

# Book Reviews

**A Nutritionist's Guide to the Clinical Use of Vitamin B<sub>1</sub>** by *Derrick Lonsdale, M.D.* Life Sciences Press, P.O. Box 1174, Tacoma, WA 98401. Paperback, 214 pages.

Some time ago, after I had finished reading this very valuable book, I concluded that only the author could give justice to an adequate abstract of the book. It is my contention that a book reviewer, if he likes a book, ought to bring this to the attention of the audience and not spend his time finding things to criticize. For this reason I requested Dr. Lonsdale to prepare this abstract, which I am pleased to publish. I simply want to add that in my opinion it is an excellent book and one which ought to be studied very, very carefully.

A. Hoffer, M.D., Ph.D.

This 214 page book is written for physicians and nutritionists. It outlines and reports the extensive clinical research carried out by the author over many years as a pediatrician in a large private subspecialty clinic. It also includes some reporting of research, using experimental animals, in which the conclusions have practical importance in human nutrition.

Doctor Lonsdale believes that the pioneer research of Hans Selye has been largely ignored and that it offers a model for health and disease which is more realistic and accurate than the present one. In particular, the Selye model abolishes the artificial separation of mental and physical disease as two distinct and separate entities.

Recognizing that Selye operated on the belief that the hypophysis was the "conductor of the endocrine orchestra", Lonsdale points out that newer knowledge places the limbic system in the master position and the hypothalamus as the bridge between brain, autonomic nervous system, and the endocrine system. Our day-to-day adaptive responses are coordinated by perception of environmental stresses, leading to computer data processing, primarily in the limbic system

(the computer) which draws on association areas to compute an adaptive, coordinated response through the endocrine and autonomic axis. Hence the adaptive mechanism invariably involves a physical or organic functional response in combination with a mental perception which is referred to as emotion. Failure in this systematic automation results in the "diseases of adaptation" proposed by Selye.

The author is careful to point out that complex adaptive responses are fundamentally dependent upon the function of the nervous system which consumes oxygen faster than any other tissue. Hence redox potential, which is in turn dependent upon the quality of nutrition, becomes the central decisive factor in determining day-to-day adaptive capacity.

Heart muscle is close to nervous tissue in consumption of oxygen, so beri-beri becomes an excellent model for studying the effect of the disease on the autonomic/ endocrine axis. Since thiamine has a central place in energy metabolism it becomes easier to understand that beri-beri represents a prototype for a disease of maladaptation. Its histopathology is compared, organ by organ, with that described by Selye in his "general adaptation syndrome" and there is a remarkable similarity.

Using this concept, Lonsdale sets out to use thiamine deficiency as one illustration of the consequences of loss of redox potential, recognizing that thiamine is only one member of a complex team of chemicals which orchestrate the appropriate control of oxygen utilization. Chapter I provides a brief review of the anatomy and physiology of the autonomic system, leading into the second chapter which describes the essential features of dysautonomia, using familial inherited dysautonomia (Riley Day syndrome) as the model. The syndrome of functional dysautonomia is outlined and some interested facets are illustrated by discussion of the prolonged QT syndrome

and sudden infant death (SIDS), both of which are dysautonomic syndromes.

In chapter 3 there is a review of the chemistry of thiamine and its role in energy metabolism. The reader will be intrigued to find information on the ecologic phenomenon of thiaminase and its ability to destroy thiamine in the bowel. The mechanism of Chastek paralysis from ingestion of raw fish containing thiaminase is described. The important class of substances known as fat soluble and alkyl derivatives of thiamine and their potential for clinical use is outlined.

Chapter 4 provides information on ways and means of studying biochemical function in the clinical laboratory. Erythrocyte transketolase in defining thiamine pyrophosphate deficiency is an excellent example of the use of enzyme cofactor saturation tests. The use of urinary creatine and creatinine is explained as a simple and cheap way of monitoring oxidative metabolism and cell membrane function.

The fifth chapter outlines a series of case histories and the conclusions drawn from each one. Three sections deal with stress, genetics and nutrition respectively, although it is emphasized that this is an artificial way of seeing a case record, and that all of them always interact. In some conditions, one of the three appears to be more important in causation.

The final chapter sums up this information to propose a revised model for both health and disease which is based on the work of Selye. Energy metabolism is viewed as the foundation; all aspects of nutrition represent the complex fuel supply which feeds it. To illustrate the clinical use of the model premenstrual syndrome (PMS) and sudden infant death syndrome (SIDS) are presented as examples.

Derrick Lonsdale, M.D.

**Prevention and Curative Intervention in Mental Illness** by *F. J. Menolascino and J. A. Stark*. Paul H. Brooks Publishing Co., Baltimore & Toronto. Hardcover, 391 pages, 1988

About one out of every 1000 babies has Downs syndrome, but this is only a small fraction of the sixty babies out of 1000 who have other congenital diseases. For most of these diseases —

including Downs syndrome — there is no generally acceptable treatment. Research is still pitifully underfunded for diseases which account for twenty-five percent of all hospital admissions. Yet there is some progress, and this is described in this valuable book by Menolascino and Stark (1988) which emphasizes prevention and treatment.

The major emphasis is on genetic defects, how they can be recognized early, on early treatment when it is available, and on possible prevention. The first chapter by Stark, Menolascino and Goldsbury provides a useful and complete outline of the diagnostic methods now available and of the role played by genes. Several etiological factors are discussed including neurophysiological, endocrine, infectious diseases, neural toxins, nutrition and socio-economic. In Chapter 2, Epstein provides a good review of the interplay between genes and their final expression, i.e. symptoms and signs. Orthomolecular treatment is referred to but a few reports are lumped together in one sentence as if one nutritional study is like every other. Dr. Henry Turkel's thirty years of research with Downs syndrome is given less attention than Dr. Ruth Harrell's more recent study, and both are refuted by referring to one paper claimed to have failed to corroborate Harrell. He neglects to show that this study did not duplicate Harrell's work as the investigator did not use thyroid. One study which had absolutely nothing to do with Turkel's work is used to denigrate Turkel.

Epstein lists three approaches he considers basic to research in mental retardation. I would suggest one more, i.e. to start from research such as Turkel's and Harrell's, really repeat these studies and then begin to dissect out the nutrients and hormones which are essential. Maybe Turkel's treatment regimen could be simplified. It does appear that thyroid is essential since Harrell's critics failed to use it and could not corroborate. Such an approach takes advantage of what is known and moves on. In my opinion, this is more productive than to wait until we know the genetics and genetic biochemistry before we initiate

therapeutic studies.

In Down's syndrome a little bit of extra chromosomal tissue in each cell increases the formation of a large number of metabolites by over 50 percent. It would be very valuable to study some of Turkel's patients who are so much better on his program and measure these same chemicals.

Some of the more common inborn errors of metabolism are reviewed by Richard Koch and five co-authors. On the basis of 167 patients with phenylketonuria (PKU) studied thoroughly over six years and more, these authors conclude: (1) that the earlier special diets are started, the better is the outcome, (2) children maintained on the diet beyond age six remain better than children who stop these diets, (3) some should continue on the diet until adolescence.

I will skip Section III which contains a valuable review of neurology and retardation so I can spend more time on the next section which deals with nutrients.

I found Dr. Donald R. Davis' contribution the most valuable part of the whole book. It is worth getting the book just for this chapter. I have not seen a more thorough review of the literature. Just a few items: animal studies have shown that giving certain nutrients at a level 50 percent of recommended doses will cause a significant increase in congenital abnormalities. Up to 25 percent of a "well fed" population fail to provide this much of several nutrients, vitamins, minerals. He points out that there is a generational effect. Changes caused by nutritional deficiencies will require two generations of a good diet to overcome these induced effects. Davis estimates that up to 50 percent of all mentally retarded individuals have become this way because of poor nutrition.

Protein/calorie deprivation has been considered with little reference to the accompanying deficiency of other essential nutrients. This type of problem, starvation, is much less common in industrialized societies. We have instead a surplus of calories and a deficiency of nutrients. It is really a form of affluent malnutrition. Davis refers to dismembered foods, which is a polite term for junk food. In many diets 50 percent of the dry

weight of the diet consists of dismembered foods. It follows that these populations ought to suffer from a variety of deficiencies. Every nutritional survey bears this out. Davis refers to good examples of how improving diets prevents disease. Thus, pre-eclampsia can be prevented by introducing whole foods into the diet. In England it has been shown that adding folic acid to the diet of pregnant women will decrease markedly the development of neural tube defects. Evidence is available to suggest that thalidomide defects occurred primarily in women whose diets were inadequate. I have seen reports that thalidomide interferes with Vitamin B<sub>3</sub> metabolism in animals when the limb buds are developing. Ample amounts of B<sub>3</sub> could have protected these babies.

A clear example which illustrates how malnutrition causes disease is fetal alcohol syndrome. Can anyone argue that pregnant women drinking alcohol are obtaining all the nutrients they need for themselves and the growing fetus?

Good nutrition can protect children from the damaging effects of toxic metals, for example lead. A diet high in fiber, containing ample quantities of Vitamin C and zinc, will decrease the damage.

Davis discusses the controversy over Harrell's report. I hope every person who has seen only the pseudo attempt to repeat her work will read it.

Sections VI, VII and VIII will be very interesting to many in the field of mental retardation, but not to me. I remain convinced, from the reading I have done and from the 1000 children under age fourteen I have treated beginning in 1960, that the answers to prevention and treatment are in the nutrition area, and that all the psychosocial interventions can only be partially successful if nutrition is ignored. Unfortunately, nutrition had been uniformly neglected in all the usual books on retardation. This is the first one to break this rule.

A. Hoffer, M.D., Ph.D.