from the standpoint of the views defended in the present work. Thus, taking the influence of the thyroparathyroid mechanism upon oxidation and metabolism as factors of the problem, we have in hypothyroidia such mental states as melancholia, hypochondria, and apathetic delusional insanity, with perhaps ideas of suspicion. What mental disorders are witnessed in the more advanced stage of myxœdema are also commonly characterized as melancholia. So marked is the depressive state in these cases, in fact, that the late Lloyd Andreizen[2] was led to assert that various insanities having a constant and distinct physiognomy grew in the soil of acromegaly and myxœdema. "In the one case (myxœdema)," he wrote, "a morbid process starting from the thyroid gland affected the whole capacity of the blood in regard to its power of taking up oxygen from the air. On examining the blood with the mercurial pump, it was found that its oxygen and carbonic acid were much diminished, and, by placing the individual in the apparatus for examining the gases of respiration, it was found out that he took in but little oxygen and correspondingly gave out but little carbonic acid during life." That this sustains my own view that the thyroid, partly through its influence on the adrenals, influences tissue respiration is obvious. But this, in itself, sustains Andreizen in his conclusion that certain mental disorders grow in the soil of myxœdema. In other words, we should look upon hypothyroidia as one of the causes of depressive mental states.

This, however, fails to account for the opposite phenomena, *i.e.*, those we associate with erethism, mania, dementia, and the various manifestations in which irritability and excitement are prominent. But these signs are also explained by my conception of the functions of the thyroparathyroid apparatus, *i.e.*, that it acts as opsonin to insure the destruction of toxic wastes. Oxidation and metabolism, particularly its catabolic phase, we have seen, being impaired by the existing hypothyroidia, we have an accumulation of toxic wastes in the blood, and it is to the morbid influence of these poisons on the brain that all manifestations, in myxœdema, such as mental excitement, persistent hallucinations or delusions, and mania, must be attributed. Here, again, Andreizen struck the keynote of the problem, in my opinion, when, in his study of the relations of autotoxin in its bearing upon insanity, to which the previous quotation belongs, he referred to the presence with weakness, dullness, and subnormal temperature "of a tendency to the accumulation of incompletely oxidized bodies (fat, etc.)" in the tissues. Correct also, from my viewpoint, were the estimates of Bruce,[3] who, in reporting the results of thyroid treatment in 60 cases, attributed the improvement noted to the production, by the remedy, of a febrile state—quite in keeping with my own view (see page 628) that the thyroid and the adrenals are active in the genesis of fever.

[2] Andreizen: Quoted by Hamilton: Medical Record, April 29, 1899.

Such being the case, we can conceive how myxœdema and hypothyroidia can become the soil not only for depressive mental disorders, but also for those in which excitement and cellular erethism prevail. It follows also that hypothyroidia should be considered as a prominent factor in the genesis of insanity. Having in myxœdema undeniable proof of the influence of the thyroid apparatus on the mental equilibrium and a rational explanation of the pathogenic process, the physiology of these, particularly the important rôle in immunity, should no longer be neglected by psychiatrists. Laignel-Lavastine[4] closed an able study of this subject by the statement that "the existence of glandular disorders in psychical syndromes has not as yet, in my opinion, attracted sufficient attention. Nevertheless the existence of such disorders in certain cases is undeniable." Frankl Hochwart[4a] also lays stress upon this connection.

Recently, L. Vernon Briggs[5] wrote more pointedly, emphasizing both the rôle of autointoxication and the neglect of this important factor in mental disease: "In many large public and private institutions those in charge receive early cases of mental disturbance, and watch the toxæmia verge into mania, catatonia, and dementia; they notice the acetone breath and are aware of the true condition; but little if anything is done to stay the progress of the disease, and to save these individuals."

As to treatment of this class of psychoses, there is good ground for the belief that, intelligently employed in suitable cases, considerable help can be gained from thyroid preparations.

[3] Bruce: Journal of Mental Science, Oct., 1895.
[4] Laignel-Lavastine: La Presse médicale, Aug. 1, 1908.
[4a] Frankl Hochwart: Amer. Jour. of the Medical Sciences, Aug., 1913.
[5] L. Vernon Briggs: Lancet-Clinic, Jan. 28, 1911.

McLane Hamilton,[6] who, with Andreizen, attributes various forms of insanity associated with disease of the thyroid gland to autointoxication, refers to his own experience and to that of Babcock,[7] Clark, and others to the therapeutic value of these agents. In cases of stuporous melancholia, cerebral exhaustion, and chronic disturbed states of an asthenic type, there was a prompt lighting up of the mental condition, elevation of the temperature, and an increase of hæmoglobin in some instances of over 20 per cent. They were also found useful in chronic insanity with erotic delusions and psychoses in which the complete symptom-complex of catatonia was present, and also in cases of climacteric despondency. Easterbrook,[8] using thyroid in 100 cases which had proved intractable by other methods, obtained 12 per cent. recoveries. He found it more effective in women than in men, the best results being obtained in mental disorders connected with childbearing. Of 22 patients treated by Leeper,[9] 12 recovered; only 1 of these required readmission subsequently, all others being, as far as he could ascertain, permanently relieved. Conversely, A. W. Wilcox[10] did not obtain satisfactory results. His beginning daily dose was 15 grains (1 Gm.), increased daily by 15 grains (1 Gm.) until 60 grains (4 Gm.) were given daily.

Very valuable in this connection is a report by Mabon and Babcock,[11] based on the results of thyroid treatment in 1032 collected cases of insanity, in which the following conclusions are reached: 1. The dose of the extract depends entirely on the individual case. In some cases 25 grains (1.65 Gm.) three times a day will be necessary to bring about a circulatory or temperature reaction, while in others the same results may be had with the use of 5 grains (0.33 Gm.) *t. i. d.* Each case must be a law unto itself. 2. It is essential that the patient should be placed in bed to obtain the best results, and he should be continued there during the entire treatment and for a week following its discontinuance. 3. The treatment should be continued for at least thirty days. 4. We should not be discouraged by failure

[6] McLane Hamilton: Medical Record, April 29, 1899.
[7] Babcock: New York State Hospital's Bulletin, Jan., 1896.
[8] Easterbrook: Scottish Med. and Surg. Jour., Dec., 1900.
[9] Leeper: Medical Press and Circular, July 5, 1905.
[10] Wilcox: London Lancet, May 20, 1899.
[11] Mabon and Babcock: American Journal of Insanity, Oct., 1899.

in the first administration, but should resort to two, three, or more trials, if necessary. 5. The most gratifying results in thyroid treatment are to be obtained in cases of acute mania and melancholia with prolonged attacks, puerperal and climacteric insanities, stuporous states and primary dementia, particularly where these forms of mental alienation do not respond to the usual methods of treatment. 6. A high temperature reaction is not essential, as the average maximum temperature in the recovered cases among men was 99.6°. 7. Physical improvement is the outcome in most cases whether mental improvement takes place or not. 8. The proportion of individuals who recover under thyroid treatment and then relapse is less than the proportion that relapse after recovery from other methods of treatment. In a series of his cases only one patient who had recovered has relapsed.

I would urge in this connection that *small* doses of desiccated thyroid, 1 grain (0.066 Gm.), increased only if necessary to 2 grains (0.13 Gm.) at most, be used in these cases. Those who have criticised the use of thyroid in psychoses erred in this direction and brought on, by excessive doses, the very untoward phenomena which they deplore, by loading the blood with pathogenic wastes.

Hypodermoclysis or enteroclysis should also be used periodically to facilitate osmosis and hasten the elimination of all wastes through kidneys, intestines, and skin.

Again, it should be remembered that—at least according to my views—the use of thyroid gland enhances the vulnerability of the organic phosphorus to oxidation, and that, therefore, the consumption of this element is greatly augmented by this agent, as is well shown by the increased elimination of P_2O_5. This loss should be compensated for, particularly in depressive mental states (the brain-cells being rich in phosphorus), by administering lecithin, or glycerophosphate. Berkeley,[12] though unaware of my explanation of the influence of thyroid on tissue phosphorus, found lecithin very valuable in catatonia when given in conjunction with small doses of desiccated thyroid.

In dementia præcox we have the opposite condition, *i.e.*, hyperthyroidia as an underlying cause. As Berkeley states:

[12] Berkeley: *Ibid.*, Jan., 1909.

"Exophthalmic goiter cases and cases of catatonic dementia præcox have usually an enlargement of the thyroid gland, the bruit over the neck, the high tension and rather rapid pulse, accentuation of the second aortic sound, wide pupils, increase of the superficial and deep reflexes, and, lastly, a moderately small cell lymphocytosis."[13] The inference is obvious: excess of thyroid activity must be the underlying cause of dementia præcox. Thyroid and other organic agents proved harmful, but partial removal of the thyroid proved curative, as first shown by Berkley. Yet, as in other forms of insanity, some cases, especially those of the hebephrenic type, have been benefited by thyroid treatment, as observed by Levison,[13a] Davidson,[13b] and others. Dercum and Ellis[13c] found in eight autopsies degenerative changes in the thyroid and adrenals.

Epilepsy.—There are certain features of the pathogenesis of epilepsy which the average textbook author seems invariably to overlook. Besides the usual array of symptoms, diagnostic points, etc., the well-worn and recognized etiological factors; intestinal worms, indigestible foods, cicatrices, dentition, fright, masturbation, alcohol, lead poisoning, syphilis, uræmia, and that great basket of iniquities, heredity, are doing duty as of old, with tumors, sclerosis, and nuclear degeneration as local lesions. The physician is enjoined to remove the cause in all conditions in which this is at all possible, to look to depressants as mainstays—with the bromides and chloral in the lead—and to think well of asylums and retreats.

The features which seem to be overlooked are peculiar in the sense that, from the standpoint of practice, they exceed all others in importance. In fact, they constitute the *deus ex machina* of the fit, as it were. Literature shows plainly, for instance, that the dominant note in the pathogenesis of the convulsions is impairment of metabolism, and that the spasmogenic agent is some toxic agent in the blood-stream. Pathological variations of vasomotor action, due more or less to a morbid condition of the blood, have also asserted themselves so strikingly in the production of fits that some observers have been inclined to regard

[13] Berkley: *Loc. cit.*
[13a] Levison: Hospitalstidende, No. 36, 1909.
[13b] Davidson: Australasian Med. Gaz., April 20, 1911.
[13c] Dercum and Ellis: Jour. of Nerv. and Mental Dis., Feb., 1913.

them as the foundation of the whole symptom-complex. Again, destruction of the spasmogenic agent has been urged by some as the only reasonable principle of cure in opposition to the use of bromides and chloral, which tend to increase its formation in the blood-stream.

As to the presence of toxic wastes, so able an observer as L. Pierce Clark,[14] after a study of 150,000 seizures, concludes that "we must see the principle of pathogenesis in an initial toxin or autointoxication," *i.e.*, "an accumulation of waste products." The relationship between the latter and the production of epileptic seizures is further shown by the fact, emphasized by Van Gieson[15] and many others, that the attacks are more frequent when gastro-intestinal disorders, especially those due to overeating, gulping, and constipation, are present than at other times. Indeed, Herter[16] caused typical tonic and clonic convulsions and death in rabbits in forty-five minutes by injecting the defibrinated blood of an epileptic overfeeder, while blood from an ordinary patient did not produce such effects. Krainsky[17] also provoked characteristic seizures in rabbits in two or three minutes and several recurrences with blood-serum obtained by cupping from a case in status epilepticus. Ceni[18] found the blood of epileptics poisonous to man also. Thus, he ascertained experimenta'ly that "non-epileptic subjects react under injections of hypertoxic serum, and present intoxication phenomena that are analogous to those observed in epileptics, though less intense and unaccompanied with psychic disorders or epileptic attacks."

The effect of such poisons on the *vasomotor mechanism* is quite as evident. The conjunctiva and face of the epileptic from whom Herter obtained the hypertoxic serum referred to were congested. That this was due to the action of the poison in his blood is shown by many facts. "Certain drugs, notably absinthe," writes Schäfer,[19] "produce, when injected into the vascular system, convulsive attacks which are scarcely distinguishable from the epileptic fits provoked by stimulation of the cortex cerebri." Now, all such drugs cause a marked rise of the blood-

[14] L. Pierce Clark: Medical News, July 18, 1903.
[15] Van Gieson: Cited by House: Buffalo Medical Journal, June, 1898.
[16] Herter: Jour. of Nerv. and Ment. Dis., Feb., 1899.
[17] Krainsky: Wiener klin. Woch., Feb. 24, 1898.
[18] Ceni: Rivista Sperimentale di Freniatria, vol. xxxi, Fasc. ii, 1905.
[19] Schäfer: "T. B. of Physiol.," vol. ii, p. 721, 1900.

pressure. This may be shown by comparison with a few of the many other spasmogenic drugs. Thus, while absinthe was found to cause intense congestion of all organs examined by Pauly and Bonne,[20] Wood[21] states that "the full dose of strychnine produces a rise of the arterial pressure which is enormously increased during the convulsions." He refers also to the observation of Bezold and Bloebaum, "that when a small dose of atropine is injected into the carotid artery near the vasomotor centers" . . . "there is an instantaneous rise of blood-pressure"—"a great rise," he subsequently remarks. Cocaine, as shown by von Anrep, causes "convulsive movements of cerebral origin" which "are arrested by section of the spinal cord." Wood also says: "Certainly the evidence is overwhelming that cocaine directly increases the blood-pressure." The fits caused by this drug are precisely those of epilepsy; the syndrome is known, in fact, as "cocaine epilepsy."

In typical epilepsy the participation of the vasomotor mechanism is none the less marked. Spitzka, over thirty years ago (1881), attributed the epileptic seizures to the "explosive activity of an unduly irritable vasomotor center," and most neurologists regard epilepsy as "a functional vasomotor disease." The blood-pressure is not only high, as observed by François-Franck and Pitres, but the speed of the blood-stream in muscular vessels, according to Leonard Hill,[22] is from three to five times greater than usual—an index of the violence of the cortical circulation. Indeed, Weber[23] found vascular lesions and extravasations in the cortex and medulla of cases of status epilepticus, so great was the force which urged the blood into the capillaries. Onuf even found, besides the capillary changes, tortuosity and aneurismal dilatations. Victor Horsley[24] has emphasized the importance of congestion of the cortical mantle in the production of epileptic seizures. Ito[25] caused them in guinea-pigs by producing traumatic hyperæmia of the cortex.

We thus have, as cause of the convulsions, a toxic in the blood capable of producing a high vascular tension, and thereby

[20] Pauly and Bonne: Gaz. hebd. de méd. et de Chir., May 13, 1897.
[21] Wood: "Therapeutics, Its Principles and Practice," 13th ed., p. 217, 1906.
[22] Leonard Hill: Schäfer's "T. B. of Physiol.," vol. ii, p. 155, 1898.
[23] Weber: Wiener med. Woch., Bd. xlix, S. 158, 1899.
[24] Victor Horsley: British Medical Journal, April 2, 1892.
[25] Ito: Deut. Zeit. f. Chir., Aug., 1899.

excessive hyperæmia of the cortex. This hyperæmia is a recognized cause of epileptic seizures; in fact, as shown by Prus,[26] even electrical excitation of the cortex cannot provoke fits after it has been anæsthetized by a local application of cocaine. This is not intended to mean that a high vascular tension due to toxics of internal or external origin will produce epileptic seizures in every one and any one; were it so, the many disorders of the gout series would likewise provoke them. What I do mean is, that in all epileptics *the exciting factor of the seizures themselves,* and irrespective of the condition which renders the subject liable to them, *is a poison formed in the tissues or food residues, toxalbumins,* and that our chief aim, if we are ever to obtain mastery of the dread disease, should be to destroy these poisons and prevent their formation. Is it by saturating the system with bromides—which lower oxidation—that this can be accomplished? Such treatment ignores nature's danger-signals, and gives the spasmogenic poison free sway.

What are the weapons at our disposal that are capable of causing the destruction of these poisons and of preventing their formation?

Textbooks on the practice of medicine, including the "last editions," continue, regardless of the admonitions of men of large experience to the contrary, to advocate the use of bromides as a *curative* measure. Percy Bryant,[27] ten years ago, emphasized the fact that the bromides had added another disease in many epileptics, namely, bromism. Spratling,[28] as the result of a close study of several thousand cases at the Craig Colony, concluded that "we must not only regard the bromides as powerless to cure epilepsy," but also "as capable of doing as much harm as they do good as they are ordinarily administered." F. Peterson[29] has reported 11 cases in which the number of attacks was greatly reduced by withdrawal or marked reduction of the drug: "in some cases," says this neurologist, "the improvement is startling."

In the light of the evidence I have submitted in the fore-

[26] Prus: Wiener klin. Woch., Sept. 22, 1898.
[27] Percy Bryant: State Hospital Bulletin, Oct., 1896.
[28] Spratling: New York Medical Journal, Aug. 19, 1905.
[29] F. Peterson: *Ibid.*, Sept. 25, 1897; and Amer. Med., June 24, 1905.

going pages, the untoward effects of the bromides are readily accounted for. The seizures are due to the presence in the blood of toxic waste products which, by powerfully exciting the vasomotor center, cause an intense rise of vascular tension and violent hyperæmia of the cortex. The logical indication is to prevent the formation of these toxic wastes by measures that enhance the oxidation processes through which they are converted into harmless, readily eliminated end-products. Now, the bromides produce precisely the opposite effect: Wood[30] states that "even small doses of bromide are directly depressant to the circulation." Again: "In mammals the bromide acts very much as on frogs, inducing progressive paralysis, depression of temperature, and death by asphyxia when given in small poisonous doses." Asphyxia here clearly points to the drug as one that impairs oxidation, a fact emphasized by the lowered temperature. Chloral, also used considerably in epilepsy, is fully as harmful; Richardson, Hammerstein, and others have found that it could reduce the temperature very greatly—6° C. (10.8° F.), according to the last-named observer.

Of major importance in this connection is the fact that the bromides paralyze a mechanism which, from my viewpoint, is the patient's sheet-anchor, viz., the adrenal system. This action is sufficiently marked in some instances to cause bronzing similar to that observed in Addison's disease. Bourneville and Chapotin,[31] for instance, refer to Echeverria,[32] who witnessed a case in which the brow and neck "were markedly pigmented brown," and to others reported by Voisin; in one of these "the skin of the face was a dark, dirty yellow"; in another it was "covered with bronze patches having no connection with the acne." Inasmuch as it is the adrenal secretion which takes up the oxygen of the air to carry on oxidation (as the albuminous constituent of hæmoglobin) throughout the entire organism, the bromides—chloral as well—inhibit precisely the function which should be activated.

On the whole, it is by preventing the destruction of the toxic wastes which provoke the fits that these drugs are harmful, and it is obvious that their use, under the delusion that they are

[30] Wood: "Therapeutics," 13th ed., p. 244, 1906.
[31] Bourneville and Chapotin: Le progrès méd., Jan. 6, 1900.
[32] Echeverria: Philadelphia Medical Times, Nov. 23, 30; Dec. 7, 14, 1872.

curative, can only serve to perpetuate the disease unless used, as shown below, with the greatest discretion.

Another very important feature in the treatment of these cases, however, is the diet. In some of my cases this alone sufficed to reduce the number of paroxysms from several a day to one or two a week, and in one instance to cause their cessation as long as the patient abstained from the use of certain foods and beverages, including coffee and tea. The general principle involved in this connection is that we are dealing mainly with poisons formed during the breaking-down of worn-out living substance, *i.e.*, during catabolism, and that these poisons form owing to insufficiency of those constituents of the blood which carry on catabolism and destroy the toxic wastes. In the majority of cases the food-intake is excessive, and the cellular elements are burdened with detritus which cannot be completely converted into eliminable products. The blood, therefore, is loaded with substances which irritate the vasomotor center and thus provoke the seizures.

The practical lesson of this principle is obvious, namely, to allow the patient only the quantity of food strictly necessary to the needs of the body. Indeed, neurologists of wide experience have found that the best results are obtained when the food consists mainly of cereals, milk, fruits, and butter. Some include eggs, but others object to them. Meat, if allowed at all, should be strictly limited to the midday meal, and even then in small quantities. Fats, fried foods, and pastry often prove harmful. Stimulants which tend primarily to cause a rise of the blood-pressure, *i.e.*, alcohol, coffee, and tea, should not be allowed. Gastro-intestinal disorders sometimes suffice to awaken the disease. Appropriate treatment, including daily colon-flushing with normal saline solution, has proven curative in such cases. The patient should also be enjoined to drink water freely, in order to facilitate the elimination of wastes.

As to the medicinal treatment, the indications are remedies which, as I have pointed out elsewhere,[34] enhance oxidation, the beneficial effects of thyroid extract in tetany, tetanus, and puerperal eclampsia affording ample proof of their value. Bourne-

[34] Sajous: "Internal Secretions and the Principles of Medicine," p. 769, 1903; Monthly Cyclo. of Pract. Med., Jan., 1903; Jour. Amer. Med. Assoc., Feb. 4, 1905.

ville[35] found that thyroid extract failed to arrest the seizures, and even increased them. But the cause of this is self-evident: he gave full doses, and thus enhanced general metabolism so actively that he increased the production of wastes. Browning,[36] on the other hand, caused the cessation of convulsions in epileptics by giving ¾-grain (0.05 Gm.) doses twice daily. He states that these cases "are due to or associated with disturbances in the general metabolism of the body" and refers to them as "pseudo-epilepsy." All cases of idiopathic epilepsy should be considered in the same light if the term "pseudo-epilepsy" were warranted at all, since, as we have seen, Pierce Clark and others ascribe the disease to "an accumulation of waste-products." Browning's cases were *bona fide* cases of epilepsy, and what they do teach is that small doses of thyroid extract are alone indicated. These, however, as my own observations have shown, must be *very gradually* increased according to the effect on the patient. I usually begin with 1 grain (0.066 Gm.) at each meal, giving a small dose of sodium bromide on retiring at first, if necessary, and gradually reducing the dose until none is given at all. Some cases are benefited by larger doses of thyroid. Thus, in a case treated by a colleague, under my supervision, the nurse, through a misinterpretation of instructions, doubled the dose; the result was considerable improvement. Söchting[36a] obtained remarkable results also in a case in which hypothyroidia was evident—the underlying cause of cases cured with thyroid.

Osborne[37] also refers to a number of cases of epilepsy treated successfully with thyroid, several cases being in women at the time of the menopause and in young girls at the time of puberty. There being no assignable lesions for the epileptic attacks, "I reasoned," says this clinician, "that they must be purely toxic, and that this toxæmia was in excess periodically when the patient should have menstruated. Many of these patients had received bromides, and, while this postponed or controlled the epileptic attacks, general debility and bromism were appearing. In the young girls I used thyroid as an emmenagogue, with the belief that normal menstruation would prevent the attack of epilepsy. I found that it did. I then used the thyroid in the menopause

[35] Bourneville: Le progrès méd., vol. xxiv, p. 20, 1896.
[36] Browning: Jour. of Nerv. and Ment. Dis., Oct., 1902.
[36a] Söchting: Medizin. Klinik, May 1, 1910.
[37] Osborne: Jour. Amer. Med. Assoc., Nov. 3, 1906.

cases, in some continuously in small doses, and in others a few days a month at a possible menstrual epoch. I found that I could control and prevent the epileptic attacks as well with thyroid as could be done with bromides, and with much better results to the system." Similar cases have been reported by A. Gordon;[37a] Sicard[37b] found that bromides counteracted actively the effects of thyroid, while Manson[37c] noted the marked value of thyroid in the epilepsy of cretins—a suggestive relationship.

Briefly, the treatment of epilepsy should have as main object, as I pointed out in 1903,[38] *to activate the catabolism of spasmogenic wastes through the adrenal system* and to avoid, *as a cure,* drugs such as the bromides and chloral, which depress this function. In some instances, the iodides serve a better purpose; the salicylates (or salicin, 5 grains [0.33 Gm.] three times daily) are also effective, as Haig, Vincent,[39] and others have shown, but both these agents stimulate the adrenal center and enhance, therefore, the oxidation processes. Such remedies, to keep the blood free of toxic wastes; dietetic measures, to reduce the quantity of such wastes formed; and finally, the free use of water, to insure the rapid elimination with the urine, sweat, etc., of all physiological wastes, constitute a therapeutic triad which soon causes the vasomotor center to lose its irritability, thus preventing the cortical hyperæmia to which the convulsions are due. A small dose of strontium bromide at night, to reduce the hypersensitiveness of the vasomotor center, may be used, but if progress is made without it no bromide had better be given.

Isolated cases in which thyroid proved of value have been reported by a number of observers, who in most instances specify that the bromides had proven ineffectual. A series of 6 cases in which hypothyroidia was discernible were treated by A. Gordon.[40] In these cases a remarkable change took place, both in the general condition and in the frequency of the epileptic seizures. On the other hand, Pierce Clark[41] used thyroid in 5 cases, which failed to be "attended with very good results." Two

[37a] Gordon: Penna. Med. Jour., Feb., 1910.
[37b] Sicard: Jour. de méd. de Paris, Nov. 19, 1910.
[37c] Manson: Med. Record, Jan. 1, 1910.
[38] Sajous: *Ibid.*, p. 769, 1903; Monthly Cyclo., Jan., 1903; Jour. Amer. Med. Assoc., Feb. 4, 1905.
[39] Vincent: Journal of Mental Science, April, 1904.
[40] Gordon: Therapeutic Gazette, Dec., 1907.
[41] Clark: Medical Record, Oct. 24, 1896.