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THIAMINE DEFICIENCY IN AUSTRALIA - A REVIEW OF METHODS OF PREVENTION

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Summary

The incidence of thiamine deficiency in Australia is unknown but the most severe clinical manifestations appear to occur as the Wernicke-Korsakoff syndrome of alcoholism. Available evidence indicates an apparently high incidence and prevalence of this medical problem in Australia. If public health action is warranted, then extensive investigation of the appropriate measures for prevention should be undertaken; these include decrease in apparent per capita alcohol consumption and increase in thiamine supply. The available alternatives are presented for consideration and discussion.

I. INTRODUCTION

Controversy regarding the thiamine status of the Australian population was examined in 1959 (N.H. & M.R.C. 1959). More recently a working party convened by the National Health and Medical Research Council (1977) was unable to reach a firm conclusion on this subject. However it noted available evidence suggesting that a percentage of the Australian population had a low thiamine intake, and that several population groups showed evidence of biochemical thiamine deficiency. The latter included consumers of excessive quantities of alcohol, some elderly people taking inadequate diets, long term residents of institutions and some aborigines and people with chronic diseases. Clinical thiamine deficiency appeared to be confined to people suffering from alcoholism.

II. INCIDENCE OF CLINICAL THIAMINE DEFICIENCY

It has proved difficult to quantitate the incidence of clinical thiamine deficiency in alcoholism; the majority of cases probably occur as Wernicke's encephalopathy or in association with the Wernicke-Korsakoff syndrome. Wernicke's encephalopathy is a serious medical emergency requiring treatment with thiamine; the presenting symptoms are ataxia, mental disturbance, nystagmus and opthalmoplegia (Jolliffe, Wortis, and Fein 1941). Treatment with large doses of thiamine by the intramuscular or intravenous route is followed within six hours by complete biochemical response to normal thiamine status but clinical response is more variable. The diagnosis of the Wernicke-Korsakoff syndrome is not always straightforward as the disease presents in varying degrees of severity. Treatment with large doses of thiamine often promote complete and dramatic resolution of Wernicke's encephalopathy leaving a residual Korsakoff's psychosis manifested by confusion, confabulation and impaired short and long term memory. It is often so severe that institutionalization is required for the remainder of life; these patients are often in middle age and the cost to the community is considerable from many points of view.

In the past few years, we have attempted to obtain better information on the incidence of clinical thiamine deficiency in Victoria and elsewhere. Amongst the most reliable data obtained was the average annual rate of first admissions with Korsakoff's psychosis to Mental

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Health Authority institutions in Victoria over the period 1972 to 1977 (inclusive). This rate was 21.4 per million of the adult Victorian population (over 18 years). This figure is several times higher than that averaged over a nine year period in New York State psychiatric centres (Centerwall and Criqui 1978) which was 8 per million adult population over 18 years and also higher than that observed in New Zealand (16.9 per million adult population over 18 years). Korsakoff's psychosis is a problem of some magnitude in this country and its aetiology is worthy of serious study.

We have been unable to obtain firm data on the incidence and prevalence of Wernicke's encephalopathy in the population. However at St.Vincent's Hospital in 1969, a study of 1,100 consecutive admissions for alcoholism revealed that 26 patients had Wernicke's encephalopathy (Horvath et al 1969). In a more recent study, 24 of 215 alcoholic patients had Wernicke's encephalopathy (Wood, Breen, and Penington 1977).

III. AETIOLOGY OF THIAMINE DEFICIENCY IN ALCOHOLISM

The relationship between Wernicke's encephalopathy and Korsakoff's psychosis has been justified by aetiological data (alcoholism is usually the predominant cause in both), by clinical data showing the continuity of the two syndromes, and by a brain histology common to both syndromes (Victor, Adams, and Collins 1971). Whilst thiamine deficiency is almost invariably associated with Wernicke's encephalopathy (Embree and Dreyfus 1963), its role in the development of Korsakoff's psychosis is unknown. Nevertheless, many other workers consider that Korsakoff's psychosis is due to thiamine deficiency and is therefore preventable. Whilst we have some doubts that this simplistic view is correct, it is certainly an important medical problem and the incidence of Wernicke's encephalopathy alone at St.Vincent's Hospital justifies our interest in methods of preventing thiamine deficiency in alcoholics.

It is also known that the nutritional syndromes resulting in alcoholic brain damage frequently present in mixed as well as pure forms (Dreyfus 1975) and may respond only to initiation of a nutritious vitamin supplemented diet over a period of time. Hypomagnesaemia potentiates thiamine deficiency in rats and affects response to thiamine in humans, and multiple Vitamin B deficiencies are frequently observed in alcoholic patients with brain damage.

The alcoholic person is at great risk of developing thiamine deficiency for several reasons. Thiamine appears to be displaced from the diet when ethanol consumption reaches 100 g per day (Wood 1972). This level of consumption over a long period of time puts people at risk of developing alcoholism (Krupinski and Stoller 1971) although the community regards it as a moderate intake. Thiamine supply is further reduced by anorexia, vomiting and diarrhoea which are of frequent occurrence in heavy drinkers (Wilkinson et al 1971) and thiamine absorption may be impaired in alcoholism (Thomson, Baker, and Leevy 1970). In the presence of severe liver disease, hepatic reserves of the B vitamins are depleted (Baker et al 1964) and thiamine may not be phosphorylated to its active form (Williams and Bissell 1944). Thiamine excretion may also be increased in association with the diuresis (Dewhurst and Morgan 1970) which occurs with alcohol consumption. Some alcoholics may have an inherited enzyme defect (Blass and Gibson 1977) rendering them particularly prone to thiamine deficiency when thiamine is low.

IV. POSSIBLE METHODS OF REMOVING 'RISK' OF THIAMINE DEFICIENCY IN ALCOHOLISM

In the short term the National Health and Medical Research Council (1977) have recommended that multi-vitamin tablets be given to alcoholics to remove them from the 'at risk' status; in the long term the Council recommends that the cause of the 'at risk' status should be removed. How this should be done is conjecture at present; in view of the complex aetiology of thiamine deficiency in alcoholic persons, it is unlikely that there is a simple answer.

Moderate alcohol consumption is common in Australia, thus we take the view that any measures introduced to decrease 'risk' of thiamine deficiency should be directed at the entire population. Although the thiamine status of the population in general is unknown, there are groups in the population who do not consume alcohol yet show low thiamine intake and biochemical thiamine deficiency (N.H. & M.R.C. 1977). This further justifies an approach directed at the entire population if treatment of the entire population is necessary to prevent severe thiamine deficiency in alcoholics, this makes preventive measures a matter of public health, to be considered carefully and at the highest level.

The possible methods of prevention include the following:-

1. Those directed towards decreasing apparent per capita alcohol consumption
2. Those directed towards increasing thiamine supply
   - (a) Nutrition/health education
   - (b) Increase in extraction rate of flour from wheat
   - (c) Thiamine supplementation of processed foods
     - (i) Restoration of thiamine lost in the processing of foods
     - (ii) Enrichment of staple foods with thiamine
     - (iii) Fortification of food items with thiamine

V. OVERSEAS EXPERIENCE WITH THIAMINE SUPPLEMENTATION OF PROCESSED FOODS

The traditional choice of food for thiamine enrichment has been the staple foods - flour and/or bread, or rice (Parman 1962). The results of enrichment programs have not been directly evaluated except for rice in the Philippines (Salcedo et al 1948, 1950; Burch et al 1952) where distinct improvement in health of the population resulted. The Japanese experience with thiamine enrichment of rice is similar (Kawasaki 1965). In Newfoundland, compulsory enrichment of flour with multiple micro-nutrients including thiamine was difficult to evaluate, but there was a dramatic fall in the number of people with low thiamine levels in urine (Akroyd et al 1949).

There is indirect evidence that beri beri has largely disappeared in American alcoholics since thiamine enrichment of flour has been a legal requirement in most States for the past 40 years (Bradley 1962).

Thiamine supplementation of foods other than staple foods have been listed (Menden 1969; Bauernfiend 1970) but there is no known evaluation of their usefulness.

In Japan, there has been extensive use of modified thiamine compounds such as the allithiamines in food enrichment (Kawasaki 1965). Compared to thiamine mononitrate and thiamine hydrochloride, these compounds are more stable, more efficiently absorbed, and following absorption, they appear to act as does thiamine in the body (Shimazono and Katsura 1965). These compounds have not been studied or used extensively outside Japan.

VI. SUPPLEMENTATION OF ALCOHOLIC BEVERAGES WITH THIAMINE OR MODIFIED THIAMINE COMPOUNDS

Dax (1968) and the Americans, Centerwall and Criqui (1978) have suggested addition of thiamine to alcoholic beverages as a method of preventing clinical thiamine deficiency in alcoholism. This suggestion is in direct opposition to American law which ruled in 1940 that there must not be any reference to vitamin content on the labels of alcoholic beverages intended for sale, on the grounds that the consumer would expect a therapeutic effect. Should alcohol become a "health food" in the eyes of the consumer?

Policy criteria have been provided to guide selection of a food source as a carrier of supplementary nutrients and to test suitability of the carrier (U.S., N.R.C. 1974). If consideration of the supplementation of any food or beverage is to be made, then this theoretical exercise is mandatory.

We have applied the criteria provided by the National Research Council to the fortification of beer with thiamine or modified thiamine compounds. Many questions remain unanswered - these include physiological availability of thiamine in beer, stability of thiamine in beer, stability of beer containing added thiamine, and the tasts threshold of thiamine in beer. Other matters of concern relate to failure of alcohol to supply other important nutrients (unless they were also added), possibly unwarranted claims for the health advantages of drinking vitamin enriched beer (which may counteract efforts to decrease apparent per capita alcohol consumption), objections to "tampering with beer", the multiple hazards of diets containing excessive quantities of alcohol, and consumption of concentrated alcohol sources other than beer which have not been enriched with thiamine. All of these uncertainties require resolution before serious consideration can be given to the addition of thiamine to beer or any other alcoholic beverage.

VII. CONCLUSIONS

The incidence of clinical thiamine deficiency in Australian alcoholics is unknown. Alcoholic patients with Wernicke-Korsakoff syndrome often present after two weeks of food withdrawal which indicates that subclinical thiamine deficiency existed prior to food withdrawal. Thiamine reserves are related to the balance between thiamine intake and thiamine requirement, and whether an increase in thiamine intake would prevent the Wernicke-Korsakoff syndrome is unknown.

Consumption of alcohol is high in Australia and it is known that the intake of staple foods such as bread is decreasing in favour of refined foods containing flour, sugar and alcohol (Turner 1973). Heavy drinkers give up sugar before they give up bread (Wood 1972), so of all the food groups, the cereal/bread group could be promoted as a carrier of thiamine for the entire population. In New York State, where thiamine

enrichment of flour is compulsory, the incidence of Korsakoff's syndrome is lower than in Victoria, but this comparison may be irrelevant unless Korsakoff's psychosis is directly related to thiamine deficiency. It is probably more difficult to add thiamine to alcoholic beverages, since many potential technical and other problems require study.

F.A.O./W.H.O. (1970) have reviewed the points leading to difference in opinion between nutritionists about the value of fortification in solving nutritional problems, and Williams (1961) has given an historical account of the political problems encountered in the introduction of thiamine fortified foods overseas. Food fortification in other countries has only been achieved in the face of objections (Bauernfiend 1970).

F.A.O./W.H.O. (1970) consider that the final decision of food fortification must be made at the national level as part of the national food and nutrition policy, because it usually means that nutrient intake is changed in the entire population for the sake of severely affected sick people.

The incidence and prevalence of clinical thiamine deficiency in Australian alcoholics should be documented immediately. If this problem warrants public health measures of prevention, there should be an extensive investigation of appropriate methods. Following implementation of any chosen program, evaluation of its effectiveness will be important.

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