# Modern Nutrition: a Clinical Symposium

MODERN NUTRITION
A CLINICAL SYMPOSIUM

PRODUCED BY E.R. SQUIBB & SONS
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*The Food and Nutrition Board, formerly called The Committee on Food and Nutrition was formed in 1940 for the purpose of improving the nutritional fitness of the people in the world crisis.*

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*This presentation is designed to clarify and simplify some of the newer aspects of nutrition for the practicing physician, with special emphasis on diagnosis and treatment of vitamin deficiency states.*
*The Council is indebted to these eminent authorities for the time and effort they have devoted to this task.*
*Dr. Norman Jolliffe will speak first on the recognition of vitamin deficiency states.*
*Dr. Tom D. Spees will follow with a discussion of therapy.*
*Dr. W. H. Sebrell and Dr. Robert Goodheart will then participate in the discussion of the immediate sociological and economic significance of better nutrition.*
*Dr Jolliffe.*

"Twenty years ago, ten years ago, perhaps only yesterday, patients with nicotinic acid deficiency encephalopathy may have been classified as "wet brain", "central neuritis" or "atypical coma". These patients almost always died. Today, such patients can nearly always be promptly and permanently cured. Many people associate the terms "scurvy", "pellagra", "beriberi", "rickets" or "ariboflavinosis" with dietary deficiency. It is not so well known however that any of these conditions may occur in the presence of a diet which, even by modern standards, is completely adequate.
Deficiency diseases may be classified into two groups: primary malnutrition and secondary or conditioned malnutrition.
By primary malnutrition we refer to deficiency states due to inadequate intake alone. Folate deficiency diseases, which come in this category, are said to have a higher incidence in the south than in most other sections. Primary malnutrition however, is found in all sections of our country.
By secondary or conditioned malnutrition, we mean deficiency states produced by factors other than an inadequate diet. We shall list and discuss these several factors.
First, interference with food intake. Many diseases interfere with an adequate intake of food. Fever, infection, surgery, special diets such as prescribed for obesity, gallbladder disease, or for diabetes or kidney disease. Improperly-fitted denture in elderly patients often prompt a selection of soft, easily masticated food.
The second factor is increased requirement. When requirements are abnormally increased, well beyond the usual or normal limits, then increased requirement may be considered a conditioning factor. Fever, hard physical exertion, hyperthyroidism, pregnancy and lactation increase requirements.
The next factor is malabsorption, that is, failure to absorb. This factor is frequently involved in conditioned malnutrition. The diet may be perfectly adequate but because of failure to absorb, deficiency disease may result. Failure to absorb may be due to achlorhydria, so

frequent in elderly persons. Diarrhea may interfere by promoting too rapid transit of ingested food through the intestinal tract. Selective absorption by substances such as mineral oil may withhold certain vitamins from absorption.

Mal-utilization. Mal-utilization is often involved as a conditioning factor in malnutrition. Liver failure, secondary disturbances of liver function from whatever cause may interfere with utilization. The inability of many diabetics to convert carotene into vitamin A is an example. Any interference with the conversion of thiamine, riboflavin or nicotinic acid into the various enzyme systems will precipitate deficiency disease as readily as an inadequate intake of food. In this boy of 15, who is a diabetic, the lesions at the angles of the mouth indicate riboflavin deficiency to be a complication.

Conditioned malnutrition may be induced by hastened destruction. Certain vitamins are relatively unstable in an alkaline medium, notably ascorbic acid and thiamine. Alkaline therapy may be instrumental in restricting availability of these two vitamins. In addition, ascorbic acid may be used by the body to detoxify certain drugs.

Next, hastened excretion. Hastened excretion may be an important factor in the production of malnutrition in patients where large amounts of fluids are forced, as in the treatment of diabetic acidosis or in the treatment of infections with sulfonamides. In uncontrolled diabetes, or in *diabetes insipidus*, hastened excretion serves to deprive the body of certain needed nutrients. It is obvious therefore that the terms “scurvy”, “pellagra” and “beriberi” do not indicate necessarily a dietary inadequacy, for these conditions may be as readily and as frequently produced by conditioning factors as by inadequate intake alone. It is a tissue deficiency whatever its cause that produces nutritional deficiency disease.

It follows that if a person has malabsorption, the physician may not correct it by giving any form of oral vitamin therapy, or if a patient has mal-utilization, any form of the vitamins may not improve the fundamental metabolic defect.

Effective treatment of subjects having heart disease, tuberculosis or syphilis requires more information than is contained in the diagnostic labels of cardiac, tuberculosis or luetic. Likewise for subjects having vitamin deficiency diseases, effective therapy requires more information then is given in the terms “niacin”, “ascorbic acid” or “thiamine deficiency” or is contained in the older terms of “pellagra” “scurvy” or “beriberi”. Therefore, as pointed out by Kruse, each diagnosis of a specific deficiency requires recognition of its velocity, its severity and its pathogenesis.

By velocity is meant whether it is acute, subacute or chronic. Of course, the acute or subacute may be superimposed upon chronic. Velocity is important for acute lesions, though severe, respond much more promptly than chronic lesions, though mild. Likewise, exact diagnosis and therapy require a statement as to severity, whether mild, moderate or severe.

Generally, mild deficiencies require less vigorous therapy then severe, though the duration of therapy is more dependent upon velocity.

Finally, the pathogenesis must be noted. That is, whether it is a primary or a conditioned, or a conditioned deficiency. Therefore, according to our hypothesis, a complete diagnosis of one of the specific deficiency diseases will include one item under each of the following heads. For example a niacin deficiency may be acute, subacute or chronic. It may be mild, moderate or severe. It may be either primary or conditioned. And, and likewise for ascorbic acid deficiency. This deficiency may be acute, subacute or chronic, either mild, moderate or severe, and either primary or conditioned. And same way for all of the other specific deficiency diseases, such as thiamine, riboflavin and so forth.

We’re dealing here with tissue deficiencies which manifest themselves in three ways. By interference with the biochemical mechanism, as functional disturbances, and finally in anatomic lesions. The resulting clinical picture may be highly confusing.

As a first step in clarification, let us chart the various degrees in malnutrition from the standpoint of relative severity.
Let this line represent a normal tissue concentration of the vitamins, the normal tissue concentration of any of the vitamins.
Let this line represent a mild deficiency. A mild tissue deficiency of any of the vitamins.
Let this line represent a severe tissue deficiency. A severe tissue deficiency of any of the vitamins.
To illustrate, let us take a mild ascorbic acid deficiency. If that mild ascorbic acid deficiency occurs over a short period of time, such as this, we will have developed then a mild acute scurvy. On the other hand, if the tissue concentration falls to this level, but over a long period of time, months or years, then we will have in this patient, a mild chronic ascorbic acid deficiency or scurvy.
Now let us illustrate the severe deficiencies by niacin deficiency or pellagra. If the tissue concentration falls over a short period of time to this level, we will have an acute severe, pellagrin or acute niacin deficiency. On the other hand, if the tissue concentrations fall to this same level, but over a long period of time, then we will have a patient who shows a chronic but severe niacin deficiency, in other words, a chronic pellagra.
Here is a patient who exhibits the signs and symptoms of an acute severe niacin or nicotinic acid deficiency. The stupor, the sucking and grasping reflexes are the only outward expressions of the ailment. Superficial examination of the tongue, the mouth, the skin, reveals nothing of diagnostic importance. Yet, the therapeutic response of this patient to niacin is almost as rapid as the response of a patient in diabetic ketosis to fluids, salt and insulin.
When the depletion of niacin in the tissues has not been as severe, there appear the ordinary signs of pellagra: the red tongue, stomatitis and dermatitis. This is ordinarily called acute pellagra but in reality represents subacute, severe niacin deficiency.
Various other combinations are possible. For example, a mild chronic deficiency may have superimposed upon it an acute deficiency.
The pellagrin here illustrates such a situation. When first seen, this patient had the satiny smooth glossitis and the dermatitis of a chronic niacin deficiency. Following abdominal surgery, failure to eat and glucose infusions, the stupor and the sucking and grasping reflexes developed. They are signs of a severe acute niacin deficiency. Treatment of this case with niacin has to be modified as a result of the development of the severe acute symptoms. With adequate dosage, and this means large dosage, these severe acute symptoms will probably respond to therapy in two or three days. The mild chronic symptoms on the contrary respond very slowly. The pigmentation of the hands and the flattened papillae of the tongue take months to respond. The principle demonstrated in the case of niacin applies to practically every nutritional deficiency.
As another example, look at this case of severe acute scurvy. Now in this first case, an acute severe deficiency, the tissue changes are largely represented by hyperemia, hemorrhage and edema. Our experience with cases of this type makes it possible to predict with full assurance that with adequate dosage, 500 to 1000 mg daily, the outstanding symptoms in such cases will disappear in about 72 hours and the gum shrink to almost normal position in about that time.
With almost equal confidence, it can be predicted that this case of mild, chronic ascorbic acid deficiency will show slow response to therapy. The tissue changes are a response to a chronic deficiency, even though mild. Hypertrophy or atrophy of the tissue has time to develop, and response to therapy is very slow.
For those who are inclined to take the diagnosis of deficiency state casually, it should be pointed out that we are faced simultaneously with one other problem, the problem of multiple deficiencies. One must acquire a facility for identifying the more common lesions of malnutrition. Let us see and name some of them.

You examine first the eyes. With malnutrition, known or suspected, you look for changes in the conjunctiva. This eye shows a typical xerosis conjunctiva. Note its position in the equators at either side of the cornea. It is usually more marked on the nasal side. Not all spots appearing in the conjunctiva on either side of the cornea represent xerosis conjunctiva. Some are synechiae and some are sclera deposits of varying composition. This lesion being chronic requires months of vitamin A therapy. These two eyes, before and after, show the results of vitamin A therapy after persistent treatment for months. Note that the spot may appear more prominent after treatment, due to thinning of the conjunctiva around the spot. In addition, the blood vessels in the sclera may also appear more prominent due to this same thinning of the conjunctiva.

The next thing to look for in the eye is corneal vascularity. One needs either the biomicroscope and slit lamp or the small hand slit lamp. This corneal vascularity may be due to riboflavin deficiency. But light or other trauma or even other diseases may produce corneal vascularity, so one again is confronted with making a differential diagnosis. If the patient gives symptoms of burning, itchy, tired eyes, and they respond fairly rapidly to riboflavin therapy, one may postulate the diagnosis of riboflavin deficiency. In this eye, one may see corneal vascularity but without the symptoms of itchy, burning and eye fatigue. Response to riboflavin is slow, and in many if not most subjects, little response may be obtained in a year therapy. This may mean either that the corneal vascularity was not due to riboflavin deficiency. It represents a relatively irreversible lesion.

We come now to the mouth. In this patient, the lesions at the corner of the mouth are typical of the cheilosis of riboflavin deficiency. Not all fissures in the angles of the mouth are due to a lack of riboflavin. In elderly patients, one often sees little fissures and cracks in the corners of the mouth. The lips cave in. She opens her mouth and one discovers the reason. Her need is not primarily for riboflavin but for some serviceable dentures that she will keep in her mouth and use.

Now let us go to the tongue. Two deficiencies are manifest in this organ. The most common is niacin deficiency. The earliest signs are red, swollen, fungiform papillae. As the deficiency continues, the filiform papillae also become red, giving the scarlet red tongue. This tongue shows what to expect in severe acute deficiency in niacin. This is the scarlet red stomatitis of pellagra. The other extreme as represented by this tongue, is that of a mild chronic niacin deficiency. See the atrophy? The papillae may be mushroomed out but are not so intensively red. They may show fusion or atrophy, and if this has gone on over several years, you see fissuring, giving a geographic or scrotal type of tongue. These tongues may or may not be associated with pernicious anemia. In any case, they often respond to niacin over long periods of time. This is not to infer of course that all tongues showing geographism or scrotal distribution of the fissures are due only to a niacin deficiency. Many, possibly most, do respond to niacin. Others are congenital and still others may be the result of *lichen planus* or other independent mouth lesions.

In riboflavin deficiency, the tongue, instead of being red and coated, may develop a purplish or magenta hue. Some observers have stated that pyridoxine deficiency will produce the same appearance in the tongue.

Now the gums. Let us briefly refresh our memories as to how healthy gums appear. Note the pointed interdental papillae. Note the healthy pink color of the gingiva. There is no recession or marginal redness. And now for contrast, we have here the acute lesions of scurvy. As previously stated, when we give adequate doses of ascorbic acid, the gums will seem almost to melt away as they return to their normal position and contour.

The lesions of mild scurvy are less definite. Also, what role infection and calculi have to do with the production of these signs is not clear. It is probable that ascorbic acid deficiency is the conditioning factor for many changes that occur in the gums, but by no means all.

For example, in this case of Vincent's infection, the primary diagnosis based upon the presence of the scarlet red stomatitis is niacin deficiency. If specific therapy, in this instance niacin therapy, is instituted, not only will the scarlet red stomatitis be blanched within 24 to 48 hours, but the Vincent's infection heals without other general or local therapy.

Certain deficiency signs in the skin should be familiar to every physician. The dermatitis of pellagra for example, although more common where the disease is endemic, may be seen in patients in any section of the United States. Typically, as in this patient, the lesions appear on the exposed parts of the backs of the hands, wrists and forearms. It may appear on the neck or about the ankles. It is usually symmetrical. The areas may progress to ulceration, desquamation and cracking and bleeding. Sunlight enhances the discomfort and the burning sensation.

The skin lesions associated with scurvy are the petechial or parafollicullar hemorrhages, often on the lower extremities or in areas where the skin is subjected to pressure. A number of observers have described various skin lesions which disappear after the administration of the whole vitamin B complex. Some of these lesions are of the nature of a chronic or acute eczema. Signs of vitamin A deficiency are often observable in the skin. Here we see the hyper-folliculitis of vitamin A deficiency.

Now we shall consider the neurologic manifestations of malnutrition. A diagnosis of mild polyneuropathy may be made when neurologic examination discloses the following signs. Calf muscle tenderness. The healthy calf muscle, if squeezed from behind so as not to include the tibia in the grip, can tolerate considerable pressure before pain is felt. Pain occurs in the presence of a mild vitamin B1 deficiency as this patient's face indicates.

Plantar hyperesthesia. We refer here not to the ordinary tickling sensation usually elicited by scratching the undersurface of the foot. Plantar hyperesthesia is a very definite hyperesthetic pain produced by drawing an object firmly but not un-gently over the bottom of the foot. How uncomfortable this becomes can be judged by the patient's facial reaction.

If there is a mild polyneuropathy, vibratory sensation, tested with a c256 tuning fork becomes diminished These three signs, calf muscle tenderness, plantar hyperesthesia and diminished vibratory sensation do not establish a positive diagnosis of polyneuropathy due to a general or systemic nutritional deficiency since circulatory disturbances may cause these or similar findings. If in addition the ankle jerks are absent, a diagnosis of mild polyneuropathy should be made. Note that the ankle jerks are absent in this patient. A diagnosis of moderate polyneuropathy may be made when knees and ankle jerks can no longer be elicited but positive signs are still limited to the lower extremities.

We shall see how this applies in this patient. Impairment of position sense in the toes is now quite definite. The gait is now frequently affected and the burning sensation, often noticed earlier, but now quite acute in the soles of the feet plus diminished position sense may compel these patients to walk carefully as if barefoot on a floor strewn with tacks. Calf muscle atrophy is now very obvious. Toe drop and foot drop are also plainly evident. Wrist drop soon appears. Walking, virtually impossible, is of a steppage type.

As in thiamine deficiency, objective signs of niacin deficiency are often preceded by mental symptoms. These symptoms involve all forms of sensation. Increased psychomotor activity, definite trends toward depression and apprehension, and wariness, fatigability, headaches and insomnia.

In patients with classic pellagra, the mental symptoms commonly seen are those found in any organic psychosis. They include memory defects, disorientation, confusion and confabulation. Periods of excitement, depression, mania, delirium and paranoia occur not infrequently. These mental disorders may appear before other signs of pellagra are evident. If the psychosis has been of short duration, response to treatment with niacin is usually prompt. Referring once again to our chart, it may be pointed out that from the standpoint of niacin deficiency, the

pellagra syndrome of stomatitis, mental symptoms, diarrhea and dermatitis represent a chronic partial deficiency of nicotinic acid. When the availability of niacin is suddenly reduced, and the restriction is virtually at complete deficiency, structural changes may not have time to develop, and a fairly characteristic neuropsychiatric syndrome occurs which has been called nicotinic acid deficiency encephalopathy.
In endemic pellagra, the encephalopathic syndrome occurs only in the most advanced and severe cases. The syndrome may occur as the only clinical manifestation of a deficiency disease or it may occur in association with polyneuropathy, scurvy or ariboflavinosis. We may thus have an acute, severe niacin deficiency superimposed upon deficiencies of almost any degree of the other water-soluble vitamins.
Nearly every one of the patients we have studied had a multiple deficiency. This must follow by necessity when we consider the fact that neither primary dietary failure nor conditioning factors are as a rule selective of any one vitamin. With the possible exceptions of vitamins D, E and K, there can seldom be a diagnosis of a single vitamin deficiency.
Sydenstricker, Williams and Wilder, Smith, McLester and other investigators have observed and described sub gross, multiple deficiency states, sometimes loosely grouped as a neurasthenic syndrome. Such deficiency states no longer need remain in the dim realm of the subclinical or sub gross. With the diagnostic methods now at hand, it is often possible to eliminate guesswork and opinion and to place our diagnosis upon a firm, objective basis.

And now, back to our conference."

*Thank you, Dr. Jolliffe. And now we shall hear from Dr. Spees on therapy. Dr. Spees.*

"Dr. Jolliffe has broken new ground for us today with his able exposition of the relationships between the various deficiency states. It is not surprising that a parallel statement as to therapy has recently appeared from my good friend, Dr. V. P. Sydenstricker. We have long applied these principles in our work at Hillman hospital. The following dosages are for adults. Suggested dosages for children are determined by proportional body weight.

BASIC FORMULA

In treating the clinical syndromes of beriberi, pellagra, riboflavin deficiency and scurvy, we use a basic formula containing 10 mg of thiamine, 50 mg of niacin, 5 mg of riboflavin and 75 mg of ascorbic acid.
When we find the symptoms of one deficiency disease predominant, we add to the basic formula more of the vitamins specific for the predominating deficiency. In beriberi, 10 mg of thiamine are added daily. In riboflavin deficiency, 5 mg of riboflavin twice daily. In scurvy, 100 mg of ascorbic acid three times a day. And in mild pellagra, 50 mg of niacin amide three times a day. If the pellagra is severe, the patient is given 150 mg of niacin amide three times a day in addition to the basic formula.
When the patient is moribund, it may be necessary to resort to parenteral injection in order to prolong and indeed even to save life. When large amounts of D-glucose are injected daily, we recommend inclusion of 50 mg of niacin amide, 7½ mg of riboflavin and 5 mg of thiamine. In a few instances, we have found it desirable to inject 50 mg of ascorbic acid.

Vitamin A deficiencies are usually of the mild chronic type and therefore require months or even years for complete regression. Vitamin A should be administered in dosages of 50,000 to 100,000 units daily.

When a specific prompt return to health is desired, vitamin therapy, either oral or parenteral, may reasonably be based on giving too much, too soon and for too long rather than too little, too late and not long enough.
We also administer a regular, daily supplement of 4-6 oz of dried brewer's yeast powder or oral liver extract. We mix this supplement with milk, tomato juice or with food on the diet tray. A careful check is made on the amount of food returned. If the supplement has not been consumed, it is added at a later meal.
In addition to the B vitamins, yeast and liver supply valuable amino acids, mineral salts and enzymes. Crude liver extracts may be administered intramuscularly with good effects. Care should be taken to distinguish between preparations made by diluting potent USP anti-pernicious anemia liver extract and the comparatively unrefined liver extracts which contain more of the active B complex principles. The therapeutic value of these liver preparations can be increased by the administration of thiamin, riboflavin and niacin amide in liberal dosage.
In less severe deficiencies, we give a polyvitamin formula supplying the daily adult allowances as recommended by the Food and Nutrition Board of the National Research Council.

RECOMMENDED DIETARY ALLOWANCES
NATIONAL RESEARCH COUNCIL

MINIMUM DAILY REQUIREMENTS
FOOD AND DRUG ADMINISTRATION

One, two or three of these capsules are given daily. These recommended daily allowances should not be confused with the minimum daily requirements set up for the package labeling purposes by the US Food and Drug Administration.

Upon initial and subsequent examinations, we endeavor to discover and correct or give due consideration to conditioning factors which may contribute to the deficiency state.
Finally but perhaps most important of all, we correct the patient's diet.

BUTTER, MILK, WHOLE GRAIN CEREAL

Diet alone cannot ordinarily be depended upon to supply the needed vitamins in corrective dosage.

MEAT, FISH, EGGS, LEGUMES

But a liberal and well-balanced diet is an essential element in this therapy.

VEGETABLES, LEAFY SALADS

In a primary dietary deficiency, there can be no favorable prognosis unless the patient permanently improves his diet.

FRUITS

This frequently presents a major management problem when the patient rebels against changes in his dietary habits.

- What you say about the importance of diet is particularly interesting to me, Dr. Spees. You will recall a demonstration made by Ebbs, Tisdall and Scott among 210 women attending the ante-partum clinic at Toronto General Hospital.

DIET OF WHOLE GROUP OF WOMENT BEFORE ATTENDING ANTE-PARTUM CLINIC

Analysis by the hospital staff had shown that these women had been consuming a poor diet containing approximately 1600 to 1900 calories, 60 g of protein, 500 mg of calcium and 10 mg of iron.
These 210 women were divided into two groups. In one group, no change was made in the diet. Those in the second group were furnished each day as a supplement one egg, 30 oz of milk, ½ oz of wheat germ, 1 oz of cheese, 4½ oz of canned tomatoes and one orange.

DIETARY IMPROVEMENT OF TEST GROUP AT ANTE-PARTUM CLINIC

This dietary improvement increased their calories from 1900 to 2600, the protein from 60 g to 100 g, calcium to 500 mg to 1600 mg and iron from 10 mg to 24 mg a day, besides materially raising their vitamin intake. The table shows the effect of the poor diet and of the supplemented diet.

TABLE 6 – EFFECTS OF A POOR AND GOOD DIET IN PREGNANCIES

| | POOR DIET | SUPPLEMENTED DIET |
|---|---|---|
| NUMBER OF WOMEN | 120 | 90 |
| OBSTETRIC ANTEPARTUM RATING “POOR” OR “BAD | 36 % | 9 % |
| MAJOR COMPLICATION DURING LABOR | 47 % | 17 % |
| MISCARRIAGES AND STILLBIRTHS | 9.4 % | 0 |
| DURATION FIRST STAGES OF LABOR FOR PRIMIPARAS | 20.3 HRS | 15.2 HRS |
| CONDITION DURING LABOR RATED “POOR” OR “BAD” | 24 % | 3 % |
| CONDITION DURING CONVALESCENCE RATED “POOR” OR “BAD” | 12 % | 4 % |
| FINAL RATING BY OBSTRETICIAN OF WHOLE COURSE FROM FOURTH MONTH THROUGH CONVALESCENCE, “POOR TIME” OU “BAD TIME” | 34 % | 6 % |
| COMPLICATION OF MOTHER DURING 6 MONTHS POST DELIVERY | 40 % | 10 % |
| BABIES HAVING BAD OR POOR RECORD DURING FIRST TWO WEEKS | 14 % | 0 |
| NUMBER OF BABIESWHO DIED UNDER 6 MONTHS | 3 % | 0 |

The 90 women whose diet had been supplemented had 30 % less major complications during labor, no miscarriages or stillbirths, better convalescence, fewer post-partum complications and lost none of their babies during the first 6 months.

Such studies indicate the prevalence of suboptimal nutrition in a population group whose health is of special importance. They are significant as a modern demonstration of what may be achieved by a better nutrition.

- Evidence of this character is rapidly accumulating. Studies on nutrition in industry, dietary surveys, hospital records, all confirm the prevalence of nutritional deficiency.

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INADEQUATE DIETS AND NUTRITIONAL DEFICIENCIES IN THE UNITED STATES

THEIR PREVALENCE AND SIGNIFICANCE

This evidence is now available to all in the literature. It has been well summarized by the Food and Nutrition Board of the National Research Council.

- Such evidence is, of course, necessary and proper. But any practicing physician who takes the trouble to familiarize himself with the objective lesions of vitamin deficiency can be convinced by his own observations. What troubles me most is the fact that some physicians earnestly inquire as to the significance of such widespread deficiency.

- Perhaps they overlook the fact that disturbances in the body chemistry are well-advanced before even microscopic tissue changes appear.

- And perhaps we've all been too prone, in the practice of medicine, to let well enough alone. I remember our records at Bellevue showing 17½ cases of malnutrition per 1000 discharges, which we all know represented only the most striking cases.

- Precisely. As Dr. Kruse has pointed out, and as we believe from our own work, the mild and chronic forms of malnutrition are accompanied by symptoms which are real handicaps. Photophobia, fatigability, emotional disturbances, the often-described neurasthenic syndrome in all its depressing personal and social implications.

- Dr. Spees can tell us something about the social importance of the nutritional problem out of his experience at Hillman.

- I would say that Dr. Jolliffe has stated the case conservatively. As you know, we in Birmingham work in a comparatively restricted area. For seven years now, we've been able to observe not only the result of therapy in the clinic, but social and economic effects in the community as well. We have to combat deeply implanted prejudice, as well as severe economic limitations. For that reason, we physicians have employed social workers and trained dieticians who have maintained continuous personal contact with our patients and their families.
Our problem is not solved. Perhaps it may never be. But the progress we have seen is unmistakable.

PELLAGRA DEATH RATE

Our pellagra death rate in severe and complicated cases, formerly 54 %, is approaching zero.

Many men who were unable to work have jobs in heavy industry or have joined the armed forces or are paying income taxes. Mothers who did not care for their families now do so. Some children who failed to keep up with their classes are now making normal progress. One of these boys is now a hotel manager, another is an engineer, and still another is a successful practicing physician.

- Could it be said, Dr. Spees, that what you have observed is peculiar to a region in which pellagra is endemic?

- I'm not speaking of pellagrins only. We do not know the incidence of pellagra in our community, and it is doubtful if it is known in any mass population group. These people around Birmingham are not different from others. They are not constitutionally inferior. Their heredity is much the same as yours and mine. Their problem is primarily one of malnutrition, with social and economic circumstances which set up a vicious circle. Our job is to break that circle.

- Your opinion is supported by experimental evidence. There was a test reported by Williams, Mason, Wyler and Smith from the Mayo Clinic, where a group of women were kept on a special hospital diet for several months. The meals were certainly appetizing and only close scientific scrutiny will detect any serious nutritional defect. Nevertheless, on this diet these women developed marked thiamine deficiency with all the classical symptoms ranging progressively from neurasthenic manifestations to polyneuritis and psychotic conditions. At this point, they were unable to care for themselves or perform any useful function in the community. When thiamine was administered, the symptoms promptly regressed.

- Other experiments now being conducted among large groups of industrial workers also support Dr. Spees' observations. In the field of industrial efficiency, the educational work of the National Nutrition Program for Industry is one method of attack. We are attempting to assure adequate feeding for every employee in the industry, a task in which we need the local support of every physician.

- The federal government is deeply concerned with this problem of better nutrition. We appeal to the medical profession for cooperation. A very definite step forward will have been made when the physician becomes accustomed to looking for gross and microscopic evidences of malnutrition in the eyes, in the mouth, on the skin, as well as for characteristic nerve responses. And when the physician gives more than a cursory glance at the patient's history as it relates to possible causes of nutritional deficiency, and when the physician prescribes diets adequate for the prevention of such deficiencies.

- And when the physician recognizes the significance of all this, for men and women engaged remotely or directly in war work.

- Exactly. We are beginning to learn that many persons without clinical evidence of disease are incompetent for the nation's need and inadequate to meet their own problems because of various forms of malnutrition. Chronic malnourishment has been found by careful medical observers among people in every walk of life, both children and adults. Through the knowledge now at hand, we can eliminate much of the half-health, half-strength and half-happiness which often results from nutrition which is not quite good enough."

THE END

Transcript: Elisabeth Fuchs