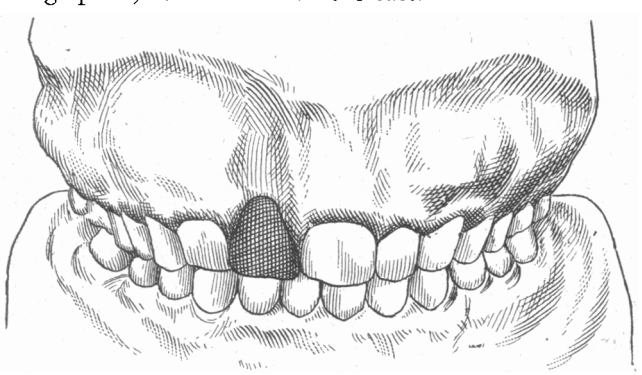
suppurative gingivitis as pyorrhea alveolaris. This I did because I could not find the egg-skin eschar, and when the blood was examined and seemed to substantiate my suspicions I refrained from giving specific treatment. To settle the question, I placed several patients suffering from suppurative gingivitis under specific treatment. This caused such unfavorable symptoms that I was soon forced to abandon it. In one case, where the alveolar process on the palatal surface of the teeth was nearly destroyed, and where it was practically in a normal condition on the buccal and labial surfaces, I was puzzled to know why this affection was not general. When septic pulps, salivary calculi and syphilis were excluded I concluded the trouble to be caused by the pressure from a vulcanite plate, to which was attached an artificial velum that had been worn twenty years.

In another case, the cast of which I have here, the disease was extreme in character. There was great destruction of the interdental process, accompanied by a discharge of pus. Many of the teeth could readily be forced by the finger 1/8 inch farther into the socket. A tumor, osseous in character, extended along nearly the entire length of the alveolar process on the buccal and labial surfaces of the upper alveolar ridge. There was, however, a break in the line of the tumor between the right central and right lateral incisor. The right central incisor had been extracted several years earlier. In this space was an artificial crown, attached to a small bridge-piece, as indicated on the cast.



The cast of the lower jaw showed by the hypertrophied condition of the gum, the extent of the pocket. At first this patient persistently denied ever having syphilis, but the evidence of it was proved by examination of the blood. After I had gained the confidence of the patient, however, he admitted that he had contracted the disease a dozen years before, but had been under treatment for it. He gave as a reason for denying the fact that he did not wish it known to any one except his physician, who had positively stated that he was absolutely cured. The patient now returned to this physician, told him my views as to the cause of the tumefaction, and telling him I said he was still suffering from the taint. The physician made light of the diagnosis, and persuaded the patient not to return to me.

I regret not having an opportunity to finish the treatment of this case, as it would have been an excellent support to my belief that this class of tumors is the result of this dreadful poison.

It is fair to state, however, that within a year the health of the patient so completely failed that he was advised to visit the hot springs for syphilitic treatment.

Where rheumatism is found to be present in a large percentage of cases, I believe it to be a coincidence, though not the cause. I believe that syphilis so reduces the resisting power of the constitution that rheumatism more easily steps in, much the same way that it may while the system is under any degenerating influence. I do not wish it understood that I believe pyorrhea alveolaris exists in every case of syphilis, nor that syphilis is found in every case of pyorrhea. But what I do believe is that some form of syphilis may exist in nearly all obstinate cases of pyorrhea alveolaris that can not otherwise be proved. As proof of the condition I mention, such cases do get well, and remain so when placed under specific treatment until all signs of syphilis cease to appear, not only outwardly, but when the blood fails to show any evidence of it whatever. The value of blood examination, which tells when to commence treatment and when to cease treatment, in this, as in some other diseases, is evident. I also regard it to be of great importance in diagnosing remote causes. Indeed, I predict that the time is not far off when examination of the fresh specimen of the blood will be the principal evidence in proper diagnosis. I have sometimes thought that locolosis or pyorrhea alveolaris may be caused by mercurial poison, but investigation does not bear out this surmise, for I have found this disease where there has been no history of mercury given.

Is it not, therefore, reasonable to conclude that this form of the disease is aggravated, if not caused, by tertiary syphilis?

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[Discussion of the Symposium of which this paper is a part will appear after the last paper, next week.]

NEUROTIC AFFECTIONS OF INTERSTITIAL GINGIVITIS.*

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The chief function of the nervous system, beside its special function, is that of regulating growth and repair. This function, as Marinesco points out, resides even in the neuron or nerve unit. While this function of regulating growth and repair is often connected with control of the vasomotor system, still, as Collins remarks, there are trophoneuroses in which there are no appreciable vasomotor disturbances, and there are any amount of vasomotor disturbances which are in no sense connected with disturbances of nutrition. The nerves regulating growth and repair are called trophic nerves, and the conditions produced by anomalies of their action are, as already stated, called trophoneuroses. It was in the domain of bone growth that anomalies of the function of the trophic nerves were first observed. Brown-Séquard pointed out certain anomalies in the joints of locomotor ataxics; later similar disturbances were observed in the jaws of patients with this disease. Another great neurosis, paretic dementia, presented similar trophic disturbances, as I pointed out twenty-two years ago.[1] Among these trophoneuroses was one characterized by looseness and falling out of the teeth, alveolar resorption, gingival ulceration and perforation, with, at times, maxillary necrosis. This condition had long been recognized by alienists and neurologists as causing that fall of the teeth which occurs in paretic dementia and locomotor ataxia. As E. S. Talbot[2] remarks, this function of the trophic nerves, however, received but little attention from dentists, albeit its influence has been recognized in dental pathology, in connection with the great neuroses in which gum disorder occurs, followed by loosening of the teeth.

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Cases illustrative of this condition have lately been described by Drs. Rawl, Baudet[3] and Chagnon.[4] The case of Dr. Chagnon was that of a man aged 34, who, about ten years previous to coming under his care, had contracted syphilis. Two years later he married and had healthy children. In June, 1895, he was admitted to an insane hospital under intense maniacal excitement, which subsided to give place to the usual symptoms of paretic dementia. The psychosis followed its course without any remarkable incident until about September, 1897. At this time Dr. Chagnon found that the two incisors, the canine, two premolars and the first molar of the left upper maxillary were very loose. The teeth on being picked out were absolutely sound. The ulceration which affected the surface of the alveoli following the loss of the teeth did not heal. About the middle of September a sequestrum, in which the work of alveolar resorption was not much advanced, became detached. The palate roof forming the anterior border of the maxillary sinus was part of the sequestrum. Two months later the ulceration had healed. In June, 1899, when Dr. Chagnon reported this case to the Quebec Medico-Psychologic Society, all the teeth in the lower jaw were sound. The two premolars and the right canine of the upper jaw were decayed. The second and third left molars as well as the first right molar were loose, but perfectly sound. There existed no alveolar pyorrhea; neither did any trace of ulceration appear, except a small opening which would not admit a probe.

Conditions like this may occur not only from constitutional neuroses, but from disturbances of the cranial and spinal nerves as well. They are frequently noticed after injuries to these nerves, but they may also occur as a consequence of the great functional neuroses like epilepsy, neurasthenia and hysteria. Their part in dental pathology is two-fold; they may cause an interstitial gingivitis, which pursues its course without bacterial infection, or they may so weaken the strength of the jaws and gums as to make these into an excellent culture-medium for pyogenic microbes. In dealing, therefore, with the question of treatment, the trophic factor should be taken into consideration, more especially as the structures involved, since they are of a transitory type, are peculiarly liable to its operation.

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INTERSTITIAL GINGIVITIS FROM INDIGESTION AUTOINTOXICATION.*

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In my work upon "Interstitial Gingivitis or So-called Pyorrhea Alveolaris," I divided the etiology into exciting and predisposing, and, again, into local and constitutional causes.

In my article published this year,[1] I discussed one form of the constitutional variety under the heading, "Interstitial Gingivitis Due to Autointoxication." This subject was discussed in a general way. Albu's classification of autointoxication was accepted:

1. Autointoxications from the suppression or disturbance of the functions of an organ, e. g., autointoxication of thyroid gland, pancreas, liver, suprarenal capsules, producing myxedema, diabetes, acute yellow atrophy and Addison's disease. 2. Autointoxications which occur from anomalies in general metabolism without definite localization, such as rheumatism, gout and oxaluria. 3. Autointoxications which are caused by the retention of the physiologic products of metabolism in different organs, such as poisoning due to extensive destruction of the skin by burning, carbolic-acid poisoning, uremia and eclampsia. 4. Autointoxications due to the overproduction of physiologic and pathologic products of the organism, such as ammoniemia, acetonuria, diaceturia, diabetic coma, etc. The most frequent source of this intoxication is the gastrointestinal tract. In this paper I propose to discuss the phase of autointoxication due to indigestion or disturbance of the gastrointestinal tract.

Autointoxication, like all intoxications, comprehends, as W. A. Evans has said: 1, production of the intoxicant; 2, absorption thereof; 3, reaction thereto. These three are embraced when autointoxication is spoken of, which is poisoning of an organism with matter produced by itself. Assimilation, or the making of tissue, is the passing of the simple into the complex, stability into instability, with the storing of the energy. This instability is a necessity of life. Disassimilation, divided into two divisions—death and energy—the last being a modification of death, is the passing of the complex to the simple; the unstable to the stable, with the liberation of the energy.

In the building-up process, the unused portions of the absorbed foods may produce autointoxication. In the breaking-up process, the ash can produce autointoxication. So long as these two processes, tissue-building and tissue-waste, are normal, intoxication can only ensue from faulty action of the destroying organs, of which the liver is the chief, or of the eliminating organs, of which the kidney is a type. This constitutes the first group, those due to faulty elimination. It applies to food-remnants and to tissue-waste, both normal and pathologic.

The second group is due to errors in cell-life. It occurs under three subtypes: 1, by some reason food elements are left unused; 2, the ash from food-burning is unusually toxic or unusually difficult to absorb; 3, the secretion of the cells is toxic.

While it may be stated in accordance with the principles just laid down that, considered from the direct standpoint of the production in the body, there can be no bacteriology of autointoxication; still it must be admitted that it produces culture-mediums in the body which would not otherwise exist and which enhance the virulency of the microbe, and hence increase the toxicity of its ptomain. Indirectly, therefore, autointoxication must be considered a factor in bacterial action. In dealing with the general question of autointoxication it should be remembered that when proteids are placed under the action of gastric and pancreatic juice they are changed into a hemi- and an anti-group. The anti-group is, as J. A. Wesener points out, broken down into antialbumose and a small quantity of antipeptone. This latter is a stable body which does not yield to the digestive juices or even to diluted sulphuric and hydrochloric acids. It is absorbed by the small intestine, but does not replace any waste of the used-up proteid of the body. Antialbumose is changed to serum albumin and is the one that furnished the body with its proteid food. The albumose, when injected subcutaneously,

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1 The International Dental Journal, February, 1900.