Nutritional Factors In General Medicine

Effects of Stress and Distorted Diets

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As the outgrowth of over forty years of clinical experience on the part of the author, this book is presented expressly and exclusively for practicing physicians and advanced medical students. It concentrates solely on those nutritional factors which are relevant to clinical medicine. That is, it focuses on material observable by practitioners and of immediate, practical value to them. Among the topics discussed are organic and inorganic macronutrients, trace elements, and vitamins.

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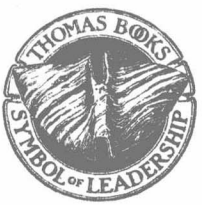

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This work is dedicated to
Eleanor Naylor Dana
whose moral and material support
led to its being written

INTRODUCTION

THIS BOOK GREW out of four decades of clinical practice. This experience was in a measure given meaning and direction by the publications of many other clinicians. The book was written, however, because colleagues and students requested it after being made to appreciate concepts and interpretations that were at variance with some advanced by some professional nutritionists. The work will, therefore, consist of material observable by practitioners and useful to them.

Some readers may be surprised or disappointed to find some matters absent from this book. For example, there will be no general statements about the present status and future developments of clinical nutrition; these will be found in the papers by Peters,[1] Williams et al.,[2] Schneider and Hesla,[3] and Butterworth.[4] More specifically, the discussions will be limited largely to *nutritional* factors rather than to *dietary* factors. Hence, formal discussions of the treatment of gastrointestinal disorders will not be found here. Similarly, no formal discussions will be made of the biochemical disorders of severe kidney and liver diseases. All of these can be covered satisfactorily only in special treatises. Another subject that requires coverage in a special treatise is the increased vitamin needs in certain inborn errors of metabolism. Medical practitioners will rarely, if ever, see any of them, but they should, of course, know about them. The subject is well covered in the excellent review by Scriver.[5]

Another topic omitted from this book is the effects of food additives. The information available on this potentially alarming topic is not only too extensive and confused for concise discussion, but it is changing almost daily. Moreover, the subject is not specifically nutritional in nature and, hence, does not belong here.

The reader will find little in the way of biochemical discussion in this book. A serious—all but fatal—defect of the science of nutrition has been supersaturation with respect to subcellular

biochemistry. If one shakes this mixture slightly, a cloud of processes consisting of electron transfers, coupled or uncoupled phosphorylation, mitochondrial and endoplasmic reticulum membrane changes—and of course DNA and RNA synthesis—all crystallize out. Since physicians rarely, if ever, are required to deal with any entity smaller than a patient, the biochemical discussions that make up much of the nutritional literature merely serve to distract and at times divert. Like the aurora borealis, they are impressive—even awe-inspiring—but not yet harnessed for use and, in any case, certain to vanish after a short existence. Accordingly, this material has been largely omitted here, only a small portion of it having been included in order to entice younger physicians into reading this book by providing them with something that sounds familiar. On the other hand, omitting all but small fragments of this vast literature has had beneficient effects: It has given us a thin book, one that can be comprehended by practicing physicians.

The exclusions listed above simplify the presentation of ideas considerably. There remain, however, two matters that require brief general discussion preliminary to the content of this clinical work. One deals with the effects of food processing. This has been discussed in two recent reports,[6, 7] but not very satisfactorily. One report[7] surprisingly states that "on an overall basis, the food preservation techniques in greatest use today do not result in major losses in the nutritive value of foods." This is quite different from what is found in the AMA monograph on the subject,[8] which cites an 80 percent loss in vitamins and minerals in the milling of grains and cereals. The Food Technologists' report[7] actually describes loss of vitamins A, B_1, C, and E during heat processing and drying. It also points out that processing plus pre-serving cooking (the latter is usually the more destructive) will remove a large part of the water-soluble vitamins such as thiamine, riboflavin, niacin, and ascorbic acid, so that only 35 to 45 percent remains. Surely these losses must be greater among those who cook carelessly at home (throwing away the cooking water) or eat in restaurants frequently. These matters are covered in the appropriate sections of this book. At any rate, these published data require modification of the statement that the American diet is the best in the world: It

probably is when it is harvested, picked, or slaughtered, but any feeling of satisfaction owing to this fact must be submerged in what we know to be the results of processing and premeal cooking, which leave our diet outstandingly rich only in calories.

Another serious problem that practitioners must deal with is that created by the publication by official agencies of recommended daily allowances for the various nutrients. Actually, the problem of how physicians are to deal with the stated recommended daily allowances is easily solved. Since they are defined as the amounts adequate for "almost all healthy people," doctors need pay little attention to them, for doctors have little to do with healthy people. The words *almost all* have been defined as 97.5 percent of healthy people, a definition made manifestly absurd by the minute amount of data that has led to the formulation of the recommendations. The marked variability of even healthy people—a fact known at least to clinicians—makes it evident that authoritative recommendations can be valid only if made on very large numbers of people, which has not been done. Physicians, as they always have, will do the best they can for their patients unless prevented by administrative or judicial obtundity. The matter of when is a vitamin a food, a food substance, or a drug is discussed with some cynicism by Norman.[9] He points out that when a vitamin is present as less than 50 percent of the recommended daily allowance, it is called a food; if between 50 and 150 percent, a food supplement; and above that, a drug. By this definition, when 15 or 20 liters of water are given in a day to a patient with severe diabetic acidosis, it is a drug! Or when a patient with severe hyperthyroidism is given 200 percent of the normal caloric intake, all his food becomes a drug! A clinician is likely to consider such administrative definitions as useless in practice and insulting to the intelligence. Rather, he would prefer to hold that nutrients of any sort, when given in whatever amounts are required to ameliorate or prevent nutritional deficiency, can never be anything but nutrients. On the other hand, when the purpose is different, and a food substance is given so as to produce an effect that is not nutritional, it is best regarded as a pharmacologic agent, as is the case when vitamin D is given for hypoparathyroidism, or vitamin E for ischemic myositis (see page 154). Similarly under some circum-

stances, a common nutrient might become a poison, as is the case with meat, cheese, or milk in severe liver disease. In short, the nutritional treatment of patients is a proper duty of medical practitioners, and medical practitioners should determine what nutrients their patients need. The aim of this book is to present information that physicians can use in treating their patients.

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CONTENTS

NUTRITIONAL FACTORS IN GENERAL MEDICINE

PART I

ORGANIC MACRONUTRIENTS

CALORIES, OBESITY, WEIGHT REDUCTION, AND STARVATION

THE PROBLEMS OF human nutrition fall into two categories: (1) How much should a person eat? (2) What kind of food should one eat? A priori we should expect the answer to the first question to be clear and definite, whereas the answer to the second might be expected to be vague, uncertain, and fragmentary. In fact, the situation is just the reverse. As the later pages of this work will show, we know a great deal about specific nutritional needs. However, we have very little information on which to base an answer to the first question. The essential ambiguity of our understanding of how much we ought to eat was brought out in a recent article by Durwin of Glasgow and Edholm, Miller, and Waterlow of London.[1] Among other things, they pointed out that current assumptions that 70 percent of the world is undernourished in terms of calorie intake may be based on ignorance. The truth may be that in fact, 30 percent of the world—the 30 percent considered the standard—habitually eats too much, and that only a proportion, an unascertainable proportion, of the rest are actually undernourished. It is evident that some people can do very well on calorie intakes much less than officially recommended. Others may gain weight on such a regimen. Moreover, some persons can be given very large amounts of food with no increase in body weight. Contrarily, it is evident that some obese persons eat much less food than some who are lean and still do not lose weight. These contradictions defy explanation by currently available concepts.

A number of authors have discussed the roles of environmental and of genetic factors. It is well known that fat parents have fat children—but they also have fat pets. The brief but weighty discussion by Garn et al.[2] appropriately emphasizes the environmental factor—at least in a population in Michigan. However, fat babies do not necessarily grow up fat.[3] On the other hand,

taking the mammalia as a whole, there is no doubt about the importance of inheritance. A recent paper by James and Trayburn[4] reviewed the evidence of decreased heat production after eating and on exposure to cold. James and Trayburn[4] suggest that there is a genetically determined variation in glucose metabolism in skeletal muscles which leads to this deficiency in heat production, thereby leading in turn to obesity when food is available. (This genetic difference favors survival when food is scarce. Accordingly, after a period of famine, many of the survivors should become obese on refeeding, as is known to happen.) Although it is too soon to accept James and Trayburn's involvement of the specific enzyme system they suggest, the concept is nevertheless a good one. The finding of decreased calorigenesis in the obese continues to be corroborated.[5] A question, however, arises: Is the enzymic disorder necessarily inborn?

Attempts have been made to calculate human calorie requirements from energy expenditure. The assumption underlying this approach is that a normal, fully grown person, not pregnant, needs enough calories in the form of food and drink to provide the energy required by his or her way of life. However, the data show that for persons pursuing similar activities, the caloric requirement needed if weight is to remain unchanged may vary by as much as 100 percent. Moreover, as Edholm[6] has emphasized, it is useless to expect to be able to balance calorie intake as food against energy expenditure as work and heat production over the short term. On the other hand, when the figures for seven days of study are made into an average, calorie intake and energy expenditure are found to be close to each other. Nevertheless, the mechanisms whereby calorie intake and energy expenditure balance each other—as they do most of the time—are not understood. For example, the authors of a recent work,[7] psychologists, reviewed the endocrine aspects of the problem and, rejecting all previous theories, formulated their own which, however, will convince few endocrinologists. It is best at this moment to leave to one side the mechanisms of food-energy balance and pass directly to a consideration of the effects of overeating that produce weight gain.

To start with, it is necessary to emphasize that overeating is not a simple or even absolute matter. There is an important time factor. The number of meals into which the total calorie intake is divided is highly important—the fewer the meals, the greater the likelihood of obesity above minimal levels of intake. The patient who has essentially nothing for breakfast and little more for lunch but makes sure to take a large meal with meat, vegetables, and dessert in the evening is an excellent candidate for obesity despite what seems not to be an excessive calorie intake. In addition to these broad considerations there are many others, difficult to define, that suggest themselves. For each nutrient, the time of ingestion, the rates of digestion, of movement through the gut and of absorption must be taken into account. Sassoon[8] has analyzed these problems largely in terms of the enzymes involved. Whatever the validity of this analysis may or may not be, it does point to the complexity and subtlety of the problem. The factors discussed by Sassoon[8] offer a partial explanation for the impossibility of describing accurately the balance between food intake and energy production.

In addition, the effects produced by variations in physical activity, of the efficiency of muscle work, and of the amounts of muscle tension while resting must be considered, but they cannot for lack of data.

The effects of overeating are not easy to study, because when obese persons come in for observation, they are at a late stage of their metabolic disorder. In fact, by the time obesity has become established, many patients have ceased to overeat. Attempts have been made to study the metabolic consequences in non-obese persons of overeating for periods of one to three weeks;[9, 10] however valuable they may be in some respects, they are too short to contribute definitive data bearing on obesity. The best study of experimental overfeeding for our purposes is that reported by Sims et al.,[11] in which weight gain was induced over a six-month period. During the study, some of the subjects doubled their adipose tissue mass, and in some all of the weight gain showed up in the calculated fat mass. Exertional dyspnea developed with the obesity. Lassitude and decreased efficiency set in. The subjects fell into two groups: one consisted of persons