The Role of Nutritional Factors in the Cause, Prevention, and Cure of Alcoholism and Associated Infirmities

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IN THIS discussion, I shall use the terms "alcoholism" and "alcoholic" simply for convenience in referring to the heavy consumer of alcoholic beverages. It is not to be inferred from this that I consider that a greater than average consumption of such beverages necessarily denotes addiction to alcohol.

That alcoholism may represent a manifestation of a metabolic defect, either congenital or acquired, that interferes with the normal utilization of certain customary nutrients is not a new concept. Recently, however, interest in this possibility has been reawakened by the work and writings of R. J. Williams who found that rats placed upon a marginal diet—marginal particularly with respect to the B vitamins—"drank heavily within a short period of time."[1] Rats on diets abundantly furnished with all the nutrients required by rats did not consume alcohol "beyond a low level." When animals that were drinking heavily on a marginal diet were supplied with an abundance of the missing nutrients, they often decreased their alcohol consumption to zero overnight and were maintained at this level as long as the required nutrients were supplied. That dietary deficiencies increase the voluntary consumption of alcohol by rats has also been reported by Brady and Westerfeld[2] and by Mardones *et al.*[3–6]

Williams found that different animals developed different deficiencies, all of which led to an increased consumption of alcohol. Why this should be has not been adequately explained, but, as Dr. Williams has stated,[1] a plausible explanation seems to be that rats on diets which do not furnish sufficient raw materials for the synthesis of various enzymes seek a source of easily derived calories. In any event, it seems clear that dietary deficiencies may lead to greatly increased alcohol consumption, at least in the rat.

Lowry, Sebrell, Daft, and Ashburn[7] found the development of polyneuropathy to be delayed in thiamine-deficient rats when alcohol was substituted isocalorically for part of the food intake. Similarly, Westerfeld and Doisy[8] found the isocaloric replacement of alcohol for fat or for carbohydrate in the thiamine-deficient ration of pigeons to delay the onset of opisthotonus and death. Butler and Sarett,[9] using human subjects, found the replacement of carbohydrate by alcohol in an adequate diet to result in an increased urinary excretion of thiamine and N_1-methylnicotinamide; indicating a sparing action of alcohol upon thiamine and niacin. A logical conclusion from these findings is that in all probability certain nutritional deficiencies, brought about primarily by inadequate diets, increased requirements, metabolic blocks, or a combination of these circumstances, in man may tend to increase the voluntary consumption of ethyl alcohol by man. But, this is a far cry from the postulation that alcoholism in man is basically a genetotrophic disease. If this be so, the fact has eluded me, although I have had years of experience with the treatment of alcoholics, both well nourished and malnourished. The recent study by Trul-

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son, Fleming, and Stare[10] sheds no light on this point, although it appears from their findings that many alcoholics may be benefited, in terms of reduced alcohol consumption, by multiple vitamin therapy. The effect of vitamin therapy in such instances, it seems to me, is more apt to be due to the correction of biochemical abnormalities and mental and somatic disorders resulting from vitamin deficiencies which, in turn, result from the poor diets and digestive disturbances associated with alcoholism, than it is to the correction of any original defect bearing a primary caustive relationship to the alcoholism. Alcoholics are a heterogeneous group and different people drink for different reasons. There is no alcoholic personality type.[11]

The chief pharmacologic effects of alcohol are well known and will not be discussed in this paper which is concerned only with chronic alcoholism.

In regard to the effects of chronic alcoholism upon the individual's mental and physical health, there is not one major disability found in association with this condition which does not occur, albeit less frequently, among total abstainers. The role of ethyl alcohol in the production of the complications of alcoholism, wherever it has been adequately investigated, has been shown to be that of a conditioning rather than a primary or precipitating cause. Let us examine, briefly, some of these complications.

Delirium Tremens: Delirium tremens not only occurs in chronic alcoholism but also frequently follows injuries, operations, and acute infections.[12–14] The primary defect is dehydration and salt depletion. Treatment consists of forcing fluids, administering sodium chloride, and providing an adequate calorie, high vitamin intake. Sedation is used sparingly and restraints no more than absolutely necessary. Flink *et al.*[15] found low serum magnesium concentrations in both alcoholic and nonalcoholic patients with delirium tremens and reported on the effective use of magnesium sulfate in the treatment of this condition.

Wernicke's Syndrome: Wernicke's syndrome is characterized by clouding of consciousness, varying ophthalmoplegias, and ataxia. Peripheral polyneuropathy is almost invariably present. As a matter of fact, I have never seen a patient with Wernicke's syndrome who did not also have a peripheral polyneuropathy. Wernicke's first case was a 20-year old woman who was not an alcoholic.

In 1940, Goodhart and Sinclair[16] reported zero blood levels of thiamine in a subject with Wernicke's syndrome. In 1941, Jolliffe, Wortis, and Fein[17] reported their conclusions from the discriminate therapy of 27 cases; to the effect that the ophthalmoplegia is a thiamine deficiency and that the other manifestations of the syndrome described by Wernicke are the results of other nutritional deficiencies or metabolic blocks. Their findings were confirmed in a 1952 report by Phillips, Victor, Adams, and Davidson.[18] These authors concluded "that the ophthalmoplegia of Wernicke's syndrome is related to a specific lack of thiamine. The nystagmus and ataxia also appear to be related to thiamine deficiency, but the evidence is less conclusive. No definite conclusions can be drawn regarding the relationship of the mental disturbances to the deprivation of thiamine or other vitamins." Obviously much remains to be clarified about the etiology of the complete syndrome described by Wernicke; however, since the syndrome occurs, in its entirety, in numerous conditions other than alcoholism, it is equally obvious that alcoholism can not be the immediate cause.

Alcoholic Encephalopathy: This syndrome is characterized by clouding of consciousness, changing cogwheel rigidities, and uncontrollable sucking and grasping reflexes. It has been repeatedly observed in nonalcoholic pellagrins.

Jolliffe[19,20] reduced the expected morality in alcoholics with this condition from 89.4 per cent to 12.5 per cent by treatment with niacin. He suggested that the syndrome should be termed "niacin deficiency encephalopathy," since niacin deficiency, not alcohol consumption, *per se*, is the cause.

Korsakoff Psychosis: As pointed out by Jellinek and Jolliffe,[21] the findings of all investigators point to much greater therapeutic success in the treatment of Korsakoff psychosis when the B vitamins are administered than

when they are not given. The improvement involves both the neurologic aspects and the mental symptoms. Bowman, Goodhart, and Jolliffe[22] have called attention to the high incidence of polyneuropathy in patients with Korsakoff psychosis in alcoholism, pregnancy, hyperthyroidism, and diabetes mellitus, which, in these cases, responds to thiamine therapy. These workers reported a threefold greater rate of recovery in patients treated with a good diet plus 10 to 50 mg of crystalline thiamine daily, by injection, than obtained with a good diet alone. They did not indicate their findings were conclusive, but recommended further studies of the relation of nutritional factors to the cause and treatment of Korsakoff psychosis, with greater numbers of patients.

Peripheral Polyneuropathy: There can be no doubt but that the polyneuropathy of the alcoholic is due to a nutritional deficiency and that, if adequate treatment is instituted before irreversible anatomic changes occur, it responds to nutrient therapy. In the great majority of such cases, thiamine deficiency is the primary etiologic factor;[23–26] however, other nutritional deficiencies also have adverse effects on the peripheral nerves. Recently, Bean, Franklin, and Sahs[27] have reported prompt improvement in nutritional peripheral neuropathy after intramuscular injection of vitamin B_{12}. Good results with the use of large doses of vitamin B_{12} have been reported by Lereboullett and Pluvinage[28] and by Menof.[29,30] Fernández[31] has employed vitamin B_6 in certain cases with good results. This simply serves to emphasize the fact that vitamin deficiencies in man generally are multiple, and that the various systems of the body are susceptible to deficiencies of not just one but of a number of nutrients.

Toxic Amblyopia: Carroll[32] divided 25 patients with tobacco-alcohol amblyopia into four groups; one group received an adequate diet supplemented with brewer's yeast; the second group received the usual hospital diet supplemented with entire vitamin B complex; the third received a definitely inadequate diet supplemented with large amounts of the vitamin B complex, and the fourth group received the same inadequate diet supplemented only with crystalline thiamine. All patients continued their customary use of alcohol and/or tobacco. All patients improved, the results being at least as good, according to Carroll, as any that he had previously obtained, even in patients who abstained from the use of tobacco and alcohol while under treatment.

A similar amblyopia has been observed in malnourished American soldiers released from Japanese prisoner-of-war camps and among malnourished children on the island of Jamaica.[33] The name "nutritional retrobulbar neuritis" has been given to this condition.

Fatty and Cirrhotic Livers: Fatty livers and cirrhosis of the liver occur in many conditions other than alcoholism. Of 129 alcoholics with fatty livers, Texon[34] found 90 per cent to have been on inadequate diets. All but one of 17 patients with cirrhosis of the liver had deficient diets, according to Texon. In the experimental animal, Best *et al.*[35] found that there is no more evidence that there is a toxic effect of pure alcohol on the liver cells than there is of a poisonous action of an equicaloric amount of sugar. They also found that the pathologic changes produced in the liver, by diets containing large amounts of alcohol or sugar, could be prevented by adequate amounts of choline, methionine, or casein. They attributed the pathologic changes found in the liver to an imbalance of calories and vitamins, particularly to a deficiency of lipotropic factors consequent to an increased caloric intake. While fully confirming Best's finding that adequate amounts of choline or methionine abolished the adverse effects on the liver of both alcohol and sugar supplements, Klatskin and his associates[36,37] found alcohol actually to have a specific effect in increasing the choline requirement, independent of the caloric intake.

In human subjects, Zieve and Hill[38] found no demonstrable relation between impairment of hepatic function and increasing degrees of alcohol consumption. Numerous studies on man indicate the importance of an adequate diet, ample in content of protein and lipotropic factors, in the treatment of the individual with a fatty or cirrhotic liver.[39–42] Phillips, Gabuzda, and Davidson,[42] conclude that their results in the treatment of fatty and cirrhotic livers in man "suggest that the improvement observed

was related to the provision of an adequate diet and that a subsidiary, if any, role was played by withdrawal of alcohol and rest in the hospital."

Cardiovascular disturbances akin to those described in beriberi have been described in the alcoholic[43] and practically all nutritional deficiencies known to occur in man have been observed in alcoholics at one time or another. It is of interest that Joffe and Jolliffe[44] have even advanced evidence strongly suggesting that the gastric achlorhydria frequently found in chronic alcoholics bears no relation to the degree or duration of alcohol addiction, and is probably due to a vitamin deficiency. Similarly, Bianco and Jolliffe[45] found the anemias of the alcoholic to be manifestations of nutritional deficiencies.

It is not to be inferred from all of this that ethyl alcohol is nontoxic. The immediate effects from the over consumption of alcohol are too well known to permit any such thesis. Incidentally, the results of acute alcoholism may differ, in some respects, in the chronic alcoholic from those observed in the normal drinker. Carroll and Goodhart[46] have described a form of total but temporary blindness associated with acute poisoning due to ethyl alcohol which occurs occasionally in chronic alcoholics, but has never been described in the normal, social drinker.

What I do wish to emphasize is that it has never been proved that any one of the so-called stigmata of chronic alcoholism is actually caused by alcoholism, *per se*. In fact, quite the contrary is true. All of those associated conditions that have been adequately investigated have been shown to be the direct result of malnutrition, with alcoholism as the conditioning or remote cause. Alcohol increases the requirements for some nutrients, and spares others. Alcohol causes gastrointestinal upsets and diminishes appetite. Also, alcoholism may induce malnutrition through the fact that money spent on alcohol is not available for the purchase of food. However, given a diet adequate to meet metabolic requirements, in a form that he can assimilate and utilize, the physical and mental health of the alcoholic can be protected.

SUMMARY

Rats, when maintained on diets deficient in certain of the B vitamins, develop a markedly increased voluntary consumption of ethyl alcohol, apparently because of the body's need for utilizable calories. When normal metabolic pathways are restored to the rat by supplying it with sufficient amounts of the missing vitamins, the animal tends to quickly lose its excessive appetite for alcohol. There is no evidence that a similar mechanism operates as a major, primary cause of alcohol addiction in man. Here, however, it must be emphasized that this possibility never has been satisfactorily investigated.

Malnutrition does play an important role in the perpetuation of excessive drinking in many alcoholics who develop deficiency states as a result of their alcoholism. As with the rat, such persons may acquire metabolic blocks and develop a reliance upon alcohol as a utilizable source of essential energy. This probability requires investigation.

More obvious is the causal relation of the painful, disabling, and embarrassing nutritional diseases in the alcoholic to the perpetuation of excessive drinking. The correction of such physical and mental ills is a prerequisite to the successful management of the alcoholism itself, and sometimes brings about a complete cure.

While alcohol is toxic to man, its known effects are acute in nature. The major, chronic, organic disorders associated with alcoholism, and often considered stigmata of alcoholism, are manifestations of nutritional disturbances. They are not peculiar to alcoholism, but occur in total abstainers who for one reason or another develop the necessary nutrient deficiencies. They can be prevented by proper nutritional management.

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