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A CLASSIFICATION OF TEETH THE DISEASED PULPS AND APICES OF WHICH ARE RELATED TO INFECTIVE FOCAL AND SYSTEMIC SEQUELAE [1]

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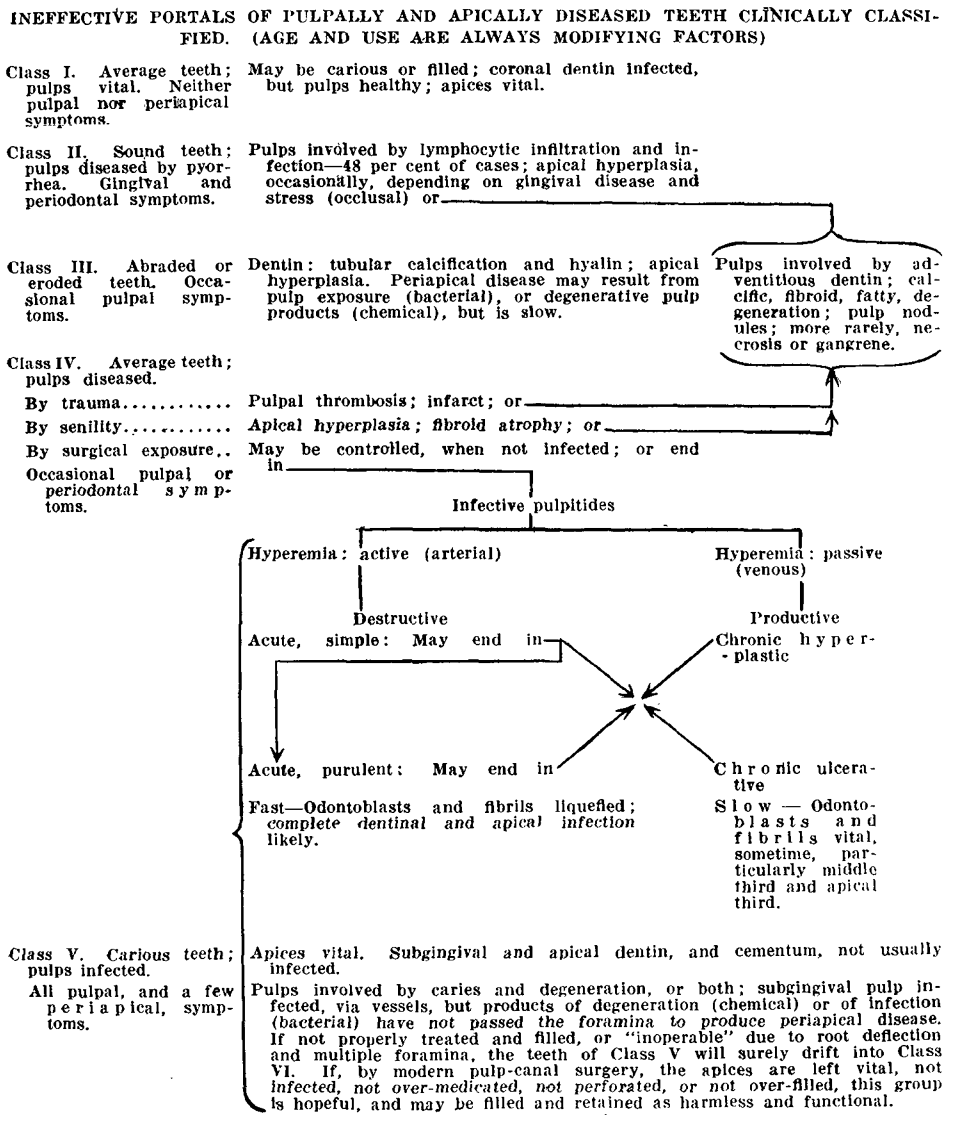
SO HEATED is current discussion of so-called "pulpless," "dead," or "non-vital" teeth; so many bodily ills of doubtful etiology, hence treated experimentally, are said to have been caused by "non-vital" teeth and cured by the removal of such teeth from the maxillae; so doubtful pathologically are these ills; so questionable is the right to the claim "cured," when applied either to the chronic *systemic* or to the equally chronic *oral* diseases that these and other reasons prompt a pause by both dental and medical professions, in order the better to classify and define the pathology, not only of "pulpless" teeth, but of all other teeth, invaded by pathogenic bacteria.

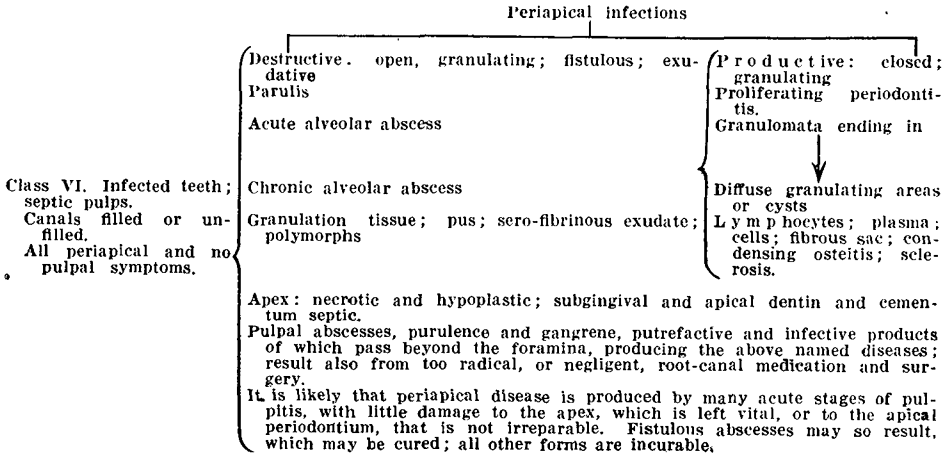
Such a working classification (16), crude but practical, was presented by the writer eighteen months ago; and, recently, at the last meeting of the National Dental Association. It will be attempted again, but it must be accepted as a clinical presentation only, for, as the matter now stands, it is next to impossible for dentists to consult intelligently among themselves upon this subject. How, then, can they expect their medical confreres to comprehend in oral pathology what, to their own minds, is

[1] This paper, to which additions have been made, was first read before the First District Dental Society, New York City, February 2, 1920.

not clear? Is it any wonder that our medical confreres, not understanding, condemn all "filled pulpless teeth," overlooking many others more virulently diseased, and doubt the judgment of any dentist who honestly insists that certain types of diseased teeth are both harmless and functional, hence should be retained?

Any intelligent discussion of this subject is necessarily based upon certain facts, which must be considered axiomatic, even tho subject to correction in the light of the constantly accruing results of research, like all other scientific data. Upon such clinical axioms we must all agree, just as we accept and apply treatment to that disease entity





known as dental caries, while disagreeing as to its etiology. These facts should be couched in the language common to general, not merely to oral, pathology, so that all pathologists may understand when we speak of the etiology and pathology of pulpal and apical diseases, all of which, if properly classified, would indicate the correct treatment and prognosis. Not only would this course greatly clarify the subject in our own minds and save countless, otherwise functional, teeth, but it would promote a better understanding, and closer co-operation, between both dental and medical professions.

If many of the facts submitted are bromidic and appear too elementary, let us remember that eventually we must return again and again to first principles; it is only by orientation of these, to recent methods of practice, that real truth will survive. Your attention is, therefore, directed briefly to the histopathology of the two main dental portals by which pathogenic organisms approach the pulp and subapical tissues, and thereby the lymph channels and blood stream, namely, (a) the dentin, pulp and root apex, and (b) the gingival crevice.

Discussion of the Classification

Class I—Average teeth; pulps vital

Class I, which may be carious, while both pulps and apices are vital and healthy, is necessarily a broad one, since all teeth that have been carious or restored by filling, within the prescribed limits of the class, belong to this group and lead to a moment's consideration of dental caries.

Many phases of tooth destruction exist which cannot be classed under erosion, abrasion, or caries. However, the disease group known under the broad term, dental caries, must be so accepted. Like many other wasting diseases, its etiology is unsettled. Whether we hold with those who consider acidophilic forms as the only cause; or believe these active in symbiosis with cocci; or again, that various streptococcal strains are the only factors, three facts are assured clinically:

(1) Streptococci constitute 50 per cent of all mouth growths of microörganisms and often are apparently normal in healthy oral and pharyngeal mucosae [Kligler and Gies (22); Hartzell and Henrici (19).]

(2) Pathogenic coccal forms have been demonstrated repeatedly, by culture and in slides, lying deep in the tubuli of vital dentin, under soft caries, and

after thoro cavity preparation. [Miller (25), Goadby (9), Hopewell-Smith (21), Hartzell and Henrici (19), Rosenow (30).]

(3) Miller (25) and others identify cocci as the deepest invaders of the dental pulp.

Attention is called to this septic factor in all stages of dental caries, hence:

Conclusion 1. Dental caries is dual in character, being a chronic tooth-wasting disease, with tissue necrosis or liquefaction superficially, and pathogenic forms irregularly invading the hard underlying dentinal matrix. This sepsis begins in the granular layer of Tomes, and is the primary infective focal portal. Therefore, because dentin is hard, or free from soft caries, it is not necessarily free from bacteria; and some effort must be made to sterilize every cavity-preparation in vital teeth.

It is probable that such latent infection exists in every well filled tooth, as it does in every carious tooth. Fortunately, the infection is merely latent and rises to the dignity of disease only when it involves the pulp. Disease is prevented (a) orally, by scientific filling-restoration, checking recurrent caries; and (b) cavally, by dentinal tubular calcification, granular hyalin formation or adventitious dentin—the usual dentinal protection, of which Hopewell-Smith (21) says: "Probably nearly every dentinal change is accompanied by some healing process of the pulp." This resistance reverses the rule, being less active in young adolescent dentin, with many tubuli or little matrix substance, than in adult or middle life. If, by reason of rapid and virulent caries, or a lack of resistance, these pulp reactions break down, much chronic pulp disease may occur under good fillings, with solid but infected dentin intervening, eventually producing periapical disease, while the dentin is sensitive and the pulp vascular.

We should be mindful of this infective element in treating children's teeth, particularly first molars. A deep cement filling or pulp capping (the proper procedure) often fails to protect the pulp because of collateral dentinal infection. Possibly, these are the most insidious and dangerous of all periapical types of infection.

What is conservative practice for Class 1, in the light of the foregoing? With dentinal infection controlled, and the wonderful pulpal resistance still functioning in a vast majority of cases, do we stand with the "devitalists," who would remove a pulp before it becomes infected, because sometime this function may fail? Or, have we such confidence in our canal technic as to devitalize and positively (?) remove every particle of pulp, and fill every root, where occurrences of apical deflection and multiple foramina are at least 40 per cent (16), with perfect root fillings in only 7 per cent of the cases (of thousands) and with 47.5 per cent of all filled roots apically diseased?

The operator who proceeds to devitalization, in spite of these facts, assumes a reparative skill more potent than the human body possesses. If he succeeds, it is usually not by his skillful technic, important tho that may be, but in spite of it—by remarkable reparative processes in the periodontium and vital apex. Hence:

Conclusion 2. The normal pulps of vital teeth should be devitalized rarely, and only when devitalization is important for the construction of such occlusal restorations as are impossible by other methods and absolutely necessary to general nutrition; recalling the 70 per cent of occlusal efficiency of bridges, as compared with 30 per cent in the case of dentures.

The possibilities of modern partial dentures and attachments, which have been so markedly improved, conserving rather than destroying attaching teeth, must be thoroly studied and exhausted before it is decided to risk devitalization of a normal pulp.

Class II—Sound teeth; pulps diseased by pyorrhea

It is not proposed, in discussing Class II, to dwell upon gingival diseases, except as they tend to produce pulpal or periapical disease.

Dewey and Noyes (7, 8) have demonstrated the association of three sets of lymph vessels with the blood supply that completely surrounds every tooth, which is seated, so to speak, in a basket of periodontal membrane, dense socket lining (lamina dura), and blood vessels, all of which tissues these lymph vessels penetrate, nourish, and drain. The largest group passes from the gingiva, centrally down thru the membrane, paralleling the cementum; it is joined by gingival and middle-third groups, the first of which carries infection from the gingival crests to the nearest lymph nodes. Thus, a superficial gingival infection would be deflected over the interseptal crests; but a periodontal infection would finally reach the subapical tissues, splitting the periodontal membrane on its way, as in a true pyorrhea pocket.

At this point we would impress you with the results of recent research by Henrici and Hartzell (20) who, in studying one hundred coronally sound, but pyorrheal teeth, found 48 per cent of the vital pulps invaded by lymphocytic infiltration and the vessel walls much damaged, with coccal forms definitely established in the tissue. These findings should settle the question of hematogenous pulp infection. The round-cell infiltration is a counterpart of the cell reaction in adjacent tissue, previously noted, by Latham (23, 24) and Talbot (31), as pulp degeneration, associated with interstitial gingivitis. But the presence of pathogenic bacteria is a new highly significant fact, about which centers many possibilities, among them the following.

(1) Periapical disease is not coincident with gingival lesions; it does not arise in the same percentage of cases, nor occur in 48 per cent of sound pyorrheal teeth, the pulps of which show lymphocytic invasion and pathogenic bacteria. On the contrary, casual clinical observation shows that many pulpless teeth, 47.5 per cent of which are apically diseased, are the only teeth remaining firm and free from gingival disease in an entire denture swaying in purulence. This may be due to sclerosis or a condensing osteitis, which often involves the apical- and middle-third attachments of such teeth, making them firmer than those adjacent. This condition, by partially destroying the lower lymph system, would present a barrier against further periodontal invasion.

(2) That this 48 per cent incidence of pulp pathology does not always produce a like amount of periapical disease may be explained by the fact that coincident with pulp disease gingivally induced necessarily runs a pyorrhea that destroys the gingival-third fibers, and creates a compensating hyperplasia of the apical-third fibers and cementum to meet extra stress. Thus, the accessory, often the main, foramina, are closed and the entire apex is encased with laminae of adventitious cementum, which may seal this portal against infection, either afferent or efferent. Further, in any serious loss of gingival tissue resulting in exposure of cementum, pulpal fibrotic and calcific degeneration is common. This interferes with complete pulp removal and root-canal filling, which, in these cases, is below the average for all pulpless teeth. In fact, the high percentage of failures to fill apices and the low percentage of periapical diseases is notable, apical hyperplasia (and often a real hypercementosis) commonly existing, having closed this whole region against both pulpal and gingival infection, before the dentist could reach it.

Be these hypotheses what they may, it is safer to assume, for teeth involved by gingival disease, that sepsis might at any time reach apical and pulp tissues, since

the main lymph flow is from gingiva to subapical tissues; and to add, to the usual conservative rule (15), that "pyorrheal teeth, gingivally damaged to the middle third and swaying in purulence, must be removed," the further practice stated in the following conclusion:

Conclusion 3. Any pulpless tooth, the periodontal membrane of which is gingivally purulent, should be removed, no matter how perfectly filled, or how well repaired subapically, whether periapically diseased or not; for, at any time, it might become diseased by the gingival pathway. If pulp devitalization is absolutely necessary, it should be practiced on only such pyorrheal teeth as are not swaying to the middle third; or those cured (?) by peridontic treatment; and then, on only those in which apical hyperplasia has closed in part the foramina as noted. (Such hyperplasia is usually demonstrable, clinically and by good radiographs.)

The fact that 48 per cent of the pulps of gingivally diseased teeth are infected will not, it is to be hoped, lead to a new fad for the destruction of these pulps—just as all pulpless teeth are now being removed because they are interstitially infected. The situations are analogous and comparable. It has been shown that real periapical disease (one of the good reasons for tooth extraction) does not result in anything like the same proportion, from demonstrated pulp disease, as it does from the best efforts to remove such pulps.

A large percentage of dentinal infection never reaches the pulp to produce pulpitis, just as, no doubt, a similar percentage of cases of pulpitis never reaches the subapical tissues to induce disease outside the tooth. These are latent infections constantly occurring elsewhere thruout the body, in connective tissue, particularly bone, from the vascularity of which they are definitely walled off by sclerosis and repair. You are referred, in this connection, to the remarkable work of Sir Kenneth Goadby (9, 10, 11), on latent infections in war wounds. Bacteria occur, isolated, in the solid matrix of sclerosed or repaired bone; and should rarely be disturbed, except when surrounded by, or enclosing, infected granulations, as in apical condensing osteitis or "loculi," constituting a real bone pathology.

Classes III and IV—(III) Abraded or eroded teeth occasional pulpal symptoms; (IV) average teeth: pulps diseased

It is in Classes III and IV (abraded, eroded and senile, coronally sound teeth: all conditions free from caries, infection, and gingival lesions, except clean gingival atrophy, which is not infective), that a most remarkable demonstration of the possibilities of the pulp as a reparative organ occurs. This is true up to a certain plane, after which, a gradual failure of function begins, the pulp displaying a singular inability to complete repair, so well begun, which results in complete degeneration.

This we recognize as inevitable, to be expected of this type of tissue, because of its peculiar vascularity, and its confined position and location as an end organ; but we must remember, according to Hopewell-Smith (21), "that nearly every degree of dentinal change is attended with hyperaemia and cell proliferation in the pulp tissue, and, generally speaking, formation of adventitious dentin." By tubular calcification and granular hyalin formation, the normal pulp responds successfully to erosive and abrasive dentinal invasion, supplementing this resistance against chemical and mechanical irritants by deposits of adventitious dentin on the approximating pulpal walls, or by pulp-nodule formation interstitially. This is typical normal defense, similar to that elicited in dental caries, but free from infection.

Such irritation in "one pulp may produce similar reactions in others of the

same denture; pulp nodules are found at all ages and stages of all pulp disease" (26). Hence, such evidences of irritation are not definitely symptomatic. Degeneration may result at any time; and, except by referred pain or the radiograph, which are not definite, or by exploration and pulp removal, both destructive, we have no sure diagnostic methods, for thermal and faradic electric tests are misleading. Eventually, a series of symptoms of subapical disease arise in the periodontium, but these are periapical not pulpal.

With the advent of the gold inlay, applied to replace gold shell-crowns covering sound teeth ground down years ago, we have been given proof of Broomell's contention (5) that tubular calcification and much adventitious dentin result possibly from dentinal irritation from grinding, exposure to thermal change, or cements. In recent cavity preparations, areas of sensitive dentin are found in such teeth; the pulps are frequently degenerate, but little pulpitis or apical disease is present, except in those previously infected thru deep carious cavities. Many such have been restored to full function by M. O. D. inlays. The shell-crown should, therefore, be condemned; if need be, not for the pulpitis it induces but for the gingival disease resulting from its careless use.

Arteriosclerosis is associated with pulp degeneration; but diseased pulp vessels are commonly sclerosed, without systemic hypertension symptoms. Advancing age is a real factor, these conditions being rare in the young. We know that senility induces, in sound teeth, fatty or mucoid degeneration of odontoblasts, the entire pulp becoming fibrotic, sclerosed, or partially or completely calcified. Any of these conditions may end in gangrene, infectious or otherwise. But we know no better way of determining the progress of this course of events, inside the tooth, save by appearance, than we have in the patient from his history and facies.

It has been the practice not only to blame pulp degeneration for causing periapical disease, but also to remove such pulps in the hope of preventing the putrefactive (not infective) products of degeneration from causing such disease. It has also been the fashion to speak of hematogenous pulp infection in these groups, and also in coronally sound teeth with the pulps non-vital from trauma. In the light of quoted research, neither practice nor theory in these respects is tenable. It is more than likely that nearly all periapical disease, except that produced by the dentist chemically—by radical canal medication—results from the arrival in the pulp of a real infection via the dentinal tubuli or by pulp exposure, or via the gingival lymph vessels, producing chronic pulpitis, the toxic products of which in turn produce periapical infection. And it seems that apical disease is just as rarely produced by chemical products of pulp degeneration, minus infection, as is pulpal infection, in sound teeth, the result of hematogenous convey.

The pulps of coronally sound teeth, which are necrosed or gangrenous, present necrotic masses entirely devoid of normal tissue-elements, and may become infected, involving the apex at anv time, with no pulpal but many peridontal symptoms. This process is slow, however, and such gangrene is often walled off, even in the dental pulp, according to Witzel and Arkövy, by a "line of demarcation" of granulation tissue, the subgingival remainder being in a passive (venous) hyperemic state, when vascular stasis usually results, with less liability of toxicity appearing beyond the foramina before the pulp can be removed.

Until better diagnostic methods are devised, we deduce the following:

Conclusion 4. Unless pulp symptoms or exposure arise in eroded, abraded or

senile teeth, it is meddlesome practice to risk pulp devitalization and defective canal filling, just because pulps *might* degenerate, the exception to this rule being pyorrheal teeth, which might be infected by the gingival pathway.

In all stages of degeneration, except the calcific, when the pulp is finally infected by opening the pulp cavity or otherwise, periapical disease is both rapid and virulent. This is to be expected and illustrates a lack of the usual resistance, resident in healthy dentin and pulp; for pulp cells are fibrotic, fatty or gangrenous, as are the fibrillae. The entire tooth is thus a perfect culture tube.

Conclusion 5. Cases in Class IV (fresh surgical exposure of symptomless pulps, made thru hard dentin, even at the risk of its being infected) are hopeful. Capping should be attempted, with frequent clinical and radiographic survey; but the slightest periodontal symptoms demand immediate pulp removal. This is especially true of young unfinished apices, which should be considered with a full understanding of the results of the recent research on apical cementum by Box (3).

No pulp exposure with the least symptoms of any disease, except calcific degeneration, should be capped, for periapical infection is inevitable, insidous, and symptomless, hence dangerous.

Class V—Carious teeth; pulps infected

Class V (carious teeth, infected pulps, but subgingival dentin not completely infected, with the apices free from disease and vital) is most interesting, because it is in this group that the profession, in the past, has done its best work.

In spite of infected and defective canal work and so-called "useless medication" (before the advent of the radiograph), it can be shown that a large percentage of such teeth were preserved, and are harmless and functional today, notwithstanding the fact that the pulpitides of this group are the principal portals for the production of periapical disease, and that pulp diagnosis was crude and inaccurate. The errors of the past lay not alone in septic canal work, nor in empirical treatment, but in the empirical dictum that every tooth must be saved at all hazards, and also in failures of both treatment and judgment in the selection of teeth for treatment, no distinction having been made between those of Class V, with vital apices, and of Class VI, which were hopelessly abscessed.

It is always instructive, even if humiliating, to review past failures. In the following statistics, instances of periapical disease, diagnosed clinically and by radiograph, are rated as failures, a percentage of which no doubt occurred prior to treatment and should appear under apically diseased teeth in Class VI. The number of rarefactions, mistaken for real bone lesions, would likewise greatly lower these ratios in favor of the apically well-filled pulpless tooth.

In all data submitted, the radiograph and its interpretation is a determining factor. While open to criticism, which will be discussed later, the radiograph, within clinical limitations, is the means by which the extensive occurrence of such lesions was first definitely discovered, not only about teeth, but in many other organs. It is still accepted as a most valuable diagnostic adjunct by the surgeon, so this is hardly the time for dentists to quibble over it.

Quoting from a previous compilation (16) of the results of the work of Callahan, Grove, Hyatt, and the writer, we find that only 7 per cent of root apices were ever perfectly filled and 53 per cent of these were periapically diseased. But of this 7 per cent, only 2 per cent showed filling material in the accessory foramina. Yet we know that these, as well as apical deflections, occur in at least 40 per cent of all teeth.

Hence, in 38 per cent of "perfectly" (?) filled apices, the accessory foramina were not filled by filling material, but by pulp-shreds and vessels, vital or non-vital. Further, available filling materials (gutta-percha usually) contract at body-temperature at least one-fourth in bulk (28), allowing seepage of exudates and bacteria. Thus very few apices, in all their ramifications, have ever been filled.

In the same report, consider the data for poorly filled roots (fillings ending short of the apical third): 43 per cent of these occurred, but only 57 per cent were periapically diseased; and these, like the perfectly filled, may have been diseased before filling. Thus, we find approximately one-half of both perfectly and imperfectly filled roots periapically diseased.

The second compilation is from Arthur Black's (2) "summary of abscesses related to root fillings," a report on six thousand dental radiographs of six hundred unselected adults. It is more fairly divided into percentages of small and large roots. It defines a good root filling as one appearing well into the apex, with no open canal beyond it; a bad root filling as not reaching the apex, projecting beyond it, or permitting the existence of a definitely open canal beyond the filling. In this report, 45 per cent of all filled roots were periapically diseased but only 9 per cent of these occurred about good root fillings, while 63 per cent involved poorly filled apices.

All of this is most encouraging to those who would save and not extract the teeth of Class V. The 50 per cent of the first report and the 45 per cent of the second report, average 47.5 per cent of all filled roots as apically diseased, leaving a balance of 52.5 per cent not visibly diseased.

What of this 52.5 per cent? If it does not represent a composite of the patient's general resistance, and a local resistance existing in and about the tooth, plus average dental skill and effort in the past, what does it indicate? Who, and what, retained these teeth to function to the patient's great comfort, if the conservative dentist did not? Surely not the "100 per cent vitality men," both doctors and dentists, who extract all filled pulpless teeth. We are not disputing the fact that the remaining 47.5 per cent, apically diseased, were a menace to the patient's maxillae and adjacent teeth, if not to his general health, for undoubtedly they were and should have been removed immediately. But, in taking thought, it is good to realize that, by modern diagnostic, clinical, and surgical methods, even a much larger percentage of the teeth in Class V may be prevented from drifting into Class VI.

Any attempt to explain this 52.5 per cent of pulpless teeth, that apparently were not periapically diseased, leads immediately into intricate questions of pulp pathology and symptomatology that can be discussed only clinically at this time.

It is recognized that various stages of pulpitis merge into each other more or less gradually and that these pathological phases may be fairly accurately defined in the laboratory, with a pulp slide in hand; but no associated definite set of symptoms differentiate such conditions in the pulp in situ. "Relative response to thermal change, dentin appearance and sensation, indefinite thermal and electric tests, or pain principally, which is rarely localized, appear to be our all, symptomatically, as indicated by the pulp. . . . . When sepsis passes out the apex, the damage we would avert has been done, and on outspoken group of symptoms present, but these are periapical, not pulpal. . . . . This is well illustrated by the treatment for pulpitis, which, notwithstanding a voluminous pathology, with few exceptions, is still pulp destruction" (16). If pulp diagnosis were accurate, periapical disease could be prevented, but with our present knowledge, "the treatment, pulp removal, unfortunately anticipates and is the pulp diagnosis" (16).

With this understanding, the prevention of periapical infection, by assisting normal tissue resistance and causing delay in extension of infection to the apex, by prompt pulp surgery and proper treatment, becomes a matter of the greatest importance. The first line of dentinal defense has been explained; it is frequently adequate, otherwise all carious and filled teeth would contain septic pulps, since caries is partially a septic process. The second occurs interstitially after actual pulp invasion. This resistance is best described by Hopewell-Smith (21), who says: "It is not at all surprising that this is a structure, which possesses great recuperative power, and is constantly exercising its functions in this respect, and undergoing repair; hence, any invasion by disease or the occurrence of an accident is succeeded by a resistance, which, in many cases, is highly satisfactory."

The chances for apical infection can best be grasped, by a comprehension of Prinz's (29) classification of pulpitides into (a) those which are *rapid*—the acute, destructive types, i.e., acute, simple, and purulent pulpitis; and (b) those which are *slow*—the chronic, productive types, i.e., chronic ulcerative and hypertrophic pulpitis. In any clinical discussion of pulpitides, Noyes' remark in this relation must always be kept in mind. He says (26): "There is the greatest difference in the rapidity with which these stages follow each other and the extent to which the inflammation spreads thru the tissue before the breaking down begins. This is probably due to the character of the invading organism and the resistance of the individual."

In acute destructive types, the entire pulp may rapidly become purulent or liquefied, odontoblasts and fibrils may become necrotic, and complete dentinal and apico-cemental infection occur, when acute or chronic apical diseases quickly follow. Or, thrombosis and infarct may induce gangrene, which has been described as slowly involving the apex; or both phases may be subacute, and result in chronic ulcerative or hyperplastic stages. But, as a rule, acute pulpitis is dangerous, producing apical disease before the pulp can be removed. In chronic productive types, the situation is much more hopeful, and dentinal and pulpal resistance more active; for the odontoblasts and fibrils are not quickly liquefied and protect the subgingival dentin for some time. Coronally, infection may progress between the odontoblasts and pulpal walls, but subgingivally and apically this layer remains well attached and more or less vital.

Below the gingivae, infection primarily follows a central course along the great vessels, attacking the pulpal-wall tissue later. Occasionally, the perivascular lymphatics convey bacteria and toxins thru the foramina, producing periapical disease, while the pulp is still sensitive and vascular, and the apex vital—a very dangerous condition. Usually, however, there is much delay, and time enough to prevent apical disease by pulp removal. Particularly is this true of hyperplastic types, where a productive cell reaction, with much granulation tissue often covered by epithelium (as in pyorrhea), is associated with pulpal tumor. Resistance to infection is high in, and infection is slow in passing thru, foramina; in these, prompt clean canal surgery should leave an uninfected vital apex and functional tooth.

The following deductions are, therefore, justified, from the standpoint of histopathology, as relating to conditions that guard the apex and prevent or delay its infection.

(A) In chronic pulpitis, and certain stages of pulp degeneration and gangrene, complete destruction of the subgingival odontoblasts and fibrillae is usually a slow process. Persistence of this layer of cells, vitally attached to pulpal walls, has been noted by many histologists, for all pulp diseases. It

guards the apical third of the dentin, possibly transmitting sensation, which often persists, indicating a vital apex. Such would not be the case, if this layer was necrotic or liquefied.

(B) Pulpal infection is generally localized centrally. Excepting the initial lesion, the carious exposure, it follows the large vessels and perivascular lymphatics centrally thru the pulp, leaving tissues adjacent to dentin and cementum intact for some time. Indeed, this pathway is more accessible for the production of periapical disease than of general infection of the apex.

(C) The presence of nerves, under these pulp conditions, sufficiently nourished and functional to record pain, denotes a persistent vascularity. The common resistance to arsenic, cocain, and other medicaments, offered by many stages of chronic ulcerating and granulating pulpitides, is hopeful, and indicates activity of nature's usual granulation methods, protecting the apex and contained vessels against infection.

(D) The results of recent research, by Box (3), conclusively demonstrate a complete circulation of tissue fluids, thru the cemental apex from the periodontium via the lacunae and canaliculi, the granular layer of Tomes and dentinal fibrillae, to the apical third of the dental pulp. This condition emphasizes the source from which the cemental matrix of a vital apex, and possibly apical pulp-shreds and vessels, are nourished; for they are thereby in touch with serologic factors, which not only fight infection, but prevent it. It is a most hopeful feature; but, when once this labyrinth is infected, these lines of communication become pathways by which sepsis passes into the apical periodontium, destroying and detaching it, and creating a thoroly septic, necrotic, apex and periapical disease.

(E) There is the further fact that this means of communication cannot be demonstrated in middle-third cementum (3), the first circumferential lamella (lying next to the granular layer of dentin) of which is unorganized, with no connecting canaliculi crossing it from pulp to periodontium. This is, indeed, a protective feature, for, if infection can be prevented from reaching the apical dentocemental communication, a definite barrier exists against it in the middle and gingival thirds. If this were not true, alveolar abscesses would commonly occur beside the root on the middle third, as pararadicular, instead of periapically on the apical third.

What part had infected-canal methods of the past in preventing periapical disease in the 52.5 per cent of teeth that were free from it? And what may we expect of future canal surgery? It has been demonstrated by Brooks and Price (4) that dentin and cementum of experimental "dead" teeth in the laboratory, once thoroly saturated with a known and virulent infection, cannot be sterilized, except by agents destructive of normal tissues. This is even more true of a tooth in situ, many statements to the contrary notwithstanding. If dentinal and apical cementum cannot be sterilized, and canal medication was worse than useless; if, as shown, very few apices have ever been filled, except by leaking fillings; then, all the canal work of this period was useless and all teeth so treated should be periapically diseased—yet this, happily, was not true in 52.5 per cent of the cases.

There is no doubt that the remaining 47.5 per cent were as thoroly saturated with infection as the "dead" test-teeth of Brooks and Price, and that all treatments in these denuded and infected apices, surrounded by infected granulations, were as useless then as we believe them to be now. There is much reason for believing that, in the other 52.5 per cent, a resistance inherent in the tissues, previously described, was so supported by canal medication and pulp-removal methods of the period, that (a) infection and putrefaction were delayed

in subgingival dentin and apical-third pulp-shreds, and that (b) complete apical infection was prevented by germicides placed directly into the pulp tissue, which is quite impossible in deeply infected dentin. Dentin was not sterilized, but apical dentin and cementum were not permitted to become deeply infected by the removal of these much medicated pulps. Infection was thus confined to the middle root-third, with its impermeable cemental laminæ, until the apex and vulnerable apical dento-cemental communication could be filled.

We believe that, by modern modifications of this method, in which greater speed and less radical medication are necessary, many more Class V cases may be saved from ultimate apical disease; hence:

Conclusion 6. For the teeth of Classes I, II, III, IV and V, diagnosed carefully and promptly: "If pulps can be removed under as nearly aseptic conditions as possible, by careful chemico-mechanical instrumentation, leaving only inaccessible vessels and vital pulp-shreds in the immediate foraminal openings of a vital apex, which has not been cauterized nor over sterilized; or, if these strong agents can be used without their passing out into the membrane (tho conservative operators prefer milder medicaments)—if all this can be accomplished without perforation and encapsulation, avoiding infection from debris, then any canal-filling method, as nearly aseptic as can be, that will close these openings well into, but not thru, the apex will insure apices infected as little, and filled as much, as possible and functional teeth" (16).

Any discussion of pulp removal raises the question of its apical boundaries. These are defined according to age. The young apex (papilla stage) presents one large trumpet-shaped foramen, with few cemental laminæ to or thru which the pulp extends. It is next to impossible to remove such pulps or fill the foramen without producing periapical disease. In the adult apex (pulpal stage), which is rounded or pointed, and built entirely of cementum about the main and many accessory vessels, forming multiple foramina (40 per cent occur in all teeth), the pulp proper is only that tissue bounded by odontoblasts, as explained by Grove (17, 18), and the apex contains only vessels.

In all poorly filled, and 38 per cent of well filled, apices these foramina still remain unfilled, because no method, in or out of the mouth, is known for detecting their number or deflected course; and, even if detected, no efficient technic is known which will either safely remove the vessels or fill the foramina. So, the tissue generally removed, except in intentional perforation, which finds or makes, and fills, one opening, neglecting all others, is that bounded by odontoblasts.

"There is some evidence for believing that remaining vessels and apical pulp-shreds, lying in touch with surrounding vascularity, either become organized into fibrous tissue or foramina are closed by deposits of cementum or osteo-dentin" (16). This can occur only in a vital apex, not infected, nor saturated with chemicals, nor perforated and over filled; and only in one to which the periodontium is physiologically attached (27).

The vital apex (13, 14) is, thus, the crux of all canal operations. Its maintenance is worth any amount of time and effort. It cannot be encapsulated because periodontal fibers are everywhere attached to it. There is, therefore, no denudation or hypoplasia in which encapsulations may lie, unless they traumatically protrude into the membrane, granulomata, or cysts. Quite the reverse: the denuded apex, necrotic by whatever means, is not worth a moment's effort, no matter how medicated nor how well filled. One of the gravest mistakes of dentistry is the stubborn belief that correct root-canal filling will cure apical disease. The most perfect canal opera-

tion is never a curative, but only a preventive procedure. It will never cure, and often does not prevent periapical disease. The warning against overmedication too prolonged or too high in oxidizing power, should again be emphasized. "There is more occasion to fear chemical tissue-invasion, than infection, because bodily resistance is better prepared against infection than against chemical necrosis; particularly is this true of connective tissues" (13, 14, 15).

"If none of the known methods really fills apices, in all their ramifications, what has protected the subapical tissues and patient in the defective work of the past and present against infection, or reinfection? . . . . It is daily becoming more evident, to many investigators, that some process other than the dentist's processes, is the final equation in root canal treatment and filling. This factor which fills, and in the past always has filled, or repaired, foramina unfilled, in so-called perfectly filled roots (preventing infection in 52.5 per cent of imperfectly filled roots), is the complicated reparative process common to all body connective tissue, namely, leucocytic function (endothelial leucocytes, etc.) plus good fibrous tissue organized by fibroblasts, cementoblasts, etc. These investigators approve any method that improves apical technic, provided it does not interfere with this reparative function" (16).

All of the foregoing cannot be used by the operator as an excuse for negligent pulp removal, or in expectation that strong medication may be substituted for the finest canal technic—it should stimulate him to even greater effort, that repair may be induced free from infection and without radical medication.

There is an increasing group of radical operators who extract all pulpless teeth because they are infected. They should proceed to the ultimate, and remove all carious and pyorrheal teeth because, as we have shown, they also are infected, if not diseased. The mere presence of bacteria in a connective tissue is not disease, to which dignity it does not rise until certain well known local cell-reactions occur. How are we to distinguish as between the periapically diseased tooth, where this has resulted in the subapical tissues, in touch with the blood stream (Class VI), and the apically well filled pulpless tooth, where it has not occurred (Class V)? The answer lies in correct diagnosis of apical disease, defining the normality of the attaching tissues, namely, cementum, periodontal membrane and alveoli; all equally as important as, if not more than, the pulp.

The laminated cementum is built by and in the fibrous periodontium. Both are developed according to age and use. The laminae and fibers of both respond to every stress thruout life by repair, which is not uncommon even in controlled infection. These are no more important, attaching the tooth, than the dense lining of the alveoli known as the lamina dura, which receives the insertion of the osteal ends of these fibers. If the cells of these tissues lie in physiologic apical contact, attaching a well filled tooth from which the pulp has been removed, that tooth is healthy, no matter whether it be called a "non-vital," "pulpless," or "dead," tooth (26). When this contact is broken, periapical infection exists beyond the tooth and can best be diagnosed by radiographic study, modified by clinical symptoms. "Principal among the clinical bony land-marks stands the character and stability of that hard lining of each alveolar socket known as the lamina dura. . . . . Showing as a dense line surrounding each tooth, uniting with its neighbor to form the interseptal crests, supported by trabeculae, which are important, it defines circumferentially the normal width

of the periodontium. Its disappearance in areas, particularly in middle and apical-thirds, with thickening of the dark line representing the periodontium, indicates undue stress or proliferating periodontitis. It may be completely fused in this region by normal cemental repair, or the hyperplasia may progress to a hypercementosis. It is missing in cemental hypoplasia and denudation, or when the periodontium is destroyed in the midst of suppuration, granulomata, unorganized granulations or cysts. . . . . Lack of continuity in the lamina is undoubtedly a most valuable symptom of infection advancing beyond the tooth; and, clinically, if it be intact, the cementum should be considered healthy" (16).

"The radiograph taken and developed according to the latest technic by those familiar with facial and dental anatomy, in form generally of intra-oral films, sometimes extra-oral plates, is simply indispensable. . . . . Radiographs do not show infection, nor disease, of facial or mandibular bones and soft parts, any more than in other body tissues. They do show, by contrasting radiopacity and radiolucence in two dimensions, in all hard and many soft tissues (as no other method will), destructive, constructive and developmental variations, when compared with normal surrounding tissue (16). Serious pararadicular disease is occasionally masked by roots paralleled or superimposed on the film; but periapical rarefaction, occurring around the apex, is always visible and diagnostic of a dead apex; about which the subapical bone can never form, for it is negatively chemotactile. Bone repair and sclerosis may occasionally present as dense as cortical bone, but the trabeculae and partitions of the adjacent normal bone, to which these should be compared, are missing.

"Sequestra occasionally seem more radiopaque than normal bone and rarefactions may result systemically from osteoporosis, osteomalacia, pregnancy, or calcic waste; in fact, many unusual factors may, but rarely do, appear. . . . . Exceptional cases cannot be compared with the usual, and should not be quoted, disturbing confidence of honest operators seeking help, nor can they be used by dishonest operators seeking an excuse" (16).

There can be no better illustration of the diagnostic accuracy of the radiograph in the radiographic study of all kinds of bones than the striking regularity with which radiographic rarefaction defines apical disease as indeed a strictly periapical and not a pararadicular disease, involving middle-third periodontium. The radiograph demonstrates in thousands of films, the usual infectious portal thru the root-canal, via the apical dento-cemental communication of Box (3), and confirms the importance of this discovery in histopathology as the cause which limits all surrounding granulations, cysts, etc., strictly to this area. The constancy with which this occurs demonstrates the efficiency of the barrier offered against this (the usual) pathway by the impermeable first cemental lamina of the middle-third, with no communication between pulp and periodontium.

"If radiographs can be trusted in abnormal tissue study, they should be trusted to show normal tissue detail and repair; and if there be a defect, it is in showing too little rather than too much tissue involved. No better diagnostic means is at hand. The whole matter hinges on the clinical features, plus the operator's attitude in interpretation, which must never be attempted unless the patient's history and tissue are under direct survey" (16).

With the foregoing diognostic, operative, and reparative possibilities in mind, we deduce the following:

Conclusion 7. If pulp removal and canal filling can be accomplished according to the conditions specified in *Con-*

clusion 6; if the lamina dura is definite, and surrounding bone cancellations appear to be normal, as in adjacent vital teeth; if there are no clinical symptoms and the apex is not hypoplastic, but contains a filling well into, but not thru it, with no visible open canal beyond; then a pulpless tooth so situated should be retained, as it is functional and harmless.

Because of inoperable canals and multiple foramina, the superior molars and canines, and the inferior molars, are occasional exceptions to this rule, which should be kept under survey. If periodontal symptoms arise, the affected teeth should be promptly extracted.

It is true that a percentage of past canal-work, still remaining, is doubtful in quality and should be kept under observation; but there is no justification for suspecting the vital apex, filled by modern methods, except as noted.

It is distinctly not true that apically well-filled teeth of the past, showing no apical disease, are a menace, and should be extracted in the ill patient, or as a diagnostic experiment in one who is under observation, just because periapical disease, even if not demonstrable, *might* sometime result.

Occasional cases are reported of the occurrence of apical disease on well-filled teeth, by hematogenous convey from other focal areas or systemic disease. Similarly, it is said to arise from chronicity or lowered resistance. The writer has observed this frequently in poorly filled, previously diseased, apices, the chronic condition becoming acute; but never except as noted in a well-treated apex. Granted such a possibility, if well filled apices have not as yet been involved in a patient suffering from metastases, toxemia, or the reduced local and general resistance of chronic disease, the chances are that periapical disease will never so result; and such teeth should be retained, for no better test could be had of the apical repair and the success of the operation.

In the writer's experience and from a complete survey of the literature, the investing tissues about very few well filled apices, free from apical disease, have ever been cultured with positive results. Quite the reverse; thousands of laboratory studies prove that all poorly filled and apically diseased teeth are infected or surrounded by both infection and pathology. Hence:

Conclusion 8. General nutrition may be seriously disturbed, and great distress caused the patient, by indiscriminate removal of Class-V (apically well-filled) teeth, for diagnostic purpose or because infection is suspected. Such operations are frequently peremptorily ordered by medical men in their anxiety to cure disease and in ignorance of oral pathology. Obviously, such teeth are extracted by a dentist, who, if he does not protest or refuse, but operates following a fad or thru fear of, or to please, his medical confrere, undoubtedly damages the patient's health and lowers dignity of his profession.

Necessarily there must be a real local pathology in the attaching tissues, a definite disease focus in touch with vascularity, before a general or systemic pathology can result from it. The term "focus" is most happily defined as a "fire-place," and such must really exist before its bacterial content may be raised in virulence, or its cell reactions permit of toxicity and general focal infection. This can and does occur in the improperly treated open canal, where the entire tooth, as well as the surrounding tissue, may well be the "fire-place," but not in a well-filled apex, protected by normal periodontium.

Class VI—Infected teeth; septic pulps

There is little use in consuming time for a consideration of the teeth of Class VI (infected teeth, septic pulp, involved by periapical disease). They have been

much discussed in former papers (12, 13, 14, 15, 16) and are really not worth while, for the reason that no adequate treatment is known which will certainly cure the suppurative, granulating, or cystic, phases of apical disease, except surgery. This statement implies recourse to *(a) apiectomy,* which is rarely successful, because the apical section of dentin and cementum, left by the operation, is not sterile, and generally, if such tissues are sterile, apical disease does not exist, hence there is no need for apiectomy; or *(b) extraction,* which obviously destroys the tooth, but does not cure the bone lesions, except when followed by *(c) curettement,* the third and most important surgical expedient.

A cure of periapical disease may be defined as regeneration of the lamina dura and associated trabeculae, comparable to adjacent areas on vital teeth or other operated areas, following extraction and curettement. It should result promptly (in four months) and the new bone should be deposited to a line the width of other periodontal membranes, in the same mouth, and the tooth must be clinically correct. Such repair is indeed rare. It has been noted occasionally in acute cases, where subapical tissues were sharply infected by acute purulent pulpitis, and were as quickly relieved of exudates by prompt drainage and canal treatment. These cases reverse the rule, which is that of a primary chronic infection, usually followed by an acute secondary parulis or fistulous abscess, associated with a necrotic apex. Any treatment of this latter type seldom succeeds; subapical bone may appear, as if repaired, but soon breaks down, invading a larger area. Hence:

Conclusion 9. With the exceptions mentioned, all periapically diseased teeth, when clinically and radiographically definite (Class VI), should be promptly extracted and sockets curetted. If there be the slightest question, it is best to err radically, and remove a doubtful tooth, than to risk inevitable damage to adjacent teeth, attaching tissues, or the maxillae and possibly the patient's health.

Many medical men, principally pathologists, believe the oral focal infection idea to be much overdone. Others do not subscribe to it at all. Whatever the outcome of this disagreement, we conclude as follows:

Conclusion 10. Dentists generally must surgically remove all such lesions, regardless of whether they be infected or not, because a real chronic bone pathology exists about the tooth and its cell reactions often simulate malignancy—it eventually proliferates, undermining the denture. So, obviously, the dentist, quite as urgently as the physician, and certainly the patient, desire prompt action to prevent further bodily invasion.

The writer is just as confirmed a believer in the doctrine that apically diseased teeth (Class VI) are infective foci, which may cause systemic disease, as he is in the fact that pulpless, apically well-filled, apically healthy teeth (Class V) do not produce systemic disease and should be retained. The relation of these lesions, to the various systemic diseases with which they are associated, has been discussed in previous papers (12, 13, 15).

The assurance of those who so confidently state the modus, by which disturbing elements of oral foci create far distant systemic disease, is disconcerting to any close student of the local oral pathology; by metastases, toxic or allergic processes, these results are said to arise, with little actual proof.

From an entire pyorrhetic denture, undermined and swaying in purulence, to a single locked periapical area walled off by condensing osteitis or sclerosis, nature's best protective procedure, is a far cry in pathology; and yet we know the former may do less systemic harm than the latter; just as the hypertrophic

pus tonsil may not be a focal factor, while the apparently normal tonsil, with its virulent crypt, may be most active.

It is the writer's opinion that granulomata may exist, well walled off for years in the young, despite the large vascularity, with little systemic invasion, until some lowered resistance, toxemia, or other infection elsewhere intervenes. This was noted frequently by the writer in the otherwise healthy young men of the naval station during the war influenza pandemic.

From many specimens and slides studied under these conditions, it is evident that the fibrous and bony protective walls break down about the many vessels with invasion of the surrounding normal bone cancellations, by fan-shaped series of granulating infected peninsulas. The radiograph shows a diffuse, blurred area with a radiolucent centre, and it is this crypt wall, which should be thoroly explored and curetted, for here cultures may be obtained, while centrally, the granuloma is sterile; again you are referred to the confirmatory work of Goadby (9, 10, 11) in war bone wounds.

Occasionally there is a proven metastasis, as in embolic pneumonia or viridans septicemia, but these are rare, we are thankful to say.

More than likely, retention of cells, which are proliferating, in a closed area causes a pressure toxemia, following Charles Mayo's idea. This is conceivably a nonspecific protein sensitization, due to cell activity and waste; or again reinfection, from open septic apices, may produce an acute, or subacute, reaction in old areas constituting a direct specific infection.

The activity of other infective areas or invasions and allergic states, elsewhere in the body, as Duke (33, 34) suggests, constitutes a definite predisposing factor to fresh, or renewed activity of oral infection; the lowered resistance of age and chronicity; direct infection from tonsils, sinuses, or gall-bladder; toxicity from gasto-intestinal stasis and disease, all create a cycle, of which oral disease is an important segment. So complicated is this cycle, that the writer would hesitate in agreeing with Duke (34, 33) that even eighteen per cent of all disease, focally induced, arises from oral infection; for he believes the percentage of primary oral foci much lower; but all the more important, because no case can be considered clear until all mouth areas are blotted out. Quoting from a recent conservative statement by L. F. Barker (1), the following diseases resulting from focal infection, in which oral areas played an important part, may be named.

"Under the locomotor system—infectious arthritis, hypertrophic osteoarthritis, osteomyelitis and myositis and fibrositis. Under the respiratory system—antritis, laryngismus, embolic pneumonia, infected tonsils and pleuritis. Under the circulatory system—endocarditis, pericarditis, myocarditis, cardiac irregularity, arterial hypertension and atherosclerosis and agina pectoris. Under the blood and hematopoietic system—the secondary and primary anaemias, Hodgkins' disease, and general sepsis. Under the digestive system—achylia gastrica and the different forms of gastritis; the possible relationship to gastric and duodenal ulcer, in some cases; also hepatitis and pancreatitis. Under the urogenital system — nephritis, pyelitis, ureteritis, cystitis. Under the nervous system and the eye—peripheral neuritis and neuralgia, neurasthenic states and recurring iritis. Under the endocrine and metabolic systems, the possible relationships of oral sepsis to hyperthyroidism and to under-nutrition."

The writer, from experience in private and hospital practice, heartily endorses the foregoing classification. It might be interesting, in this connection, to state that the results reported by Dr. Barker in clearing up oral focal infection were obtained by radically removing all periapically diseased pulpless teeth (Class

VI) and curetting, when necessary, the adjacent diseased areas; and by leaving all apically well filled pulpless teeth (Class V) not so diseased, in the mouth; further, they have been so obtained in the last seven years, with as many, if not more, recoveries to the credit of this method than to "100% vitality" procedure.

But it must also be noted that all other focal areas, elsewhere in the body, were likewise radically blotted out. Microscopic oral infection was not removed, and other macroscopic bodily foci ignored. Thus, the focal "hobby" was not over-ridden.

The closure of oral infective portals, if attempted at all, should be uncompromising. Only by promptly ruling out all systemic foci will "the fight long continued against a minute but daily toxic invasion be won; and these principles should be applied equally to all who consult us, either sick or well" (15).

The dentist who, without medical assistance of the highest order, attempts to distinguish between the sick and well patient, ignores recent experience of the Medical Advisory Draft Boards, where it required ten specialists, of whom one was a dentist, to diagnose an ambulant man, to say nothing of a sick one.. The dentist presumes by a glance and a few routine questions to determine, instantly, matters which might keep hospital laboratories busy for days. We dabble superficially in internal medicine, when we do not know the actual etiology of or sure treatment for a single dental disease.

One says the complement-fixation test for a streptococcus is as definite as a Wassermann test, when we know it is not; and proceeds to treat apical disease, retaining teeth when it is negative, and extracting when it is positive. Others speak vaguely of dyscrasias, diatheses, and susceptibility to streptococcus. Again another assures us that, if the tonsils are normal, the kidneys or heart and all blood counts correct, we may, without looking further, experiment with septic apices. Many criticise the internist for ordering out abscessed teeth, yet few profess actually to cure them; and, withal, none admit that they are internists. The circle of modern group-medicine always centers in the judicial diagnostic-mind of the skilled internist, who surveys the reports of all associated specialists, writes the final diagnosis and dictates the treatment.

The position of modern dentistry in this combination is now assured and is most honorable. We, of all specialists, are deeply interested, and should be most active, in clearing up all the present oral sepsis; and we are doubly interested in its prevention for the future.

If an amount of energy similar to that which is now being expended by many dentists in heralding recovery from systemic disease caused by oral sepsis could be put into the oral hygienic effort or into further research into dental caries and pyorrhea, for instance, we might eventually prevent these diseases and then there would be little oral sepsis to treat. This would be a much more becoming and modest pose; and, furthermore, it would be attending to our own business—in our own field of pathology, of which we know so little.

Bibliography

1. Barker, L. F., "Oral Sepsis and Internal Medicine," *Jour. Dental Research,* II (1919), 43.

2. Black, A. D., "Preventive Treatment of Chronic Infection Involving the Alveolar Process," *Dental Summary,* XXXIX (1919), 721.

3. Box, Harold, "The Dento-cemental Junction," *Jour. Nat. Dental Assoc.* (in press).

4. Brooks, Matilda M., and Price, W. A., "The Relative Efficiency of Medicaments for Sterilization of Tooth Structure," *Jour. Nat. Dental Assoc.,* V. (1918), 273.

5. Broomell, I. N., "The Adventitious Effect of Large Masses of Gold in Contact with Tooth Tissues," *Dental Cosmos,* LII (1910), 389.

6. ———. "A Plea for the Removal of Healthy Rather Than Diseased Pulps," *Dental Items of Interest,* XXXIX (1917), 762.

7. DEWEY, KAETHE, AND NOYES, F. B., "A Study of the Lymphatic Vessels of the Dental Pulp," *Dental Cosmos,* LIX (1917), 436.

8. ———. "The Lymphatics of the Dental Region," *Jour. Amer. Med. Asso.,* LXXI (1918), 1179.

9. GOADBY, KENNETH W., *Mycology of the Mouth.* London: Longmans, Green & Co., 1903.

10. ———. "Bacterial Flora of War Wounds," *British Med. Jour.,* Part I (1918), 581.

11. ———. "Latent Infection in Healed Wounds," *Lancet,* Part I (1919), 879.

12. GRIEVES, C. J., "Systemic Pus Poisoning Associated with Diseased Dental Apical Regions," *Dental Items of Interest,* XXXIII (1911), 339.

13. ———. "Dental Periapical Infection the Cause of Systemic Diseases," *Dental Cosmos,* LVI (1914), 52.

14. ———. "The Relation of the Vitality of the Periapical Cementum and Ajacent Tissues, etc.," *ibid.,* LVII (1915), 1.

15. ———. "The Clinical Status of the Gingivo-peridental and Dento-alveolar Infective Focal Portals," *Jour. Nat. Dental Assoc.,* V (1918), 775.

16. ———, "A Review of the Clinical Data Relative to Efficiency of Various Root-Filling Methods," *ibid.,* VI (1919), 113.

17. GROVE, C. T., "The Biology of Multicanaliculated Roots," *Dental Cosmos,* LVIII (1916), 728.

18. ———. "Some Causes for Periapical Infections with Pathology," *Jour. Nat. Dental Assoc.,* VI (1919), 669.

19. HARTZELL, T. B., AND HENRICI, A. T., "Pathogenicity of Mouth Streptococci and Their Role in the Etiology of Dental Disease," *ibid.,* IV (1917), 477.

20. ———. "The Bacteriology of Vital Pulps," *Jour. Dental Research,* I (1919), 419.

21. HOPEWELL-SMITH, ARTHUR, *Normal and Pathological Histology of the Mouth* (Philadelphia: P. Blakiston's Son & Co., 1918), II, 174.

22. KLIGLER, I. J., AND GIES, WILLIAM J., "Chemical Studies of the Relations of Oral Micro-organisms to Dental Caries," *Jour. Allied Dental Soc.,* X (1915), 282, 445.

23. LATHAM, V. A., "Neoplasms of the Pulp," *Jour. Amer. Med. Assoc.,* XLIII (1904), 534.

24. ———. "Some Pathological Features of the Pulp," *ibid.,* XLVII (1906), 916.

25. MILLER, W. D., *Micro-organisms of the Human Mouth.* Philadelphia: S. S. White Dental Manufacturing Co., 1890.

26. NOYES, F. B., *Dental Histology and Embryology.* Philadelphia: Lea and Febiger, 1915.

27. NOYES, F. B., AND DEWEY, KAETHE, "A Study of the Pathology of the Peridental Membrane," *Jour. Nat. Nental Assoc.,* IV (1917), 375.

28. PRICE, W. A., "Report of Laboratory Investigations on the Physical Properties of Root-filling Materials, etc.," *ibid.,* V (1918), 1260.

29. PRINZ, HERMANN, "Diseases of the Dental Pulp," *Dental Cosmos,* LXI (1919), 382, 641, 718.

30. ROSENOW, E. C., "Studies in Elective Localization," *Jour. Dental Research,* I (1919), 205.

31. TALBOT, E. S., *Interstitial Gingivitis and Pyorrhea Alveolaris.* Philadelphia: S. S. White Dental Manufacturing Co., 1913.

32. WAUGH, L. M., "Deviation from the Normal in the Teeth and Their Supporting Structures," *Jour. Allied Dental Soc.,* XII (1917), 437.

33. DUKE AND DIVELEY, "Multiple Infection; A Study of the Relationship between Infections," *Dental Cosmos,* 1919.

34. DUKE, W. B., "Oral Sepsis in Its Relationship to Systemic Disease," *Jour. Amer. Med. Assoc.,* 1918.