substance is disposed in the form of granules on the threads of the achromatic reticulum, and not in that of the larger aggregations which constitute the Nissl-bodies. It is on account of this disposition of these granules that his method, which does not alter their normal arrangement, reveals the achromatic reticulum both in the cell body and in the larger protoplasmic branches. It will be seen that this view of the arrangement of the chromatic substance of the protoplasm is very similar to that of van Gehuchten, who has maintained that the Nissl-bodies are essentially constituted by a granular incrustation upon the achromatic fibrils.

Changes in the Nerve-cells of the Cœliac and Mesenteric Ganglia during Digestion.—G. B. Pellizzi (*Annali di Freniatria*, 1898, f. 4) has made a series of observations upon the changes in the nerve-cells of the cœliac and superior mesenteric ganglia of the dog at different stages of digestion. He finds that the chromatic particles of the protoplasm become entirely or partially consumed with greater or less rapidity when these nerve-cells exhibit their functional activity, and that they are gradually formed again when this activity ceases. The process of reintegration is associated with certain changes in the nucleus. The results of this investigation probably constitute the most important evidence that has yet been obtained in confirmation of the much-disputed conclusion of Mann, formulated in 1894, that the chromatic substance is stored up in the protoplasm of the nerve-cell during rest, and consumed during functional activity.

ALCOHOLISM AND ALLIED NEUROSES.

By G. R. Wilson, M.D.

Three years' numbers of the *Journal of Inebriety* contain contributions which may well be repeated in summary form, and brought before readers of the Journal.

Alcoholic Neuroses.—Dr. Howard, Baltimore, contributes a short article on "Alcoholic Maniacal Epilepsy" (July, 1897). He properly emphasises the importance of *petit mal* in alcoholism. Distinction must be drawn between the "drunken stare" which persists during consciousness, and which may accompany conversation, and the "epileptic stare," which is sharp, sudden, with fixation of the oculi-motor organs, and which ceases with a return to consciousness. The minor attacks may last only a few seconds, may be accompanied by the sudden grasping of an object near at hand, but are compatible with the erect attitude and may pass unnoticed. Such attacks are common, and many of them may precede a violent epileptic *furor*—"a period of anger preceded by a calm attitude; then comes the sudden period of ferocity during which the deed is done; almost immediate subsidence of the furor, followed by partial or complete ignorance of the act."

"*Epilepsia Alkoholika*" (January, 1898, Dr. Stern, New York.)—As in Dr. Howard's paper, it is assumed that alcoholism "causes" epilepsy.

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That may be,—"(1) loss of consciousness with tonic-clonic convulsions of one or more muscles or of the whole body—*grand mal.* (2) Loss of consciousness without, or with only very slight convulsive movements—*petit mal.* (3) Certain phenomena acting as equivalents to the typical symptoms—epileptic equivalent." Such epilepsy is considered as—"(1) *Symptomatic epilepsy.*—(*a*) Epilepsy caused by anatomical changes—molecular epilepsy. (*b*) Epilepsy caused by toxic influences—toxic epilepsy. (2) *Idiopathic epilepsy*, epilepsy not traceable to anatomical sub-states of pathological changes or to toxic influences." This last is attributed to vaso-motor disturbance, an irritability of the vaso-motor centre in the medulla, and probably in the cortex cerebri. (But why not "molecular"?)

The interest of the paper attaches chiefly to its chemical considerations. Dr. Stern is convinced that epilepsy is most usually toxic. An interesting case quoted revealed repeatedly the presence of acetone and diacetic acid in the urine.

Dipsomania (Dr. Howard, Baltimore, July, 1898).—In this paper a much-needed discrimination in the use of the term dipsomania is insisted on. Dr. Howard's conception of dipsomania is very important.

In dipsomania there is no habitual drunkenness. Contrary to all habit and usual character, the patient drinks to great excess as the result of an attack of restlessness and specific craving. Such an attack usually lasts about three weeks. Subsidence is often sudden, and followed by considerable forgetfulness and "psychical contentment." The incidence of the attack is marked by insomnia, anorexia, and often vomiting if food is attempted, "precordial anxiety," great dread, and a hurried pulse. If secluded the patient may drink huge quantities of water, yet void only a little highly phosphatic urine. The throat is parched, the skin hot and dry; then comes the frenzied desire for alcohol. Convalescence may be much more prolonged and much less happy in patients who have been prevented taking alcohol. When the "nerve-storm is over" alcohol in any form is repugnant.

"Alcoholism never leads to true dipsomania, although alcoholism and pseudo-dipsomania are allied. . . . The pseudo-dipsomaniac is an intermittent drunkard. He will drink to excess whenever opportunity occurs."

Cases are cited, and the subject ably discussed, but even here is a great confusion as to terms. In this paper we are told that "the conditions existing in dipsomania are so different from those in inebriety." But in his former paper, following Norman Kerr's lead, he tells us "inebriety is . . . an intoxication mania of such furor, intensity, and force." Norman Kerr says "inebriety is an overpowering morbid impulse, crave, or craze, which tends to drive certain individuals to excess in intoxicants." Obviously in his later paper Dr. Howard means inebriety in its classical sense—a morbid alcoholic habit. That is a condition clearly to be distinguished from the epileptic onset of dipsomania.

Dr. Agnes Sparks, on *Alcoholism in Women*, October. 1898, gives expression to opinions which seem to the present writer very sound in a comparison of the disease in men and in women. Dr. Sparks finds that in women heredity is less important and somatic conditions more

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important, notably some kind of neurasthenia, and the distress incident to disorders and crises of the reproductive organs; that vicious fondness for alcohol is rare, that the disease is slower, that ovulation is not so commonly suspended as in morphinism, that dementia is rarer than in men, and periodicity more marked; that prognosis is better. The most valuable therapeutic agents in Dr. Sparks' repertoire are confidence, "gastro-alvine" remedies, abrupt withdrawal of alcohol, strychnine, arsenic, electricity, and hypnotism. Treatment must be very prolonged.

Dr. Herter contributes to the April number. 1897, a very interesting case of *acute alcoholic intoxication* in a child aged three years. The boy swallowed twelve ounces of whisky in the afternoon, fell to the floor, was stuporose for thirteen hours, and had a convulsion the following morning. Up to that time there was no paresis. The boy was admitted to hospital three weeks later, on December 13th. The following is a summary of the case:

December 13th.—Loss of sensation and of some power in arms; left leg spastic, the great toe nearly at right angles; right patellar reflex gone, the left slight; urination difficult; right kidney enlarged. 14th.—Vomiting; stupor; pulse 160, fairly good; slight vertical nystagmus; slight rigidity of neck; feet dropped; knee-jerks gone; convulsion, beginning in right arm and face. 15th.—Constant drowsiness; slight rigidity of right hand and forearm. 16th.—Three slight convulsions like the last; marked general rigidity, especially on left side; neck slightly stiff; swallowing difficult. 23rd.—Severe convulsion; left leg has become acutely flexed on thigh; right foot extremely extended; some fibrillary twitching of tongue, and tremor of left side of body; head rolling. 24th.—Tremor; conjugate deviation of eyes to the right; miliary eruption on skin. 25th.—Repeated convulsions. 26th.—*Tache cérébrale* pronounced. 28th.—Hypostatic pneumonia.

January 8th.—Stupor; right arm paralysed; right leg weak; left leg in extreme contracture; left hand in bird-claw position. 11th.—Eyes examined show no *optic neuritis*. 23rd.—Mental state improved; contractures continue; wasting muscles tender; head still rolling; complete consolidation of lower lobe of right lung. 30th.—Some purulent discharge from right ear.

February 3rd.—Condition bad; 4 lbs. 4 oz. weight lost; weakness and pallor; contractures and atrophy; no strabismus. 6th.—Left leg and thigh give no response to strong faradism, nor the abdominal muscles, nor the left biceps. Most of the other muscles react only slightly, the neck and the right interossei well. Under galvanism the C.C. contraction is greater in all muscles of extremities than the A.C.C.

March 2nd.—5 lbs. 12 oz. lost; contractions less; muscular power returning. 18th.—Speech returning.

April 6th.—Hands mobile; lower limbs mobile in bed. 28th.—Apparently entirely recovered.

There was irregular fever, not very high, from December 13th till February 7th, 1896. Dr. Herter, assuming a multiple neuritis, does not think it necessary to assume a meningitis to account for the cerebral symptoms, which he attributes to probable vascular and cellular lesions in the cortex. There is no mention of treatment.

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Dr. Kinney (January, 1896) reports a case in which there occurred some most interesting symptoms (first described, I think, by Magnan)—*pseudo-intoxication* in a man who had not been recently drinking. The case was distinctly alcoholic, but abstemious of late. On receipt of the news of a calamity he became drunk on emotion, and the interesting point is that he became not rowdy, but staggering and stammering, with a temporary sottish appearance. The condition lasted half an hour.

Another case, not of alcoholism, showed a prolonged trance-state with excitement and irritability, ending instantaneously and followed by complete forgetfulness of the excited period.

A third case is one of somnocyclism (cycling in sleep). The patient, asleep, sometimes in cycling costume, and sometimes in an undershirt or less, got up, mounted his wheel, and rode about town and in the country. He generally awoke from a fall. On one occasion it was at the foot of a hill, his head on the edge of a pond, and his wheel about thirty feet distant. Another night he found himself suspended by his shirt on a pear tree in his father's garden. It is not known whether he had descended thither from the roof or was trying to ascend. At other times he would go to his office and work. Once, having stuck over a balance in the afternoon, he found next morning that he had completed it, correcting an error previously not observed. There is no subsequent recollection of what has transpired. He has been observed in hospital to hold a conversation through an imaginary telephone, and, again, to sit atop a wardrobe with an umbrella up over him. Evidently this man's disease included a sense of humour. Later, while under treatment, his chair collapsed, and he fell backwards, striking his head. Thereupon he promptly fell asleep. During his convalescence he discovered that music, of which he had been very fond, had become impossible for him, inducing great distress. The patient's trouble began after excessive cycling in heat. Recovery was chiefly attributed to doses of *natrum muriaticum*, "which was given in the thirtieth potency every three hours."

The last case is one of abeyance of speech for seven weeks after a fall on the head, with survival of reading and writing, coherence, occasional violence, and much headache. In the midst of a severe headache, with face flushed and pupils dilated, speech suddenly returned. From that instant he was again consecutively conscious, but there remained the hiatus of seven weeks of which no recollection was possible.

The *fundus oculi in delirium tremens* is reported upon by Dr. Davis (April, 1896). Of sixteen cases eight were delirium tremens proper, the other eight "acute drunks." The paper is important because records of such examinations are rare. The result of it is to establish the prevalence of vascular changes, enlarged and tortuous vessels in the fundus, often with pulsation in the veins. Even when these facts are not noted congestion is common. Dr. Davis suggests the importance of these factors in the ætiology of visual hallucinations.

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