POISONING BY WOOD ALCOHOL.

A CASE OF COMPLETE BLINDNESS (TRANSITORY), WITH RECOVERY OF VISION.

By CARL KOLLER, M.D.
NEW YORK.

THE knowledge of the poisonous properties of wood alcohol, and especially of its deleterious effects on the vision, is of very recent date; in fact, it is within only about five years that it has become generally recognized by the medical profession. Of the fifty-four cases collected from literature and tabulated by Buller in his and Casey A. Wood's valuable monograph on "Poisoning by Wood Alcohol" (read at the Fifty-fifth Annual Session of the American Medical Association, in 1904), only four cases antedate the year 1899. These four were respectively in 1879, 1888, 1897, and 1898. The other fifty cases were published between 1899 and 1904. Upon inquiry instituted among the profession in the United States and Canada, Wood has been able to add one hundred and eighty-one cases of partial or complete blindness, blindness followed by death, or death without history of previous blindness—all of recent years. It is very likely that sporadic cases occurred in former years (and were perhaps attributed to other causes). The present alarming increase in blindness and death caused by poisoning with wood alcohol is due directly to refinement in its manufacture by which it is rendered odorless. The nauseous odor adhering to the product by the old process limited its use to purposes of the arts and trades, for instance, as a solvent of shellac, etc.; and although poisoning by the fumes *occasionally did* occur, poisoning by drinking was quite out of the question.

Since an odorless product under the name of "columbian spirits," "colonial spirits," or "eagle spirits" has been put on the market, its uses have been largely increased, and not only its legitimate use, but it has to a very large extent entered into the adulteration of alcoholic beverages, into the manufacture of flavoring substances meant for consumption, and into the preparation of proprietary remedies in which only ethyl alcohol should have been used. Many cases of blindness and death have followed the ingestion of Jamaica ginger, lemon extract, essence of lemon, essence of peppermint, bay rum, Cologne water, witch hazel, etc.

The sensation produced in this city quite recently, through the sudden death of a considerable number of men (variously stated at 17 to 25), in a certain district, who had fallen victims to the consumption of whiskey adulterated with wood alcohol, is still fresh in everybody's memory. Under these circumstances, the publication of the following case, which presents some interesting features, appears sufficiently justified.

S. G., a Russian Hebrew, forty-two years of age, presented himself on October 28, 1904, at Dr. I. Abrahamson's clinic for nervous diseases in the Mt. Sinai Dispensary. He was totally blind, but perfectly rational. He had no idea what had caused his condition, and the history which he gave lacked, therefore, the most important element. He stated that he used tobacco and alcoholics very moderately, drank tea excessively, and enjoyed, in general, very good health, occasionally suffering from rheumatic pains. There was no history of syphilis.

He was referred to the eye department for an examination of his eyes, and, the true state of affairs being here suspected, the following history was elicited from him:

On Saturday, October 22, after the morning service, he took at the house of some friends a small

quantity (about two ounces) of whiskey, but did not feel anything unusual until after eleven o'clock in the evening. When returning home from a meeting which he attended the same evening, he felt chilly and nauseated, and vomited. He drank some tea and went to bed. The next day (Sunday) he went, according to his every-day custom, to morning and also to evening service, but neither ate nor drank. During the night he woke and noticed that the gaslight seemed dim. On Monday morning he rose at seven o'clock and as it appeared rather dark to him he asked if it was raining. Then he noticed that his vision was at fault, but ascribed it to his abstaining from food the previous day, and drank some tea. He could not read the ordinary print of the newspaper although he was able to distinguish the large letters. He felt chilly and extremely weak and his hands were cold. As the day wore on, vision became worse, but toward evening of the same day he could indistinctly see, though not recognize, human figures. On the next morning (Tuesday) he was completely blind, all perception of light being gone. Various physicians were consulted but the condition remained unchanged. Total blindness persisted for the following four days. On Friday, October 28, he came to the Dispensary. The status præsens was as follows:

"The pupils are widely dilated and absolutely without reaction. The optic nerves of both eyes present the picture of a neuroretinitis of moderate intensity. The outlines of the discs are indistinct; the radiating nerve fibers are opaque and somewhat edematous; the blood-vessels (arteries and especially the veins) congested. In the macular region of both eyes are dispersed numerous yellowish, bright-shining little spots, similar to the picture described as choroiditis guttata."

On his way home from the clinic he saw with his right eye the first ray of light. This caused him to change his attitude toward our request for a sample of that whiskey of which he had partaken two days before the visual disturbances began, and at a subsequent visit he brought a quantity of it sufficient for chemical analysis. Dr. Carl Goldmark, chief of the Dispensary pathological laboratory, made a qualitative test which showed the presence of methyl alcohol in the whiskey. As the test is very decisive and easy of execution, it might be useful to describe it in a few words. Dr. Goldmark says regarding it:

"The test depends upon the synthetic production of methyl salicylate (artificial oil of wintergreen). Beringer devised the method of producing methyl salicylate by taking half an ounce of salicylic acid, dissolving it in two ounces of absolute methylic alcohol, and then gradually adding one ounce of sulphuric acid. On this basis the whiskey was tested. To about two drachms of the suspected fluid, half a drachm of salicylic acid solution and one drachm of sulphuric acid were added in a test-tube. The odor of the oil of wintergreen was at once apparent, and became more pronounced the longer it stood."

Later, Dr. S. B. Bookman, of the Mt. Sinai pathological laboratory, made a quantitative analysis of the sample by fractional distillation, and found that it contained 34 per cent. of pure methyl alcohol. Our patient had therefore taken about 20 c.c. of pure methyl alcohol. 1.36 Tablespoons

Potassium iodide was prescribed and the patient was directed to take hot baths to induce free perspiration, and to nourish himself well. As a result of this course of treatment there was a gradual improvement in vision for the following two months. He did not present himself for an eye examination until December 21, when sent for; but on November 18 we heard of him, that the right eye had steadily and rapidly improved since the day of his first visit, and that the left eye had begun improving about two weeks later. When he was examined on December 21, the vision of the right eye was 15/20, and that of the left 15/30. The outlines of the fields of vision were normal. The ophthalmoscopic examination showed a decided change. The discs were well outlined, and in their temporal halves very pale. The big vessels showed no abnormal caliber, perhaps the arteries were somewhat narrower than they should have been. The bright yellowish spots in the macular region of both eyes were decidedly increased in number.

The patient was lost sight of until sent for at the end of April, that is, six months after the onset of the affection. His examination on April 23, 1905, gave the following result: The pupils were more than average size, about 6 mm. in diameter, in an ordinarily bright room. When his face was turned toward the strong light of the window, they became slowly narrower, 3 mm. in diameter. The reaction to strong changes of light was very sluggish. The vision of the right eye was 4/12 and with +1 sph. cyl. +0.5 ax. hor. it was 4/8, this improvement of the vision with a convex correcting glass in a man of forty-two years, who had not used glasses before, evidencing a weakness of the accommodation which is in keeping with the weakness of the pupillary reaction. The vision of the left eye was 6/36, the refraction the same as in the right eye, but the glass caused no improvement. The field of vision of the right eye was normal in its outlines, no relative central scotoma. The field of the left eye showed moderate concentric contraction (about 15 degrees); no perception of green color; red recognized, and no relative central scotoma could be ascertained with this color. The ophthalmoscopic examination showed the optic discs opaque and very pale, bluish, edges fuzzy; lamina cribrosa visible; arteries narrow, especially those of the second order; those of the third order were hardly visible. A connective tissue sheath accompanied the arteries a short distance from the nerve head. The veins were larger than usual, especially in the left eye. The retina was very thin, appeared atrophic. In the macular region of either eye were very numerous yellowish-white dots.

That methyl alcohol is a poison dangerous to eyesight and to life is now generally concerned, although not long ago it was disputed, sincerely by some and from interest by others, the chief contention being that not the methyl alcohol itself, but admixed "impurities" produced the poisonous results. Of such, acetone w chiefly accused. The uncertainty was largely due to the fact that taken in small quantities methyl alcohol is innocuous to a great many persons, although taken in large quantities or habitually in moderate quantities it must be acknowledged as poisonous without exception. On the other hand, there are a great many people for whom a very small quantity (half a teaspoonful) would constitute a dose sure to bring on the gravest consequences. This difference, or "idiosyncrasy," well known in regard to every poison, in the case of methyl alcohol is most likely due to the difference in secondary chemical compounds formed in the alimentary canal and in the blood of the different persons by whom it is ingested. One of these secondary compounds is formaldehyde, but whether it is or is not the chief factor of methyl alcohol poisoning is not decided.

In reviewing all the cases on record, Casey A. Wood comes to the conclusion that there are three

or 4,08 Teaspoons

or 0.68 oz. (Fluid)

degrees of wood alcohol poisoning: "(1) An ordinary, mild intoxication, with perhaps some dizziness, nausea, and mild gastrointestinal disturbance, terminating in perfect recovery within a few days, but occasionally followed by more or less serious damage to vision. (2) A toxic effect more pronounced in every way—dizziness, nausea, and gastroenteritis being conspicuous symptoms. Dimness of vision, often increasing to total blindness, is characteristic of this degree of poisoning. (3) An overwhelming prostration which terminates in coma and death."

The most conspicuous and characteristic feature of wood alcohol poisoning is complete bilateral blindness, appearing sometimes suddenly, but in most cases gradually after a period of failing vision. The disturbance of vision will sometimes come after a few hours; in most cases it lasts from one to several days, until a dimness of the sight is noticed. (In the case above reported it took about forty hours.) This progresses until the blindness is absolute. The interval between the ingestion of the poison and the onset of the eye symptoms is very characteristic, and may in some cases, as for instance in ours, hide the real cause of the blindness from the patient. The blindness lasts for a period of several days or even weeks, after which sight returns gradually. The improvement is frequently very considerable, sometimes full restoration takes place; but the improvement is not lasting. After several weeks the vision begins to fail again, and in the great majority of cases this progresses to final and irreparable blindness. Very few cases preserve useful vision; early treatment seems to be favorable to this termination.

Of the objective symptoms the only one constant from the beginning is widely dilated pupils without any reaction whatever. The ophthalmoscopic picture varies. Either it is entirely negative and thus corresponds with the usual assumption of a retrobulbar neuritis, or it shows a papillitis or neuroretinitis of low degree—slight swelling of the nerve-head, blurring of the outlines, radiating appearance of the slightly edematous nerve fibers, and congestion of the large blood-vessels, especially the veins. This condition was present in our case in a rather marked degree. A third variety shows pallor of the disc from the onset, with contracted blood-vessels, so that, although not so intense, it is similar to the appearance in quinine poisoning. But whatever the picture in the beginning, in all cases we ultimately find atrophy of the optic nerve and retina, an extremely pale, bluish or grayish disc, with outlines distinct but fuzzy; the large blood-vessels, especially the arteries, contracted, for some distance contoured by a connective tissue sheath; no smaller blood-vessels or capillaries visible— in a word, the picture of postneuritic atrophy. Nowhere do I find mention made of the presence in the whole macular region, of the yellowish, bright spots described in the above case. I might consider them an accidental circumstance if I had not once seen the same condition in a man whom I treated for alcohol-and-tobacco amblyopia.

The field of vision shows anomalies in all cases. It is nearly always concentrically contracted, and generally there is an absolute or relative central scotoma present. In our case, after six months the right eye showed complete recovery of function (except a moderate diminution of the acuteness of vision), probably owing to the small amount of poison ingested and the early institution of treatment. The left eye showed marked although moderate concentric contraction of the field, considerable diminution of central vision, and absence of perception of the green color. A central scotoma for other colors at that time could not be established.

As to the pathology underlying these visual disturbances, opinions are divided, some ascribing it to a retrobulbar neuritis, whereas others believe that the ganglion cells of the retina are the structure primarily affected, followed by ascending—or, rather, descending—atrophy of the optic nerve fibers. The writer is inclined to accept the latter view, although it must be admitted that a number of weighty arguments can be adduced for either of them.

With regard to the course of therapy to be followed in a case of wood-alcohol poisoning, sweating by hot bath or pilocarpine, potassium iodide internally, and sufficient diuresis (perhaps by means of a milk diet), suggest themselves as rational, and have been successful in some cases.

The utmost importance belongs to prophylaxis. The public in general must be enlightened as to the poisonous nature of this substance, and laws should be enacted forbidding its use in the manufacture of "essences" and proprietary remedies which may be the means of poisoning persons not addicted to the use of intoxicating drinks and who are entirely ignorant of the danger to which they are exposed.