THE OCULAR MENACE OF WOOD ALCOHOL POISONING*

BY

DR. S. LEWIS ZIEGLER

PHILADELPHIA

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Whether the ganglion cells of the retina or the fibres of the optic nerve are first attacked has never been decided. Birch-Hirschfeld, Holden, and de Schweinitz endorse the former view. Most of the conclusions reached have depended upon animal experimentation, but post-mortem degeneration of these delicate structures is so rapid that a satisfactory study is difficult to make. As I have elsewhere stated it is most unfortunate that so much excellent human material has recently gone to waste without a single microscopical report being made. According to Fridenberg, both of these highly energized tissues are seriously injured by contact with the formic acid, which like its congener, the bee sting, creates strangulation by the sudden tissue swelling or oedema which follows exposure to such a corrosive poison. The corrosion does harm and so does the swelling. Whichever tissue is touched first will be injured first. As the serous infiltration subsides and strangulation is removed, the vision improves, but if the nerve fibres are corroded and later undergo shrinkage, vision is finally lost. The injury is modified, therefore, by the degree of concentration of the circulating poison which seems to have a selective affinity for these delicate tissues.

A critical study of the phenomena occurring in cases of wood alcohol poisoning and more especially the characteristic symptoms noted in Case VI, herein reported, have led me to the conclusion that the primary and fundamental lesion in all cases of methylic poisoning is a *profound injury of the pituitary body*. The acute toxic symptoms are typical of such a lesion, while the chronic symptoms are equally significant. A similar lesion of the pituitary may possibly be found in quinine and other toxæmias. The tentative diagnosis of encephalitis lethargica in my case is also suggestive, since the symptoms of that disease indicate a pituitary lesion. The changing but steadily contracting fields, the fugitive scotomata, the visual loss and recovery, the sclerosed or atrophic nerveheads, the fixed and dilated pupils, the temporary paresis of the extraocular muscles, the ptosis, the ataxic gait and the mental hebetude are all characteristic of pituitary involvement.

The fields of vision usually reveal a concentric contraction for both form and colour. Central scotomata are often present but not always permanent, as many recoveries have been recorded. Tyson

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noted in his chronic cases, multiple scotomata scattered over the fields. Occasionally, both the fields and the central vision are preserved in spite of the apparent sclerosis of the nervehead.

History of Cases

I will briefly report a few cases :

CASE I. Housewife, 49, seen by me 10 years ago and reported before the Pennsylvania State Medical Society in 1910. Indulged in a single alcoholic drink on Christmas eve, 1909. Jamaica ginger was suspected. This was followed by sudden and complete blindness the next day. Sclerotics were yellow. Had a spasmodic "stomach" cough for several days. Now blind in both eyes; pupils wide and fixed; feeble light reflex in O.D. Nerve heads sclerosed, dull white, but no shrinkage; vessels attenuated.

Thyroid extract given; negative galvanism advised. Acidosis not tested. Electrical treatment was begun January 19, 1910. One month later right disc became vascularized and left slightly hyperaemic. At the end of second month she regained P.L. and gradually continued to improve until on October 1, 1910, vision was O.D., 20/50 J-10, O.S. 20/100 J-14. The fields began with a very small central area and broadened out proportionately. This vision was maintained for several years, but recent reports are not so favourable. If her acidosis had been treated in the early stages and she had persisted with her galvanic treatment I believe this vision could have been maintained.

CASE II.—Male, 46, seen by me at about the same period; sudden blindness followed the drinking of a bottle of fortified cider bought at a country grocery store. When first examined the acute symptoms had subsided and vision had returned to about 2/200 in each eye. The nerve heads were pale, edges distinct, slight central cupping. Fields were concentrically contracted. A trial of galvanism was made but without improvement in vision.

CASE III.—Male, 40, previously referred to as resulting from inhalation, was under treatment for several months with failing vision, slight pallor of disc and contraction of fields without apparent cause. I finally directed him to keep a diary of his daily activities, hoping to discover something definite. One day I noticed the record of a visit to a china cement factory for one hour. He said he went there regularly every day but forgot to record it previously. On examination of the cement formula wood alcohol was found to be a constituent. By cessation of his visits to the factory and the application of negative galvanism he slowly recovered normal vision and fields which he still retains.

CASE IV.—Marine, seen in June, 1919, by Dr. Connole, of Wilkes-Barre, Pa. (to whom I am indebted for the notes), and by Dr. Daland in consultation. After a bay rum debauch "two sailors

died and one went blind." Thirty-six hours after ingestion vision was reduced to P.L. and wholly lost two days later. Typical papillitis, 1.5 D. increasing to 2 D. and gradually receding.

The patient was immediately placed on alkaline treatment:—

1. Lime water in milk. 2. Enteroclysis of 5 per cent. dextrose containing sodium bicarb. 3ij to the pint. 3. Sodium bicarb. by the mouth, gr. x every three hours. 4. Strychnia Sulph. gr. 1/3 in 24 hours, later gr. 1/5.

P.L. regained in two weeks and vision improved to practically normal after two months, at which time the margins of disc were distinct, reddish on nasal side, vessels contracted, with central cup showing lamina. Colour perception normal. Concentric contraction of fields; left, 10°, right, 20°; blind spot slightly enlarged. Patient was seen in France one year later by Dr. Dunbar, and claimed vision was excellent. Present condition not known.

CASE V. Recently reported by Dr. Mongel before the College of Physicians, of Philadelphia. Male, 21, had three drinks in a saloon on Christmas Eve, 1919, and three the next day. Remembers that liquor had a rusty, foul, ether-like odour and taste. In 48 hours vision was reduced to P.L. Pupils slightly dilated, respond feebly to light. Conjunctivae deeply injected. Anterior scleral vessels distended. Media clear. Severe neuro-retinitis. Papillae swollen, hyperaemic, margins blurred, oedema extending into retinal tissue. Retinal vessels dark, engorged, and tortuous. Urine highly acid, Sp. Gr. 1012.

Treatment:—1. Gastric lavage; 2. Salines through enteroclysis; 3. Pilocarpin; 4. Hot packs; 5. Strych. sulph. gr. 1/30 t.i.d.; 6. Calcium chloride gr. x, q.i.d.

P.L. regained in two weeks, reached 20/50 in one month and became normal at the end of two months. Fields showed slight concentric contraction with central scotomata. The nerve, retina, and blood-vessels gradually cleared up. Calcium chloride and strychnia were continued for three months.

CASE VI.—Painter, aged 20, inhaled fumes while varnishing the engine room of a submarine for three days, in March, 1920. Dizzy the first day, hilarious the second, and nervous the third. Gastric pain and insomnia. Diplopia from paresis of external rectus. Ptosis and blindness soon followed. In three weeks began to improve. Diagnosis of encephalitis lethargica made by attending physician.

Now vision O.D. 20/70 J-14, O.S. 5/200 J-20. Pupils unequally dilated, O.S. wider; react to light and accommodation. O.D. nerve-head pale, central excavation, venous pulse, fine vascularization of disc. O.S. nerve pallor more marked, less vascularization. Excavation more extensive, venous pulse, veins overfull. Slight nystagmic movements. Exaggerated patellar reflexes. No static ataxia. Curious gait; lack of co-ordination suggests pituitary injury. Fields

limited for form and colour; no central scotomata. Marked mental hebetude.

Acidosis of early stages had changed to alkalosis, 113 per cent. This was gradually reduced by treatment to 65 per cent., which is practically a normal balance. Some backache, but urine is normal. Engorged nasal tissues interfere with breathing.

With negative galvanism and correction of refraction, central vision has improved to O.D. 20/15 pt. O.S. 20/20. Accommodation paralyzed. Add Sph. + 2.D = O.D. J-2, O.S. J-3. Fields have widened out. Less pallor of disc; vascularization has increased. Less mental hebetude and greater agility following the use of pituitrin.

The Biochemistry of Methyl Alcohol

The biochemistry of methyl alcohol as it passes through the system is somewhat complex. It is essentially a protoplasmic poison. The bulk of this poison is eliminated through the lungs, skin and kidneys, while the alimentary tract gets rid of a large portion. The remainder undergoes oxidation into formaldehyde and formic acid, both of which are corrosive poisons. The latter, however, will not oxidize further, but is slowly eliminated by the kidneys. Pohl, in 1893, showed that after ingestion of wood alcohol, the excretion of formic acid was increased in the urine. In wood alcohol workers this increase is so marked that Fehling's solution is promptly reduced. This chemical fact should always be borne in mind or sugar will be suspected and a false diagnosis of diabetes made by the inexperienced.

Acidosis seems to be a constant condition in the early stages. Tyson has demonstrated acidity of the aqueous humor in some of his cases. Judging from my experience in Case VI, alkalosis may appear in the later stages. These chemical reactions will be revealed by Van Slyke's test for carbon dioxide in the blood. If the attack of acidosis is acute, and of a severe type, it may manifest itself by the Kussmaul type of breathing.

Treatment

Acidosis should be overcome by the early administration of alkalis. Harrop reports good results obtained in a case of acute acidosis of great severity treated by him at Johns Hopkins Hospital, in which he made intravenous injections of from 400 to 500 cc. of a 5 per cent. solution of sodium bicarbonate on succeeding days. He governed his indications by Van Slyke's test. We should always bear in mind, however, that excessive alkalosis may cause grave irritation of the kidney, and that as soon as tests show that the plasma bicarbonate has returned to normal no more alkali should be given. We already know that the oedematous swelling

caused by a bee sting can be reduced by alkalis. It will, therefore, be a great advance in the treatment of methylic poisoning if we can relieve this acute acidosis by the early use of alkalis, and thus lessen the destruction of nervous tissue.

Connole's case shows the value of comparatively small doses of sodium bicarbonate, given by the mouth and by enteroclysis. The vision promptly improved, and was maintained for at least a year. Mongel secured equally good results with calcium chloride, but not sufficient time has elapsed for us to pass final judgment in his case. It will be interesting to watch whether these results are permanent.

According to Bongers, much of the wood alcohol in the system is returned to the stomach and can be removed by gastric lavage. He recovered in this way three times as much on the second and third days as on the first day. He also found that formic acid appeared in the washings as long as 27 hours after the ingestion.

Many observers have noted that those who vomit early and freely are not so seriously injured by the poison and recover with less impairment of vision. It will be necessary to decide, therefore, whether emesis through mustard or apomorphin is indicated, or whether daily lavage will prove of greater utility. The stomach pump should be used early if the patient is seen early and methylic poisoning is suspected; but most victims are so secretive in the beginning that this is seldom possible.

Diaphoresis through hot packs, vapour baths or pilocarpin has frequently been employed with great success. Hot drinks also encourage this effect. Apomorphin and pilocarpin are both excellent lymphagogues in small doses. Jalap and saline purgatives have accomplished much good, but an alkaline enteroclysis may prove more useful because the absorbed water helps to dilute the poison and wash it out of the system, while the added alkali will help to neutralize the toxicity.

Oxygen has been used in methylic toxaemia to relieve cyanosis and support the heart, and yet it is an undetermined question whether this oxidation would not increase the virulence of the poison by converting the formaldehyde into formic acid. Harneck in a study of this question concluded that "Methyl alcohol by slow oxidation is converted into formic acid, while in rapid oxidation carbon dioxide and water were formed." Rapid oxidation may, therefore, be a valuable suggestion in the chemistry of metabolism. In a former paper on wood alcohol I recommended the use of potassium permanganate in order to oxidize rapidly the abnormal chemical compounds in the stomach, just as is done in opium poisoning, but I would prefer to have this suggestion tested out by animal experimentation before putting it into practice among humans.

It has been suggested that since deafness often ensues and there is uncertainty of gait together with certain head movements, the

middle ear is involved. I would rather believe that the pituitary body had suffered injury, since many of these symptoms point to such an origin. Case VI is very suggestive of this. In that event the use of pituitrin should prove of service, as it did in this case. Thyroid extract, however, was more useful in Case I.

If marked nervous symptoms develop the use of hyoscin hydrobromate may be indicated, but it should be used with caution as it will interfere with elimination unless pilocarpin is used to overcome this tendency. While potassium iodide has been freely used to eliminate the toxins during the more chronic stages, I would prefer to use Donovan's solution for this purpose. The tonic effect of large doses of strychnin must always be considered, but I have not observed success from the use of this drug.

To revascularize the disc and restore the lost function of the nerve, no measure can equal the stimulating effects of negative galvanism. The case of partial blindness through inhalation that I have previously referred to (Case III) recovered practically normal fields and vision after prolonged treatment with negative galvanism. The patient with sudden sclerosis (Case I) who was totally blind for two months, recovered half vision and fields under the use of negative galvanism for one year. Case VI also regained normal central vision from galvanic stimulation although the accommodation is still paralyzed.

Negative galvanism should be administered with great care, using a high voltage and low amperage. Sixty volts should be passed through the main shunt controller, with the amperage reduced to one milliampere by a secondary carbon controller. The current is passed for ten minutes, and then reduced to one-half a milliampere and passed for a second period of ten minutes. These seances are continued on alternate days, as a rule. I believe that electricity is the most efficient therapeutic measure we have for the milder cases of toxic injury where there has not been complete destruction of the nerve fibres. If this has already occurred, as in my case of poisoning from bottled cider (Case II), galvanism will have no effect whatever.

In Conclusion

1. Wood alcohol is the most deadly poison used in daily commerce.
2. One teaspoonful has been known to cause blindness and one ounce to cause death.
3. The port of entry may be through the mouth, nose, or skin.
4. Wood alcohol should be identified by Robinson's test.
5. It is a protoplasmic poison possessing a selective affinity for the delicate nerve tissues of the eye.

6. Its biochemistry is modified by oxidation, first to formaldehyde and then to formic acid, both of which are corrosive poisons.

7. Formic acid is the end-product excreted by the kidneys.

8. If formic acid is present in the urine it will promptly reduce Fehling's solution, thus suggesting to the inexperienced a false diagnosis of diabetes.

9. Van Slyke's test will reveal acidosis in the early stages and alkalosis later.

10. Sudden blindness with vomiting and abdominal pain should always arouse suspicion of methylic poisoning; especially if diplopia or ptosis is associated.

11. Papillitis, sector-like atrophy, and sudden sclerosis of the nervehead are equally typical fundus lesions.

12. Symptoms of pituitary injury are most suggestive in pointing to this as the primary and fundamental lesion.

13. Contracted fields and central or paracentral scotomata are usually present.

14. Treatment should include early neutralization by alkalies, elimination by lavage, emetics, diaphoretics and rapid oxidation, together with stimulation of the optic nerve by negative galvanism applied directly to the eye. Thyroid extract and pituitrin may be indicated.

15. The manufacture and sale of wood alcohol should be prohibited or regulated by law.

16. If sales are permitted, safeguards and warnings should be required and the public instructed as to the great danger to vision and life.

17. A special revenue tax with registered "Poison Sales" would regulate and record its distribution and in cases of poisoning reveal the source.

18. This tax should equalize the cost of denatured alcohol and methyl alcohol, and thus remove the temptation to adulteration because of cheapness.

19. All wines, whiskies, toilet articles and patent medicines imported from foreign countries should be tested for wood alcohol before passing through the Customs inspection.

20. The name "Methanol" specifically designates this product and yet avoids the tempting suggestiveness of the word "alcohol."

SOME LATE GLAUCOMA RESULTS

BY

H. HERBERT, Lt.-Col. I.M.S. (Retd.)

BRIGHTON

In the ordinary course of work, during the last two years in Nottingham, I came across eight examples of return of plus tension