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Important

* Phacolytic hemorrhage in optic chiasma

Pair of

extrinsic flattened nuclei 564

A Study of Addiction to Nonethyl Alcohols and Other Poisonous Compounds¹

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Paraldehyde 576

INTRODUCTION

AMONG the alcoholic patients admitted to the Boston City Hospital, we have been impressed by a small group of men who stand out because of an unusual willingness to ingest alcohols other than ethyl. Since these patients resemble alcoholics in other respects but seem more intense in their addictiveness, we have taken to calling them "superalcoholics." We are reporting these cases because of their intrinsic interest and in the hope that the study of extreme forms may throw light on the somatopsychic pathology of alcohol addiction generally.

Brain
Methanol
Optic Chiasma
Hangover
Alcoholics

LITERATURE

1. Statistics.—There is relatively little statistical information concerning the ingestion of toxic (nonethyl) alcohols in the United States or elsewhere. A report in 1935 (1) pointed out that "a disturbing amount of drinking of methyl alcohol" was occurring in Great Britain. The author stressed the uniformly poor prognosis in methyl alcohol intoxication. In a Massachusetts study of deaths

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(412)

568 Hangover effect?

* maybe the adaptive mechanism involves the destruction of the mechanism that metabolizes the methanol by formaldehyde.

from poisoning (2), toxic alcohols were an important factor in the causation of death. This study covered the 10-year period 1928-1937 in which 8,661 deaths were certified by the medical examiners as due to toxic substances. Alcohol ingestion (ethyl alone or in combination with other toxic substances) was responsible for 52 per cent of all such deaths. Within this group, alcohols other than ethyl caused 108 deaths, and the compounds named included Alcorub, amyl alcohol, "auto" alcohol, antifreeze, bay rum, solidified alcohol, "canned heat," denatured alcohol, jamaica ginger, methyl alcohol, Prestone, Sterno, and witch hazel.

A total of 908 cases were reported in which death resulted from the ingestion of "nontherapeutic chemicals," defined by the authors as "substances never presented by physicians to be taken by mouth or by ingestion." This group included the previously mentioned nonethyl alcohols and a large variety of other toxic agents. Of all such cases, 55 per cent were suicidal and 40 per cent accidental, others being homicidal or unknown. In this over-all group of cases, 14 per cent were alcoholics. However, it was not reported whether the group which ingested the toxic alcohols were alcoholics. Moore (3) reported a series of 1,195 cases admitted to the Boston City Hospital for attempted suicide during the period 1915-1939, among which only 143 (11 per cent) were alcoholics. He concluded that suicide is not a frequent complicating factor among alcoholic patients in the Boston area. It would appear that the alcoholic usually ingests toxic alcohols either accidentally or for beverage purposes but in any case for reasons other than to commit suicide.

It is well known that the alcoholic will often take extreme measures to obtain alcohol. The usual motivation ascribed for the ingestion of toxic alcohols is lack of funds to secure regular wines, beers or liquors. Myerson (4), for example, states that "in their destitution, bay rum, vanilla extract and muscatel were luxuries. For the hearty, even rubbing alcohol could be used if taken with crackers and grass to prevent vomiting. On a Sunday, when the stores were closed, Sterno (canned heat) served as a source of supply for these desperate men." Though the ingestion of toxic alcohols as described above is regarded as commonplace in the "Skid Row" alcoholic, no actual statistics on its frequency or on the resulting complications have been reported.

Two reports (5, 6) have appeared recently on addiction to "methyated spirit" in Great Britain. "Methyated spirit," which is

ethyl alcohol with the addition of 4 per cent methyl alcohol, is used commercially as a paint thinner. In the cases reported, the use of this substance was ascribed to "an exclusively economic reason." The typical addict was described as possessing "emotional blunting, a tendency to vagrancy and pessimism of outlook and depression." No toxicology, physical examinations, or laboratory studies were reported. Treatment was described as being similar to that for other alcoholics, and the prognosis for cure was considered good. Details of therapy, period of follow-up, classification of type of alcoholism or complications from alcoholism were not mentioned.

2. *Toxicology.*—The great variation in individual susceptibility to methyl alcohol has long been recognized. Simons (7) notes the large differences in the amounts of methyl alcohol which produce amblyopia. General individual variations have been pointed out by Sroka (8), who calls them "enormous." Two recent reports of pure methyl alcohol poisoning, by Bennett and his co-workers (9) and Tonning, Brooks and Harlow (10), and the review by Røe (11), attest to the toxic nature of methanol, though until 1923, when Rief (12) published his findings in the Hamburg outbreak, there was doubt about its toxicity. Studies in mice, rabbits and dogs had led to confusing results, and the validity of the Richardson rule (in a homologous series of alcohols toxicity increases as the number of carbon atoms increases) was not questioned by earlier workers.

The symptoms of methyl alcohol poisoning usually begin after a latent period of from 12 to 19 hours following its ingestion. The earliest are weakness, anorexia, headache, nausea and, somewhat later, vomiting; these are followed by dyspnea, pain in the back and extremities, and severe abdominal pain. Simultaneously or shortly thereafter amblyopia appears and may go on to amaurosis. In the most severe cases, where death occurs, respirations are slowed and eventually stop in full inspiration, with the heart continuing to beat for a few minutes or for as long as 2 hours.

Laboratory findings are remarkable for the marked depression of the plasma bicarbonate; in about half the cases reported by Bennett and his co-workers (9) it was below 20 mg. per liter. Fatalities are most frequent in those with the most severe acidosis. Other electrolytes are only slightly abnormal except for potassium,

which is frequently found to be low, and this latter deficit is reflected in abnormal serial electrocardiograms. Serum amylase was elevated in the Bennett and co-workers series, but was not reported in other series. Other laboratory findings, except for a mild rise of cerebrospinal fluid pressure, are usually not remarkable.

The specific pathology (9, 13, 14) associated with methanol poisoning is characterized by the marked changes in the ganglion cells of the retina. These are enlarged and globoid in shape, and their flattened and distorted nuclei are displaced to the periphery. The dendrites are difficult to see even with silver staining, and there are only sparse remnants of tigroid substance. The cellular changes are clearly irreversible. Punctate hemorrhages are seen in the optic chiasm, and minute extravasations of red cells are seen on microscopic examination. The optic nerves are edematous and hyperemic. The brain is grossly edematous and there may be herniation of the cerebellar tonsils. Microscopically the ganglion cell changes are less pronounced than those in the retina and are probably reversible.

The other organs, as reported by Bennett and co-workers (9), revealed congestion of the lungs, epicardial hemorrhages, mild fatty infiltration of the liver, gastritis, and general congestion of the abdominal viscera. One additional finding in this series was pancreatic necrosis in 13 of 17 cases, consistent with the elevated serum amylase levels.

Therapy is well outlined by both Bennett and Tønning. It is directed toward the rapid correction of the acidosis and other electrolyte abnormalities, especially potassium. Ethyl alcohol (15) is occasionally used in addition to bicarbonate replacement, with varying success. When the therapy is vigorous, in favorable cases, recovery occurs within 24 to 72 hours.

The toxic effects of the higher alcohols vary somewhat from those of methyl alcohol (16, 17). The symptoms of poisoning with isopropyl alcohol are similar to those in methanol poisoning. Small doses cause very little exhilaration but, instead, dizziness, nervous and muscular disturbances, and later headaches of long duration. Large doses result in a marked fall in blood pressure, gastrointestinal hemorrhage, coma, and acute renal insufficiency resulting in anuria and uremia, with subsequent enlargement of the liver and secondary anemia. Laboratory studies (18) reveal acetonuria with-

out acidosis in contrast to the situation in methanol poisoning where acidosis usually occurs without acetonuria.

Fusel oil, a mixture of higher alcohols, is found in crude brandies and whiskies. In small amounts it is toxic to the mucous membranes, and produces coughing, dyspnea and headache. In larger amounts it can lead to deep coma, cyanosis and respiratory collapse. It is thought that its toxicity is increased by ethyl alcohol, whereas the toxicity of methyl alcohol is decreased by ethyl alcohol.

Ethylene glycol (antifreeze) ingestion is followed in several hours by moderate exhilaration, paresis of certain muscle groups, mydriasis, diminution or elimination of reflexes, and stupor and coma. Vomiting occurs frequently. The pulse is usually rapid, and respirations slowed. Anuria and uremia are seen terminally. The urine frequently contains large amounts of oxalic acid which may be responsible for the renal lesions.

Paraldehyde ingestion produces nonspecific symptoms of nausea and vomiting, headache and dizziness in the acute phase. In autopsied cases, where fatal amounts have been taken, the chief pathology is inflammation of the stomach and congestion of the viscera. Chronic ingestion of paraldehyde leads to disturbances of digestion, emaciation, muscular weakness, confusion, hallucinations and, in extreme instances, delirium tremens.

The metabolism of methanol can be summarized briefly as follows: It is absorbed through the gastrointestinal tract or through the lungs within a few minutes or hours. Its distribution through tissues is according to water content. Some excretion occurs through the lungs and kidneys. Elimination takes place chiefly by oxidation to formaldehyde and formic acid, with further slow oxidation to carbon dioxide, probably by the same enzyme system, alcohol dehydrogenase, as is used by ethanol (19). The toxic effects of methanol have been attributed to the oxidation products formaldehyde and formic acid, though formaldehyde has never been implicated directly, and the rate of formation of the formic acid does not coincide exactly with the clinical symptomatology.

Thus far we have discussed the effects of poisoning with single substances. When methanol is ingested together with ethanol, the development of acidosis is inhibited and the latency period is delayed until the ethanol is metabolized, the usual toxic symptoms of methanol beginning only after the elimination of ethanol. In vitro studies by Zatman (20) reveal that methanol oxidation by

impure alcohol dehydrogenase is inhibited by ethanol, even with amounts as low as one-sixteenth the concentration. Bartlett (21) confirmed these findings in vitro with liver tissue, and in vivo with rats, using the excretion of labeled $C^{14}O_2$ as the measure of inhibition of methanol by ethanol. Thus if the concentration of ethanol remains not far out of proportion to the methanol, then the slower metabolism of methanol, about one-fifth that of ethanol, may proceed sufficiently to prevent any of the sequels of methanol poisoning. The rationale for repeated ingestion of ethanol during the period of high methanol level is therefore clear. The confusion of earlier workers concerning the toxicity of methanol was due to a lack of understanding of this mechanism.

3. *Psychiatric aspects of alcoholism.*—The available literature is devoid of references to psychiatric aspects of nonethyl alcoholism. Many volumes of material have been written on the psychiatric aspects of ethyl alcoholism and it is not intended to attempt even a brief review here. We do wish to emphasize certain features of the literature which bear on the data and conclusions of the present study.

Not all writers on alcoholism believe that it is a form of addiction. Diethelm (22), for example, separates alcoholism from drug addiction. He believes that, while the typical alcoholic develops tolerance to drink, he remains at this level of tolerance. If alcohol consumption is rapidly increased the individual gets sick and tolerance actually decreases. Diethelm questions whether the alcoholic experiences the typical craving, tolerance and physiological changes which are characteristic of addiction.

Most writers, however, believe that alcoholism is a form of addiction. Psychological addiction to alcohol is said to occur because alcohol depresses the cerebral cortex and reduces anxiety. Thus the less an individual is able to tolerate anxiety the greater is his vulnerability to psychological addiction. Physiological addiction occurs when alcohol becomes an essential part of the body's metabolism. Presumably this happens if the subject ingests alcohol long enough (23).

Various theories have been put forward to explain alcohol addiction. Williams (24) postulates a genetotrophic factor in some individuals that requires the presence of alcohol in their bodies for proper metabolism. He has been unable to identify the responsible

substance. Roe (25) seems to have ruled out the possibility of a specific hereditary factor. Experiments with laboratory animals (26) have not settled the question whether animals can be addicted to alcohol.

There is no agreement regarding the psychodynamics of alcoholism. Psychoanalysts note the action of alcohol in releasing inhibitions and repressions and they stress, variously, infantile cravings, oral-dependent needs, latent homosexual and heterosexual drives, and hostile impulses. Kant (27) emphasizes emotional immaturity, insecurity, and chronic anxiety. Alexander (23) describes a cyclic phenomenon in alcoholics: Phase I is elation and release of inhibited tendencies, phase II is depression, and phase III is the hangover. The alcoholic now reexperiences the emotional stress which originally caused him to drink and has, in addition, the guilt and shame due to the drinking itself. He tries to escape by returning to phase I and the euphoric effect of alcohol. "This at the same time removes both inhibitions and self-criticism by dulling the higher faculties of discriminatory judgment."

Knight (28) divides alcoholics into two groups: essential alcoholics who drink to achieve gratification of their intense oral needs, and reactive alcoholics, who drink because of unpleasant events in their environment. The latter seek in drink escape from unpleasant internal and external stress by dulling the higher discriminatory functions of the central nervous system, thus alleviating anxiety. The former use alcohol to regress to the state in which infantile oral impulses are gratified and the associated feelings of childlike omnipotence are achieved, all the realities of life becoming temporarily dissolved in alcohol. A vicious cycle establishes itself in this group. They feel frustration and rage toward the adult world which cannot satisfy their intense oral cravings. The rage causes guilt, need for punishment and self-debasement, which in turn bring on even greater oral strivings and alcoholism as relief from these intolerable feelings. The more they drink the worse they feel, and the worse they feel the more they drink.

CASE REPORTS

Case 1.—V. C., a 56-year-old white divorced man was admitted to the Boston City Hospital with the diagnosis of acute alcoholic hallucinosis following a 9-week episode of heavy drinking. He developed delirium tremens and lobar pneumonia while in the hospital. During the period of drinking prior to his admission he had eaten little food and his major

beverages were cheap wines and Sterno.⁶ For 3 days prior to his admission his only intake was Sterno and it was estimated that he drank three or four large (4-oz.) cans per day.

The patient gave a history of 25 years of alcoholism. During the past 10 years he had been drinking large quantities of Sterno and occasionally smaller quantities of rubbing alcohol. He was a well-developed and well-nourished white man. His vital signs were within normal limits and his chest and heart were normal. His liver was palpable one and a half fingers below the right costal margin and was slightly tender. He had no abnormal cranial nerve signs and no motor or sensory deficit. His deep tendon reflexes were normal and there were no abnormal cerebellar signs. The urine and blood constants were within normal limits. Blood non-protein nitrogen was 29, chlorides 102, cephalin flocculation 0, formol gel 0, bilirubin 0.6, bromsulfalein retention 9.4 per cent, and Hinton negative. On admission the plasma CO₂ was 15.5 mEq per liter but after adequate intravenous fluid therapy it rose to 23.5 within 24 hours. The delirium tremens was controlled with bed rest, intravenous fluids, vitamin therapy, and thorazine. The pneumonia which developed responded well to penicillin and the patient was discharged in good condition 2 weeks after admission.

The patient was at all times cooperative and pleasant. He stated that he had started drinking heavily at 18 and by 23 considered himself a severe alcoholic. He said, "I liked the feeling I got out of it. It made my work easier, it gave me more ambition." He had begun drinking large quantities of Sterno about 10 years ago. When asked if he had any ideas of its toxic properties he stated, "I had an idea it wasn't fit to drink but I saw other people drink it and so I did." He said that it was not always economic necessity that prompted him to drink it but rather the greater feeling of obliteration which it produced. He added that Sterno had greater sedative properties when he was tapering off his drinking.

Case 2.—G. R., a 60-year-old white single man was brought to the Boston City Hospital in a semicomatose condition by the police. He had been well known to the hospital since 1947 as a severe chronic alcoholic. The police brought in substances which the patient had ingested during his last 5-week drinking spree. They included the following:

1. Big Chief (Isopropyl rubbing alcohol 70 per cent).
2. Lavender Refresher (Isopropyl alcohol 70 per cent).
3. Dickinson's witch hazel.
4. Sea Breeze antiseptic for skin.
5. Roma sherry wine.
6. Petri California muscatel.
7. LeMello California muscatel.
8. Extract of vanilla (41 per cent alcohol).

⁶ Sterno is the trade name of a canned cooking fuel which contains 15 per cent methyl alcohol.

Tomato
Juice
contains
Methanol

9. Septive mouth wash (25 per cent alcohol).
10. Methyl salicylate.
11. Menthol.
12. Benzoic acid.
13. Thymol.
14. Eucalyptol.

The patient was effusively polite, cooperative and apologetic. He constantly reiterated that he hoped he was not taking too much of the physicians' time and efforts. He reported a history of alcoholism since the age of 20. He began drinking toxic compounds when he was 22 years old. He said these compounds "gave more of a kick." When questioned about the toxic nature of methyl and isopropyl alcohol he replied, "They make you sick in the stomach and they make you blind." When asked if he ever became aggressive or combative when drinking he replied, "I never hated anybody; I only hate myself." There was a past history of subtotal gastrectomy for intractable bleeding due to alcoholic gastritis.

On admission the patient had a temperature of 100 F. and a pulse rate of 120. He was poorly nourished and dehydrated. The liver was palpable three fingers below the right costal margin and there were moist râles and increased breath sounds in the right lung base. Physical and neurological findings were otherwise negative. X-ray of the chest revealed a pneumonia. This cleared rapidly with penicillin therapy.

On admission the examination of the urine for salicylates was positive. (On discharge this test was negative.) The cephalin flocculation was 1-plus, formol gel and bromsulfalein retention were 0.

The patient responded well to intravenous fluids, antibiotics, and nutritional therapy, and was discharged in good condition 10 days after admission.

Case 3.—A. D., a 46-year-old white man, separated from his wife, had been admitted 26 times to the Boston City Hospital since 1944, mostly for alcoholic hallucinosis or delirium tremens. His history included severe diabetes, controlled with insulin, duodenal ulcer, and chronic pancreatic insufficiency. He had begun drinking heavily when he was 26 years of age. The history of ingestion of toxic alcohols (mostly isopropyl) dated back 10 years.

Prior to the present admission the patient had indulged in a 6-week episode of heavy drinking during which he had consumed large amounts of rubbing alcohol. The patient was in delirium tremens on admission and his diabetes was out of control. There were no abnormal cardiac, pulmonary or neurological findings. The liver was palpable two fingers below the right costal margin. The NPN was 33, the cephalin flocculation was 1-plus and bromsulfalein retention 12 per cent.

The patient was ingratiatingly polite and always answered with a characteristic "yes, sir" or "no, sir". During the latter phases of his recovery he volunteered to assist with odd jobs on the ward. He said he

was quite aware of the toxic properties of rubbing alcohol but denied any suicidal intentions. In regard to combativeness or aggressiveness he said, "I never hated a person in all my life. Hate is only a dictionary word. I never had a fight in my life. I am just the type who won't fight."

The patient responded well to intravenous fluids and vitamin therapy. His diabetes was controlled with diet and insulin and he was discharged in good condition 4 weeks following his admission.

Case 4.—P. F., a 38-year-old white man, separated from his wife, was admitted to the Boston City Hospital in a comatose and severely toxic state following the ingestion of a large quantity of paraldehyde. Since his condition was critical, he was transferred to the Peter Bent Brigham Hospital for dialysis via the artificial kidney.

The history was that of heavy drinking since the age of 15. The very first drink taken by this patient had been rubbing alcohol. He vividly recalled that the label of the bottle stated, "causes serious gastric disturbances." This first episode, according to the patient, was prompted by the wish to be "a wise guy" and to "show off" before his friends. He recalled that he became extremely ill with severe gastric pain but he did not consult a doctor and recovered spontaneously. During his 23 years of alcoholism he had continued to drink rubbing alcohol or hair tonic (Vitalis) from time to time. Five years prior to his present admission he had started drinking paraldehyde, with which he had become acquainted in a hospital during an episode of delirium tremens. He gradually developed a preference for paraldehyde over whisky and during the past 5 years had gone to any extreme to obtain it. He had continued to drink rubbing alcohol as well.

In describing his feelings when drinking paraldehyde, the patient said, "I got to like the sensation it gives you. It works faster than alcohol and calms your nerves down quicker." He used any possible means to secure paraldehyde including the forging of prescriptions. As for rubbing alcohol, he admitted knowing that it was toxic but vehemently denied that he ever wished to commit suicide. In describing himself the patient used terms such as "I hate myself, I am disgusted with myself," but in dealing with others he rarely expressed overt hostility.

When admitted to the Boston City Hospital the patient was severely acidotic, with a plasma CO_2 of less than 5.5 mEq per liter. The NPN was 49, and an arterial blood pH was 7.01. This rose to 7.23 after 2½ hours of dialysis. On admission the blood ketones were 2-plus undiluted, but fell to 0 after dialysis. The blood paraldehyde level was 100 mg. per 100 ml. in serum and the acid aldehyde level was 10.6 mg. per 100 ml. in serum. Twenty-one hours following his admission, before dialysis had been instituted, the patient's serum paraldehyde level had fallen to 27 mg. per 100 ml. Before dialysis the patient's urine revealed a 2-plus albumen and a 2-plus sugar; following dialysis the urine was normal. On admission the patient had a leucocytosis of 26,000. The leucocyte

count became normal after penicillin therapy for pneumonia of the lung bases.

The patient's liver was enlarged three fingers below the right costal margin. After dialysis the patient developed gastrointestinal bleeding from alcoholic gastritis. This was successfully treated with a dietary and antacid regimen. The bromsulfalein retention was 3 per cent and the cephalin flocculation 2-plus.

It is of interest to note that the patient's blood paraldehyde level on admission was in a range usually considered fatal and that his spinal fluid was positive in an aldehyde test, yet he was discharged in relatively good condition in 3½ weeks with no apparent hepatic, renal or other consequences.

Case 5.—J. L., a 60-year-old white man, separated from his wife, was brought to the Boston City Hospital from the Long Island Hospital in a confused and disoriented state following the ingestion of four large (4-oz.) cans of Sterno and half a bottle of tincture of green soap. The patient was a known alcoholic of over 40 years' duration. He was a spree drinker with episodes of heavy alcohol ingestion lasting 3 to 4 months followed by intervals of abstinence of 1 to 2 months. During his drinking periods he would consume large quantities of beer and cheap wine and, in addition, large amounts of hair tonic, Sterno, rubbing alcohol and bay rum. He had been living a Skid Row existence for the past 20 years, with many hospitalizations for delirium tremens. During his recent stay at the Long Island Hospital he had managed to obtain large quantities of alcohol from his friends in the form of cheap wine. When these resources failed he secretly took alcohol or tincture of green soap from the surgical carts in the hospital.

When admitted the patient was tremulous and disoriented in time and place. He had chemical burns of the mouth, tongue and pharynx. The vital signs were normal. There were no abnormalities in the cardiovascular, gastrointestinal or pulmonary systems. The liver was enlarged two fingers below the right costal margin. The neurological examination was within normal limits. Urine and blood examinations revealed no abnormalities. The NPN was 20, the bromsulfalein retention 3 per cent, and the plasma CO₂ 51 mEq per liter. Following intravenous fluid therapy, the CO₂ reverted to 25.

Two days after admission the patient was oriented, cooperative and effusively polite. He improved rapidly on conservative therapy and after a week volunteered to leave the hospital, saying that he did not wish to deprive some other patient of a bed. He said that he never showed anger and rarely felt dislike for any person or situation. His behavior in the hospital was in accord with this statement. It is remarkable, considering the severity and duration of the patient's alcoholic history and the large quantities of toxic substances he had ingested, that his physical and mental state were essentially intact.

Case 6.—C. L., a 47-year-old white divorced man, had a history of 33 prior admissions to the Boston City Hospital, all for complications of chronic alcoholism. His heavy alcoholism had begun at age 21. In the subsequent 26 years the patient had had delirium tremens more than 30 times. He was in the habit of drinking approximately 5 cans of Sterno per week. Prior to his current admission he had ingested 4 or 5 cans of Sterno in one day and when brought to the hospital he was disoriented, confused and agitated.

The patient was well developed and well nourished. The vital signs were normal. The liver was palpable three fingers below the right costal margin. There was a nutritional peripheral polyneuropathy, with decreased sensation to pain, touch and vibration in both legs, and absent ankle jerks. There were no specific cardiac, pulmonary or gastrointestinal signs or symptomatology. The cephalin flocculation was 2-plus, bromsulfalein retention 12 per cent, and the NPN 25. The blood methyl alcohol concentration was 0.014 mg. per 100 ml. and the blood acetaldehyde level 3.5 mg. per 100 ml. The patient responded well to intravenous fluid and vitamin therapy. He was discharged 4 weeks after his admission in relatively good condition, though with a residual peripheral polyneuropathy.

This patient was extremely polite and cooperative. He was always a quiet, solitary drinker, who never engaged in any form of violence. He professed only the most pleasant and congenial feeling for all members of society and denied any feelings of anger or hatred even when provoked. In describing his feelings about Sterno he said it had a definite advantage over ethyl alcohol. "It makes you feel crazy and it makes you feel foolish. It deadens your nerves better." The patient also reported occasional drinking of rubbing alcohol during the past 17 years, and described its effects. "It makes you sick as hell, makes your stomach swell up." Despite these symptoms and the realization of the toxic potency of rubbing alcohol, the patient continued to drink it.

Case 7.—F. S., a 46-year-old white divorced man, was admitted to the Massachusetts General Hospital following a 3-month period of heavy drinking during which he consumed large amounts of wine and beer and several large cans of Sterno each week. Two weeks before admission his left foot had become painful, red and swollen. On admission he was febrile and had a leucocyte count of 17,500. He was tremulous, agitated and somewhat depressed. Two days after his admission he began having visual and auditory hallucinations, became progressively more confused and developed frank delirium tremens.

This patient presented a history of 27 years of alcoholism. During all this time he had consumed large amounts of Sterno. He said he used the Sterno not because he could not afford to buy whisky but because the feelings he obtained from it were far more pleasurable than those from regular alcohol. He would drink as much as four or five large (4-oz.)

cans at one time. During a period when he was working as a woodcutter in the northern Maine woods, Sterno was his only alcoholic beverage. He described the effects as follows: "It makes you feel light. You think you are on top of the world. It gives you courage." Later he modified this statement by saying, "It gives you false courage."

During the patient's long history of alcoholism he had had many episodes of delirium tremens and "rum fits." He also presented a long history of tuberculosis. The chest X-ray and sputum examinations were negative at this time.

The patient never became aggressive or belligerent while intoxicated. He said, "I've been too easy going, I've tried to keep calm. I never hated anyone. I try to make friends with everybody." He realized that his large intake of Sterno could be poisonous but he rationalized by saying, "I know plenty of people who drink it and it never hurts them." When confronted with the possibility of his dying of poisoning he said, "If I die within a half hour I'd be just as happy. The best time of your life is when you're dead." Yet the patient vigorously denied any frank suicidal desires and felt he would never "have the courage" to take his own life.

On admission the patient had an oral temperature of 100 F. and a pulse rate of 115. His skin and tongue were dry. There were diminished breath sounds at the right apex of the chest and cardiac examination revealed a grade 1 apical systolic murmur. The liver was palpable two fingers below the right costal margin. There were spider angiomas on the upper thorax. The right foot was hyperemic, red, tender and edematous, with moderately advanced varicosities. The neurological examination was within normal limits. The infectious cellulitis of the left leg responded well to penicillin and streptomycin therapy. He improved steadily and was discharged in good condition 10 days following his admission. On discharge the NPN was 25, cephalin flocculation, urine, and blood findings were within normal limits, and the bromsulfalein retention was 8 per cent.

Case 8.—F. T., a 50-year-old white male, separated from his wife, had had over 40 visits to the emergency wards of both the Massachusetts General Hospital and the Boston City Hospital during the past 10 years. He had been admitted to each hospital on numerous occasions because of overt or impending delirium tremens. The patient was a confirmed alcoholic of 25 years' duration and during the past 10 years he had become addicted to paraldehyde as well. He visited numerous hospitals in the Boston area presenting a somewhat confused mental status, with tremulous psychomotor activity, and always requested paraldehyde for sedation. He usually managed to receive paraldehyde, and during the past 2 years had used it almost as his sole beverage. The patient would remain intoxicated for periods of 2 to 3 months, during which time he managed to eat fairly well. He would occasionally take

a job as a dishwasher for 2 to 3 weeks but in the past 10 years had never held a job steadily. The patient's physical and nutritional status was good and he showed no abnormalities in the cardiovascular, pulmonary, gastrointestinal or genitourinary system. On admission he was usually disoriented, confused and tremulous, and during his many hospitalizations it took progressively larger amounts of paraldehyde to sedate him.

The patient was always extremely polite and cooperative. He insisted that he would do anything to rid himself of his illness. He was quite sophisticated, when sober, in his discussions of alcoholism, and was always extremely complimentary to the physicians and nursing staff who were aiding him. He became excellent at feigning symptoms of hallucinosis and delirium tremens and it was difficult to know whether his symptoms were real or merely acted out in attempts to obtain paraldehyde.

Except for varying degrees of dehydration and mild acidosis which responded well to intravenous fluid therapy, the patient presented no specific abnormal physical findings on his present admission to the Massachusetts General Hospital. His blood and urine were within normal limits. The NPN was 23, cephalin flocculation 1-plus, and bromsulphalein retention 8 per cent.

Case 9.—W. T., a 42-year-old white married man, was admitted to the Massachusetts General Hospital after a 5-week period of heavy drinking. He drank all varieties of alcohol but during the latter weeks he had consumed mostly wine. He continued drinking until an hour before entering the emergency ward.

Except for his alcoholism the patient had been in relatively good health. He had had his first drink at age 14. From then until he was 23 he drank sporadically; at that age he embarked on longer periods of intoxication. He did most of his drinking alone and ate very little food during his bouts, which lasted 3 to 4 months. He described his goal in drinking as "just drink to get blind." The patient said that when he drank he often became belligerent and engaged in violent fights. This report, however, was contradicted by his wife. She stated that the patient became quiet and docile when drinking and described the patient's own account of his behavior as a figment of his imagination. The patient had periods of abstinence ranging from 3 to 14 months, during which he worked regularly as a longshoreman.

Fifteen years ago the patient had had his first drink of isopropyl alcohol and was aware that it had toxic properties. He said that he saw other individuals drinking it and thus minimized its toxic potentialities. He realized, however, that isopropyl alcohol was a poisonous compound and clearly recalled the statement on the bottle, "not to be taken internally." He described the feeling achieved from drinking isopropyl alcohol as, "It just deadens you, controls you. You just feel dead." He vigorously denied suicidal thoughts.

When admitted the patient was acutely tremulous and had visual and auditory hallucinations. In addition, he had severe cramps in both legs, which were hyperemic, slightly edematous, and extremely sensitive to touch. The knee jerks were hypoactive and the ankle jerks could not be obtained. There were a few râles in both lung fields. The liver was enlarged two fingers below the right costal margin and was tender at the edge. The patient's urine and blood were within normal limits. The cephalin flocculation was 3-plus and the bromsulfalein retention 10 per cent. The patient improved rapidly with bed rest, vitamins, and nutritional support. He was discharged 5 days after admission in good condition but with a persistent peripheral polyneuropathy.

RESULTS

Charts I and II demonstrate the following salient facts: All the patients were adult white males. All had been addicted to ethyl alcohol for periods ranging from 19 to 40 years. All were addicted, in addition, to toxic alcohols for periods ranging from 10 to 40 years. In four of the nine men the drinking of toxic alcohol began at the same time as ethyl alcohol ingestion.

CHART I.—Summary of Clinical Data in Nine Patients Addicted to Nonethyl Alcohols

CASE, AGE (ALL W. ♂)	DURATION OF CHRONIC ALCOHOLISM (YEARS)	DURATION OF TOXIC ALCOHOL INGESTION (YEARS)	TYPES OF TOXIC ALCOHOLS & AGENTS	HEPATIC STATUS				NEUROLOGICAL STATUS
				HEPATO- MEGALY	ICTERUS & ASCITES	B S P %	CEPH.FLOC.	
(1) V.C. 56	25	10	METHYL ISOPROPYL	1+	0	9.4	0	DT'S
(2) G.R. 60	40	38	METHYL ISOPROPYL METHYL SALICYLATE	4+	0	0	1+	DT'S
(3) A.D. 46	20	10	ISOPROPYL	2+	0	12	1+	DT'S
(4) R.F. 38	23	23	ISOPROPYL PARALDEHYDE	3+	0	3	2+	COMATOSE
(5) J.L. 60	40	40	METHYL ISOPROPYL	2+	0	3	0	ACUTE INTOXICATION
(6) C.L. 47	26	26	METHYL ISOPROPYL	3+	0	12	2+	PERIPHERAL NEUROPATHY
(7) F.S. 46	27	27	METHYL	2+	0	8	0	DT'S HALLUCINOSIS
(8) F.T. 50	25	10	PARALDEHYDE	0	0	8	1+	DT'S
(9) W.T. 42	19	15	ISOPROPYL	3+	0	10	3+	HALLUCINOSIS PERIPHERAL NEUROPATHY

CHART II.—Summary of Laboratory Data in Nine Patients Addicted to Nonethyl Alcohols

CASE	TOXIC ALCOHOL INGESTED PRIOR TO ADMISSION	LAB. STUDIES			CLINICAL TOXICOLOGY
		CO ₂ mEq/L	ELECTROLYTES mEq/L	NPN mg. %	
(1) V.C.	METHYL	DAY 1 = 15.5 DAY 2 = 23.5	NORMAL	29	.
(2) G.R.	METHYL SALICYLATE	DAY 1 = 23.2	NORMAL	21	URINE + FOR SALICYLATE SERUM KETONE 2+ (UNDILUTED)
(3) A.D.	ISOPROPYL	DAY 1 = 25.0	NORMAL	33	
(4) P.F.	PARALDEHYDE	DAY 1 = 45.5 DAY 2 = 14.2 DAY 4 = 25.8	DAY 1 Na = 135 K = 8.8 DAY 4 130 3.2	24	SERUM PARALDEHYDE DAY 1 = 100 mg. %
(5) J.L.	METHYL	DAY 1 = 15.0 DAY 2 = 25.0	NORMAL	20	
(6) C.L.	METHYL	DAY 1 = 25.9	DAY 1 Na = 140 K = 2.7 DAY 2 134 3.8	25	
(7) F.S.	METHYL			25	
(8) F.T.	PARALDEHYDE	DAY 1 = 24.5	NORMAL	23	
(9) W.T.	ISOPROPYL	DAY 1 = 25.2	NORMAL	29	

All the patients were hospitalized following an episode of heavy intake of ethyl alcohol along with the ingestion of one or more toxic organic substances. None had attempted suicide. While a wide variety of toxic substances was ingested, the two most common were methyl alcohol and isopropyl alcohol.

Five of the nine patients were in delirium tremens when admitted or developed it shortly thereafter. Two had alcoholic hallucinosis, one was comatose, and one was acutely intoxicated. All but one had evidence of hepatomegaly but none was jaundiced, or had ascites or splenomegaly, six had normal liver function tests and three tested only slightly abnormal. None showed any evidence of permanent kidney damage.

Three of the patients had pneumonia when admitted. All nine responded well to treatment and were discharged recovered in from 5 days to 4 weeks.

The electrolyte abnormalities in this group of patients were remarkable for their lack of severity. In six cases, sodium, potassium and chloride were within normal limits. In one patient, who had

ingested paraldehyde, the serum potassium was increased, and in one who had ingested methyl alcohol it was decreased. In only two of the four cases of methyl alcohol ingestion were low plasma CO₂ levels found, and these returned to normal by the second day after therapy was instituted. Severe acidosis was found in only one patient, who had probably ingested paraldehyde contaminated with formic acid. This individual responded promptly to electrolyte replacement, and after dialysis on the artificial kidney, his condition returned to normal. In the remaining cases involving paraldehyde, isopropyl alcohol and methyl salicylate ingestion, there was no evidence of uncompensated acidosis. In all cases, renal studies were within normal ranges. In Case 4 the initial paraldehyde level was over 100 mg. per 100 ml., within the fatal range. This fell to 27 mg. per 100 ml. after therapy but before dialysis was begun. The urine was positive for salicylates in the one instance of methyl salicylate poisoning.

There were no evidences of neurological deficit at the time of discharge except in two patients who had peripheral neuropathy. None had Korsakoff's syndrome, Wernicke's syndrome, overt psychosis, or amblyopia.

The personality characteristics of these patients exhibited no uniform or common general pattern. What was striking during their hospital stay was the pleasant, cooperative, effusively polite manner in which they conducted themselves. In addition, all were aware of the poisonous nature of the substances they ingested, but vehemently denied any suicidal intent. All said they preferred the toxic alcohols to ethyl alcohol because of the greater effects produced. They all characterized themselves as individuals who neither felt nor expressed hostile impulses.

DISCUSSION

At first glance it may seem surprising that patients as heavily intoxicated as the men reported on herein, with both ethyl and toxic alcohols, should recover so rapidly and so completely. The explanation probably lies in a number of factors. All our patients were heavily addicted for many years, and presumably had developed increasing tolerance to both ethyl and toxic alcohols. It is possible that cases with fatal outcomes do not reach our wards and were thus selectively omitted from our series.

Our patients drank ethyl alcohol in addition to toxic alcohols. The protective effect of ethanol in methanol intoxication has been discussed above in the section on toxicology. Our patients were spree drinkers; between drinking bouts they maintained themselves in good nutritional status with reasonably well balanced diets. Their livers and nervous systems were thus spared. Nevertheless, hepatomegaly was present to some degree in all but one case, and peripheral neuropathy existed in two.

Another possible explanation should be mentioned, though we have no evidence bearing on its validity. The known individual variation in tolerance to toxic alcohols is great and may become greater in chronic imbibers. Can it be that adaptation occurs through the establishment or reactive overgrowth of specific enzyme systems for the breakdown and metabolism of the offending substances? Organic studies in cases of withdrawal may one day throw light on this hypothesis.

From the psychiatric point of view, the data on these extreme cases favors the view that alcoholism is definitely a form of addiction, contrary to Diethelm's hypothesis. The craving and tolerance of these patients for ethyl and toxic alcohols did not remain static. Such statements as, "It deadens your nerves better," and "It gives you more courage," typify their attitude toward the toxic alcohols and are characteristic of the psychologically addicted. Their history of increasing dosage until amounts were ingested that might have been fatal to a dozen ordinary men, is indicative of their increased physiological tolerance and addiction. The speed with which the toxic substances were metabolized and the great immunity of their body organs are further evidence for the same conclusion.

How can the turning toward toxic alcohols which our patients demonstrated be explained? That the reason was not economic necessity, or ignorance of the danger, or desire for suicide, seems clear. The answer, we believe, lies in the personality of these patients and in the psychodynamics of their alcoholism at the outset. Without going into extensive psychiatric histories, enough clinical data have been presented above to demonstrate that these patients, in spite of their ethnic, cultural and developmental differences, had one characteristic in common: a striking degree of submissiveness and compliance. They seemed to be utterly devoid of overt hostility and aggression. Each of them fitted perfectly into Knight's classi-

fication of "essential alcoholics," with marked dependency and unconscious guilt, self-debasement and need for punishment. We believe that for them ethyl alcohol—which indeed these patients consumed in copious amounts—became too tame; their unconscious needs demanded severer measures, more complete obliteration, and a nearer tread toward the fearful and tantalizing brink between life and death.

SUMMARY

Nine case reports of patients who were addicted to ethyl and other alcohols have been presented. The literature on the statistical, toxicological and psychiatric aspects of nonethyl alcoholism was reviewed. The surprisingly good physical condition of the nine patients, and their unusually submissive and compliant behavior, were discussed in relation to the literature and to the possible psychodynamics of nonethyl alcoholism.

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