ACUTE METHYL ALCOHOL POISONING*

RAPHAEL ISAACS, M.A., M.D.
Instructor in Internal Medicine, University of Cincinnati College of Medicine
CINCINNATI

The increasing incidence of poisoning with wood alcohol has shown the necessity for an efficient method of treatment. A study of a series of cases in the medical wards of the Cincinnati General Hospital has led to a plan of treatment that promises to be of value in saving life and in preventing blindness, the most serious of the complications of the convalescent.

The methyl alcohol is taken either in the form of denatured ethyl alcohol, or as commercial wood alcohol, usually with the intention of taking a substitute for whisky. Cases of suicidal poisoning or accidental and occupational forms are also encountered. The symptoms vary with the amount taken, the type of the mixture and the length of time before treatment is begun.

SYMPTOMS AND PATHOLOGY

In the extreme cases the patients are brought in in a comatose condition, usually with cyanosis which sometimes is very intense. In a fatal case, in which the patient was admitted to the wards in a moribund condition, the color was a deep blue-black. The cyanosis is more marked over the upper part of the body—chest, arms and hands, neck and head—than over the lower part, although the feet may be blue and cold in severe cases.

Methyl alcohol appears to act as a respiratory poison, the rate of breathing in severe cases being reduced to six or fewer times per minute and quite shallow. In the mild cases the respiration rate may not be affected, and sometimes, especially if complicated by other substances, as ethyl alcohol, Jamaica ginger, lemon extract and other similar whisky substitutes, the rate may be more rapid, commonly 24. One case, complicated by strychnin poisoning and bronchopneumonia, developed a rate of 36. The breath usually has the odor of methyl alcohol, showing pulmonary excretion of the poison.

The temperature is subnormal at times, but usually reaches 99 some time during the first twenty-four

* From the Department of Medicine, University of Cincinnati and the Cincinnati General Hospital.

hours, remaining normal after this in the uncomplicated cases.

During the acute stages the pulse is accelerated, reaching 100 and over (120). In a fatal case the heart continued to beat for several minutes after respiration had ceased. With improvement, there is a slowing of the pulse. The heart sounds are usually clear, but not very forceful.

A characteristic feature is dilatation of the pupils, which, however, may react somewhat to strong light. With return of full consciousness, the patients usually complain of blurring of vision, noted in from one to twelve hours. Often the ophthalmoscope fails to show any abnormal appearance, although serous exudates along the course of the blood vessels, hyperemia of the disk, with blurring of the edges, sometimes of the nasal side only, and finally optic atrophy, with contraction of the retinal vessels and peripheral limitation of the field of vision, are sometimes noted. The conjunctiva may be congested.

Headache is usually experienced, being quite constant on the following day, with sometimes a feeling of numbness in the head. At postmortem there is a marked edema and congestion of the brain and meninges, with increase in the cerebrospinal fluid. On spinal puncture, the fluid is clear and under increased pressure. There may be no increase in globulin or cell count. Vertigo is frequently present.

Nausea is usually present, vomiting, frequently. The vomitus, sometime after gastric lavage, may still have the odor of methyl alcohol. Pain, at times cramplike, may be present in the epigastrium, with tenderness, or in the lower part of the abdomen. During the toxic stage there may be incontinence of urine and feces. Postmortem there is some edema of the gastric mucosa, with increase in mucus, and numerous deep red areas of ecchymosis. The liver is brownish, and the cut section is lusterless and very friable, and easily crushed. The kidneys show injection of the surface and internal vessels, and there is some cloudy swelling. During life the urine may show a trace of albumin, with or without casts, and is usually acid to methyl red. Acetone bodies are usually absent. A volatile compound, which reduces Fehling's solution, may be present. Krol [1] found an increased ammonia output in dogs. There may be a preliminary period of anuria. The blood often is a deep chocolate color, such as is seen in conditions in which methemoglobin is formed, and is spectroscopically similar. Harrop and Benedict [2] report definite findings of acidosis in a case of methyl alcohol poisoning, with the bicarbonate content of the blood plasma equivalent to 36.4 per cent. by volume of carbon dioxid.

The mode of action of the toxic agent is still problematic. Wassermann and Keysser [3] considered the effect to be due to "toxic peptids" resulting from the action of the methyl alcohol on the proteins of the gastro-intestinal tract. Stadelmann [4] suggested that the slow oxidation of the alcohol resulted in accumulation, with resulting toxic action. Magnus-Levy [4] pointed out a possible similarity between the action of the poison and that of the toxins and ptomains, and suggested the possibility of methylization of compounds found in the body. Kobert [5] noted the selective poisonous action of the drug on different people, describing it as due to idiosyncrasy. Others consider the poisonous action to be due to the impurities, as furfurol and oxalic acid. The action of the pure and impure wood alcohol, however, does not seem to differ materially (Hunt,[6] Buller and Wood [7]), and the impurities themselves give other or less marked symptoms, in the amounts present (Baskerville [8]). Pohl [9] considered that the toxicity was caused by the formation of formic acid from incomplete oxidation of methyl alcohol. The resulting "acidosis" was considered by Krol [1] to be the cause of the syndrome.

The symptoms seem to group themselves into a paralysis or depression of the medulla and the cranial autonomic system, sometimes the sacral, with an additional selective action on the optic and possibly other nerves. Symptoms may be severe after the drinking of small amounts, or light even after large amounts have been taken, depending on the individual and his previous experiences, as well as the conditions under which the methyl alcohol was taken, as after heavy excesses with ethyl alcohol.

TREATMENT

Various treatments have been recommended. These consist of stimulants, as caffein, epinephrin, strychnin, digitalis, camphor, oxygen, pilocarpin and also potassium iodid. Gettler and St. George [10] suggest saline or sodium bicarbonate infusions and phlebotomy, with repeated gastric and rectal lavage. Fenton [11] reports a case of blindness in which the patient recovered after the instillation of ethylmorphin hydrochlorid (dionin) into the eyes. The patient was taking sodium bicarbonate freely by mouth. Harrop and Benedict [2] report the recovery of a patient following 5 per cent. solution of sodium bicarbonate intravenously.

The plan of treatment is based on alkalization and elimination. If the patient is not comatose, and is received within twelve hours after taking the wood alcohol, it is well to pass a stomach tube and wash out the contents with a 1 or 2 per cent. solution of sodium bicarbonate in warm water, as experience has shown that some of the alcohol is excreted into the stomach. Three or four ounces of a 50 per cent. solution of magnesium sulphate solution are then poured in through the tube and left in the stomach. Sometimes an hour or more after washing, the patient will vomit a considerable amount of food débris having a marked odor of methyl alcohol. The patient is put to bed, kept warm if his temperature is low, and is given 3 gm. of sodium bicarbonate, with about 250 c.c. of water every two hours, for about six doses, being awakened for his medication if asleep. Sometimes a whiff or two of aromatic spirits of ammonia will serve to awaken the patient enough to make him swallow. The dose of bicarbonate may be doubled without apparent ill effects.

1. Krol, J.: Ueber das Wesen der Methylalkoholvergiftung, Arch. f. exper. Path. u. Pharmakol. 73: 450 (July 3) 1913.
2. Harrop, G. A., Jr., and Benedict, E. M.: Acute Methyl Alcohol Poisoning Associated with Acidosis, J. A. M. A. 74: 25 (Jan. 3) 1920.
3. Wassermann, M., and Keysser, F.: Ueber Toxopeptide, Folia Serolog. 7: 243, 1911.
4. Stadelmann and Magnus-Levy: Ueber die in der Weihnachtszeit vorgekommenen Massenvergiftungen in Berlin, Tr. Berlin med. Gesellsch., Oct. 1, 1912.
5. Kobert, R.: Kompendium der praktischen Toxikologie, Ed. 5, 1912, p. 219.
6. Hunt, Reid: The Toxicity of Methyl Alcohol, Bull. Johns Hopkins Hosp. 13: 213 (Aug.-Sept.) 1902.
7. Wood, C. A., and Buller, Frank: Poisoning by Wood Alcohol, J. A. M. A. 43: 972 (Oct. 1), 1058 (Oct. 8), 1117 (Oct. 15), 1213 (Oct. 22), 1289 (Oct. 29), 1904.
8. Baskerville, C.: Air Impurities: Dusts, Fumes and Gases, New York M. J. 96: 1061, 1119, 1912.
9. Pohl, J.: Ueber die Oxydation des Methyl- und Aethylalkohols im Thierkörper, Arch. f. exper. Path. u. Pharmakol. 31: 281, 1893.
10. Gettler, A. O., and St. George, A. V.: Wood Alcohol Poisoning, J. A. M. A. 70: 145 (Jan. 19) 1918.
11. Fenton, R. A.: Use of Dionin in Wood Alcohol Blindness, Northwest Med. 19: 22 (Jan.) 1920.

After this the patient is given 3 gm. of sodium bicarbonate with a glass of water, three times a day, one hour before meals, until the symptoms have disappeared. A safe guide as to the dose is to keep the fresh urine alkaline to methyl red. Fluids are forced, and a liquid diet is given until the acute symptoms are over. Then the diet may be as varied as the patient wishes.

If the patient is comatose, or if the cyanosis is very marked, with respiration much depressed, it is well not to wash out the stomach at first. In this case, or if medication by mouth is not retained, 1,000 c.c. of Fischer's [12] sodium carbonate (0.37 per cent.) sodium chlorid (1.4 per cent.) solution at 99 F. is given slowly intravenously. *In case of any doubt, it has been our custom to give the intravenous injection.* No ill effects have been noted. If there is time and there seems to be congestion of the venous circulation from embarrassment of the right side of the heart, it may be well to draw off from 100 to 300 c.c. of blood before giving the intravenous injection. Half may be given at one time, and the other half later. It has usually not been necessary to give a second intravenous injection on succeeding days. A spinal puncture may be made if there is much restlessness and if there are signs of cerebral compression. After Fischer's solution there appears to be a dehydration of the nervous tissue, with increase in spinal fluid. The breathing usually improves rapidly, the mental state clears, and in from six to twelve hours the cyanosis has virtually disappeared. There is rapid improvement in the eye signs, the blurring of vision disappearing in from twelve to twenty-four hours, or sometimes a slightly longer period. The abdominal tenderness soon disappears.

It is well for the patient to remain in bed until the cyanosis has disappeared and the mental confusion has cleared. The bowels should be kept open with magnesium sulphate. The average stay in the hospital was about five days, with treatment for two or three days. It is well to have the patient return at intervals to note whether or not there has been any permanent damage. All our patients have had hæmatropin or atropin sulphate dropped into their eyes for ophthalmoscopic examination.

The tables show some of the symptoms and effects of alkalization as opposed to the method of stimulation only. The toxic symptoms are produced by widely different amounts in different individuals. The blurring of vision varied from inability to count fingers held about a foot in front of the eyes to inability to read ordinary newspaper print, but with enough light

TABLE 1.—EXAMPLES OF CASES TREATED BY ALKALIZATION

Case	Material Taken	Condition	Blurring of Vision	Treatment Begun (After Taking)	Treatment	Comment
E-4829	½ pint denatured alcohol (with methyl)	In coma; cyanotic; pupils dilated; blood deep chocolate color	+	4 to 10½ hours	1,000 c.c. Fischer's solution intravenously; later, gastric lavage; sodium bicarbonate, etc., by mouth	Improvement within 8 hours; after third day all symptoms had gone; recovered with no impairment of vision
E-4837	1 pint denatured alcohol (with methyl)	Semicomatose, cyanotic, "head felt numb"	+	Over 12 hours	Gastric lavage; magnesium sulphate; 900 c c of Fischer's solution intravenously; later, sodium bicarbonate, etc., by mouth	Rapid recovery with no impairment of vision; discharged after 5 days
E-4908	½ pint denatured alcohol (with methyl)	Recovered consciousness on reaching hospital; slight cyanosis; pupils dilated, nausea, headache	+	About 4 hours	Gastric lavage; magnesium sulphate; sodium bicarbonate, etc., by mouth	No symptoms remained after second day; recovered with no impairment of vision
E-678	1 pint of whisky; 4 ounces of bay rum	Conscious, "going blind," face flushed; abdominal pain; conjunctiva congested	+ Within 1 hour	2 to 3 hours	Gastric lavage; magnesium sulphate; enema; sodium bicarbonate and citrate, 40 grains of each, every hour, with 8 ounces of water	Complete clearing up of vision in three days
E-4963	Lemon extract, 24 oz.; 3 or 4 drinks of supposed "whisky" (with methyl alcohol)	Cyanosis; vomiting; headache; vertigo; conscious	+	4 to 5 hours	Gastric lavage; magnesium sulphate; 1,000 c.c. Fischer's solution intravenously; spinal puncture; later sodium bicarbonate, etc., by mouth	After 12 hours, vision was "as clear as before" the attack; complicating syphilis, chronic alcoholism, aortic dilatation; discharged with no bad effects
E-5165	1 ounce of supposed "whisky" (with methyl alcohol)	Felt weak; dizzy; stuporous; pain in abdomen; vomiting; slight cyanosis	+ Congestion of nasal halves of both disks	8 hours	Gastric lavage; magnesium sulphate; colonic flushing; 800 c.c. Fischer's solution intravenously; later sodium citrate and bicarbonate by mouth	Vision clear by third day; discharged recovered

TABLE 2.—CASES NOT RECEIVING ALKALIZATION

Case	Material Taken	Condition	Blurring of Vision	Treatment Begun (After Taking)	Treatment	Comment
D-4440	8 ounces of "wood alcohol"	Dyspnea, cyanosis, cough and rusty sputum in last 8 hours; tremors	?	About 20 hours	Gastric lavage; magnesium sulphate; later, sodium bromid, scopolamin, tr. digitalis, caffein sodium benzoate; atropin sulphate (each when indicated)	Developed pneumonia and died
F-4804	4 or 5 (?) ounces of clear solution containing wood alcohol	As in Case F-4829, marked respiratory depression with cyanosis; pupils dilated; unconscious	?	Not known; probably within 4 hours	Caffein sodium benzoate; epinephrin	Respiratory paralysis; patient died within ½ hour after reaching ward

12. Fischer, M. H.: Oedema and Nephritis, New York, Ed. 2, 1915, p. 549.

perception to count fingers. These conditions had cleared up entirely at the time of discharge.

SUMMARY AND CONCLUSIONS

1. Wood alcohol appears to vary in its toxicity to different individuals.

2. The symptoms are those of depression of the medulla and the cranial autonomic system and at times the sacral. There is usually a marked and early effect on vision.

3. There is a rapid disappearance of the symptoms (unless the anatomic injury is beyond repair) with intensive alkali therapy.

4. Many of the toxic symptoms probably accompany the acidosis, which may be associated with the acid production (formic) and with methemoglobinemia.