is the functional activity of the tonsil glands; 3, the excitant of purulent amygdalitis is never "cold;" it is always poison contained in the air inhaled (possibly, to include the suppurative amygdalitis which sometimes complicates scarlatina, I should add "or exhaled").

Holding this belief, and holding it not as an unproved hypothesis but as a theory established beyond reasonable doubt, I inquired concerning the patient's exposure to poison-bearing air. Where had he been? What had he been doing? Not the smallest light could be obtained from his answers. He had indeed, again visited the country. But he had avoided the house in which he had stopped during his previous absence from town; had not even been in the same region. And the visit to the country, in itself, could not be accepted as the cause of the amygdalitis; especially as he had frequently gone to the country in former years, and had never had the disease.

At length there was found a single similarity in the antecedents of his two attacks of tonsillar inflammation. Each had been preceded by an absence from his domicile for some weeks, and a return home. This one thing, the return home, was clearly shown by further inquiry to be that sole common antecedent which must be regarded as the cause. The quinsy poison was then in the house which the man had inhabited for years. Yet during these years he had had no quinsy. How was this? It was explainable by the fact that, while he was at home, his rooms were always well ventilated; whereas during his absence they were tightly closed, and he had on both occasions returned at night, and had slept on the infected pillows in the confined air of a month. (Have we not here, by the way, an explanation of the fact that purulent amygdalitis is more prevalent in winter, when houses are shut, than in summer, when they are open?)

The first etiologic insight being obtained, the view expanded rapidly. The patient was now, in spite of his quinsy, bright or vivacious. Though unable to talk before the evacuation of the pus, he said in writing that he "felt well," was "hungry," and that, while it hurt him to swallow, he got down "lots of milk and oysters." Sharply in contrast with this was his condition during his first attack. Then he had been "sick;" dull, drooping; he had had no appetite, swallowing a little soup or gruel occasionally with reluctance; he had been, in a word, prostrated mentally and physically. Equally marked was the contrast in recovery. Now, when the pus escaped, he was "well;" that is, without any great amount of felt indisposition; before, for days after evacuation he had been "sick," and had remained "indisposed" for weeks; not, certainly, confined to bed, but markedly prostrated.

What was the cause of the difference in the accompaniments of the two attacks? In both there was approximately the same amygdalitis and pus formation. The inflammation produced, in both cases, systemic disturbance. But, since the inflammation was approximately the same in both cases, there was quite certainly something else which intensified the systemic symptoms of the first attack. Eliminating, then, the similarities of circumstance of the two attacks, I sought the difference of circumstance. The only marked difference found lay in the place where the disease was incubated and endured. The first or prostrating attack came on after the patient had been at home three or four days, and was endured at home. The second or non-prostrating attack came on and was endured away from home (the patient having

gone home after a month's absence, remained one day, and departed again). The prostration of the first attack was then quite certainly due, like the quinsy itself, to the house in which the patient lived.

The problem was solved. The etiology of the dental caries was at once transparent. The man had slept in a quinsy house; hence his quinsy. The atmosphere of the house (more strictly, of the rooms which he occupied) produced not only quinsy, but also general prostration; hence, in my judgment, the excessive "sickness" which accompanied the first attack. Did the cause which produced prostration when the man was ill also produce it, less conspicuously but no less really, when he was "well?" In other words, was the foul air of his domicile the cause of his atonic condition, and did this in turn produce his long continued digestive indisposition? I answered, with confidence, yes.

The step to the causation of his dental caries was easy. All dentists, probably, hold that the etiology of dental caries extends beyond the field of the microscope. All, necessarily, regard the saliva as a factor in the problem of dental decay. The evidence of an "epidemic constitution of the atmosphere" is as nothing in comparison with the evidence of an epidemic constitution of the saliva. How the saliva acts is an unsettled question. It is not even settled whether the saliva is, as to the teeth, a destructive or a protective fluid. Some dentists, not having seen any clinical evidence to the contrary, hold that acid saliva favors solution of the calcium salts of the teeth, and that alkaline saliva protects the teeth by neutralizing decay-producing acid. Others hold, with Dr. Black, the more elaborate hypothesis that normal saliva is protective, and that it acts germicidally; or, perhaps more properly, that this saliva modifies microbial life, diminishing the microbial secretion or so changing its character as to render it harmless. Others, with Dr. Palmer, regard the saliva as decay-producing, by reason of its being an electropoison fluid of greater or less power. Others, perhaps, without denying that there may be other features of salivary preservative or erosive action, believe that, the saliva being essentially protective to the teeth, one of its main prophylactic functions as regards dental caries is to dissolve and wash away such matter as tends to undergo acid fermentation—in a word, to cleanse the teeth. But whatever the hypothesis as to the mode of action, the fact, if not of salivary preservation of the teeth from caries, at least of the dependence of dental caries on the state or character of the saliva, must be universally admitted—if not explicitly declared, then implicitly held. It follows plainly that any state of the system in which the oral digestion or salivary function is impaired, is a state predisposing to dental caries. And, since the prostration of our patient impaired the gastrointestinal digestion, I considered it reasonably certain that it impaired also the oral digestion—that it gave rise to saliva deficient in caries-preventing power.

Feeling satisfied now that I understood the case, if not in all its details, at least sufficiently for practical purposes, I felt myself in a position to endeavor to effect the radical cure which was the patient's great desire. My remedial measure will be evident without statement. It was, as the patient was only a lodger, an immediate change of domicile.

The sequel showed that my advice was probably

correct. The man left the haunted house and remained under my eye for fully three years. His improvement in tone, in the feeling of "general health," was noted by himself, and the improvement in his appearance, though not very marked, was perceptible. The general digestive difficulty, I think, disappeared; it certainly diminished, for it ceased to be noticeable. But what was of special interest to me and is of special interest in the present connection, was the improvement in the oral digestion; the fact that though the patient became comparatively lax in the local hygienic care of his teeth, he ceased to suffer from dental caries. It is true that two (I think three) cavities of decay were found in his teeth shortly after his change of residence; but these cavities were residual to the period of decay, and they were not followed by others appearing year after year. In a word, after removal from the poisoned house, the man became, as he had been before his sojourn in it, "proof against dental microbes."

Such are the facts of the case as far as I saw them. I sought to extend or supplement them by a more minute inquiry into the aerial conditions which produced the atony—the incipient cachexia. But my supplemental facts proved meager. The unhealthy house was a comparatively modern structure, with large rooms, plenty of sunlight and good ventilation; it was, in fact, as I have already stated, prepossessing. The rooms occupied by the patient were a handsome suite in the second story. The only elegance lacking in them was that which, if it had been present, might have been blamed as the source of "sewer gas"—the stationary washstand. There was, indeed, no apparent connection with any sewer; nor was there, at first glance, any apparent source of quinsy-poison. The source of this poison was not, however, very difficult to find. Not only did green or rot-inviting lumber appear to have been used in the construction of the building, but the apartments of our patient were directly over a room and cellar in which usually provisions and sometimes articles not free from decomposition were kept, and the apartments communicated sufficiently with the lower rooms to allow air to pass. The patient's apartments were always "close," and "smelled badly" after being shut up for a time. The quinsy, then, was readily accounted for; a month's concentration gave a large stock of putrefactive germs. But the prostration, and its consequence, the oral indigestion? Was it due to the same poison, acting in small quantity for a long time? Or was it due to some gaseous accompaniment of the poison which produced the purulent amygdalitis—some chemic accompaniment of the physical poison? I hold the latter hypothesis because of familiarity with the effects of chemic poisons on tissues and consequently on organisms, plant and animal; but confess that I do not know. It may possibly have been due to something which had no connection with the amygdalitis, *e. g.*, to a leak of illuminating gas, whose depressing effect on the human organism may be seen by inspecting the workmen in almost any gas factory; but this supposition, infracting Occam's caution, "*Entia non sunt multiplicanda praeter necessitatem*," is rendered highly improbable by the fact that the systemic prostration accompanying purulent amygdalitis is usually very great, usually exceeds what one would expect the abscess (which constitutes the disease) to produce.

I have now laid a foundation for a statement of generalities. I have reported a case wherein, if my interpretation is correct, dental caries was caused by systemic error and was cured by systemic treatment. But before proceeding to state generalities, and to make, finally, the plea which I propose to make for a more intimate working together of the general and the dental practitioner, it will be well to clinch one nail which I have driven. It will be well, especially since the presentation of the nature and the clinical origin of purulent amygdalitis is so recent that it has probably not yet received general consideration, to justify by the facts of this case itself, as far as these facts go, my immediate certainty, on finding purulent amygdalitis, that the patient had been breathing foul air.

The facts are these: Before lodging in the house herein referred to, he had returned to San Francisco several times and had never had quinsy; while lodging in this house he returned twice (and only twice), and had the disease both times. Confirmation is furnished by the supplemental fact that, after abandoning this house, he returned to San Francisco three times at least (probably more), and did not have quinsy at any time. Clearly, then, the cause of the quinsy was not San Francisco; it was the house; in other words, it was not cold; it was poison in the air.

While I am speaking parenthetically, it may not be amiss to make another digressive remark; to call attention to the difference in the period of incubation of the two attacks of amygdalitis in the case reported. The first attack appeared probably three days and certainly not more than four days after exposure; the second attack did not present its initial symptoms till at least the fifth and perhaps the sixth day. The reason of this difference in time of incubation, though it seems fairly plain, as it has no relation to my present subject, I can not here discuss; I merely note the fact in passing, because it is seldom that we have an opportunity of observing two different periods of incubation of the same disease in the same individual at the same time of life.

Returning now to our subject we are ready for generalities. First, I may state that dental caries is not usually idiopathic. In this disease the general is not dependent on the local; it is the local which is dependent on the general. We speak of "frail teeth" as "prone to decay," or, more flatly, say that teeth decay because of their frailty. But when we speak thus we do not, or at least should not, mean literally what we say. If the salivary function (which is directly dependent on the general or systemic condition) is normal, the teeth, however frail, are safe from caries. And, contrariwise, the strongest teeth, though more resistant than frail teeth, will, under adverse systemic conditions, succumb to decay.

Next let me, as well as I can in a brief space, discriminate between idiopathic and systemic or symptomatic dental caries. Generally, I may say that cavities of decay on the morsal aspect of a denture are not to be considered as of systemic origin. To this rule, however, the exceptions are exceedingly numerous; they are based on this consideration: Rapid decay on any aspect of a denture, is to be regarded as due to systemic disturbance. Generally, again, it may be said (the exceptions are few) that decay on the proximal or buccal aspects of the teeth is symptomatic or of systemic origin, and calls for

something more than local treatment. It is true that we sometimes find cases in which we can feel reasonably certain that there is a direct salivary defect; cases of idiopathic salivary deficiency. But such cases are so rare as to throw the presumption on the other side. We may safely say, then, that proximal decay is to be generally regarded as a result of distant disorder or systemic depression.

Let me repeat here, to avoid possible misinterpretation and, incidentally, to be more explicit:

1. Dental caries is commonly due to fermentation of food, and occurs only where food lodges or remains for a sufficient length of time to undergo fermentation; it occurs, that is, only where there are lodging-places for food, *i. e.*, on the proximal faces of teeth which stand close together, on the buccal faces where the gum forms a pocket for the retention of food, and on any face where deep sulci, pits or fissures hold food till it undergoes fermentation. Those exposed or smooth surfaces of teeth where food can not lodge are always free from decay.

2. Normal saliva prevents dental caries, either by preventing decay-producing fermentation, or by overcoming the effects of this fermentation. It is in fact preventive of this fermentation or its results to a greater extent than one would, *a priori*, think possible. The saliva has scanty access to interdental spaces more or less filled with food; yet, in spite of uncleanly habits, normal saliva prevents proximal decay. The saliva can scarcely reach the filth-covered cervico-buccal portion of the teeth of the uncleanly; yet the dirtiest teeth are often free from buccal caries. Again, fissures and pits penetrating the enamel would be expected invariably to become seats of decay; but where the saliva is normal such fissures and pits are usually innocuous; they do not, in health, become centers of caries.

3. Anatomic (*i. e.*, macroscopic) faults of the teeth, such as pits and fissures, and errors of location, such as over-lapping, are sometimes so great as to lead to decay in spite of normal saliva. Decay due wholly to this cause is, however, rare except in teeth which, besides having the gross defects mentioned, are microscopically (histologically) defective; so-called "frail" or "soft" teeth—teeth wherein the mineral matter is lacking in solidity of structure, either as a whole or in spots, or, as may be the case, both as a whole and in spots; and even in teeth which are decidedly frail, pits and fissures of moderate size and depth, and irregularities of moderate extent, are, with normal saliva, innocuous.

4. "Frailty" or histologic defect of the dental tissues, even to the extent of what might not improperly be called idiocy of dental structure, rarely if ever produces idiopathic dental caries.

5. Decay-producing error of saliva is undoubtedly sometimes idiopathic. We find differences in the saliva of different persons, both in quantity and in quality, under circumstances which render us reasonably confident that they are not due to what we may for brevity, call systemic disturbing causes.

6. Idiopathic dental caries is relatively rare. The presumption is therefore always that the disease is secondary or symptomatic. The least attentive observer will see that it is symptomatic when, after many years of complete dental soundness, suddenly every fissure becomes a seat of caries, while the disease at the same time attacks the proximal faces of the teeth in many places. From cases like this we

may descend step by step to those wherein the most observant can not assert with confidence the nature of the disease. The pathognomonic signs of symptomatic dental caries may be stated as follows: *a*, its residue is "moist" or "leathery" in character; *b*, it is rapid in its progress; *c*, cavities are found to have originated simultaneously in different parts of the mouth. The severity of the systemic disturbing causes may be determined by noting which faces of the teeth are attacked. If the systemic cause is mild the decay will be confined to the fissured morsal surfaces, or will at most attack the buccal pits of the molars. If the systemic cause is pronounced, the proximal faces of the teeth will be attacked, perhaps also the buccal faces close to the gum margin.

7. Beyond symptomatic dental caries, using the term in its strictest sense, there is usually, after removal of the ultimate cause, some further decay of the teeth—what may not improperly be called residual decay. This is a phenomenon which we might perhaps have anticipated, *a priori*, for (apart from the probability that incipient dental caries is sometimes overlooked, to be discovered in an examination at a later date) a return of deranged salivary function to complete normality might be assumed to take time, even after full removal of the cause of the derangement; but as a fact I never thought of it till I observed it in practice, when it occurred to me, first, that I had seen it in other cases without noticing it, and then that it was a natural thing to expect.

I do not know that this residual decay has been noticed or commented on by others and therefore speak of it only from my own experience. It has appeared to me, in cases wherein I have felt able to fix the date of removal of the fundamental cause, that there was a period of some months, possibly a year or more, wherein dental caries continued to appear, though with diminished severity. The disease seemed to subside, as a rule, gradually, not instantaneously. I think it well to make a note of this both for confirmation or disproof by others, and also because residual decay if one did not expect it might prove misleading.

Having discriminated between idiopathic and symptomatic dental caries, it remains to ask on what other diseases or on what fundamental causes the symptomatic dental disease depends. Primarily, symptomatic dental caries is plainly due to disorder of the oral digestion, fault of the fluids of the mouth. Unfortunately, having said this, we have almost reached the limit of present knowledge. The sources of oral apepsia are incapable at present of being pointed out with even an approach to definiteness. We naturally first look to the other portions of the digestive tube as theoretically likely to be the over-shadowing source, by extension of derangement, of oral mal-function. But, clinically, we find in many cases that gastric or intestinal disorder is not accompanied by decay of the teeth. The oral function does not seem to sympathize universally with that of the lower portion of the digestive tract. Gastric or gastro-intestinal error (atonic dyspepsia particularly) does indeed frequently accompany the oral error, but it is probably only a joint result of a common cause. In a word, we do not find any one great or especially probable source of salivary derangement. We find merely that dental caries seems to spring from the most diverse causes with equal readiness, in fact, from almost anything which ap-

pears likely to diminish the vigor of the ultimate cells or to drain or to enfeeble any portion of the organism.

This general statement, vague as it is, presents nearly all our present knowledge on the subject of the fundamental etiology of the disease. A little definiteness may be given to the statement of causation, but only a little. We can say, cautiously, that with our present limited clinical knowledge, in male subjects not suffering from any evident organic defect, lack of good air and exercise seems the first subject of suspicion, the most probable cause. We can say that, secondarily, in these subjects, our limited experience would lead us to look to the nitrogenous diet as a fairly probable cause. For the avoidance of possible misinterpretation, I must explain that while it is the carbohydrates which, by fermentation in the mouth, directly yield the (acid) agent, which, corroding the teeth, produces dental caries, and while therefore a strictly nitrogenous diet could probably not cause dental decay because it would be devoid of acid forming material; on the other hand, the proteids, when ingested in large quantity by persons of sedentary habits, seem to give rise, probably through their effects on the greater glands, to decay-producing error of the salivary secretion; in other words an ill-advised largely-nitrogenous diet may and apparently does produce an oral or salivary condition favorable to that fermentation of carbohydrates which causes caries of the teeth.

In young men our observation would lead us to say that, even where it does not produce perceptible gastro-intestinal error, irregularity of life (sexual excess particularly) should be first suspected, it being a common, if not the most probable, cause of dental caries. In the female subject, the "organism round the uterus," the center of the organism appears to be the most suspicious point. Experience has shown that, not only pregnancy but also almost everything amiss in the female generative system, is accompanied or followed by dental decay. Caries of the teeth comes on so frequently between puberty and the menopause, ceasing when the cervical canal is rendered patulous or some other change for the better is wrought at the center of the organism, that we are amply warranted in first suspecting, in females, that the cause of our dental disease is a generative trouble.

But having said all this, we who are practicing dentistry must confess ignorance—ignorance on the one hand and inability to act satisfactorily (by reason of the burdens imposed on us by the necessary local treatment) on the other. We find that we can not yet say with certainty that any given general cause will always produce, or that any given pathologic condition will be always accompanied by caries of the teeth. We find (to take an actual case) the same person suffering at three different periods with gastric or intestinal dyspepsia or both; the first and the third attack were accompanied by dental caries (which ceased with the disappearance of the gastro-intestinal difficulty); the second attack, though it continued for two or three years at least, was not at any time accompanied by caries of the teeth. We find ourselves, in a word, when we come to the fundamental etiology and therefore to the radical treatment of dental caries, unable to answer any but the simplest questions; to solve any but the simplest problems; to cure any but the simplest cases. We

are able, certainly, sometimes, to see through a case of dental caries almost at a glance, and are able occasionally to point with ease the way to positive cure. But the cases wherein the cause of dental caries is obscure outnumber those wherein it is easily discernible. The one which I have reported for the sake of illustration may be taken as a sample of those which we daily encounter; and in cases like this, in the diagnosis of the fundamental difficulty, we who are practicing dentistry usually need assistance. Again, when the cause of the dental disease is readily perceptible, we usually need the aid of the general practitioner in its removal.

It is idle to say that he who undertakes to treat a disease should go to the bottom. As a practical fact, the dental practitioner, however thorough his understanding of the nature and the treatment of disease in general and of those extra-dental pathologic conditions with which dental diseases are connected in particular, finds that the burden of attention to local manifestations is usually all that he can bear; he must practically, as a rule, refer his patient for the radical cure of a dental difficulty dependent on a general cause, to some one else; perhaps to the special practitioner in some distant field, but usually to the general practitioner. Hitherto, owing to inattention on the part of the general practitioner to the matter which forms the subject of this paper, the reference has been made by the dentist with comparative infrequency; and when made has not always received attention. In the future it is to be hoped that it will be made more frequently, and that it will meet with a more general and a more thorough response. We who are practicing dentistry are not content to merely temporize. And if we were content to do no more than, locally, to "meet indications," our patients would not always contentedly acquiesce; for they are, some of the more intelligent at least, awaking to the fact that dental caries has more than a local meaning; and, in the interest of their general as well as of their local welfare, they are beginning to desire not merely treatment of symptoms, but cure. This, the actual cure of dental caries, we are as a rule unable to effect alone. We need the aid of those who are practicing in the general field. My aim, in this paper, has been to show that this aid can be frequently effective. And I close with an appeal for the assistance which will accomplish the end; an appeal like that of the man in the vision to the apostle: "Come over into Macedonia and help us!"

DISCUSSION.

DR. DUNBAR—The etiology of caries of the teeth is difficult to discuss, and I am quite ready to accept the views of the essay as to systemic influences as a general cause of decay. Original defective development of the tooth, Dr. Dean touched upon as one of the principal predisposing causes of decay. There is no question that it is an acid in some form mysteriously supplied which causes the first breach, and people moving into malarial districts are liable to many conditions which develop into loss of teeth. I heard many years ago a prominent dentist, who lived in a miasmatic district, declare that he could not save pulps when they were once diseased. So, also, the references to quinsy poisoning are certainly verified in this malarial environment. There is no question that we are on the right track for the discovery of the mysterious acid which makes the first breach in sound teeth, and opens the way for decay. We are on the verge of an important discovery to add to and supplement those of Miller, of Berlin.

DR. DEAN—I present here two teeth for inspection, and I would like an opinion as to whether the trouble with them is decay or erosion. The history is this: The teeth are

baby teeth. The child during its second summer was very much troubled with digestive difficulties—it is not fashionable to think there is any connection between such difficulties at this time and teething. The four central incisors above and below were present, but no other teeth. The upper ones presented on the points when erupted, what I thought was caries. Now I want to know what it is. The cavities approached to pulp so close that I could not attempt to fill. The four cuspids presented some appearance of erupting. I lanced over the two which were nearest to eruption, but it did not check the diarrhea. Then I cut for the others, and within twenty-four hours the digestive difficulty improved, and within a week the child had entirely recovered.

DR. L. A. TEAGUE, San Francisco—I would ask Dr. Dean why lancing was resorted to?

DR. DEAN—My belief was that the digestive difficulties were due to nervous reflex by pressure of the gum on the advancing tooth, that pressure being reflected on the papilla underneath. I lanced for the purpose of relieving the tension.

DR. TALBOT—I have had it in mind to write the same sort of a paper as this of Dr. Dean's for five or six years. We are indebted to Miller for the establishment of the relation of bacteria to the causes of caries, but we have to go outside of dental practice to find the causes of decay. I don't think the writer of the paper meant to say that the poisonous air in the home of the patient acted directly, but that it produced a neurosis which caused the lesions with which he was troubled. Neuroses, eruptive fevers, cause mal-development of the teeth and bones. It is a fact that these troubles are due to lack of tonic in the nervous system, as is nicely demonstrated in the case of the pregnant female, whose loss of teeth is due not to a lack of lime-salts in her food, but to the lack of tonic in her nervous system which prevents their proper assimilation. It is to this cause we have to look for many of the troubles which we as dentists are called on to combat.

DR. DUNBAR—It has often been said that we as a race are going to lose our teeth; that we as Americans are most prone to diseases of the teeth. Those who make these assertions cite old country districts and say they don't have these things there. It has been said that the cause of the difference is our American environment, our climate, the mixture of races, etc. They forget, in enumerating the causes of the state of affairs they describe, the circumstances under which our forefathers took up their residence here. Almost universally they had plenty of hard work to do and they raised large families, for which the mothers did all the work. Isn't it possible that a broken-down mother may transmit to the tenth or the twelfth child, especially if it is a female, her own exhausted nervous system and that that is in some cases, at least, the secret of the "poor American teeth"? Then our American haste, our constant-rush for money, make us nervous and we can't transmit, under such circumstances, an unimpaired nervous system to our children. On the other side, they are more contented with their lot; here we are dissatisfied with our income and always trying to increase it. We are a neurotic people and we are begetting neurotic children. More of the difficulties against which we have to contend are due to this cause than is generally understood.

DR. A. C. HART, San Francisco—My father had good teeth; my mother poor ones. I have my father's denture. He had but one lateral incisor; I have none, but my other teeth are like his in form and quality. My brother has teeth like our mother, of poor structure, and decay progresses among them in the same way as in hers. My preceptor taught me if I found decay on one side of the mouth, to look for it in the same teeth on the other side, but while this is a good rule, it does not hold in all cases. I confess I am puzzled to account for many things, and I have to refer them to a principle which I call life-force. For instance, why is it that there are only boys born in one family and why all girls in another? I have reached the conclusion that when the man is stronger the child is apt to be a boy; when the woman is stronger, the child is generally a girl. Whichever of the parents is stronger makes the impression on the embryo which determines its sex. There are many things in which if we change the environments we change the results.

DR. TALBOT—Two points have been referred to by Dr. Dunbar and Dr. Hart upon which I want to say a word. Dr. Dunbar said there was more decay and more deformity of the teeth here than in Europe. If you take the class of people in Europe who can be fairly compared with the people here, their teeth do decay as much as here, and deformi-

ties are more prevalent and more marked there, especially among the aristocracy. I have just read a paper, before the Neurological Section of the Association on this subject. The Spanish people have the most difficulty from the neuroses which produce deformities among the aristocracy of Europe. Dr. Hart brought up the point of heredity, but it is not quite fair to give a conclusion on one case only. The tenth child may be the best of all. Heredity works both ways.

DR. LUNDBERG, San Francisco—I differ from Dr. Talbot as to the condition of the teeth of the aristocracy of Europe, especially as to the part where I came from, Sweden, as I am sure they have good teeth.

DR. TALBOT—Do they not have a great deal of decay and many deformities?

DR. LUNDBERG—Not more so than here. There is no other nation of Europe where the teeth are in a more excellent condition, both in the aristocracy and the mediocracy. This is attributable to their mode of living. Their food is not so generous as here. The caries and defective teeth of Europe may be traced to gestation. You may find good teeth among people who live luxuriously, if the mothers take exercise and live on nourishing food. I don't think you can fairly say that the people of Europe have as bad teeth as the people have here.

DR. DEAN—I have asked a question as to abscess unconnected with the gingival margin. I understood Dr. Younger to say that there is always some opening to or connection with the margin. I would like to ask if any one has seen a calculus unconnected with the gingival margin? (Receiving no reply, Dr. Dean said that he had specimens which seemed to be of that nature.)

THE DISEASE OF INEBRIETY.

ITS STUDY FROM THE STANDPOINT OF THE EXPERIENCE
OF AMERICAN PHYSICIANS OF EMINENCE WHO HAVE
WORKED IN THIS FIELD OF RESEARCH.

BY EDWARD C. MANN, M.D.

NEW YORK CITY.

(Continued from page 853.)

SPREEING AND TIPPLING.

A not uncommon form of the tendency to inebriety is manifested, especially in young men, in whom the propensity is not fully developed. This consists in the practice of occasional "spreeing," as it is sometimes termed. This forms one of the earliest stages of the habit or attack, and may be properly termed the formative stage. It makes its appearance on occasions of rejoicing, at festivals and celebrations, on Fourth of July and during the holidays. At other times the propensity is dormant, and the individual is in no way affected by it. There is a feature in this malady, which may be properly noted in this place, and that is that in the advanced stages of the disease nothing satisfies but complete intoxication. So completely is the system under the influence of the morbid propensity, that the desire to get drunk is irresistible. No lesser degree of exhilaration or excitement answers—nothing but total obliviousness satisfies the craving propensity.

Inebriety makes its appearance as other diseases make theirs, sometimes by the fault of the individual attacked and at other times from causes over which he has no control. The propensity is quite often hereditary, and transmitted from one generation to another, in accordance with the same law by which any constitutional taint, like scrofula or tubercular disease is handed down. The law of development which, from the germ, fashions and matures an individual, and by which it is made to resemble its prototype or parents, will also in due time bring forth the defects which may have existed in a previous generation. The fact is a familiar one, that children resemble