

stance is broken down and granular. Other cells show no nuclei or nuclei very irregular in shape. In other cells again the chromatic substance appears fused so that it forms an almost complete ring around the nucleus, in which case the cells are more or less irregularly contracted and the parts where the processes leave the cells are also shrunken. This change is suggestive of chronic cell degeneration. Some ganglion cells in which the cell body has disappeared, leaving a faint nucleus, suggest the acute degeneration described by Nissl.

It is very significant that the ethyl and methyl alcohols produce similar changes in chronic poisoning. I shall not venture to say whether these changes in the retina are primary or secondary to optic nerve lesions, though I incline to the former view. The question can not be answered until more extensive experiments which the writer has in contemplation are concluded. But it must be admitted that in producing these retinal changes by *chronic alcohol poisoning* we have made an important advance in the solution of the same chronic poisoning in man. I shall give a detailed report of these experiments elsewhere.

In concluding this paper I desire to point out the review given by Uththoff¹¹ at the last International Ophthalmological Congress in Paris in August, 1900, on "Toxic Neuritis." He divided this into two groups: first those toxic amblyopias that present a central scotoma and normal periphery of the field; these are the partial retinobulbar forms due to alcohol, tobacco, bisulphid of carbon, arsenic, iodoform, stramonium and hashish. The second group shows changes in the blood vessels with secondary ischemic necrosis, and in addition there is direct poisonous action on the nerve cell. He notes that the constriction of the vessel alone can not explain the affection, for ergot with its powerful constrictive action is not known to produce such destructive changes.

Uththoff is one of the most formidable champions of the neuritic theory, so far as the alcohol tobacco group is concerned. His arguments are based on the pathologic findings in his eleven cases of alcohol and tobacco blindness, in which the optic nerve was subjected to microscopic examination and in all of which, evidences of neuritis beside the atrophy of the nerve fibers were found. He therefore refuses to regard the condition as a simple degeneration.

Siegrist¹² has likewise recently written a strongly argumentative article opposing the view that the optic nerve lesions in alcohol and tobacco amblyopia are due to primary retinal lesion with ascending degeneration. It is difficult to harmonize the results of experimentation on animals and the pathologic findings of partial neuritic atrophy of the optic nerve in man.

At present the only possible way is to assume that, as Heilbronner, and recently, Nuel, have endeavored to prove, the neuritic changes are secondary to a primary degeneration of the nerve fiber. But it is wiser still to await the results of more extensive experimental and pathological research.

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ATROPHY OF THE RETINA.*

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It may be sometimes quite impossible to discover the cause of disease, and it is not always easy to determine whether a lesion is dependent upon some other morbid process, or of primary origin. I have hesitated to believe it possible for atrophy of the retina to come on as a primary condition, without any discoverable preceding inflammation, or even hyperplasia; but, it is certain that, whatever disturbances may have preceded the beginning of atrophic changes in the two cases herein reported, there was no manifest hyperemia before failure of sight. I am not sure that some occult inhibition of the functions of the vasomotor nerve force may not cause primary atrophy.

Miss M. B. J., aged 19, came to me July 12, 1882, with a note explaining that she had been unable to pursue her studies without headache. After suspending her accommodation, I found that, with +1/30 C. ax. 90° for each eye she saw 20/XX, Snellen. She returned home and got along satisfactorily with her studies until April, 1888, when I received a letter telling me she had suddenly experienced difficulty in reading at night, and could not distinguish the color of roses.

She returned to Louisville, when I found this difficulty had increased progressively until she was no longer able to read on cloudy days. Her fields were irregularly contracted, and she had no perception of either red or green. Her general health seemed to be perfectly good. The menstrual function was normal. Her sight was now reduced to 20/LXX, Snellen, in the right eye, and 20/C in the left. She remained with relatives in the city until June, when she was unable to see the test type, even at 10 feet with any glass. She could not differentiate colored lights. Ophthalmoscopic examination showed nothing definite. Her fields were extremely contracted, she had central scotomata in both eyes. She went with her grandmother to New England to spend the summer; and in August consulted Dr. Hasket Derby, of Boston, who found "the central vessels of the retina extremely small, with no abnormal appearances of the discs. She is unable to distinguish any test object."

The first week in September she returned to Louisville and I again mapped her field of perception. Both fields showed increased peripheral contraction, with central scotomata of irregular outline. She described everything as "smoky and blurred." In brilliant illumination, a book held before the face seemed "covered with a fine gray network." November, she was unable to distinguish any objects. On bright days everything

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seemed so dark and indistinct, she felt as if she were in a dense fog. Looking at any bright light, she was annoyed with a gray network before her. Artificial lights seemed smoky, and she said "it seemed covered with a dark mosquito net."

The ophthalmoscope showed at this time, faint grayish striations in the region of the macula, and near the larger vessels around the disc. The larger retinal vessels were scarcely visible beyond the discs, while the latter seemed perfectly normal.

She has remained in this condition until the present time. No other member of this young lady's family has suffered with blindness in any form, although a large number of her close family relations have various kinds of optical defects, for which I have prescribed; yet, none of them have retinal, or choroidal disease. There was no evidence of hereditary blood taint, and no sign of toxemia. I felt obliged to consider this a case of primary atrophy of the retina, advancing in both eyes to the extinction of useful vision, probably due to excessive exposure of the eyes to the bright sunlight, as she was accustomed to read in the sun.

Miss H., aged 17, of Frankfort, Ky., consulted me on Nov. 12, 1892. She had been having headaches, so severe as to interrupt her studies at school. With accommodation suspended, she saw with $+1/60$ C. $90^\circ = 20/XX$ in the right eye, and with $+1/48$ C. $90^\circ = 20/XX$ in the left. There were no ophthalmoscopic signs of disease. She returned home and resumed her studies. March 6, 1895, amblyopia had appeared to a degree sufficient to make reading impossible. Sight in the right eye = $6/XVIII$, and in the left = $6/IX$. I was astonished to find that now no glass improved the sight. Her fields of vision were irregularly contracted, with comet-shaped scotoma in the field of the right eye. The left was obscured by a veil-like mist with incomplete central scotoma. She had no color perception in either eye. Ophthalmoscopic appearances at this time were great narrowing of the retinal vessels, the larger ones only being easily traced. Light grayish striations in different portions of the field seemed to occupy the vicinity of the contracted vessels. May 17, 1895, the retinal vessels were empty, and not visible except in the discs, and faded away within two millimeters of the papillæ. She had always enjoyed good health, and there had been at no time any sign of inherited blood taint or toxemia. The discs were at each examination perfectly distinct in outline, and apparently normal. March 5, 1900, she came with her sister, and on careful examination I found it very difficult to trace even \dagger outlines of the larger vessels of the retina, even to the margins of the discs, while numerous pale grayish striations occupied the region of the macula, and, radiary streaks of gray following the direction of the closed vessels around the discs. The discs appeared entirely sound and normal. Both eyes appeared alike. The pupils were moderately dilated, and responded but slightly to concentrated light. She could see shadows of objects passed between the eyes and a strong light. Artificial lights appeared pale gray, and seemed covered with a heavy mist or fog. I could find nothing as to any probable cause of the atrophy in this case.

Drasche and Weinlechner Reach the Age Limit.—Professors R. von Drasche and Weinlechner took leave of their charge in the Vienna public hospital recently, as they had reached the age limit. The occasion was rendered memorable by tributes from their colleagues and pupils.

A CASE OF BLINDNESS FROM DRINKING BAY RUM, COMPARED WITH THE REPORTED CASES DUE TO METHYL ALCOHOL AND TO ESSENCE OF JAMAICA GINGER, ETC.*

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For some time the idea has been entertained that wood alcohol is the toxic agent in cases of amblyopia following the drinking of essence of Jamaica ginger and similar preparations. Dr. Hiram Woods thought of, but seems to have rejected the idea which Dr. Jackson more lately advocates.[†]

We will remember that reports of these cases began to appear nearly at the same time with reports of the cases due to wood alcohol. While bay rum and various essences have been long used as substitutes for whisky in prohibitory districts without accident, there have recently occurred several fatalities. I personally know this fact to apply in the Indian Territory, where the United States prohibition is very rigid. Within a year or two there have been several deaths in that country from drinking these things as well as wood alcohol. Ordinary wood alcohol has a characteristic odor and taste so disagreeable as to have formerly prohibited its use in pharmacy, but the more recent method of preparing it, under the name of "Columbian spirits," removes to a large degree its objectionable features, so that such use is possible. Its cheapness, too, recommends it as a substitute in part at least for ordinary alcohol, in such preparations as are sold at cheap price in country stores.

Moreover, it seems in some quarters to be considered non-toxic in this purified form. (Prof. J. H. Long, personal communication.) This is, however, refuted and shown to be dangerous by the reports of Gifford and of Patillo. Professor Long (same communication) states that it is rapidly coming into use in pharmacy, especially in making bay rum and kindred preparations. Dr. Harlan had a chemist demonstrate by analysis the presence of wood alcohol in the essences of ginger and peppermint drank by the patients whose cases he reports.

It is worthy of note that we know of no other substance which when swallowed selects for attack with such uniformity the optic nerve and retina. Those who record cases of blindness due to this cause mention in all thirty persons who drank from one to two drams to an ounce or more of the substance, and were made sick by it. Fifteen, or 50 per cent., lost their sight. One of the first cases on record was published by the present writer in 1899. Four preceded and others have followed, till now fifteen or more are described in literature. A dozen or more cases from essences also are reported, a sufficient number to afford reliable data for comparison. An analysis of fifteen cases of wood alcohol blindness, and of twelve cases of blindness due to the essence of Jamaica ginger, etc., proper references to which are given in the appended bibliography, shows the striking identity of important symptoms.

In each group, unless the dose was large enough to produce coma, no unusual symptoms presented on the first day. Usually on the second or third day, sometimes later,

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[†] Dr. Woods afterwards accepted this idea.