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A CASE OF METHYL-ALCOHOL AMAUROSIS, THE PATHWAY OF ENTRANCE OF THE POISON BEING THE LUNGS AND THE CUTANEOUS SURFACE.

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The toxic action of methyl-alcohol in recent times has attracted much attention, and its influence upon the eye has been the subject of many papers and some experimental work. The report of the first case of methyl-alcohol amblyopia is usually attributed to Mengin,[1] but as Harold Gifford[2] points out, Mengin's case had previously been reported by Viger,[3] so that to him really belongs the priority of describing this agent as a cause of amblyopia. The next case[4] on record appears to be one described by Dr. F. Van Fleet,[5] followed a year later by the case of MacCoy and Michael.[6] In 1899 the literature of the subject has been enriched by the cases recorded by Kuhnt,[7] Moulton,[8] Gifford,[9] Callan,[10] Patillo and Casey Wood,[11] J. F. Raub,[12] and others. More recently additional cases of this form of amblyopia have appeared in this country and abroad.

In most of the instances thus far reported the methylic alcohol has been consumed by man as a substitute for ordinary alcoholic drinks. In a few instances, however, for example, the two reported by Patillo

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[1] Recueil d'Ophthalmologie, 3 serie, 1, 1879, p. 663.

[2] The Ophthalmic Record, VIII, 1899, p. 441.

[3] L'Anne medical, 2, 1877, p. 105.

[4] I think the report of the first case of methyl-alcohol amblyopia in this country was made by Dr. J. M. Ray, of Louisville, Ky. He examined his patient in March, 1896, and described the case to a local medical society at the time, but the report never appeared in any journal. He has been kind enough to send me the notes of this case, and another one that he saw in 1899, which has not yet been published.

[5] Manhattan Eye and Ear Hospital Reports, 1897, Vol. IV, p. 15.

[6] Medical Record, May 28, 1898

[7] Zeitschr. f. Augenheilk., Vol. I, 1899.

[8] The Ophthalmic Record, VIII, 1899, p. 335.

[9] Loc. cit.

[10] Quoted by Ward Holden, Archives of Ophth. XXVIII, 2, 1899, 129.

[11] Ophthalmic Record, VIII, 1899, p. 599.

[12] Ophthalmic Record, VIII, 1896, p. 619.

and Casey Wood, the toxic agent found entrance through the lungs. Sometimes the methylic alcohol has been taken by itself, or diluted with water, and sometimes mixed with other substances. The quantity has varied exceedingly, and it would be difficult to estimate exactly what is the smallest quantity that can produce blindness, or what is the greatest quantity which may be consumed with impunity. It would further seem that patients may be exposed to the influence of methyl-alcohol as it is used in the arts for long periods of time, even for years, without experiencing deleterious effects, and then, apparently without changing their work, become affected. So, also, the patient may drink in moderate quantities of the drug at times, as did Viger's patient, and suffer no ill effects, or at least, no serious inconvenience, but become blind suddenly when on one spree a rather excessive quantity is taken.

Preceding the blindness the following symptoms have been noticed by various observers: Great muscular weakness, defective heart's action, nausea, vomiting, profuse sweating, intense headache, giddiness, coma and delirium. Usually within twenty-four hours, and perhaps earlier, after the methyl-alcohol spree and the subsidence of the symptoms previously described, the patient has noticed obscuration of vision, which rapidly, sometimes within a few hours, as in one case is entirely lost. Under the influence of treatment there may be temporary improvement, which, with perhaps the exception of a single case, is succeeded by return of the blindness. This in most instances has been practically complete, or at least, vision has been reduced to counting fingers. The pupils are widely dilated and irresponsive to light, although they may respond to the convergence and the orbicularis test.

Ophthalmoscopically there have been noted blurring of the edges of the disc, positive optic neuritis with exudations into the retina, and complete atrophy, with great diminution of the calibre of the vessels. The field of vision is contracted, and where it has been sought for carefully in the earlier stages an absolute central scotoma and color blindness will be found. An interesting symptom noted by Gifford and Kuhnt is pain on movement of the eyes, or upon pressing them backward into the orbit.

The orbital pain, the complete blindness followed by marked but temporary improvement, and the positive optic neuritis which has been noted by a number of observers, for example, by MacCoy and

Michael, Hotz, Ray and Gifford, seem to indicate a primary affection of the optic nerve that is to say, a retrobulbar neuritis, a conclusion which is still further established by the presence of the central scotomas which have been noted in at least two of the instances. Ward Holden, however, on the basis of experiments on dogs, concludes that methyl-alcohol causes an amblyopia which depends upon nutritive changes in the ganglion cells of the retina. His observations have been confirmed by Birch-Hirschfeld[1] who uniformly found degeneration of the ganglion cells with breaking-down of the chromatin bodies, development of vacuoles, shrinking of the cell and the nucleus, and finally, destruction of the cell body. F. C. Hotz does not attach a great deal of weight to these experiments, and after describing a case of methyl-alcohol amblyopia which he himself had seen and in which there was well-marked optic neuritis, points out that if the drug primarily affects and directly destroys the retinal elements, it is difficult to account for the temporary return of sight which has been noted in a number of instances, while on the other hand, a violent inflammation of the connective tissue of the optic nerve could primarily prevent the conduction of visual impressions which could subside when the swelling of the nerve subsided, but would again be blocked when the later atrophy came on. Ray, in describing his case, makes a similar suggestion.

It is probable that several substances which have been credited with the power of producing toxic amblyopia depend for their activity upon methyl-alcohol. The investigations of Herbert Harlan[2] strongly indicate if they do not absolutely prove, that all the cases of blindness from drinking essence of ginger, peppermint, etc., have been due, not to the activities of the ginger and the peppermint, but to the methyl-alcohol with which they are mixed.

With this imperfect résumé of our knowledge of methyl-alcohol blindness, I desire to place a case on record which contains a number of interesting features and in some respects differs from any of the cases thus far reported.

John M., married, born in America, aged 39, came to the Jefferson College Hospital on January 4th, 1901, with the statement that he had two weeks previously, after a brief illness, waked up entirely blind.

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[1] Klin. Monatsbl. f. Augenheilk., October, 1900, p. 682.

[2] The Ophthalmic Record, February, 1901.

History.—The patient is a varnisher and painter by trade, but for the last three years has confined his work to the varnishing business. He has always been in good health, and with the exception of the ordinary diseases of childhood and an attack of malaria three years ago has been free from illness. For the past two years he has been subject to attacks of nose-bleed. These, however, never have been severe and have never been followed by any untoward consequences. He uses tobacco in moderate degree, and has also used alcohol. There is no history of syphilis. He is married, and has four living children in good health. His wife is healthy. His father is living, past seventy years of age, and in good physical condition. His mother died when he was a child from apoplexy. He has two brothers alive and one sister, all in good health. For five years he worked in white lead. He has also used what he calls "alkaline," as well as oxalic acid, muriatic acid and bichromate of potash.

For the last three years he has pursued the occupation of a varnisher, and since the introduction of wood spirits, or, as it is ordinarily called in the trade, Columbia spirits, which is a liquid containing about 95 per cent of methyl-alcohol, he has employed this substance in the same way in which turpentine is used in paint, that is to say, the methyl-alcohol is mixed with the shellac in such proportions as may be needed to make it either thick or thin. The patient positively denies that he ever drank any of the wood-alcohol. As he expressed it, "the smell of it was enough." He has frequently, however, when obliged to varnish the inside of small articles of furniture, for example, a closet, become so confused with the fumes that, again to use his own expression, he "staggered like a drunken man." This peculiarity he observed particularly in hot weather. He was accustomed every day to wash his hands and arms and sometimes his face at the close of his labors with wood-alcohol in order to remove the varnish which was sticking to the skin. For two months prior to his attack of blindness he had worked daily as a varnisher, employing the shellac and methyl-alcohol in the manner already described. This is the longest period during which he had pursued this work uninterruptedly; formerly he worked at varnishing for three or four days at a time and then changed to some other kind of labor connected with his trade which did not entail the use of methyl-alcohol. For some time he had noted attacks of misty vision, which usually came on in the evening after leaving the shop, but which always disappeared in fifteen or

twenty minutes. He had especially observed that the lights on the streets appeared blurred during these temporary obscurations of sight.

On the morning of December 22, 1900, after a hasty breakfast consisting only of about a pint of cold milk, he started to his work, but on his road began to feel ill and when he reached his shop he had a chill, so that he was unable to continue his labor. This chilly sensation was particularly manifested in his lower extremities and was followed by numb and shooting pains, so much so that the doctor who was called in told him he had sciatic rheumatism, and in order to relieve him gave him two morphia pills and subsequently a hypodermic of the same drug. The chilliness and pain, however, were not entirely subdued. There was no nausea, no vomiting and no headache. He dozed a little during the day and towards evening fell asleep, waking at midnight, and to his great alarm found himself so completely blind that he could not see the gas light in the room. In the morning a physician was hastily summoned and prescribed for him remedies the nature of which I do not know. The blindness continued absolute, so far as his own observation is concerned, for two weeks, when he reported at the Jefferson College Hospital and was examined by Dr. Howard F. Hansell.

The notes on the book are as follows: Pupils dilated to 6 mm. and irresponsive to light, but reacting to accommodation. Media clear; nerves pale, their outlines distinct; arteries and veins both reduced in size, the latter tortuous and filled with dark blood.

He was immediately ordered pilocarpine, and for fourteen nights in succession was subjected to vigorous sweats. After the second sweat there was a slight return of vision, and by the end of two weeks he was able to distinguish objects so that he could walk alone upon the street. He never, however, recovered sufficient vision to recognize faces. At the end of three weeks this return of vision began to fail until the present time, that is, the 12th of April, 1901, when the blindness again became complete.

Examination.—The patient is a well-built man of the blonde type, with a somewhat pallid skin and a typical amaurotic stare. The circulation, heart and blood vessels appear to be normal and there are no external signs of disease. An examination of the urine yields the following result:

Color: Amber.
Reaction: Faintly acid.

Microscopic examination:
Crystals: None.

Albumin: None.	Amorphous urates.
Sugar: None.	A few epithelial cells.
Urea: 1.9 per cent.	A few leucocytes.
	No casts.

The examination of the blood was as follows:

Erythrocytes: 4,950,000.

Leucocytes: 6,400.

Hæmoglobin: 75 per cent.

Dr. William G. Spiller, who also examined the patient, reports as follows: The knee-jerks, especially the left, are exaggerated. The Achilles jerk is about normal on each side. There is no Babinski reflex, and no girdle sensation exists about the waist. The sense of smell is not impaired. No weakness in the limbs is present, He has no intention tremor and no ataxia. There is no history of brain tumor, i. e., vomiting, headache or vertigo.

Eyes.—V. of R. E. faint light perception in a small area, about 5 degrees in diameter, 20 degrees below the fixing point; the rest of the field is completely dark. The pupil is dilated to 8 mm., and when a beam of light is directed slightly downward and outward through a condensing lens there is just a suggestion of contraction in the iris. When the patient converges to a point a few inches in front of his eyes, there is prompt response both of this pupil and the pupil upon the opposite side. In other words, the convergence reaction is preserved, as is also the reaction on forced closure of the lids, that is to say, the orbicularis reaction. The media are clear, the nerve-head irregularly oval in outline and partially surrounded with a choroid ring. There is a deep physiological cup exposing the lamina cribrosa and the general nerve-head is of a greenish, atrophic color. The arteries are small and are bordered by faint thickening of the perivascular lymph-sheaths. The veins are also somewhat contracted, distinctly tortuous and contain very dark blood. The general fundus gives no evidence of the pathological lesions, except perhaps a slight mottling in the region of the macula. The movement of the eyeball is normal in all directions.

V. of L. E. absolutely gone, not the faintest trace of light perception being demonstrable in any portion of the field. The pupil is dilated like the other one, to about 8 mm., and the iris is absolutely irresponsive to the influence of light, but like its fellow, contracts on efforts of convergence and to the influence of the orbicularis test.

The appearances of the fundus in all respects repeat those upon the opposite side, except that the disc is unsurrounded by a choroid ring. If anything, the veins are a little darker and more tortuous upon this side than upon the opposite, and many of the finer twigs are decidedly twisted.

While of course it is impossible to ascribe this case of sudden blindness with absolute certainty to the toxic influence of methyl-alcohol, this theory affords a satisfactory explanation.

Dr. H. F. Hansell, however, who examined this patient soon after the blindness first began, is inclined to think that a hemorrhage into the chiasm might explain the sudden loss of vision. He is confirmed in this opinion because soon after the amblyopia set in, but when there was still sufficient vision to take a light field, a bitemporal hemiopic defect was evident. There was also at this time slight optic neuritis.

Dr. William G. Spiller, who also saw the case, thinks that this is a possible explanation, but tells me that he knows of no case of hemorrhage in the chiasm with autopsy. The clinical history of the blindness, however, is like that of all other cases of methyl-alcohol amaurosis, that is, great suddenness of onset, complete loss of vision lasting for a given period of time, followed by a partial restoration of sight, to be again succeeded by a return to the original total loss of vision. The visual field defect which Dr. Hansell noted might be explained by an action of the drug on the ganglion cells of the retina.

The prodromal symptoms are somewhat different from those which have occurred in other cases, where usually there has been serious constitutional disturbance, for example, violent headache, unconsciousness, delirium, etc., but it must be remembered that in these cases the agent was taken in large doses into the stomach and produced an acute poisoning and naturally more florid symptoms. In Patillo's patients, who became blind as the result of shellacing beer vats and who therefore inhaled the poison, the symptoms prior to the blindness were not nearly so severe as those which occurred in men who drank the liquid. They did, however, have severe frontal headache, nausea and dizziness. My patient has on frequent occasions, as before noted, had attacks of dizziness, vertigo, and, as he expressed it, "drunkenness," when he has shellaced enclosed spaces, particularly in warm weather, but the interesting point in his clinical history is that although he had for some years been a varnisher and had frequently used methyl-alcohol, he had never worked at his trade for

long periods of time continuously, never more, in fact, than three or four days at a time.

For two months prior to his blindness he had been daily occupied in shellacing and had been constantly exposed, as he expressed it to me, to the fumes of the liquid. He had not, however, for two weeks prior to his blindness had one of his attacks of "drunkenness." I have already pointed out that in addition to his exposure to the fumes, he was accustomed to soak his hands and forearms in the methyl-alcohol for the purpose of getting rid of the shellac stains, and sometimes washed his face in the same liquid. Therefore we may reasonably assume that there was a gradual absorption of the poison, and it is probable that, like many other poisons, it has a cumulative action in the system and finally bursts forth with sudden force. Whether the attacks of blurred sight which he was accustomed to have were prodromal of the ultimate entire blindness I cannot say. He tells me that he knows of another shellacer who was obliged to discontinue his work because of disturbed vision, and who was told that he would certainly go blind unless he found some other occupation which freed him from the influence of methyl-alcohol.

If this case is correctly classified as one of methyl-alcohol poisoning, it has a very important bearing, because it demonstrates that varnishers are liable to a slow poisoning by this drug, which gradually accumulates and finally produces, with great suddenness, complete blindness, just as the drug does when it is consumed in large quantities in place of some other alcoholic drink. In other words, it indicates that varnishers who employ either the methyl-alcohol or the Columbian spirit, are liable to dangers similar to those which surround workers in lead, in india rubber where bisulphide of carbon is employed, and in dinitrobenzol. If this is the case, then it is most important that workmen should be warned of the dangers of these fumes of methyl-alcohol, and also of the dangers of washing their hands, forearms, etc., in the same liquid.