Cases illustrative of this condition have lately been described by Drs. Rawl, Baudet³ 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 parietic 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 transitional type, are peculiarly liable to its operation.

BIBLIOGRAPHY.

1. Journal of Nervous and Mental Diseases, 1878.
2. Interstitial Gingivitis.
3. La Presse Médicale, 1898.
4. American Journal of Insanity, October, 1899.

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

INTERSTITIAL GINGIVITIS FROM INDIGESTION AUTOINTOXICATION.*

EUGENE S. TALBOT, M. D., D. D. S.

FELLOW OF THE CHICAGO ACADEMY OF MEDICINE
CHICAGO.

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 disturb-

ance 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, acetoneuria, 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 virulence 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,

*Presented in a symposium on Interstitial Gingivitis, to the Section on Stomatology, at the Fifty-first Annual Meeting of the American Medical Association, held at Atlantic City, N. J., June 5-8, 1900.

¹ The International Dental Journal, February, 1900.

causes death; the blood fails to coagulate, by reason of the fact that the lime salts are precipitated by this body. If for any reason the epithelium of the intestine fails to perform its functions of changing this body into serum albumin, toxic symptoms will arise.

While uric acid is charged with being the chief factor in autointoxication, its importance has been overestimated. It is a thermometer of the extent of autointoxication rather than its chief factor. Recent investigations, as J. A. Wesener remarks, show that uric acid represents the metabolism of the nucleins of the body, and is in no way related to the albumins taken in as foods, for these last bodies of the group are very poisonous. The necrosis arises as follows: The leucocytes break down easily because the carbon dioxide remains too long in the tissue spaces; the nucleinic acid which is liberated in this way attacks the connective tissue, etc., irritates it, and this forms a good basis for destruction of nucleins.

A factor in autointoxication is non-performance of the process of elimination by the various excretory organs. In the urine alone, as Bouchard has shown, there are present each day, sufficient toxins in a normal individual to cause death if not excreted. This condition is notoriously increased after prolonged nervous explosions like those of epilepsy or hysteria. This was pointed out 30 years ago by Meynert, who showed that the status epilepticus or condition of repeated convulsions was due to accumulation of a proteid or nitrogenous body in the system. This status epilepticus is preceded by an increased amount. The same is true as to the influence of non-elimination from the excretory organs—the bowels, lungs and oral cavity—as well as the non-exercise of its poison-destroying powers by the liver. The non-elimination factor, moreover, interferes with ordinary digestive functions, and hence increases its own extent. The other factor in autointoxication is the production of toxic products in such quantity as to prevent their destruction by organs like the liver and consequent elimination, since a product to be properly eliminated must be reduced to a certain chemical type.

Autointoxication of the gastrointestinal tract takes place during any period in the life of the individual. Especially is this true in degenerates, in disease and in later pregnancy. In these states the eliminating organs do not perform their functions. The amount of bile excreted is an important factor in aiding digestion: 1, It is a solvent, a large amount being necessary; 2, it is an alkaline substance, producing alkalinity of food after it leaves the stomach, thus aiding the pancreatic and intestinal juices to neutralize the acidity of the chyme; 3, a purgative, its presence in the intestine stimulates the peristaltic action; 4, it is an antiseptic, thus preventing the growth of bacteria; 5, its alkalinity causes the emulsification of fats; 6, it assists absorption; Pavy's experiments have shown that moistening an animal membrane with bile accelerates osmosis; 7, when excreted in large quantities bile passes through the intestinal tract, coloring the feces.

The pancreatic juice is likewise an important factor in digestion. It is coagulated readily, being rich in albumin. Owing to its alkalinity, when brought in contact with chyme containing gastric juice, it neutralizes the latter's acidity. Kuhn, Chittenden and others have shown that pancreatin is made up of two or three ferments, each having an individual action: 1. It mixes with the bile, causing emulsification of fats; 2. trypsin, which is analogous to pepsin, acts in neutral or slightly acid solution; 3, amylase or diastase, which resembles

ptyalin, is more powerful than the other two, and converts the remaining starch into maltose. This, however, is aided by the previous action of ptyalin converting the starch into dextrin. It will be seen, therefore, that secretions of the liver and pancreas prepare the food for absorption after it has left the stomach.

Stockton, Elsmer, Boas, Ewald and others have shown that indigestion may be due to the following: 1. Disorders of the senses of smell and taste; 2, insufficient mastication; 3, alteration of saliva in quantity or quality; 4, absence or excess of hydrochloric acid, absence of pepsin, insufficient muscular power; excess of lactic acid in the stomach; 5, alteration in the biliary or pancreatic secretions; 6, excessive motility or torpidity of intestinal muscles, together with abnormal fermentative processes; 7, torpidity of the liver; 8, general poisoning, toxic influences; 9, altered blood-supply, in amount or quality; 10, nervous irritation, causing some of the symptoms of any of the above conditions. The influence of emotional states in the secreting process, and thereby directly on autointoxication, is illustrated in the fact long ago pointed out by Tuke, that pleasurable emotions increase the amount of gastric juice secreted, the opposite effects being produced by depressing passions.

As Bichat showed decades ago, sudden emotion may produce marked effects on the secretion of bile, and occasion jaundice. Cases are far from infrequent where emotions like jealousy produce mimicry of gall-stone colic in neuropathic individuals. Murchison, Christison and Thompson have traced attacks of biliary colic to jealousy. I have a number of patients in whom grief, worry from family or business troubles and overwork have caused such mental depression as to interfere with the excretory functions of the eliminating organs, and thus produce indigestion autointoxication.

Other liver changes from sudden nervous disturbances, whether of a mental type or not, are not rare. As mental impressions are communicated to the central nervous system purely through mechanical changes in the nerves, such influence must be purely material in operation. As the brain exercises a checking influence on the operations of the liver it is obvious that these mental influences can produce two effects: 1, The mental shock might increase the checking action of the central nervous system on the local ganglia of the liver; 2, the mental shock might destroy the checking action on the liver ganglia, and in consequence these may work too fast and exhaust themselves. Either of these two conditions would interfere with a poison-destroyed action of the liver, and accumulation of waste products would be the result. What is true of the liver is true of the other organs.

No one condition will so change and affect the secretions of the body as to produce autointoxication to such an extent as extremes in temperature. These sudden changes have been most marked in Chicago in the past winter. A change in temperature from 20 to 45 degrees in twelve to twenty-four hours was not uncommon. The result is that trophic changes and metabolic action were most marked. It was with difficulty that the excretory organs, especially the liver, pancreas, skin and kidneys, could adjust themselves in many cases to the freaks of temperature, as the result of which autointoxication was very noticeable, especially in those persons over 30 years of age and those who were suffering from previous indisposition. Sick headache, dizziness, indigestion, a sense of fullness, itching of the skin, eruptions, inflammation of the mucous membrane of the mouth and

gums, peeling of the fingers and odor in the axillæ and groins are quite common.

In healthy people a natural movement of the bowels every day may take place and yet decomposing food may be retained in the small intestine. It is not known that the bile prevents decomposition. If, as is frequently the case in degenerates, the liver and gall-bladder are not large enough, or, if in disease, a sufficient quantity of bile is not secreted, fermentation will take place. Again, one may eat more nitrogenous food than can be digested, or than can be taken care of by the bile, in which case decomposition will ensue. After one has obtained his growth this is not infrequently the condition. When fermentation and decomposition of the carbohydrates take place we have the various fatty acids produced. When the albuminous substances decompose, ammonia, sulphuretted hydrogen, indol and skatol, leucin and tyrosin are found. Aaron and others have demonstrated that ptomains and toxins are constantly found in the intestinal canal owing to bacterial action. In many people, fruits, cooked, as well as uncooked, will produce fermentation in the stomach and intestines, thus producing the most violent forms of autointoxication.

It is claimed that the symptoms of autointoxication are manifested, first, in the nervous system; next, in the circulatory; then in the tegumentary and, finally, in the kidneys. From my own studies, one of the first symptoms, I believe, is—especially in the young—sick headache. The relief which follows a thorough evacuation of the bowels is a familiar phenomenon. Some more marked psychic symptoms are depression of spirits, cranky notions, distaste for society and melancholia. In most cases of autointoxication or dyspepsia, as these cases are called, neurasthenia results. In the neurasthenic condition, the skin and mucous-membrane poisoning occurs. The most common of these is urticaria or hives, after eating lobsters and other shell-fish, or strawberries, or after the use of tobacco.

The mucous membrane throughout the alimentary canal, especially that of the mouth, is frequently involved. Changes in secretions, thickening of the mucous membrane, and, finally, inflammation of the gums and alveolar processes are noticed. I have called this interstitial gingivitis. So sensitive are the alveolar processes, gums and mucous membrane, owing to their transitory nature, that their inflammation and absorption might be considered one of the first, if not the first, symptom of autointoxication. Two predisposing factors assist greatly in influencing the disease: previous inflammation and age. When the gums have once become affected by drugs or other causes, indigestion, by producing autointoxication, will quickly set up irritation and inflammation.

People over 30 years of age, especially those of sedentary habits, do not require either an excessive nitrogenous or starchy diet. In such cases the food is not digested, and ptomain poisoning results. Autointoxication with gum and alveolar-process inflammation and absorption takes place.

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

South African Hospitals.—Evidence taken on the opening day of the Commission on the South African Hospitals pointed out the efficient method in which the army medical service was conducted. A varied array of witnesses from medical, military, and civil life were unanimously in favor of the completeness of the medical arrangements. A full report is given in the *British Med. Journal* of August 4.

THE THERAPEUTIC USES OF THE THYMUS GLAND.*

SOLOMON SOLIS-COHEN, M.D.

PHILADELPHIA.

Mr. President and Gentlemen:—It is always a pleasure to meet my colleagues of the New York County Medical Association. I only regret that what I have to offer for your consideration is not very satisfactory. If at the close of my remarks you shall think that they have been vague and inconclusive, even contradictory, I shall have left on your minds a truthful picture of the state of our knowledge concerning the subject on which you have honored me by asking me to address you.

It has long been known that children are liable to sudden death, attributed in some mysterious way to enlargement of the thymus gland. Authors have written learnedly on the subject, explaining, or rather failing to explain, these cases. In his classic study on diseases of the thymus gland, your honored fellow-townsmen, Dr. A. Jacobi,¹ points out how a slight increase in bulk of the tissues occupying the comparatively small space of the mediastinum may produce fatal compression of the trachea.² But why should the thymus suddenly enlarge to this fatal point? Some writers attribute the symptoms of thymic asthma to toxemia, whether from hyperthymization or hypothyminization. But, again, why this sudden change in the secretory activity of the gland?

Whether these cases are really examples of disease of the thymus or of an unfortunate effort of nature to overcome some other disease, I, at least, do not know. The function of the gland will have to be better known before we can decide; and until we know both the physiology and pathology of the organ better than we now do, we shall lack a sure basis for thymic therapy.

Clinically, too, we are confronted with the contradictory reports of equally competent observers upon the therapeutic action of the thymus gland in various cases of disease. One reason for this is to be found in the very varying nature of the preparations administered. In the beginning the fresh glands of recently slaughtered animals were used. Later, commercial preparations, desiccated powders, tablets, glycerin extracts and solid extracts have been employed.

I wrote to the leading manufacturers of America and England in order to get some idea of the manner in which the preparations on the market are made. Messrs. Burroughs & Wellcome, to my surprise, excused themselves from replying to my query. Messrs. Parke, Davis and Company said they used only the glands from sheep, usually young ones, such as the butchers sell for lamb. Messrs. Armour and Company said that the glands were taken from calves, 3 to 4 weeks old, after these animals had passed the government inspection. Neither of these houses mixes the glands of different kinds of animals. They both courteously and fully described their process of selection and desiccation. The Phospho-Albumen Company replied that they used the thymus gland from young lambs (young sheep) only, and described the process by which they make an extract instead of a desiccated preparation. These three American preparations, as we see, differ from each other either as to the source or method of preparation. If we could have the active agent or agents, our therapy would be more scientific and our observations more definite.

The dose of thymus preparations at present, is

*Address to the New York County Medical Association, May 21, 1900.