POSTULATING A NEW CONCEPT OF THE ETIOLOGY, PATHOLOGY AND TREATMENT OF CHRONIC IDIOPATHIC ULCERATIVE COLITIS — IS IT A HEMORRHAGIC DISEASE CAUSED BY MULTIPLE NUTRITIONAL IMBALANCES?

N. Philip Norman

Note to the Reader: Because of present Federal laws we are not permitted to mention certain commercial products and trade names which are blocked out in this book. For the latest specific information as to his experience with any special product we suggest that you write the author: N. Philip Norman, M.D., 19 East 88th Street, New York 28, New York.

Other Books

By

N. Philip Norman

Tomorrow's Food

Constructive Meal Planning

POSTULATING A NEW CONCEPT OF THE ETIOLOGY, PATHOLOGY AND TREATMENT OF CHRONIC IDIOPATHIC ULCERATIVE COLITIS — IS IT A HEMORRHAGIC DISEASE CAUSED BY MULTIPLE NUTRITIONAL IMBALANCES?

N. PHILIP NORMAN, M.D.

Publication of the Lee Foundation for Nutritional Research
Milwaukee, Wisconsin

Printed in the United States of America

Contents

Part I

PREAMBLE

I

PREAMBLE

"A rather rare malady occurring in nervous women" — thus did Osler describe colitis of the mucous variety in his first edition of MEDICINE. Studies during the past thirty years have contributed to a better understanding of all forms of colitis. The subject of this report is the variety of colitis which is now designated as chronic, idiopathic or non-specific ulcerative colitis. Despite increased progress and understanding, two fundamentals of this disorder — its etiology and cure — still elude us.

Chronic non-specific ulcerative colitis has been called a disease of civilization, a phrase implying that primitive people were not troubled by it. Is colitis as old as the colon? Are any of our modern *itises* as old as the organ to which these inflammation-denoting suffixes are respectively appended? Speculation provokes some interesting conjectures.

Evidently, man appeared on the earth about a half million years ago and presumably he had a colon similar to ours. Little is known of this man except the fact of his existence.

First, survival necessitated eating. Next, note his diet. He subsisted on fruits, berries, seeds, nuts, roots, and succulent portions of wild plants; also grubs, snails, worms, birds, birds' eggs, mice, lizards, frogs, snakes, scorpions, centipedes, insects, shellfish, and such animals and fish as he could catch — hunting and fishing implements had not been invented. Honey appears to have been a valuable source of nutriment for our early forebears. This dietary was omnivorous; it was not constant — being either seasonally abundant or insufficient. During periods of season-

This study was made possible by annual grants for the years 1945-1949 from the Lee Foundation for Nutritional Research, Milwaukee, Wisconsin.

al food scarcity, he may have escaped starvation through the biologic expediency of hibernation. Anthropologic reconstructions depict early man as half-animal and half-man. He was endowed with a large paunch and beneath the skin of this paunch there may have been rudimentary hibernation glands, which atrophied as his food supply became more constant and secure.

As long as man lived in family groups his dietary was composed exclusively of natural, unprocessed foods. Little or no culture existed within the framework of this primitive family group. Behavior was instinctive and concerned primarily with two basic objectives — survival and reproduction. Reproduction was prolific, consonant with modern nutritional research demonstrating that the reproductive rate is stimulated among animals fed a natural dietary. In time, with population increases, survival pressures became exigeant and family groups consolidated to fight the constant seasonal threat of famine. Primitive tribes were organized on a communal basis which afforded a greater protection against the common survival hazards. These primitives learned to conserve a portion of their seasonally abundant food supply to tide them over seasonal food shortages. Thus, population pressures continued to increase, became explosive and scattered these family groups over adjacent areas. This enlarged their sense of geography, and new environmental situations and conditions had to be met. Concomitantly, primitive cattle, goats, sheep, pigs, etc. were domesticated, providing a more secure food supply. With the use of the horse, the raft and the dug-out, travel became less arduous and people were no longer indented within circumscribed geographic areas. About this time each tribe began to evolve a set pattern of culture, conditioned largely by its environment. The two important aspects of this culture were concerned primarily with food and improved reproduction facilities. Later, we shall again refer to these primitive tribes and their evolution, to outline the conditions

that altered their original food cultures and economies as they approached the early stages of civilization.

When in the history of civilization chronic ulcerative colitis afflicted man, or *where* among the earth's tribes and peoples, remains unknown.

It is with the dietary aspects of chronic non-specific ulcerative colitis that this study is primarily concerned. These dietary aspects suggest the key to its etiology and the keystone for its rational management.

Present concepts of the etiology, pathology, prognosis and treatment should be reviewed first. *Civilization brought incorrect eating habits* — worse on the whole than the nutritional aberrations one encounters among primitive peoples, ancient and modern. It also brought food deficiencies and excesses through attempts to improve on nature's foods by milling, refining and processing.

Belatedly came some realization of the enormity of these mistakes. Now hasty and haphazard attempts are employed to fortify factory-foods whose known and unknown elements have been removed and/or destroyed through meddlesome processing.

Chronic non-specific ulcerative colitis was unknown until Cruveilhier,[1] reporting work done between 1829 and 1842, drew colored pictures of this pathologic entity. In the following hundred years significant accounts of sporadic occurrences of the disease were reported. Two reports have described it well.[2,3] Others included worthwhile statistical information, but apparently there was little interest in the problem until 1919 when Logan[4] reported a series of 117 cases. Thereafter, more study was devoted to this disease. Nutritionists were among the first to recognize that this disease was possibly a disease of civilization because of the fundamental, cumulative effect of a universally inadequate dietary. Only within the last two decades has an extensive knowledge of this clinical syndrome been acquired, although its exact cause remains a matter of debate and its treatment

a matter of the empirical administration of any particular miracle drug in current vogue.

For many years chronic non-specific ulcerative colitis was confused with the amoebic and bacillary dysenteries, and it was naturally assumed that all three were disorders of bacterial origin. Progressively, idiopathic colitis was differentiated as a separate entity. A general review of the diverse and contradictory concepts which were held, and are still held, regarding the etiology, pathology, prognosis and treatment of this type of ulcerative colitis is pertinent.

Part II
ETIOLOGY

II

ETIOLOGY

Although credible reports have appeared implicating various bacteriologic, allergic, neuro-psychogenic, and nutritional deficiency factors, causation of the disorder by any one of these alone has yet to be established on a definitive basis.

Bacteriologic Factors:

In 1924, Bargen[5] suggested that a *diplostreptococcus* of definite morphologic, cultural and biologic properties was probably the cause of some cases of chronic ulcerative colitis. Four years later additional studies confirmed this and he claimed to have proven that this organism was the etiologic agent of this definite disease entity. Daniels,[6] in 1942, stated that attempts to prove a specific organism as the primary cause of ulcerative colitis had failed and a stalemate had attended progress in the basic knowledge and treatment of this disease.

That the *diplostreptococcus* reported by Bargen plays a part in the etiology of some cases is supported by convincing evidence. Two groups, working independently and without knowledge of the other's results, concluded that this organism played a predominant role. It has been found by culture and smear in 80 per cent of a large series of patients with the disease, some of whom have been found harboring it in periapical dental abcesses, tonsils and sinuses. Organisms isolated from these sources, when injected into animals, produced pathology in their colons closely resembling ulcerative colitis as found in humans. Commenting on bacterial implications, Mackie[7] has this to say:

> Hurst has long considered the disease to be the result of continued infection by *Shigella dysenteriae*. The proponents of this hypothesis base their views largely on the results of the

agglutination reaction. Dysentery agglutinins, however, are frequently present in the absence of demonstrable homologous infection. Furthermore, there is evidence to indicate that these anti-bodies appear in response to heterologous infections by certain strains of *Escherichia coli.* Dysentery bacilli have been isolated in approximately 20 per cent of the cases that I have seen in New York City. Bargen and his associates hold that a specific diplococcus is responsible and that the organism can be recovered in 80 per cent of the cases. The specificity of this organism has been denied. Strains obtained from the Mayo Clinic have been shown to differ among themselves in their behavior in culture and in heat resistance. Their serologic characteristics are not uniform and there is close immunologic relationship with strains of Enterococcus. Other investigators have failed to recover the diplococci in a large proportion of their cases. More recently an anaerobic bacterium has been advanced as the primary agent. Most of the bacteria recoverable from the human colon have, at one time or another, been suggested as important etiologic factors.

In my opinion, the many types of bacteria observed in smears, or recoverable in cultures, from the colonic tract in idiopathic ulcerative colitis *are concomitants, rather than etiologic factors associated with this morbid process.* With specific cultural technics and media it is possible to grow almost any type of bacteria from a toxic stool. In fresh fecal smears one observes numerous *streptococci, staphylococci,* usually a marked reduction of *B. coli,* large numbers of red blood cells, white blood cells, plaques of disintegrating colonic epithelium, and mucous which is seemingly disproportional to the severity of the pathology. The more acute the disease, the less mucous one finds. When ulcerations and hemotumescent (a swollen bleeding mucous membrane; more commonly called ecchymotic or "paint-splotched") areas begin to resolve, then the mucous content of the stools increases. When complete resolution takes place, the amount of mucous secreted is normal. For many years I assumed that the excessive exfoliation and disintegration of colonic epithelium, the paucity of mucous, and the decrease in the amount of *B. coli* were due to a lysogenic substance.

The abnormal intestinal flora characterizing the stools of ulcerative colitis patients presumably elaborated this lysin.

The Lysozyme Hypothesis:

In 1922, Fleming[8] shed some light on the subject when he observed the action of a bacteriolytic and mucolytic enzyme which was apparently present in most surface mucous membranes subject to inflammation. He named this enzyme lysozyme. A decade later, Fleming[9] presented additional facts about lysozyme. Thereafter, it received little attention until Karl Meyer and co-workers[10, 11] showed that this mucolytic enzyme, lysozyme, was greatly increased in chronic non-specific ulcerative colitis — sometimes being 75 times more than normal. Moreover, they have shown that the lysozyme titre is normal in other colonic diseases. Lysozyme is a protein enzyme found to be secreted by the mucous cells lining the walls of the stomach and intestines. It is also a normal constituent of our digestive juices and is present in many tissues and fluids of the body. Egg white is a rich source of lysozyme — probably accounting for the frequent observation that eggs and food products containing eggs are not well tolerated by ulcerative colitis patients. When present in high concentration, lysozyme is capable of dissolving the protective mucous from the mucosa of the intestinal tract.

As a result of these investigations Meyer and his co-workers postulate two stages in the pathogenesis of chronic ulcerative colitis: first, the removal by lysozyme of the surface mucous with dissolution of the mucous cells; and second, an ensuing necrosis of the denuded tissue by proteolytic enzymes presumably elaborated by the invading organisms. The discovery that lysozyme apparently initiates the lesions of the disease stimulated a search for some "anti-enzyme" which might prevent or cure chronic ulcerative colitis by inhibiting or neutralizing the effects of lysozyme. Two chemical agents, now available but far from perfect, are *para*-nitrosulfathiazole and sodium hexadecyl sulfate. It

clusive. It is another interesting biochemical laboratory observation which merits further investigation. Several pertinent questions come to mind — does the lysozyme titre of tears and nasal secretions parallel the stool titre during emotional situations and in ulcerative colitis? Is it a localized or a generalized reaction to increased psychic tension?

From the laboratory standpoint it also raises some interesting questions. What relationship exists between the lysozyme titre and the sedimentation rate? Do they parallel one another — when the lysozyme titre is high, is the sedimentation rate greatly increased? My observation has been that as the patient improves, the sedimentation rate becomes progressively less rapid and it would be interesting to determine whether or not the lysozyme titre decreases correspondingly. Furthermore, research studies should be conducted to determine the relationship of increased titres of this enzyme to prolonged prothrombin times and depressed platelet counts, as well as its effect upon the functional integrity of the intercellular ground substance of tissue.

Hyaluronic Acid, Hyaluronidase and Capillary Permeability:

Hyaluronic acid is a viscous mucopolysaccharide found in almost all animal tissues, particularly in connective tissue and the pericapillary sheath which forms the "bed" for the capillary system. This viscous material is normally resistant or impermeable to invasion, and functions as a simple tissue cement and as a binder of intercellular fluid. The viscosity of the gels, formed by the union of this acid with the intercellular ground substance of tissue, plays a major role in the exchange of electrolytes, various metabolites, and fluid.

At strategic points throughout the organism, the gels of this acid maintain viscous barriers which act in the regulation of metabolic processes as well as in the localization of infections. However, variations in this resistance do occur and determine the degree of permeability of the tissues. Therefore, the function of hyaluronic acid is to

maintain the viscosity of the intercellular ground substance of the tissues.

Recent work with the "spreading" factor, hyaluronidase, has demonstrated that this substance antagonizes or inhibits the hyaluronic acid activity on the ground substance, thus permitting fluids to diffuse more freely. Later, the role which these factors play in the pathology and treatment of idiopathic ulcerative colitis will be discussed.

Allergic Factors:

In 1925, Andresen[15] postulated the theory that allergic sensitivity to specific food substances was the underlying cause of ulcerative colitis. He stressed the resemblance of mucosal lesions in ulcerative colitis to allergic reactions in the skin; the similarity of the early pathology of this disease to that produced in experimental gastro-intestinal allergy; and the ease with which the entire symptomatology of ulcerative colitis might be explained on an allergic basis. Andresen's contribution is plausible but not definitive.

Fecal smears, when fixed and stained by Wright's method, usually reveal a high percentage of acidocytes, hematologically implying the presence of an allergy. They also reveal large numbers of lymphocytes and what are probably mast cells.

Upon considering the nutritive inadequacies of food substances prescribed for ulcerative colitis, it is not surprising that allergic manifestations occur. The orthodox dietary for this condition is strikingly similar to the usual dietary prescribed for youngsters. It is composed mainly of pureed vegetables, cooked fruits, an abundance of sugar and other highly processed carbohydrates, processed proteins and excessive quantities of eggs and milk, *per se,* and as ingredients in food dishes. There is an almost complete absence of raw fruits, berries and melons, raw salads, properly cooked vegetables, whole grains and cereals, and fresh meats of good biologic quality. It is a recognized fact that allergic manifestations are prevalent among our youngsters. These

allergies are manifestations of nutritional deficiencies and excesses which disturb the body chemistry, chiefly the acid-base balance, the calcium-phosphorus and the sodium-potassium ratios.

Therefore, these allergic manifestations should not be considered etiologic; rather, they should be considered accessory phenomena attending prolonged subsistence upon inadequate dietaries.

Neuropsychogenic Factors:

Professionals familiar with the psychosomatic principles of medicine believe that many cases of ulcerative colitis are primarily neuropsychogenic in origin.

The concensus is that a colitis personality or situation is usually present. A careful history often reveals a severe emotional conflict antedating the onset of the disorder. Obvious remissions and exacerbations often parallel the solution or frustration of these conflicts. Considered from a purely neurogenic standpoint, the work of Lium and Porter[16] is an outstanding contribution. According to these investigators there is a possibility that ulcerative colitis may be a specific reaction to a number of influences which can initiate smooth muscle spasm of the colon. The emotional disturbances (part of the disease of civilization concept) frequently cause parasympathetic stimulation resulting in hyperperistalsis of the colon and digestion of its mucosa. Bacterial invasion and ulceration follow. This phenomenon has been induced in dogs by the stimulating effect of parasympathetico-mimetic drugs. Repeated spasm of the colonic musculature damages the mucosa and results in persistent hemorrhage and ulceration. Thus, neuropsychogenic implications are clear and have led to the use of antispasmotic drugs and psychotherapy.

The Dutch physician, J. Groen[17] claims to have found profound psychological disturbances in patients suffering from chronic ulcerative colitis and concludes that the disease is the result of "psychogenic" factors. Therefore, Groen

is possibly the chief supporter of the ulcerative colitis "personality" or "situation" theory. Groen's psychosomatic philosophy was excellently reviewed in an editorial by Beaumont S. Cornell.[18] Cornell approaches the subject from an unbiased standpoint and substantially contributes to an understanding of Groen's views. To a large extent, I concur with Groen. As I see it, however, the psychosomatic factor is not the basic cause — rather it operates as a "trigger" mechanism to set-off a series of events in inadequately nourished and adjusted persons, thus precipitating a full blown attack of ulcerative colitis. In other words, the colitis is present in latent form until an intervening psychosomatic crisis becomes an exacerbatory factor.

It is impossible to discount the effect of the tremendous physical and psychic strain attending a morbid state of monotony induced by repetitious routines. To this must be added the pitiful state of continuous frustration, the severe mental and emotional conflicts engendered by economic insecurity, and the multiplicity of other factors which increasingly complexify an already overly-complex civilization. Under these circumstances the human machine is likely to crumble. No comprehensive colitis regimen should fail to consider the correction or amelioration of physical strains and mental conflicts from which the patient may be suffering.

Undoubtedly, a constitutional emotional instability predisposes the individual to the disease. In all of my cases the psychosomatic factor was present and active. A significant per cent were severe psychoneurotics. Five cases traced the onset to a period of reactive depression. Schizoid personalities were frequently observed. Unfortunately, many physicians lack training in neurology and psychiatry which is a handicap in the management of these cases. On the other hand, something more than psychotherapy is needed. One cannot escape the question — why are these people emotionally unstable?

None of these etiologic concepts are definitive. They represent only partial aspects of the pathogenesis of the disease. When considered in the light of recent researches concerning the hemorrhagic diseases and nutrition, a new concept can be postulated.

Nutritional Deficiency Aspects:

From year to year the concept of deficiency diseases has broadened. These diseases were originally thought to be a direct manifestation of a specifically inadequate (deficient) diet, and were limited to a few well-defined clinical syndromes, as scurvy, beri-beri, rickets, etc. Today many more clinical syndromes have been added to the list and as research proceeds it is increasingly recognized that practically all, if not all, of the systemic diseases are direct or indirect manifestations of nutritive failure and improper nutrition.* This list comprises the primary and secondary anemias, the hemorrhagic diatheses, tropical and non-tropical sprue, gastro-intestinal diseases, pellagra, the several non-specific dermatologic and neurologic diseases, allergic states, endocrine disturbances, and the many masked, immature, poorly defined and border-line manifestations of improper nutrition and/or nutritive failure.

The result of this study justifies the postulation that there is an eye-to-eye correlation between faulty nutrition and ulcerative colitis when it is considered as a hemorrhagic disease. This postulation, from an etiologic standpoint, involves:

1) The prolonged subsistence upon a dietary containing simultaneous excesses and deficiencies of multiple nutrient factors.

2) The presence of the *accessory etiologic factors* — bacteriologic, psychoneurotic, allergic, the lysozyme hypothesis and a disturbance of the hyaluronic acid-hyaluronidase

*I prefer the terms "improper," "faulty," or "inadequate" nutrition to the term malnutrition. "Improper," "faulty," or "inadequate" nutrition is not exclusively concerned with deficiencies of essential nutrient factors. Nutritional deficiencies are always associated with nutritional excesses, except in the case of starvation.

balance — is the end result of this biologically inadequate and imbalanced dietary.

3) This hemorrhagic disease is characterized by a profound disturbance of the blood clotting mechanism and a demonstrable increase of capillary permeability and capillary fragility due to major nutritional deficiencies of the total vitamin C complex, vitamin K and the vitamin P complex, and of the minerals, chiefly calcium and iron. Accessory, conditioned deficiencies involve other nutrient factors — notably vitamin A, the total B complex, and proteins of good quality.

4) Collectively, the foregoing factors are responsible for the genesis of the ulcerative colitis syndrome.

Part III
PATHOLOGY

III

PATHOLOGY

In contrast to the divergent views regarding the cause of ulcerative colitis, there is more agreement in regard to its pathology. Intensive study of hundreds of cases has been aided by *in vivo* observation of the pathology by proctoscope and sigmoidoscope.

It will be remembered that Andresen[19] postulated that the pathology and symptomatology of ulcerative colitis could be explained on an allergic basis. He presents a concise description of the pathology.

> The pathological lesions of ulcerative colitis have been quite definitely described and may occur in a part or the whole of the colon at one time. In the early stages of hyperemia, hypersecretion and mucosal edema are present and a punctate rash, resembling herpes, is usually seen. As a result of localized ischemia, areas of necrosis develop and slough out, leaving ulcerated areas, which, when they coalesce, cause often extensive areas of denudation of mucosa, with small islands or tabs of mucosa still visible. These denuded areas may bleed upon slightest trauma or the actual laceration of small blood vessels may produce more profuse bleeding. Very deep sloughing may produce single or multiple perforations, with resulting local or general peritonitis and later adhesions, deformities, kinks or obstructions. Induration, with infiltration by leucocytes, often demonstrable as eosinophiles, may disappear with healing or may result in fibrosis, with marked narrowing or stiffening of the colon. The mucosal lesions may disappear entirely, or with long continued irritation, may result in polypoid changes, in which carcinoma may, but does not often develop. More or less bacterial invasion may be found in the tissues and lymphadenitis may be demonstrable in the mesenteric glands.

Andresen's presentation is consuetudinary and perhaps slightly colored because of his views on the role which allergy plays in this disease. For many years I have felt that the pathologic descriptions were static — nothing of signifi-

has been reported that *para*-nitrosulfathiazole controlled symptoms in about 50% of cases; sodium hexadecyl sulfate is reported beneficial but frequently aggravates the condition when first administered.

Another challenging observation concerning lysozyme is that in high concentration it seems to inhibit or prevent the satisfactory absorption of vitamin A.[12] For many years the vitamin A levels of my patients have been checked with a bio-photometer and in all instances, unless the patients were taking a high potency vitamin A preparation or physically improving, the bio-photometer readings revealed a lack of this substance. Therefore, the low vitamin A levels are probably a conditioned deficiency. There is also some likelihood that a high concentration of this enzyme conditions the absorption, and intestinal bacterial synthesis of vitamin K; and it may be responsible for the almost universally low ascorbic acid level found in these patients.

Grace and his associates[13, 14] undertook a study of the lysozyme concentration in the stools of human subjects living under diverse circumstances and situations in an attempt to discover whether or not increased lysozyme production was a concomitant of certain psychic factors. The lysozyme titre in a group of healthy persons without complaints was determined and found to be relatively low, ranging from 0.3 to 1.7 units per gram of wet stool. It was observed, however, that emotional situations (guilt, anxiety, resentment, etc.) in normal subjects and subjects with diarrhea without ulceration, provoked transitory elevations of the lysozyme titre. Lysozyme titres fell to lower figures when the psychic tensions in these individuals abated. Grace's observations tend to support the contention that psychosomatic factors do positively influence the condition of the ulcerative colitis patient. His findings seem to forge another link connecting unfavorable psychic situations with a biochemical phenomenon of adverse significance. In my opinion, the lysozyme concept while intriguing, is incon-

cant interest appeared after Cruveilhier described the disease. Andresen's contribution adds little besides the observation that "a punctate rash, resembling herpes, is usually seen."

To date, the pathologic descriptions of idiopathic ulcerative colitis have fallen short of supplying the clinician with an adequate concept of the fundamental disturbances of structure and function of the intestinal tissues which occur as a consequence of this disease. Pathologic pictures are too frequently adduced from autopsy studies instead of *in vivo* observations. This would color the pathologic picture of idiopathic ulcerative colitis because the autopsy findings reveal the tremendous amount of tissue wreckage that has occurred in the intestinal tract of people who died from the disease.

When one begins to correlate the diverse aspects of the etiologic factors with the pathology, then one begins to see the light. Pathologically, we must take a new view of the reason for congestion, hyperemia, punctate rashes, small and large ulcerations, so-called "paint-splotched" or ecchymotic areas, tubulation and uncontrollable diarrhea which characterize this disease in its active phase.

Recent studies in the diathetic hemorrhagic diseases have brought to light some new concepts concerning their etiology, pathology and treatment. These justify my postulation that chronic idiopathic ulcerative colitis should be included in the hemorrhagic group of diseases.

Chronic idiopathic ulcerative colitis *should no longer be considered a non-specific disease.* Data will be presented to support the thesis that it is a nutritionally-induced disease involving the consumption of foods containing excessive amounts of the ternary elements and noteworthy deficiencies of specific nutrient principles, simultaneously. Fundamentally, the ulcerative colitis patient is sick because our food culture has been degraded to a dangerously low level. Concomitant with this debasement, dislocations in our food

economy occurred and have had a far-reaching effect upon our total economy, and the health of every person.

The cumulative effect which all the previously enumerated etiologic factors have upon the development of the pathologic lesions, perhaps would never take place if the nutritional factor was adequate. All these factors are but partial aspects of the total problem. In appraising the disease, it is imperative to consider faulty, improper nutrition as the chief etiologic factor. Co-related and accessory factors must be elicited and added to the damage done by the nutritional factor. When this disease is appraised on this basis, it is possible to formulate a clear cut concept of the disease in its totality. New etiologic and pathologic horizons emerge concerning three major factors:

1) Alterations in the blood clotting mechanism.

2) The presence of increased capillary fragility.

3) The presence of increased capillary permeability.

In my series of cases, the presence of extensive hemotumescent areas preindicated a morbid condition. The blood loss through extravasation in these hemotumescent areas is constant and quantitatively more than the intermittent blood loss which occurs in the ulcerated areas. Therefore, these cases are not as responsive to treatment as those in which the ulcerated areas predominate. The foregoing observations have led me to classify my series of cases into three categories. Briefly, these are:

1) The granular, hemotumescent type with small, round shallow ulcerations, and an occasional large ulcer.

2) The ulcerative type in which the ulcerations are large and frequently involve the muscularis. The large ulcers predominate. Interspersed between the ulcers are hemotumescent areas.

3) The mixed type, in which both deep ulcerations and granular, patchy, hemotumescent areas are about equally present.

economy occurred and have had a far-reaching effect upon our total economy, and the health of every person.

The cumulative effect which all the previously enumerated etiologic factors have upon the development of the pathologic lesions, perhaps would never take place if the nutritional factor was adequate. All these factors are but partial aspects of the total problem. In appraising the disease, it is imperative to consider faulty, improper nutrition as the chief etiologic factor. Co-related and accessory factors must be elicited and added to the damage done by the nutritional factor. When this disease is appraised on this basis, it is possible to formulate a clear cut concept of the disease in its totality. New etiologic and pathologic horizons emerge concerning three major factors:

1) Alterations in the blood clotting mechanism.

2) The presence of increased capillary fragility.

3) The presence of increased capillary permeability.

In my series of cases, the presence of extensive hemotumescent areas preindicated a morbid condition. The blood loss through extravasation in these hemotumescent areas is constant and quantitatively more than the intermittent blood loss which occurs in the ulcerated areas. Therefore, these cases are not as responsive to treatment as those in which the ulcerated areas predominate. The foregoing observations have led me to classify my series of cases into three categories. Briefly, these are:

1) The granular, hemotumescent type with small, round shallow ulcerations, and an occasional large ulcer.

2) The ulcerative type in which the ulcerations are large and frequently involve the muscularis. The large ulcers predominate. Interspersed between the ulcers are hemotumescent areas.

3) The mixed type, in which both deep ulcerations and granular, patchy, hemotumescent areas are about equally present.

The term hemotumescent (a swollen bleeding mucous membrane) more adequately describes the condition than such terms as ecchymotic or "paint-splotched."

In the hemotumescent type, it is almost impossible to determine the nature of the hemorrhage — is it the result of capillary fragility or capillary permeability? Apparently, these two conditions are concurrent, co-existent and co-related and the hemorrhage may be due to a rupture of the capillary wall or to an oozing or extravasation of blood through the intercellular spaces of the vascular epithelium, or to both processes occurring simultaneously.

In the ulcerative type, hemorrhages are due to increased capillary fragility and to the necrotic erosion of the mucous membrane involving the capillaries and small blood vessels which constitute the vascular supply of the ulcerated areas. In this type there are patchy hemotumescent areas in which the presence of capillary fragility and permeability can be demonstrated. Moreover, a massive hemorrhage, the result of the eroding effect of the necrotic process upon a sizeable artery or vein, is frequently encountered.

In the mixed type, all types of hemorrhage occur.

If treatment is ineffectual, a chronic condition ensues. According to Boyd[20]:

> The tissue reaction in chronic inflammation is often said to be productive in character, but the cells which collect in response to the irritation either come from the blood stream (lymphocytes, etc.) or are derived from those wandering tissue cells which go by the alternative names of histiocytes, mononuclears and macrophages. The only cells which proliferate are the fibroblasts . . .
>
> The fibroblasts proceed to lay down collagen fibers. The collagen fibers form wavy bundles of *scar tissue*. By this process of fibroblastic proliferation and immigration together with the formation of fibrils of increasing density the two surfaces of the wound are firmly sewn together. When the fibers are fully formed they shorten, and this contraction continues for some months, so that the scar which was at first raised becomes puckered.

Marked tubulation (pipe-stemming) of the colon takes place when the scar tissue contracts. As a consequence of this process the *reservoir* or *storage capacity* of the colon is reduced proportionally to the severity of the scarring. The result of this is a continuous and uncontrollable diarrhea. Nature attempts to compensate for this cicatricial crippling of the terminal two-thirds of the colon with a dilation of the caecum and ascending colon. This may have the effect of diminishing the number of stools, but the compensation is never sufficient to relieve the patient of diarrhea, even after all signs of the disease have disappeared.

If the physiologic function of the colon is properly understood, then the validity of the above concept becomes clear. Unfortunately, to date, concepts concerning colonic peristalsis are not definitive. In an article published by International Clinics in 1923,[21] graphs were presented illustrating the peristaltic cycle which occurs when a colonic irrigation is properly administered. A study of these graphs will portray, by implication, the disturbance of the normal peristaltic cycle which pipe-stemming produces. Incidentally, the nutritional, etiologic perspective of gastrointestinal diseases, incorporated in this article 28 years ago, will reveal that the thesis then postulated is sounder today than it was at that time.

Analogous pathologic lesions occur in scurvy involving a different portion of the alimentary tract — the gums are swollen, tender, hyperemic and the bleeding may be spontaneous or traumatic. Subsequent to swelling, a characteristic atrophy may result accompanied by a retraction of the gingiva. This cicatricial retraction which occurs in the gingiva, in my opinion, is analogous to the cicatricial crippling of the colon in many cases of idiopathic ulcerative colitis.

Hemorrhagic Aspects of Idiopathic Ulcerative Colitis:

It is necessary to consider all factors, local and constitutional which play a part in the genesis of this disease. A

better understanding of these constitutional factors — pathologic, biologic and physiologic — is in order.

Five new factors were introduced and merit discussion:

1) Lysozyme.
2) Hyaluronic acid and hyaluronidase.
3) Alterations of the blood clotting mechanism.
4) Increased capillary fragility.
5) Increased capillary permeability.

The first two factors are just emerging from the experimental laboratories but from what is known of them, it seems that they play an important role in maintaining the integrity of the capillary system, the capillary bed and the intercellular ground substance which are so necessary for maintaining barriers to invasion, or to block leaks. Sufficient evidence is at hand to connect a hyaluronic acid-hyaluronidase imbalance directly with a deficiency, or a disordered metabolism of the flavonols, especially rutin.[22, 23] More about this when capillary friability and permeability are discussed.

The other three factors presented stressed the concept that several factors favoring hemorrhage are involved. When considered from this point of view, idiopathic ulcerative colitis emerges as a hemorrhagic disease. Hemorrhagic diseases are characterized by a disturbance of the blood clotting mechanism and by structural and functional changes in the capillary system, the capillary bed and the intercellular ground substance, which results in increased friability and permeability. Spontaneous or traumatic hemorrhage may occur in the skin, mucous membranes, serous membranes, or the viscera — each disease entity having its special characteristics. In idiopathic ulcerative colitis, the spontaneous, extravasative or traumatic hemorrhage is confined to one or more segments of the gastro-intestinal tract. Therefore, the intelligent approach to this disease is predicated upon a thorough understanding of the simultaneously active factors which produce the local pathology and the disturb-

ance of the biologic and physiologic mechanisms concerned with blood clotting and which maintain the normal integrity of the tissues of the capillary system.

Recently, blood clotting, hemostasis and the phenomena of capillary fragility and permeability have been intensively studied. With a more comprehensive understanding of the part which the aforementioned factors play in the physiological aspects of blood clotting and capillary fragility and capillary permeability, we have revolutionized our therapeutic approach to the management and control of the thromboembolic diseases.

Literature concerning these factors is voluminous, academic, controversial and often confusing. The excellent contributions of Quick, Tocantins, Howell, Seegers, De Takats, Wright, O'Neill, the Josiah Macy reports and other references too numerous to mention, have been reviewed. So far as I know, no one has sponsored the idea that idiopathic ulcerative colitis belonged in the category of the hemorrhagic diseases. This pigeonhole seems to have been reserved for a few diseases like purpura, hemophilia, occasionally Eale's disease, and lately the thromboembolic diseases. I feel that scurvy and idiopathic ulcerative colitis should also be added to this list.

It is the concensus that the three commonest contributory causes of thromboembolic diseases are:

1) An increase in the number of platelets.
2) An increase in the agglutinability of the platelets.
3) An accelerated rate of conversion of prothrombin.

A fourth factor, which frequently plays an equal and sometimes a predominant role in these diseases, is the presence of a degenerative process involving the vascular system itself. It is obvious that this factor may be more dynamic than the factors which reside in the blood. In the thromboembolic diseases, we now possess two drugs, heparin and dicumarol, which depress the conversion rate and

III

PATHOLOGY

In contrast to the divergent views regarding the cause of ulcerative colitis, there is more agreement in regard to its pathology. Intensive study of hundreds of cases has been aided by *in vivo* observation of the pathology by proctoscope and sigmoidoscope.

It will be remembered that Andresen[19] postulated that the pathology and symptomatology of ulcerative colitis could be explained on an allergic basis. He presents a concise description of the pathology.

> The pathological lesions of ulcerative colitis have been quite definitely described and may occur in a part or the whole of the colon at one time. In the early stages of hyperemia, hypersecretion and mucosal edema are present and a punctate rash, resembling herpes, is usually seen. As a result of localized ischemia, areas of necrosis develop and slough out, leaving ulcerated areas, which, when they coalesce, cause often extensive areas of denudation of mucosa, with small islands or tabs of mucosa still visible. These denuded areas may bleed upon slightest trauma or the actual laceration of small blood vessels may produce more profuse bleeding. Very deep sloughing may produce single or multiple perforations, with resulting local or general peritonitis and later adhesions, deformities, kinks or obstructions. Induration, with infiltration by leucocytes, often demonstrable as eosinophiles, may disappear with healing or may result in fibrosis, with marked narrowing or stiffening of the colon. The mucosal lesions may disappear entirely, or with long continued irritation, may result in polypoid changes, in which carcinoma may, but does not often develop. More or less bacterial invasion may be found in the tissues and lymphadenitis may be demonstrable in the mesenteric glands.

Andresen's presentation is consuetudinary and perhaps slightly colored because of his views on the role which allergy plays in this disease. For many years I have felt that the pathologic descriptions were static — nothing of signifi-

economy occurred and have had a far-reaching effect upon our total economy, and the health of every person.

The cumulative effect which all the previously enumerated etiologic factors have upon the development of the pathologic lesions, perhaps would never take place if the nutritional factor was adequate. All these factors are but partial aspects of the total problem. In appraising the disease, it is imperative to consider faulty, improper nutrition as the chief etiologic factor. Co-related and accessory factors must be elicited and added to the damage done by the nutritional factor. When this disease is appraised on this basis, it is possible to formulate a clear cut concept of the disease in its totality. New etiologic and pathologic horizons emerge concerning three major factors:

1) Alterations in the blood clotting mechanism.

2) The presence of increased capillary fragility.

3) The presence of increased capillary permeability.

In my series of cases, the presence of extensive hemotumescent areas preindicated a morbid condition. The blood loss through extravasation in these hemotumescent areas is constant and quantitatively more than the intermittent blood loss which occurs in the ulcerated areas. Therefore, these cases are not as responsive to treatment as those in which the ulcerated areas predominate. The foregoing observations have led me to classify my series of cases into three categories. Briefly, these are:

1) The granular, hemotumescent type with small, round shallow ulcerations, and an occasional large ulcer.

2) The ulcerative type in which the ulcerations are large and frequently involve the muscularis. The large ulcers predominate. Interspersed between the ulcers are hemotumescent areas.

3) The mixed type, in which both deep ulcerations and granular, patchy, hemotumescent areas are about equally present.

economy occurred and have had a far-reaching effect upon our total economy, and the health of every person.

The cumulative effect which all the previously enumerated etiologic factors have upon the development of the pathologic lesions, perhaps would never take place if the nutritional factor was adequate. All these factors are but partial aspects of the total problem. In appraising the disease, it is imperative to consider faulty, improper nutrition as the chief etiologic factor. Co-related and accessory factors must be elicited and added to the damage done by the nutritional factor. When this disease is appraised on this basis, it is possible to formulate a clear cut concept of the disease in its totality. New etiologic and pathologic horizons emerge concerning three major factors:

1) Alterations in the blood clotting mechanism.

2) The presence of increased capillary fragility.

3) The presence of increased capillary permeability.

In my series of cases, the presence of extensive hemotumescent areas preindicated a morbid condition. The blood loss through extravasation in these hemotumescent areas is constant and quantitatively more than the intermittent blood loss which occurs in the ulcerated areas. Therefore, these cases are not as responsive to treatment as those in which the ulcerated areas predominate. The foregoing observations have led me to classify my series of cases into three categories. Briefly, these are:

1) The granular, hemotumescent type with small, round shallow ulcerations, and an occasional large ulcer.

2) The ulcerative type in which the ulcerations are large and frequently involve the muscularis. The large ulcers predominate. Interspersed between the ulcers are hemotumescent areas.

3) The mixed type, in which both deep ulcerations and granular, patchy, hemotumescent areas are about equally present.

Marked tubulation (pipe-stemming) of the colon takes place when the scar tissue contracts. As a consequence of this process the *reservoir* or *storage capacity* of the colon is reduced proportionally to the severity of the scarring. The result of this is a continuous and uncontrollable diarrhea. Nature attempts to compensate for this cicatricial crippling of the terminal two-thirds of the colon with a dilation of the caecum and ascending colon. This may have the effect of diminishing the number of stools, but the compensation is never sufficient to relieve the patient of diarrhea, even after all signs of the disease have disappeared.

If the physiologic function of the colon is properly understood, then the validity of the above concept becomes clear. Unfortunately, to date, concepts concerning colonic peristalsis are not definitive. In an article published by International Clinics in 1923,[21] graphs were presented illustrating the peristaltic cycle which occurs when a colonic irrigation is properly administered. A study of these graphs will portray, by implication, the disturbance of the normal peristaltic cycle which pipe-stemming produces. Incidentally, the nutritional, etiologic perspective of gastrointestinal diseases, incorporated in this article 28 years ago, will reveal that the thesis then postulated is sounder today than it was at that time.

Analogous pathologic lesions occur in scurvy involving a different portion of the alimentary tract — the gums are swollen, tender, hyperemic and the bleeding may be spontaneous or traumatic. Subsequent to swelling, a characteristic atrophy may result accompanied by a retraction of the gingiva. This cicatricial retraction which occurs in the gingiva, in my opinion, is analogous to the cicatricial crippling of the colon in many cases of idiopathic ulcerative colitis.

Hemorrhagic Aspects of Idiopathic Ulcerative Colitis:

It is necessary to consider all factors, local and constitutional which play a part in the genesis of this disease. A

ance of the biologic and physiologic mechanisms concerned with blood clotting and which maintain the normal integrity of the tissues of the capillary system.

Recently, blood clotting, hemostasis and the phenomena of capillary fragility and permeability have been intensively studied. With a more comprehensive understanding of the part which the aforementioned factors play in the physiological aspects of blood clotting and capillary fragility and capillary permeability, we have revolutionized our therapeutic approach to the management and control of the thromboembolic diseases.

Literature concerning these factors is voluminous, academic, controversial and often confusing. The excellent contributions of Quick, Tocantins, Howell, Seegers, De Takats, Wright, O'Neill, the Josiah Macy reports and other references too numerous to mention, have been reviewed. So far as I know, no one has sponsored the idea that idiopathic ulcerative colitis belonged in the category of the hemorrhagic diseases. This pigeonhole seems to have been reserved for a few diseases like purpura, hemophilia, occasionally Eale's disease, and lately the thromboembolic diseases. I feel that scurvy and idiopathic ulcerative colitis should also be added to this list.

It is the concensus that the three commonest contributory causes of thromboembolic diseases are:

1) An increase in the number of platelets.
2) An increase in the agglutinability of the platelets.
3) An accelerated rate of conversion of prothrombin.

A fourth factor, which frequently plays an equal and sometimes a predominant role in these diseases, is the presence of a degenerative process involving the vascular system itself. It is obvious that this factor may be more dynamic than the factors which reside in the blood. In the thromboembolic diseases, we now possess two drugs, heparin and dicumarol, which depress the conversion rate and

amount of prothrombin and the agglutinability of the platelets.

In other types of hemorrhagic diseases the two commonest defects responsible for abnormal bleeding are to be found in the blood itself — a decrease in the prothrombin level and in the number of blood platelets. In the majority of my ulcerative colitis cases, the prothrombin times were greatly prolonged and the number of blood platelets were considerably decreased. Here we seek to accelerate the rate of formation of blood platelets, to increase their agglutinability, to normalize the prothrombin time, and to make available a sufficient amount of calcium ions for restoring the normal fluidity of the blood stream.

Fortunately we have a potent weapon for combating low prothrombin levels — vitamin K. On the other hand, thrombopenia (depressed blood platelet count) does not respond so readily to vitamin K therapy. This may explain the transitory beneficial effect of a fresh blood transfusion (rich in platelets) in thrombopenic cases.

But there are two other factors connected with this disease which cannot be ignored. These are concerned with capillary fragility and capillary permeability. In my opinion, there seems to be too much hair-splitting controversy regarding the meaning of these two terms. In ulcerative colitis we must consider conjointly these two capillary or vascular factors as evidence of a profound systemic disturbance in which the blood clotting mechanism is also involved.

Capillary fragility is characterized by a change in the structural integrity of the capillary wall which renders it extremely vulnerable to spontaneous hemorrhage or trauma; whereas, capillary permeability is a condition in which there is an alteration of the intercellular ground substance which cements the tissues of the capillary system together, thus permitting blood to extravasate, or ooze. In ulcerative colitis it appears that these factors are simultaneously present and interrelated. We do not need to employ the Rumpel-Leede

test or the Gothlin modification of it to determine the presence of capillary fragility. One may measure it directly during a sigmoidoscopic examination — spontaneous hemorrhages are observed and it is possible to rupture the capillaries in the hemotumescent areas with the slightest trauma. Nor do we need any tests to detect the phenomenon of capillary permeability — if one will lightly touch a portion of the inflamed colonic mucous membrane with a swab stick, one will find that blood is constantly oozing.

Significance of Abnormal Blood Clotting Mechanism:

Some readers may be critical because tests relating to bleeding time and blood clotting time have been omitted. These two tests are simple but time-consuming procedures and the determinations may be profoundly influenced by the following:

Site of puncture — ear, finger, vein.

Chemicals used to cleanse the skin.

Pressure applied before, during and after puncture.

Vasoconstriction or dilatation due to emotional factors.

Method of puncture.

Depth of puncture.

Clumsy venipuncture or finger puncture.

Sharpness or dullness of needle or blood lancet.

Dry or wet syringe.

Contamination with tissue juice, sweat or cleansing chemicals.

Turbulence or foaming during withdrawal and transfer of blood.

Type of paper or kind of receptacle (respectively) used for the test (glass, silicon-treated glass, plastic material, etc.)

It must be kept in mind that the *bleeding time* measures the activity of the platelet factor in hemostasis. The *clotting time* measures the rate of fibrin formation in clotting.

Quick, a well-known authority on this subject, states that clotting time and bleeding time tests should be discarded from clinical work, particularly in the otolaryngological field, because these tests frequently sponsor a false sense of security. I feel that Doctor Quick's remarks also hold true for idiopathic ulcerative colitis.

In this study, bleeding time and clotting time tests were not employed. Five tests are at hand for the determination of the activity of the platelet factor in hemostasis, the rate of fibrin formation in clotting, and the prothrombin level. These are:

1) Platelet count.
2) Prothrombin time.
3) Sedimentation rate.
4) Clot retraction pattern.
5) Quick's prothrombin consumption test for eliciting a deficiency of thromboplastin.

The vascular reaction of the patient's tissues cannot be anticipated or entirely controlled. Therefore, the collection of blood samples should be done in conformity with a standardized technique. In this study an effort has been made to do this. All hematologic and chemical determinations have been done on untreated and oxalated blood with the exception of the finger punctures necessary for obtaining blood drops on slides to determine the clot retraction pattern. Another important factor in collecting blood samples is the use of a sphygmomanometer which precisely determines the amount of pressure necessary to cut off the blood flow. This cannot be done with a tourniquet. Moreover, during the withdrawal of blood the sphygmomanometer facilitates a constant control of the applied pressure and patients are more comfortable and relaxed. I have used this method for many years and it is surprising to find that the tourniquet method is still so widely used in modern hospitals and routine office practice.

There are several theories concerning the mechanism of blood coagulation. One which is widely accepted is the Howell theory,[24] postulating that in normal blood a substance called antiprothrombin inactivates prothrombin. When thromboplastin is released from disintegrated platelets or injured tissue cells, the antiprothrombin is neutralized, thus permitting prothrombin to combine with calcium ions to form thrombin which in turn reacts with fibrinogen to form fibrin, or the clot.

More recently, Seegers[25] has modified the Howell theory by adding an accelerator factor.*

There are several other factors involved in the mechanistic pattern of blood clotting. The apparently important factors among these are:

1) Blood platelets. It is not well understood whether or not blood platelets have a direct action on thrombin. They apparently have a favorable influence on thrombic activity in reference to the elaboration of fibrinoplastic agents and substances.

2) Calcium ions. In optimal concentration these are essential for the physiologic activation of prothrombin. Apparently, the action of calcium is interrelated with the thrombic activity of the blood platelets.

3) Ac (accelerator) globulin is a plasma protein which seems to be essential for the *rapid potentiation* of prothrombin. A deficiency of this globulin may result in a bleeding tendency.

4) Antiprothrombin and inactive thrombin. These are the "balancers." A depression or an elevation of their levels may seriously affect the phenomenon of blood clotting.

Regardless of the theory to which one may subscribe, it appears that the significance of prothrombin in the mechanism of blood clotting is preeminent and predominant.

An analysis of the literature concerning the elaboration of adequate prothrombin directs our attention toward a

*This factor is variously known as Ac globulin, Factor V, labile factor or thrombogen.

nutrient essential — vitamin K. Unfortunately, vitamin K has been popularized as the anti-hemorrhagic and/or coagulation vitamin which places it in a false role. Vitamin K, *per se,* does not arrest hemorrhage in normal persons, the purpuric states, hemophilia, or in other hemorrhagic diseases but apparently acts as a biocatalyst for providing prothrombin. It is not definitely known whether or not vitamin K supplies a building stone necessary for the formation of prothrombin. There is no evidence that vitamin K is a component of the prothrombin molecule, because pure prothrombin preparations contain no naphthoquinone structure. It seems more likely that vitamin K is a stimulant to liver function for the synthesis of normal prothrombin levels. From purification studies we know that prothrombin is a glycoprotein. It follows, therefore, that unless constituent protein factors are available in the blood stream, the liver may be unable to synthesize sufficient prothrombin, despite the fact that vitamin K may be present in adequate amounts.

Quick is of the opinion that prothrombin is composed of calcium ions and two separable components — one of which is stable and appears to be related to the oxidation-reduction system of the blood, and the other being heat labile and inactivated by dicumarol. Other investigators have suggested that vitamin K takes part in an oxidation-reduction system in which -SH groups are oxidized to -S-S-, upon which the transformation of fibrinogen to fibrin has been postulated.

Summarizing: prothrombin, activated by calcium ions, thromboplastin, Ac globulin, platelet derivatives and other factors, forms thrombin which reacts with fibrinogen to form fibrin, or the clot. It is also important to consider the opposing mechanisms, the inhibitors, which may favorably or unfavorably condition the action and function of the activators in the consumation of the blood clotting mechanism.

Vitamin K is available from two sources:

1) raw fruits and vegetables, and
2) by intestinal bacterial synthesis.

This vitamin enters the stomach and the intestinal tract as a component of food. During the digestive process and after the food passes into the small intestines, the function of the gall bladder is stimulated and bile flows into the duodenum. In the presence of bile, the vitamin K content of the food and the amount synthesized by intestinal bacteria is absorbed by the intestinal mucosa for transport to the liver. If there is a deficiency of vitamin K in the diet it will be absorbed in subminimal amounts. The intestinal bacterial synthesis of this vitamin may be conditioned by the degree of the dietary deficiency. Another factor, hypermotility of the gastro-intestinal tract, may seriously interfere with the absorption and bacterial synthesis of this substance in the intestinal tract. As a result of this deficiency of vitamin K, the liver cannot synthesize an adequate amount of prothrombin. It should not be overlooked that the sulfa group of drugs, especially sulfadiazine, will inhibit the synthesis of vitamin K from available material by the intestinal bacteria. It is also known that if there is a disturbance of liver function, vitamin K cannot be used for the synthesis of prothrombin, thus resulting in a hemorrhagic state which is resistant to vitamin K therapy. An analysis of the average colitis dietary reveals that it is notoriously deficient in foods containing vitamin K — raw fruits and vegetables. Obviously, in idiopathic ulcerative colitis, where the dietary has been seriously restricted in foods containing vitamin K and where the hypermotility of the gastro-intestinal tract seriously impairs its absorption and bacterial synthesis, we are dealing with an *avitaminosis K.* To date, no relationship has been established regarding the effect which ulcerative colitis has on liver function although it probably exists. As previously mentioned, sufficient proteins of good biologic quality are necessary to complement vitamin K in the synthesis of

normal prothrombin levels. In idiopathic ulcerative colitis, the usual deficiency of proteins of good biologic value, the accelerated intestinal rate which frequently reduces the nutrient absorption, and the constant loss of blood are all factors which seriously condition the ability of the liver to elaborate adequate amounts of prothrombin. Therefore, in this disease, it seems possible to establish a correlation between the profound disturbance of the blood clotting mechanism and an induced and/or conditioned deficiency of vitamin K.

Capillary Fragility and Permeability

Different mechanisms of hemorrhage are observed in the hemorrhagic diseases in which capillary fragility and permeability are present. These have been classified as *pathologic,* involving the factor of diapedetic oozing, rupture of capillaries and corrosive and/or ulcerative perforations of the blood vessels. Other aspects have been classified as *physiologic,* in which there may be an alteration of the capillary endothelium, or the pericapillary connective tissues and/or the consistency of the intercellular cement, the result of physiochemical processes. It appears that increased capillary fragility is *pathologic* when a chemical agent (bacterial toxins, metabolic toxins and nutritional deficiency factors) causes a disintegration of the intercellular substance.[26] This limits the concept of increased capillary fragility to a condition where chemically produced lesions are present in the capillary wall. Just where capillary fragility ends and capillary permeability begins is a controversial matter. According to the above classification, increased capillary permeability is *physiologic* when there is an alteration of the capillary endothelium or the pericapillary connective tissue as a result of the physiochemical processes. Burrows[27] lays great stress on the presence of increased capillary permeability in inflammatory diseases. He considers that the essential vascular factor in inflammation is the increased permeability of injured cells which permits substances to flow more

freely in and out of the cytoplasm. If Burrows' opinion is correct, the process may have a physiologic significance — in the sense that it is probably a *compensatory metabolic mechanism* which facilitates the concurrent disposal of toxic products and the entry of nutrient repair substances into the cytoplasm.

It is not amiss to quote from Boyd[28] concerning scurvy:

> The obvious *lesions of scurvy* are hemorrhages and changes in the bones. But the essential underlying lesion is an inability of the supporting tissues to produce and maintain intercellular substances. The effect is on cells of mesenchymal origin in contrast to the ectodermal and entodermal effects of vitamin A deficiency. The intercellular substances concerned are the collagen of all fibrous tissues, the matrix of bone, dentine and cartilage, and all non-epithelial cement substance including that of vascular endothelium. The weakening of the capillary walls is responsible for the hemorrhage which forms so prominent a feature of the disease.

It seems that each hemorrhagic disease has its individual characteristics. Fundamentally, however, the factors of a disturbed blood clotting mechanism, of capillary fragility and capillary permeability, are almost always present.

It appears to me that the hair-splitting controversy concerning these two terms is inapplicable to the pathologic manifestations which are observed firsthand in chronic idiopathic ulcerative colitis. These two pathologic states (capillary fragility and permeability) are concurrent and closely interrelated in most cases. Therefore, they shall be discussed conjointly. Perhaps the reason for the inability to satisfactorily estimate which process predominates in non-specific ulcerative colitis may be traced to the numerous etiologic factors which may play major or minor roles in the genesis of this disease — psychoneurotic, psychosomatic, neurologic, allergic, bacteriologic, chemical (chiefly lysozyme and hyaluronidase), faulty food habits, and the adverse effect of simultaneous nutritional excesses and deficiencies. This disease is a complex problem in which probably each of the aforementioned factors plays a part, and if envisioned as

such, it is always the result of more than one of these factors. Irrespective of the theoretical considerations regarding etiology and pathology, we are faced with the necessity of realizing that fundamentally we are dealing with four major factors which are co-existent, concurrent, co-related and inextricably woven into a basic clinical pattern. These are:

1) A disturbance of the blood clotting mechanism.
2) Increased capillary fragility.
3) Increased capillary permeability.
4) Nutritional deficiencies in relation to the above three.

Foremost investigators of the problem of capillary fragility, permeability and the intercellular cement substances, postulate a new concept concerning the capillary system. They contend that the orthodox anatomical concept of a capillary as being merely a single-layered endothelial tube must be revised and expanded to explain capillary physiology and pathology. The function of a capillary is manifold. It may dilate or contract in response to a number of diverse stimuli. Through its walls pass nutrient materials, metabolites, oxygen and other substances for tissue consumption. Waste products also pass through its walls to enter the blood stream for transport to the excretory organs. The consensus among modern investigators is that a capillary is composed of at least five distinct layers, each having a highly specific function. From the lumen outward, these layers are:

1) The protein absorption layer.
2) The capillary wall itself.
3) The pericyte layer.
4) The pericapillary layer.
5) The connective tissue stroma layer which serves to imbed the capillary in one general area.

The protein absorption layer contains no fixed cells *per se.* It is extremely elastic and it apparently plays an important role in maintaining the normal fluidity of the

blood. When conditions are normal the blood cells can glide through this "lubricated" plastic tube without damage. If damage occurs to this inner layer, then the cellular elements of the blood will not flow smoothly through the capillary and the interaction of the various components necessary for the coagulation phenomenon (prothrombin, thromboplastin, Ac globulin, thrombin, etc.) takes place.

The next layer is composed of a single layer of endothelium which is imbedded and fixed by the intercellular cement substances. The cement acts not alone as a binder but also as a physical filter (permeability factor). The viscosity of the intercellular cement substances is due to a mucopolysaccharitic acid which forms a viscous gel wall in the ground substances between the mesenchymal cells. The consistency of this gel wall varies under different circumstances. Some investigators believe that it is in this second layer that vitamins C and P play important roles.

The pericyte layer, the capillary layer and the connective tissue stroma layer are composed collectively of reticuloendothelial cells, nerve cells, fixed macrocytes, histiocytes, fibroblasts, and the hyaluronic acid gel which regulates the normal or abnormal function of the capillary system.[29]

Part IV
PROGNOSIS

IV

PROGNOSIS

Exacerbations, remissions and recurrences are traditionally associated with the clinical course of every case. Many doubt that chronic idiopathic ulcerative colitis can be cured permanently.

Willard, Pessel, Hundley and Bockus[80] made a formal study of the prognostic aspect of the problem. They used the case records of 66 patients with chronic non-specific ulcerative colitis whom they had observed for more than three years (or who had died while undergoing treatment and observation). In addition to obvious prognostic factors such as acuteness of infection versus resistance of the patient, these investigators found duration and severity of symptoms significant. For example, in the series of cases studied, the highest mortality occurred among those with the shortest history, although this may have been due to the inclusion of many acute fulminating cases. They go on to state that although patients having mild symptoms of short duration had the best prognosis, the greatest number of deaths occurred among those who had their symptoms for six months or less. From seven months to a year, there were fewer deaths and a greater incidence of improvement and remission. From two to five years the mortality rate again goes up — perhaps the debilitating effect of the disease had begun to take its toll during this period. After five years the mortality rate starts to go down and the patient who has had the disease as long as ten years seldom dies from it. Remissions at this late stage are uncommon but improvement can sometimes be obtained.

The prognosis in any case of chronic ulcerative colitis should be extremely guarded. The disease is chronic: it may suddenly improve or retrogress for no apparent reason.

There may be remissions; long symptom-free periods, but out of a seemingly clear sky there may be a serious recurrence. If no emotional upset can be elicited, one should investigate the dietary errors of omission and commission to which persons, habitually prone to dietary indiscretions, so frequently succumb. In the absence of any emotional disturbance, one usually discovers that the patient has been on a food or beverage binge.

No hope of permanent cure can be entertained before three to five years of observation, and even then *hope should not pass to conviction unless the patient adheres strictly to a dietary composed almost exclusively of natural foods.*

Bassler[31] in a recent article concerning chronic nonspecific ulcerative colitis makes this statement:

> I agree with Kiefer that the term control as good, fair and poor is all that is permissible. By good is meant where both the local and general constitutional symptoms have been absent for at least five years, the patient being active and capable to stand strains, physical and emotional, eating any foods, no treatment, etc., without ill effect. By fair is meant that marked improvement had taken place, there is practically no disability, with but mild and easily controlled symptoms. Poor are those in whom continuation of symptoms has produced a severe degree of disability or complications.

This statement is open to question. Moreover, the blanket statement that the ulcerative colitis patient is never cured is also questionable. I agree with Doctor Bassler concerning the five-year period but I disagree that the patient should be able to endure unusual physical and emotional strains, eat any kind of food, and have no treatment. We need a better perspective of this situation.

Once the patient becomes symptom-free he craves reindulgence in former faulty food habits, and to resume subsistence upon a dietary which is notoriously inadequate for the maintenance of health. Too often, the physician fails to impress upon him the hazards of such a course. It

follows that a return to this inadequate dietary undermines his physical status and again he becomes sensitive and susceptible to physical and emotional strains that otherwise would not harm him.

The consensus is that the ulcerative colitis patient is rarely cured. If symptom-free, it is assumed that the disease exists in a latent, subclinical form which may react positively to an exacerbatory factor. Peculiarly enough, the ulcerative colitis patient is supposedly a victim of this disease for the rest of his life. This is inconsonant with our prognostic perspective regarding other diseases. For example, many people have scarlet fever, pneumonia, typhoid and other diseases two or three times during a lifetime. Repeated attacks of these diseases in the same individual are considered separate, independent events, having no connection with previous morbidity periods. It is largely the fault of the physician or the patient that recurrence follows a cure. I do not consider a patient cured until there is a complete disappearance of ulcerations and hemotumescent areas; a normalizing of the sedimentation rate; platelet count; prothrombin time; hematologic status and blood chemistry* over a two-year period. It is reasonable to assume that many patients are discharged when the majority of their symptoms subside. Too frequently, residual symptoms are considered a part of the colitis personality which will persist, treatment or no treatment. These cases are not cured and treatment should continue, especially proper dietary treatment.

*Blood glucose, cholesterol, cholesterol-esters, total protein, calcium (total), calcium (ionized), phosphorus, calcium-phosphorus ratio, chlorides as NaCl, and vitamin C.

Part V
TREATMENT

V

TREATMENT

Perhaps in no other disease is there more need for the admonition: *treat the patient, not his disease!* It must be kept in mind that the specific manifestations of this disease are the result of a complex of dietetic and hygienic factors, including mental and emotional strains and indisciplines. If this is the case what is done *for* the patient, is automatically done *against* his disease.

Treatment divides itself into four main categories: (1) nutritional, (2) psychoneurogenic, (3) anti-allergic, (4) anti-bacteriologic, in the order of their therapeutic importance. To these may be added general symptomatic, or supportive measures and surgery. It is stressed that the selection of any or all of these measures for application in a given case should be based on a definite need for them as revealed by thorough study of the probable causes of the disease in that particular case and of the patient's symptoms, some of which may be obviously the result of the disease and some of which may be contributing to its causation or prolongation.

Nutritional Aspect:

In reviewing the literature concerning the nutritional aspect and the dietary treatment of ulcerative colitis, one is struck by the many contradictory statements that have been made.

In chronic ulcerative colitis restrict fruits, whole grains and cereals, and vegetables for they contain too much residue. Thus admonishes most authorities. Others, equally competent, are of the opinion that fruits and vegetables supply a nonirritating amount of bulk that is beneficial. Most of the professionals feel that the high protein, low residue diet is best, that milk is not generally well tolerated,

that enough carbohydrate should be given to satisfy the patient's caloric requirement and that — rather vaguely — there should be some sort of proper balance among proteins, carbohydrates and fats.

Pennington[32] states the proposition well:

> Emaciation, always present in ulcerative colitis, may be extreme and is attributable not only to decreased caloric intake as a result of a poor appetite but also to loss of blood and protein in the stools, disordered digestion and increased katabolism from toxaemia. Too often the caloric value of the food has been wrongly reduced by direction of the attending physician, who unwisely restricts the diet to unpalatable, uninteresting, stodgy meals, low alike in calories, proteins and vitamins, in an attempt to provide, unnecessarily, a diet with no residue.

Bercovitz'[33] comments are also noteworthy:

> From the standpoint of understanding the complex nature of chronic ulcerative colitis, the fundamental problem is to determine, if possible, what metabolic or nutritional factors may underlie the entire situation and may have existed for a long period of time prior to the onset of ulcerative colitis. The question that has yet to be resolved is whether the circumstances which seemed to cause the disease are incidental, or whether a failure over a considerable period of time of the metabolism of food substances, including vitamins, carbohydrates, and proteins, is responsible for the condition.

Specific and sometimes conflicting recommendations are made here and there with reference to the value or harmfulness of various foodstuffs. Administration of vitamin and mineral supplements is invariably urged. Numerous "diet lists" are available but radical discrepancies are found in their respective directions. Such directions lack qualitative precision in any case because of the adulterated and characteristically deficient qualities of available processed foods. Even unprocessed foods, because they may be grown on deficient soils and subjected to significant nutrient losses in transport, storage and cooking, share this ambiguity, although to a lesser degree. Hence one is moved to applaud the statement that recovery from chronic ulcerative colitis depends preponderantly on the availability of

the best possible quality of food. Emphasis on the *quality* of foods, of course, raises the question of vitamin and mineral deficiencies and imbalances.

Unfortunately, despite recent advances in our knowledge of nutritional science, commercially motivated advertisers and propagandists continue to feature this and that vitamin as the specific cause and cure of this and that human misery. Even the academic nutritionists, for the most part, cling unconsciously to the obsolete concept of "deficiency diseases" in which the vitamin synthesizers and patentees, the pharmaceutical houses, the drugstores and the food processors (greatly helped by the government sponsored enrichment program) have all acquired a vested interest. Hence the findings of nutritional research and the commercial indoctrination of the people by the food and the pharmaceutical advertisers are increasingly in conflict.

No one questions the profound influence which vitamins play in the maintenance of good health; nor does one question the far-reaching effect which minimal or subminimal amounts of these substances can play in the production of ill-defined and well-defined clinical syndromes. But we must remember that *all* nutritional factors (proteins, carbohydrates, fats, minerals, vitamins, natural roughage and water) are essential for the maintenance of good health; also that the consumption of excessive amounts of the ternary factors (carbohydrates, proteins and fats) is equally as harmful as a deficiency of one or more of the indispensable, imponderable, accessory* food factors. Actually the damage caused by vitamin and mineral deficiencies is almost always associated with the excessive consumption of the more concentrated foods — an excess not only harmful in itself but damaging through its intensification of the actual or relative deficiency of vitamins and minerals.

*Unfortunately, these food factors have been designated as accessory, implying that their role in nutrition is subordinate to the ternary food factors. This is not the case because no single food factor is more important than another and in no event should these nutrient factors (vitamins, minerals and roughage) be considered "accessory."

Almost always we find ourselves dealing not with deficiencies *or* excesses but with deficiencies *and* excesses *occurring simultaneously.* Moreover, this condition encountered routinely in practically every patient is very difficult to treat because it represents not so much a disease syndrome of the patient as of his environment: *the planless and perverted food economy and culture that limits, conditions and indoctrinates him.* Hence in treatment the first job is to undermine the patient's faith in the unconscionable propaganda of the food and pharmaceutical hucksters.

Specific Therapeutic Measures Employed:

Because this investigation was concerned primarily with the therapeutic effect of a precise dietary, no patient was treated with antibiotics, sulfa drugs, miracle glandular substances, or transfusions. One case required surgery due to a recto-vaginal fistula following childbirth. The therapeutic measures employed were:

(1) Constructive Meal Planning, as outlined in the book of that title.*

(2) Vitamin Therapy:

a. the oral, subcutaneous, intramuscular or intravenous administration of vitamin K in appropriate doses. The dose of vitamin K is determined by the severity of the clinical picture, prothrombin time, platelet count, sedimentation rate and the profundity of the disturbance of the blood drop clot retraction pattern. When the clinical and laboratory pictures improve, the dose of vitamin K is correspondingly reduced. Formerly, the oral administration of vitamin K was ineffectual unless combined with bile salts and this frequently aggravated the condition. Fortunately, for ulcerative colitis patients, several water soluble and rapidly absorbed synthetic compounds, possessing antihemorrhagic prop-

*Norman, N. Philip: Constructive Meal Planning; Phototone Press, Passaic, New Jersey. 1946.

erties, are now available. In this work Roche's Synkavite was used. When the intestinal flora normalizes itself after the motility of the gastro-intestinal tract approximates a normal pace and the degree of hemorrhage becomes progressively less, the absorption of the vitamin K in food and the amount provided by intestinal bacterial synthesis, returns to normal and supplies the liver with an adequate amount for the synthesis of prothrombin — perhaps the key substance in maintaining a *normal blood clotting status.*

b. the oral administration of rutin, 60 or more mgs. (or the equivalent in a natural product) per day. In this study dehydrated buckwheat juice ita[illegible]

c. [illegible]dministration of a combination of hesperi- [illegible] d vitamin C (National Drug Co.) 50 mgs. each, 6 to 8 tablets daily.

d. if there is a demonstrable deficiency of vitamin A and/or B, these supplements should be prescribed in adequate doses.

(3) A combination of:

Bismuth subgallate	60. gm.
Paregoric	90. cc.
Elixir lactopeptine qs. ad.	180. cc.

Teaspoonful every 2-3 hours, diluted in ¼ glass water.

(4) A combination of:

Syntropan (Roche)	0.05 gm.
Novatrin	0.006 gm.
Charcoal	0.65 gm.
Phenobarbital	0.016 gm.

Ft. Capsule: i every 4 hours.

(5) Sun's cholera mixture ć opium (NF)
one half to one teaspoonful every 2-3 hours, diluted in ¼ glass water.

(6) For the anemias; iron and copper, orally and/or intravenously, and intramuscular injections of liver extract. Intravenous and intramuscular injections are frequently indicated because the ulcerative colitis patient does not always tolerate the oral administration of these drugs. The hematologic picture, especially the reticulocyte count, the hematocrit, and the sedimentation rate are the yardsticks for determining the intensity of antianemic treatment.

(7) Colonic irrigations are sometimes prescribed for this condition. No one but a tyro would administer an irrigation to a patient in an acute phase of this disease. Colonic irrigations are indicated only upon the disappearance of ulcerated and hemotumescent areas plus a normalizing of the sedimentation rate, prothrombin time and platelet counts. Frequently the injection of two ounces of medicated vaseline into the rectum relieves the patient of tormina and tenesmus. The following formula has been used:

Menthol	
Camphor	
Eucalyptol	
Methyl salicylate aa	1.0
Boric Acid	20.0
Salol	20.0
White vaseline qs. ad.	1000.0 gms.

Therapeutic Aspects of Citrin and Vitamin P:

In postulating this new concept the phenomena of increased capillary fragility and permeability have been correlated with some specific nutritive factors. This new concept opens an interesting field of thought in respect to the local pathology and merits ampliative discussion regarding the drug and dietetic treatment of idiopathic ulcerative

colitis. As previously stated, vitamin K is demonstrably deficient in this disease but the disease is not solely due to this deficiency. Experimental and clinical studies with vitamins C and P link a deficiency of both of these vitamins with the capillary permeability and fragility factors.

To date, unfortunately, the biochemical analysis of the total factors composing the vitamin C and P complexes is incomplete. There is much controversy regarding the role which a deficiency of these substances plays in the production of hemorrhagic diseases, and also in their therapeutic effectiveness. From my point of view this controversy is concerned more with academic aspects than with practical clinical application.

In 1928, Szent-Györgyi and his co-workers[34] isolated vitamin C from the adrenal gland. In the report he states: "even in the earlier days of the isolation of this acid, one of us was looking for the substance which bound ascorbic acid with the peroxidase system in a coupled oxidation system." His attention was directed to a group of substances called benzopyrones which play an important role in the cellular permeability of plants. In 1936, he succeeded in isolating a compound from lemon peel and Hungarian red pepper which he first called "citrin." Since that time much study has been devoted to this group of substances which is found in raw fruits, plants and vegetables. Unfortunately, the term citrin has been loosely applied to the plant, fruit or vegetable extracts which may contain several flavonols in varying quantities. This is particularly true of an extract of lemon peel which, while rich in crude hesperitin, a vitamin P component, also contains other flavonols, chiefly eriodictyol, which is almost but not identical with hesperitin. Eriodictyol is evidently the citrin fraction of the total vitamin C complex. It has been shown that unripe lemon peel is composed almost entirely of hesperitin while the eriodictyol content is scarcely present. As the fruit ripens, the hesperitin content decreases and the amount of erio-

tictyol increases. Therefore, these two flavonols represent a portion of the vitamin C and P complexes.

The flavonols which are important are the following:

1) Hesperitin and its rhamnose *glucoside,* which is hesperidin. It is also available as a methyl chalcone derivative which is a pure chemical compound.

2) Eriodictyol is the same flavonol as hesperitin except that the methoxyl group on the C ring is replaced by a hydroxyl group.

3) Quercitin and its rhamnose *glycoside,* quercitrin.

4) Rutin, which is the rhamnose *glucoside* of quercitrin.

It seems that the total components of the vitamin C complex are closely akin to, or part of, what is considered the vitamin P complex. It is the consensus that these flavone substances have a profound effect upon the health and structural integrity of the capillary system. It is also thought that in combination they seem to complementary potentiate one another.

It is not known precisely how these flavonols act to increase the resistance of the capillary to the fragility and permeability factors. It has been suggested that vitamin P-like substances function indirectly by sustaining the activity of adrenalin. There is some evidence that these substances may prevent or inhibit anaphylactic shock and serous inflammation, thereby performing an anti-allergic role.[35] It seems well established that abnormal capillary permeability is present in many allergic conditions. Quoting W. H. Manwaring, et al:[36]

> Increased specific capillary permeability will be shown to be the dominant fundamental physiologic change in protein sensitization in which all other anaphylactic reactions are secondary.

This viewpoint will be hailed by the disciples of Andresen's school of thought which postulates that allergy is the major, and frequently the chief etiologic factor in this disease.

Over 5,000 scientific articles on the relationship of the total vitamin C complex and the vitamin P-like factors have been published within the decade that followed Szent-Györgyi's discovery. This extensive literature contains reliable evidence that the total vitamin C complex (ascorbic acid, eriodictyol and perhaps other unidentified substances) plays a significant role in transforming the collagenous material surrounding the capillaries into a firm supporting bed.

If there is a deficiency of hyaluronic acid, the periarterial colloidal buffer coat becomes less viscous and the capillary wall becomes more fragile and permeable. The same thing happens with an excess of hyaluronidase. The vitamin P-like substances (hesperitin, quercitin and rutin) and the total vitamin C complex seem necessary to maintain a physiologic equilibrium between hyaluronic acid and its enzyme hyaluronidase, thereby maintaining normal capillary fragility and permeability. In the treatment of this characteristically hemorrhagic disease, which has its own clinical pattern, the biologically therapeutic team consists of vitamin K, the total vitamin C complex (including citrin) and the vitamin P flavonols (quercitin, hesperitin and rutin). The response of patients receiving this combination has been prompter than before these substances were available — previous specific vitamin therapy was confined to vitamins K and C. This is evidence that these substances have a potentiating effect upon each other and that their action increases platelet counts, increases the agglutinability of platelets, normalizes prothrombin and calcium levels, and either stimulates hyaluronic acid production or inhibits the activity of its enzyme, hyaluronidase.

There is a possibility that the function of the total vitamin C complex, especially the citrin and/or the eriodictyol fraction, is to bind hyaluronidase. Hyaluronidase in this bound state then seems to be vulnerable to the action of the

component fractions of the vitamin P complex, in which case there occurs an actual inhibition of hyaluronidase activity.

A few observers believe that the vitamin P-like substances strengthen the capillary wall through a process of "tanning" the intercellular cement, thus making it less permeable or almost impermeable to protein and other substances.

Quoting Beiler and Martin[87]:

> Hyaluronidase has been considered as a factor in accentuating capillary fragility rather than in inducing direct changes in capillary permeability . . .
>
> Of the vitamin P substances tested, only rutin showed an inhibitory action on hyaluronidase and this only at high concentration. The only compounds possessing activity at the lower concentration were ascorbic acid and dicumarol. However, a combination of ascorbic acid and the vitamin P compounds showed a marked potentiation of inhibitory action, especially in the case of hesperidin methyl chalcone . . .
>
> It appears possible that a hyaluronidase-ascorbic acid combination is susceptible to inhibition by these compounds, while hyaluronidase itself is not. It seems that its action is due to an inhibition of hyaluronidase, and that it is two-fold; first, a direct inhibition of hyaluronidase, and second, a potentiation of the action of vitamin P . . .

In partial substantiation of the rationale of the vitamin therapy presented, I shall make reference to the following:

In 1937, Zacho[88] noticed an outstanding result with vitamin P in controlling capillary permeability in ulcerative colitis.

Vacek[89] reports his experience with two cases of ulcerative colitis. One of these had been ill for four months with three to five bloody stools a day. After four months of treatment with orthodox therapeutic measures (transfusions, vitamin B and C) the condition of the patient was unchanged. Treatment with vitamin P was instituted. After the first injection of 20 mg. the active bleeding stopped. After ten injections occult blood tests were negative. A

total of 14 injections was administered. When the injections were discontinued, lemons and vitamin A were prescribed. The patient did not adhere to the prescribed diet and had a relapse. Ten injections of 40 mgs. of vitamin P (Roche) plus 20 50 mg. citrin tablets (Roche) checked the bleeding completely. This author stresses the importance of the patient adhering to a precise dietary. In his experience, the patients who did not adhere to the dietary suffered recurrences. I wholly concur with him on this point. His conclusions were:

1) Vitamin P reduces the capillary permeability factor considerably.

2) It shortens the blood clotting time.

3) Its effect upon the platelet count and the bleeding time were negligible.

Kushlan[40] treated one case, hereditary hemorrhagic telangiectasia with associated abnormal capillary fragility and hemorrhage, with rutin. This patient had repeated and chronic hemorrhage from the nose, gums and gastrointestinal tract for 15 years. Bleeding was rapidly controlled following administration of 40 mg. of rutin three times daily.

Between 1940 and 1947, Z. Maratka[41] had 62 cases of ulcerative colitis under observation. He used citrin in 33 patients. It was administered in series from 10 to 20 doses in daily intravenous injections. He states that citrin had a favorable influence in mild forms (inflammatory, hemorrhagic type) of ulcerative colitis, especially in the initial stage. He is of the opinion that it is of much less value if the condition exists for a considerable time and in advanced ulcerative cases. He assumes that citrin has a pharmacologic action, diminishing the permeability of the capillary vessels and increasing their resistance, and perhaps suppressing the tendency to pathologic reactions. He states:

> The use of citrin in the therapeutics of ulcerative colitis is motivated on one side on account of the vascular nature of the

initial lesions and on the other side on account of the effect of citrin on the blood vessels.

Psychotherapeutic Aspects:

As previously stated, psychoneurotic and psychosomatic factors were present in all of my patients. These factors are not essentially etiologic — rather they are accessory or contributory and in all cases the history revealed these neurotic traits to be present long before the ulcerative phase of the colitis.

The clinical and dietary histories of my series of cases also disclosed that they were, for many years, victims of improper or inadequate nutrition of varying degrees. Recent studies in nutrition suggest that psychoneurotic and psychosomatic syndromes are frequently the result of chronic malnutrition — therefore, it seems more logical to consider psychoneurotic and psychosomatic states the result of an inadequate nutritional status extending over a period of years rather than to consider them autogenic psychopathologic syndromes.

The psychoneurotic, psychosomatic, psychiatric and psychoanalytic schools of thought consider this disease a result of conflicts, social problems, maladjustments, anxieties, tensions, conversions, substitutions, rejections, compensations, indecision, sensitivity, hostility, immaturity, etc. I cannot subscribe wholeheartedly to this or to the theories invented by specialists to justify their respective viewpoints.

The physician should attempt to elicit all of the personality disorders which may be present in the ulcerative colitis patient and do his best to assist the patient to solve his psychic problems. Frequently, this can be done without extensive psychiatric or neuropsychiatric investigations. In my experience, many of the patients who came to me with this disease had been previously convinced by neuropsychiatrists that their ailment was due solely to their unstable personalities and their failure to make satisfactory adjustments to their problems, conflicts, etc. Moreover, they were

also told that the cure for their diseases was not predicated upon better nutrition or intelligent treatment — it would only occur by employing modern methods of psychiatry and psychoanalysis.

The foregoing remarks are not intended to convey the impression that I am nihilistic in respect to the broader aspects of psychotherapy. I fully appreciate the significant part which these disturbed emotional and personality states play in *precipitating the onset* of this disease in chronically malnourished patients.

On the other hand, it has been my observation over more than a score of years, that proper nutrition is more potent in its effect, and more practical in its application, than the various forms of psychotherapy in treating psychoneurotic and/or psychosomatic syndromes.

Allergies and Antiallergic Therapy:

Here again, we confront another paradox in the basic philosophy of modern medicine. In common with psychoneuroses and psychosomatic states, the profession assumes that allergies are, *per se,* definite clinical syndromes. But the significant fact is that most allergies are traced to some particular food or foods. Therefore, by more than implication, in the majority of the allergies the nutritional relationship is clear and unequivocal.

In discussing the many factors which altered or impaired the quality of our food supply, it was stated that allergic phenomena were frequently the result of the excessive consumption of processed foods. Therefore, all food products which contain white flour (plain or enriched); eggs and dried egg products; sugar, candy, mints, cocoa, chocolate, malted milks, ice cream, sweet beverages, pies, cakes, pastries; milk and dairy products; fresh shellfish; the tinned and processed (preserved) types of proteins (ham, shellfish, Spam, Treet, Readi-meat, tinned, pickled, salted or smoked fish; and all forms of sausages) were omitted from the dietary. If the patient gave a positive history of a

sensitization to certain foods, then these foods were also excluded.

After the hemotumescent areas and ulcerations healed, and the sedimentation rate, prothrombin time and platelet count normalized, a restricted amount of eggs, milk, butter and fresh shellfish were allowed.

Antibacteriologic Factors:

Thirty years ago I became greatly interested in intestinal bacteriology and spent hours culturing stools and standardizing the dose of autogenous vaccines. I also wrote several papers on the subject.[42, 43] After six years of investigation, I found that the intestinal flora could be more effectively "reformed" with a dietary composed preponderantly of natural foods than it could be with autogenous vaccines, *B. acidophilous* and *B. coli* implantations. Therefore, the administration of these therapeutic agents was discontinued. In chronic ulcerative colitis the concurrent colonic infections are not basically etiologic — they should be considered accessory phenomena. It is my considered opinion that antibacteriologic agents play minor, unimportant roles in the therapeutics of this disease.

Analysis of Clinical Material:

Seventy-four patients suffering from ulcerative colitis consulted me for diagnosis and treatment during the past 30 years.

Of these 74, 19 cases (25.67%) could not or would not renounce the mixed meal and their faulty food habituations for highly processed foods.

Thus, only 55 (74.3%) patients of the total number (74) wholeheartedly cooperated in respect to specific and precise dietary instructions.

Of these 55 patients, 53 (96.3%) are reported as cured.

Two (3.6%) of these cooperative patients had unsatisfactory results. One case, a female 34 years old, bedridden, afflicted with the disease for 20 years, had a com-

plicating generalized non-malignant polyposis of the colon. Her death, within three months, resulted from progressive nutrient failure and uncontrollable bowel movements due to a progressive diminution of the reservoir capacity of the colon. The other, a male of 23½ years, who had the disease for two years, achieved excellent results within three months and remained symptom-free for four years. He felt well enough to get married. Unfortunately, his wife took an antagonistic attitude regarding his meal planning scheme and refused to prepare what she called "his grass diet." Six months on mixed meals resulted in an acute recurrence of the disease and I understand that he is still unwell.

The youngest was a female of 20 years; the oldest a female of 57 years.

Fourteen females (48.2%) were between 20 and 30 years of age.

Five males (19.3%) were between 20 and 30 years of age.

Twelve females (41.3%) were between 30 and 40 years of age.

Eleven males (42.2%) were between 30 and 40 years of age.

Three females (10.5%) were between 40 and 57 years of age.

Ten males (38.5%) were between 40 and 53 years of age.

The duration of the disease, prior to consulting me, averaged four and one-half years; one patient had the disease for only six months, and four had the disease for more than ten years.

Twenty-four (43.63%) patients of this group of 55 consulted me between 1920 and 1938. This group of 24 is to be considered separately because the laboratory investigations were limited to what is called complete blood counts (CBC), stool examinations and routine urinalyses.

Eighteen (32.74%) patients of this cooperative group of 55 consulted me between 1938 and 1943. The laboratory investigations were more thorough and included stool examinations, urinalyses, and a complete hematologic examination (hematocrit; icterus, volume, saturation indexes; cell diameter, reticulocyte and platelet count, sedimentation rate, and determination of prothrombin time, etc.).

Since 1943, 13 (23.63%) patients have been intensively studied from a clinical and laboratory standpoint. The hematologic examination consists of the following: red blood cell count; hemoglobin per cent (100%=14.2 gms.); hemoglobin (gm. Hgb. per 100 c.c.); hematocrit (3500 r.p.m.); percent cell volume; cell diameter; mean corpuscular volume; mean corpuscular hemoglobin; mean corpuscular hemoglobin concentration; color, volume, saturation and icterus indices; reticulocyte count; sedimentation rate; prothrombin time; platelet count; white blood cell count and differential. Routine blood chemistry determinations: blood glucose, cholesterol, cholesterol esters, total protein, calcium (total), calcium (ionized), phosphorus, calcium-phosphorus ratio, chlorides, Bolen Test for malignancy,[44] and the vitamin C blood level. Urine and feces examinations were also routinely performed.

All the above mentioned hematologic tests are essential to establish what type of anemia is present. The routine "complete blood count" (consisting of RBC, Hgb, WBC, and differential), serves only as a screen test for anemia — the data are too meager to formulate differentially, with conviction, the type of anemia involved.

The 53 cured cases were supervised for a minimum three-year period. During this time there were some recurrences, occasioned by a resumption of improper eating habits. Usually three or four recurrences firmly convince patients that a resumption of mixed meals adversely affects the *status* of their colitis. In other words they are convinced that *they must permanently renounce the mixed-meal, and*

its processed food content, to remain cured. Then, I insist on an additional two-year recurrence-free period to teach these patients the necessity for a *continued* subsistence on a natural, unprocessed dietary. I have yet to see a recurrence in cooperative patients after this five-year period. This evidence of the superiority of this concordant dietary, over the emasculated, orthodox, colitis dietary, convincingly supports my thesis — chronic non-specific ulcerative colitis should not be considered an exclusive result of a deficiency of one or more essential nutritional factors. *It is more than that.* Nutritional imbalances in respect to the ternary elements, simultaneously condition the relative or absolute requirement for the other nutrient factors.

Now is an opportune time to review and correlate the laboratory and clinical data with the postulation that idopathic ulcerative colitis is a hemorrhagic disease, predominantly the result of simultaneous dietary excesses and deficiencies.

Major Factors in Ulcerative Colitis:

1. Invariably the dietary of the ulcerative colitis sufferer is composed preponderantly of the highly processed starches, sugars and fats, and to a lesser degree, processed proteins. This characteristic pattern may be attributed to two factors:

(a) the perverse food habituations which are concurrent with increased mass food production and processing technologic innovations within the past 65 years.

(b) the indoctrination of the patient's mind to the belief that eating natural foods will profoundly worsen the condition because the "roughage content" would denude the ulcers of their protective coverings.

2. In the active phase of the disease, the presence of a hypochromic anemia (84.61%) which may be normocytic, microcytic or macrocytic, due to the following factors:

(a) a constant loss of blood from the ulcerative areas and the unremitting "leakage" of blood in the hemotumescent areas of the mucous membranes. It is

my impression that more blood is lost from these "leaking" areas than from ulcers.

(b) subsistence upon the orthodox, nutritionally inadequate "colitis diet."

(c) nutritive failure due not alone to the inadequacy of the dietary but also to the insufficiency and inability of the gastro-intestinal secretions to digest and promote the absorption of essential nutrients, which in turn is accentuated by the hypermotility of the intestinal tract.

(d) a perversion and exhaustion of the hematopoietic function of the blood forming organs — due to hemorrhage, "leakage," and an imbalanced dietary which simultaneously contains excessive amounts of the processed ternary food elements and deficiencies of the accessory, but indispensable, nutritional factors.

3. Laboratory studies of the hematologic status of these 13 patients reveal the following significant data which closely parallel the foregoing observations in the preceding paragraphs:

(a) the presence of an acidophilia (84.61%) which may persist for two or more years after the intestinal lesions have disappeared.

(b) an increased sedimentation rate (84.61%) — the sedimentation rate is a reliable criterion by which one may judge the progress of a patient. An increase in the sedimentation rate may presage the onset of a remission, although the sigmoidoscopic picture may not reveal the presence of a worsened condition of the colonic mucosa.

(c) an increased prothrombin time in 84.61% of cases actively bleeding, reflecting a deficiency of vitamin K.

(d) a decreased platelet count in 76.92% of actively bleeding cases, reflecting a disturbance of the blood clotting mechanism.

(e) a slight increase of the icterus index in all active cases.

(f) adequate amounts of vitamin A and/or its precursors are necessary for maintaining a normal level of visual acuity. It follows that the dark adaptation curve will be unsatisfactory if the dietary is deficient in these nutrient essentials. Lerner and Rapaport[45] performed biophotometer tests on 30 cases of chronic idiopathic ulcerative colitis and found that 41% of these had a subclinical deficiency of vitamin A. In my series, biophotometer tests revealed a deficiency of vitamin A in 53.85%. These data indicate that there is more than a casual relationship between avitaminosis A and idiopathic ulcerative colitis. Moreover, biophotometer tests may indicate deficiencies of other vitamins. Recent investigations have stressed the importance of riboflavin and ascorbic acid in the utilization of vitamin A.[46] Conversely, a deficiency of vitamin A and its precursors could conceivably alter the physiologic action of riboflavin and ascorbic acid.

(g) subminimal blood plasma saturation levels of vitamin C in 69.23% of cases.

(h) a decrease in the vitamin B level in 65.54% — determined chiefly by clinical signs and symptoms.

(i) low total protein levels (84.61%).

(j) low ionized calcium levels (69.23%).

(k) low phosphorus levels (61.53%) because of the consumption of excessive amounts of processed carbohydrates, sugars and fats and a paucity of proteins of good quality (usually scraped or ground meat).

(l) low chloride level (as NaCl) in 30.76% of cases, the result of an increased excretion of NaCl due to diarrhea and hemorrhage. When the chloride level is low, the consumption of additional table salt is indicated. If the salt consumption exceeds physiologic requirements, the diarrhea may be worsened.

(m) a disturbance of the blood drop clot retraction patterns (Bolen Test) in all active cases.

(n) no definite relationship between the B.M.R. and this disease could be established. In the psychosomatic and psychoneurotic groups, the metabolism was found to be slightly elevated but normalized itself as soon as the patient improved.

Comments Regarding These Data:

The foregoing represents mostly the first examination findings of 13 ambulatory cases of chronic idiopathic ulcerative colitis. Prior to consulting me, all 13 had been under treatment; had been taking the miracle drug in current vogue; multiple vitamin supplements; hematinic preparations, chiefly in the form of liver and iron, both orally and by injection; ten had been or were taking vitamin K; and three had been hospitalized for transfusions.

All of this therapy modified the laboratory picture. For example, reticulocyte counts were often found to be 2.5%, the result of active liver and iron therapy; hematologic values were frequently enhanced by the use of antianemic drugs; vitamin C levels approached saturation; prothrombin times may have been reduced by vitamin K therapy; and large doses of vitamin A are known to influence the photochemical phase of vision.

Therefore, all of these therapeutic agents may condition, modify and/or distort the true laboratory picture. A true picture would necessitate the omission of all treatment and continued subsistence upon *the orthodox colitis diet* for two weeks before performing laboratory and clinical investigations. This, however, is obviously impractical because the patient's condition usually makes some form of treatment imperative.

In evaluating the laboratory data which have been presented, due importance has been given to all of the aforementioned factors. It is my impression, that if these

factors could have been eliminated or normalized, the percentages adduced from a breakdown of the laboratory data in relation to these 13 cases, would have been greatly increased — perhaps approximating 100% more nearly than they did.

Reliable results in clinical and laboratory methods necessitate the employment of a uniform and precise technic. As far as possible, all tests should be performed under identical conditions. After a 15 hour fast, patients reported for their laboratory work and the examination of the specimens began immediately after withdrawal of the blood. This is important — for example, if the blood, obtained at 9:00 A.M. from a fasting patient and immediately processed, is compared with the same patient's blood, obtained at 1:00 P.M. and immediately processed, the likelihood is that significant differences will be noted. If processed several hours later, the differences will be even greater.

Other factors which greatly influence the clinical and laboratory picture have to do with the competence of clinical and laboratory technicians. With rare exceptions, technicians graduated from commercial laboratory schools (not approved by the A.S.C.P.) are unfamiliar with the kind of blood chemistry or the number of hematologic determinations which have been a routine part of this study.

Laboratory solutions must be dated and constantly checked to detect deteriorations. For colorimetric determinations a photo-electric colorimeter should be used, thus eliminating the human element of error. Oxalated venous blood was used for all hematologic tests. It is necessary to emphasize the importance of the anticoagulant. For each 10 c.c. of blood, I use four drops each of a 3% ammonium oxalate solution and a 2% solution of potassium oxalate. These solutions are placed in a bottle and slowly evaporated to dryness. I have found this to be the best anticoagulant because less crenation, fragility and other changes in the blood elements occur. Recently, a few comparative tests

justify the impression that this anticoagulant is also better suited for chemical determinations.

Summary:

Evidence has been presented to support the postulation that what is now known as chronic non-specific idiopathic ulcerative colitis is a hemorrhagic disease, the result of specific nutritional deficiencies and excesses. Vitamin K, the total vitamin C complex and the vitamin P flavones are the major deficiencies. Conditioned deficiencies of vitamins A and B are also encountered. Faulty food habits and a sick food culture and economy have resulted in the excessive consumption of foods grown upon poor soils, damaged in transport and distribution centers, and the almost universal practice of excessively processing foods, thus degrading and debasing their nutrient value. The chief foods processed are the fats, carbohydrates, sugar and sugar-rich foods and beverages, and to a lesser degree, proteins.

My limited experience leads me to believe that the aforementioned dietary factors play an important role in the etiology and treatment of idiopathic ulcerative colitis. To date, the dose of ascorbic acid, rutin, hesperidin, hesperidin methyl chalcone and vitamin K has been standardized. As research proceeds, the likelihood exists that standardization will also include eriodictyol and quercitin. Moreover, the other non-specific polyphenols so widely distributed in vegetables, plants and fruits, may be proven under experimental conditions to exert a protective effect on the capillary itself and its supporting intercellular matrix. It seems to be well established that these substances are non-toxic and I know of no contraindications to their use in this disease.

Part VI
MIXED MEALS AND IMPROPER NUTRITION

VI

MIXED MEALS AND IMPROPER NUTRITION

The foregoing postulation that chronic non-specific ulcerative colitis is a nutritionally induced hemorrhagic disease, has necessitated challenging the scientific propriety of the traditional mixed meal, balanced or unbalanced, as well as an overall criticism of what is wrong with our total food culture and economy.

The validity of the accepted, traditional nutritional perspective sponsored by the food industry, their hucksters and propagandists, to perpetuate the unsound doctrine that proper nutrition necessitates employing the mixed meal, will be challenged and data cited to support this challenge.

When one's nutritional perspective is fully developed, one immediately realizes how the traditional mixed meal sabotages all attempts on the part of the clinical nutritionist to provide his patient with a dietary containing adequate amounts of all nutritional principles of good quality. This circumstance necessitates a brief discussion of all dimensions of our food culture as they condition or modify our daily fare.

It is necessary for the clinical nutritionist to fully acquaint himself with the many factors which influence the food supply before it reaches the table. It should always be kept in mind that proper nutrition goes back to the seed and the soil plus WHAT IS DONE TO FOOD before it reaches the shelves of your local food distributor, and WHAT YOU DO TO IT before it is served on your table. This should be the basic philosophy of APPLIED NUTRITION — NUTRITION IN ACTION.

Whole Food Patterns:

Nature has balanced the chemical composition of all natural foods and every component principle or element

in these foods is necessary for their proper digestion, absorption and metabolism. To destroy or to rob any natural food of one or more of its component elements is to impair the biologic value of the food. For example, when a grain of wheat is milled and separated into five parts, each of which is destined to be used separately, one of Nature's original food patterns has been destroyed. The fractions (flour, bran, middlings, gluten, wheat-germ oil, etc.) which are sold separately and used separately, *represent only parts of the total food pattern.* The elements which have been separated from the starch (flour) are essential for the proper digestion, absorption and metabolism of the starch fraction. Beriberi can result from a diet based on white flour bread that has been robbed of these elements. On the other hand, feed for livestock which is rich in most of the elements removed from the wheat berry during its processing, is almost devoid of the starch fraction. What effect the removal of the starch fraction from this food pattern has, or will have, upon the nutrition and health of these animals has yet to be determined.

No addition of milk or eggs to bakery products can compensate for the minerals, fats and proteins which the miller has segregated during his mechanical processing. Adding *processed* bran to the diet of an excessive consumer of white bread does not compensate for the *natural* bran which has been removed from the flour.

The Processing of Cereals:

Grains and cereals are our most economical and important sources of energy-forming foods. Unfortunately, these are dispensed to the public in the form of highly processed flours, meals, breakfast foods and cereals. Few professionals and laymen realize the nutritional deficiencies and the chemical adulterants which debase the quality of the commercial flour and meal "*mixes,*" breakfast foods and cereals. For several years, nitrogen trichloride (agene) has been an "improver" ingredient of all commercial white flours

Sir Edward Mellanby of England demonstrated that this food adulterant was poisonous to dogs.[47] Later experiments proved that dogs were not the only animals which were susceptible to this poison. Despite the proof adduced by E. Mellanby in England and by some nutritionists in this country, of the potent toxic properties of agene when combined with wheat protein, the Food and Drug Administration allowed millers and bakers to continue the use of this chemical "improver" until the latter part of 1949, because its immediate abandonment would have inconvenienced many units of the baking and milling industries. To justify this leniency the authorities point to a few hurried tests the results of which suggest that human beings may be immune to the toxic effects of nitrogen trichloride. The studies of Dees and Lowenbach[48] have shown that encephalographic tracings of allergic youngsters were abnormal in an unexpectedly large percentage of cases. In all likelihood, these youngsters had consumed large quantities of agenized white flour products — by implication, does this not have some significance?

At the recent (1949) hearing held by the Food and Drug Administration to determine the standard of identification for white bread, the millers presented their case for the substitution of chlorine dioxide for nitrogen trichloride. Also, because bread products stale prematurely, a section of the industry and its chemical suppliers sought legal sanction for the use of a group of fatty synthetic compounds employed to off-set this inherent propensity. Seemingly it was only the strong adverse report of the Council on Food and Nutrition of the American Medical Association that prevented the inclusion of these dubious chemical softeners and emulsifiers in the standard. The interest of the industry, of course, is in the salvaging of stale products, which are for the most part *undated.* Labels rarely convey useful information to consumers about either the adulterants and "improvers" used in flour and meal "mixes," or the quanti-

ties and qualities of the genuinely valuable food ingredients used in them.

Bakery products not only become stale; they spoil because of the presence of fungi and molds. To offset this tendency, two chemicals are ordinarily used—calcium or sodium proprionate, sold under various trade names. The public does not know that one of these chemicals is a common ingredient in solutions, powders and ointments which are used to treat athlete's foot, tinea cruris and other mycoses.[49]

Whole corn meal will not keep unless refrigerated. It is fertile food for weevils and corn borers. Tests which I have conducted have shown conclusively that when commercial packaged corn meal is inoculated with these pests, they will not grow — they die within 48 hours. The processors of this kind of corn meal state that the label on their product indicates that it has been "degerminated." This means that it has been robbed of its nutrient amino acids. Unless the label defines what is meant by a technical term like "degerminated" the consumer ignores what is in effect a tacit warning. Heat treatment is necessary to destroy the eggs and larvae of the pests which live on whole corn meal. This is one reason why the corn meal is so brittle. One large processor denies the use of a chemical like calcium or sodium proprionate to poison weevils or corn borers. Yet this much I do know — the pests promptly die when transplanted from infested whole corn meal to freshly opened packages of commercial corn meal. It does not seem reasonable that they should be quite so short-lived merely because of a lack of essential amino acids.

The Pasteurization and Homogenization of Milk:

Practically all commercial food processing impairs the value of the natural food pattern. For example, pasteurizing milk, a simple thermic process which our health authorities have tried to make compulsory everywhere. This process removes vitamins, hormones and enzymes which are indispensable for the proper digestion, absorption and utilization

of its chemical constituents. Moreover, it disarranges the mineral elements and a good proportion of the calcium (as milk stone) is precipitated in the pasteurization vats.

This processing procedure is designed to try to prevent the transmission through milk to humans of undulant fever, tuberculosis, and other bovine diseases. The health authorities have not concerned themselves with the reasons why cows develop undulant fever, tuberculosis, hoof and mouth disease, etc. So far as the public is concerned, these diseases are immutable and unpredictable happenstances. Yet Sir Albert Howard, William A. Albrecht, Oscar Erf and others have contended and demonstrated that these diseases are not biologic happenstances but the result of improper nutrition, chiefly concerned with a deficiency of the trace mineral elements. The researches of Doctor Francis M. Pottenger, Jr.[50] of Monrovia, California have clearly demonstrated the adverse effect of heat processed food and metabolized vitamin D milk on the dento-facial structures of experimental animals. This research was concerned chiefly with the effect of pasteurized milk and cooked meats upon the skeletal development of cats. Melnick and Oser[51] have shown also the effect of heat processing on the function and nutritive property of proteins.

It is improbable that the impairment of the food value of milk which attends pasteurization can be compensated properly by the addition of natural or synthetic vitamins. Recently the large corporate units of the milk industry have been ballyhooing the "advantages" of homogenized milk. They have presented considerable research data, of a too-familiar polemical type, to support their contention that homogenization of milk and milk products is a beneficial process. Certainly it benefits the larger milk distributors who use it, because it helps them to salvage stale milk. Homogenized milk keeps better than pasteurized or certified raw milk. The enthusiasm of the larger distributors for the process is also explained by the fact that small dairies

cannot afford the expensive homogenization equipment. If the large units of the milk industry are permitted to impose homogenization on the consumer, then the small dairy units will disappear as quickly as did the old stone-mills, and with comparable results.

With the disappearance of the picturesque stone-mills from the American landscape went the disappearance from the market of the whole flours or meals which they produced. Similarly, with the economic liquidation of the small dairy, raw, approved or certified milk will be increasingly difficult to obtain. More and more we shall be obliged to subsist upon the products which can be made from stale, post-dated milk.

Processed Vegetables and Fruits:

The preparation of food by cooking, irrespective of whether it is done in a cannery or a kitchen, can easily be a most destructive form of food processing. Fortunately, on the whole, the pressure cooker is now displacing the old-fashioned frying pan and cooking pot. If properly used, the pressure cooker reduces nutrient losses in the kitchen. It is regrettable, however, that the directions which accompany the pressure cooker in respect to cooking time are so frequently incorrect. Foods cooked under steam pressure for the number of minutes which are recommended for each type of food, are no better than foods cooked by the old methods. They are thermically degraded to non-nutritious "pap." Steaming time should be reduced by one-half or one-third, under moderate steam pressure. If this is done, foods will be properly cooked to retain most of their vitamins and minerals.

Commercial food processors use heat, of course, and often excessively, to destroy the activity of bacterial contaminants. They also bleach vegetables by several methods, thus destroying the chlorophyl and greatly impairing the mineral and vitamin content.

Fruits are processed by cooking, drying, canning, and treatment with sulphur or combined with large quantities of sugar in the manufacture of jams, jellies, preserves, etc. Citrus fruits are dyed or subjected to gas treatment to make them appear tree-ripened. Most fruits (including berries, melons, etc.), except when grown in the locality in which they are marketed, are harvested when unripe and allowed to ripen during transit and in the market.

Animal Proteins:

Proteins — animal proteins especially — are indispensable in man's dietary. Because of their indispensability and their seasonal supply, they have been subjected to a number of processing practices. Meats and fish are dried, salted, pickled, smoked, soaked in salt solution, saturated with sugar or pumped with gelatin, fat or "smoke" solutions. Frequently, a tough meat is ground or subjected to partial putrefactive decomposition to make it tender. Two chemicals, sodium nitrate and sodium nitrite, are frequent additions to processed meat to facilitate uniform dispersion of salt and sugar. In addition to this, chemical preservatives are added to fish or tinned meats to give these a healthy color or to prevent the growth of bacteria.

Eggs are an important contribution to our protein requirement. When produced under commercial poultry-farm conditions for public markets, they are non-fertile and devoid of an essenial hormone. "Spotted" eggs may be salvaged and used in the production of commercial egg powder and this may account for some cases of botulism. What effect hormone-deficient eggs play in the ever-increasing allergic states should be a matter of investigation. In my experience, two patients, allergic to commercial eggs and suffering from chronic asthma, were relieved of their allergy and their asthma, when they substituted fertile eggs for commercial eggs. The explanation for this reaction is not at hand. Perhaps it involves the sex-hormone present in fertile eggs and a better biotin-avidin balance.

Cheese is an important item in the dietary. Its importance is appreciated when other complete proteins are scarce, costly or rationed. Unfortunately, the consumer is not too familiar with the food value of cheese. Most people are more familiar with imported cheeses than with domestic cheeses. Possibly our best domestic cheese is the round loaf of American cheese weighing 70 to 80 pounds and known to the trade as a "Cheddar." A "Daisy" weighs 20 to 25 pounds, and "Young Americas," "Long Horns," and "Flats" are smaller sizes and shapes of American cheddar.

Five ounces of American cheese have the same calcium, vitamin A, and protein content as a quart of milk. Therefore, it is a cheap, economical source of protein; cheddar cheese contains almost 25 per cent protein; round steak 17 per cent. Discriminating buyers should find out whether or not a cheese is natural or processed. Processed cheese has been unofficially described by the Food and Drug Administration as "the modified cheese made by comminuting (grinding up) and mixing one or more lots of cheese into a homogenous plastic mass, with the aid of heat, and with or without the addition of water, and with the addition of not more than 3% of a suitable emulsifying agent."[52] The agency fails to mention why it is necessary to grind, mix, pasteurize and add an emulsifying agent to one or more lots of cheese — or why this plastic mass has to be "aerated" with compressed air. People sometimes complain of digestive discomfort after eating processed cheese.

Hydrogenated Fats:

Fats are another class of foods which are indispensable for proper nutrition. However, the biologic quality of the fats consumed today are not the same as those which our grandparents consumed. For the past 20 years a great number of commercial fats have been extended into the dietary — chiefly hydrogenated fats. Moreover, natural fats have undergone a number of induced changes. For example, by force-feeding concentrated food fractions it has been

possible to increase the cream and butter content of cow's milk. Animals destined for the slaughter house have also been force-fed on concentrated food-fractions. This is done to increase the weight of the animal. Not infrequently two phenomena may result from this force-feeding practice — first an infiltration of fat into their muscle tissues and then, if the force-feeding process is continued long enough, an actual fatty degeneration within the muscle tissues. Just what biologic effect this has, outside of increasing the fat content of these foods, no one seems to know. Fats and oils are derived from many sources and they are frequently processed to decrease the spoilage factor. Butter-yellow, a coal tar derivative now well known to be a carcinogenic factor, was used for almost 50 years to color oleomargarine as well as butter. Inferentially, there are many fats on the market which are of poor biologic quality. These fats have increasingly become a component part of many of the staple food articles which are obtained at the grocery store.

From the foregoing it is apparent that there are many factors which may impair, alter or sabotage the biologic quality of our food supply. Unfortunately, the average consumer knows little or nothing of all this. Hence in selecting his food he is at the mercy of the advertiser, the commercially pressured food columnist and the radio huckster.

Allergy, Food Poisons and Perverted Nutrition:

Disciples of the allergic school feel that milk, eggs, fish and shellfish should be eliminated from the dietary. Andresen, the leading proponent of this school of thought, feels that a few other commonly used foods should also be eliminated. He summarizes his observations by stating that milk has been found to be one of the offending foods in over 80% of cases and the only one in nearly 40%; wheat contributed 18%; tomatoes 15%; oranges and potatoes 12% each; and eggs 9%.[53] The observations of this competent clinician are provocative and cannot be dismissed

lightly. On the other hand, many ulcerative colitis patients displaying colonic lesions, apparently allergic in nature, are negative to allergic tests for these commonly accused offenders. Possibly most of these allergic manisfestations are more closely related to the excessive consumption of processed starches, sugars and proteins, than they are with milk, eggs, fish and shellfish, although it is a frequent observation that these foods may worsen the local condition. The quality of the milk supply has progressively been debased in the past few years — largely because of the use of salvaging processes — pasteurization and homogenization. The quality of the egg supply has also declined, being influenced by modern poultry farm practices to increase the production of eggs and to decrease the spoilage factor of these during refrigeration — thus the non-fertile egg. Therefore, the cow and the hen have been converted into a milk and egg factory, respectively; quality does not count — quantity being the yardstick for production and profits. These allergic manifestations are probably an individual constitutional reaction to faulty foods and food habituations which play an important role in debasing the tolerance threshold for these foods (milk, eggs, fish and shellfish).

From my point of view, Andresen's success with his cases is predicated largely upon an improved dietary regimen, containing sufficient natural residue to promote normal bowel function. I concur that milk, eggs, fish and shellfish should be restricted until the patient has made sufficient progress to warrant their cautious inclusion without an unfavorable reaction.

A rarely mentioned faulty food habituation, most important in the production of allergic states, is the consumption of food products containing large quantities of refined sugars and syrups combined with cocoa or chocolate to make various confections — the same confections which Wesbrook Pegler's Eleanor-The-Great and Quartermaster-General Edmund B. Gregory plugged on a coast to coast network

broadcast on December 9, 1942. This broadcast was sponsored by the Council on Candy as a Food in the War Effort, a propaganda creation of the National Confectioners Association. These two celebrities turned in some neat plugs for the sugar and candy industries. Of course, what Mrs. Roosevelt and the Quartermaster-General neglected to say in behalf of candy and sugar was fully covered by the announcer.

Most likely their "plugs" induced many doting parents to send their loved-ones in the armed forces excessive amounts of sugar and candy to fortify their energy and morale requirements. No one knows how many dental cavities resulted from this increased consumption of sugar and candy products or how much this contributed to the prevalence of gastro-intestinal disturbances and ulcerative colitis amongst servicemen.

Molecules of Sugar:

From the inception of the flour and bread enrichment program, the sugar industry eyed the zooming sales and the flattering press notices of the millers and bakers with envy and concern. If bread and breadstuffs, why not sugar? It is a universally consumed food staple. Moreover, the intelligent consumer knows that white sugar is destitute of minerals and vitamins. The "take out and put back" pseudo-scientific philosophy sponsored by the processors soon gained sufficient momentum to induce manufacturers of emasculated food products to attempt to obtain approval of this vicarious program for their products. If sugar and sugar-rich foods could be enriched, they would achieve a quasi-nutritional respectability and thus lend themselves to a too familiar type of hucksterism. This would offset the "carping" criticisms of nutritionists who were not "playing ball" with the food processors. The fortification of sugar and sugar products was proposed to the Food and Nutrition Board shortly after Roosevelt's celebrated Nutritional Conference had convened and sold the consumer

down the river on the "enriched" bread deal. Fortunately, it was decisively rejected. The Board saw through this scheme and was perturbed about what effect any increase in the already excessive consumption of sugar would have upon the general health of the public.

Thwarted in their attempt to impose this contradictory program upon the American public, some 77 producers and processors of cane and beet sugar in the Continental United States, Hawaii, Puerto Rico, Cuba, Canada and Haiti announced their own entry in the field of food science by the creation of the Sugar Research Foundation which was incorporated on June 10, 1943. The avowed objectives were to sponsor both pure and applied scientific research on sugar in the fields of chemistry, biochemistry, microbiology and medicine. It was to promote scientific studies of sugar as a human food, the industrial application of sugar and its derivatives, and to *disseminate factual information about sugar.*

This Research Foundation was organized with appropriate fanfare. Most scientific men were captivated by the idea that at last the sugar industry was going to settle the sugar controversy, come what may. A few non-logrolling nutritionists posed this question — can a great industry, burdened by vast obligations to its stockholders and its employees, and at the same time vested with a major public responsibility to the nutritional and health status of the world's peoples, follow the light of science wherever it may lead, even if it should lead to its own detriment and loss? To offset this needling attitude on the part of some honest but "carping" nutritionists, the Board of Directors appointed Doctor Robert C. Hockett of the Massachusetts Institute of Technology as Scientific Director. Doctor Hockett was a scientist of impeccable reputation and high achievement. For more than four years he has directed the destiny of the Sugar Research Foundation. There were a few "heretical" nutritionists on the sidelines who con-

tinued to question the validity of the aims and purposes of the Sugar Research Foundation, and Doctor Hockett finally settled all doubts when in the SUGAR MOLECULE, Volume III, Number IV, October 1949, he uninhibitedly crashed through the saccharine curtain and stated:

> Because the Sugar Research Foundation has undertaken to support several studies in the field of dental caries research, certain members of the dental profession appear to have leaped to the conclusion that the sugar industry is interested only in trying to disprove evidence that high-sugar diets may be conducive to tooth decay.
>
> Nothing of this kind has ever, in fact, been contemplated. Our program has always had very positive objectives which, if briefly summarized, might be stated in the following words: *The purpose of our dental caries research is to find out how tooth decay may be controlled effectively without restriction of sugar intake.**
>
> To deny that there is self-interest involved in this program would be fatuous in the extreme. It is obvious that attainment of this goal would be beneficial to all industries that produce and process sugar or utilize it in their products. However, if this commercial interest were the only consideration, the whole undertaking would be far less significant than it is in fact.
>
> The important point is that the public interest is well served by the same program. Sugar has many values as an ingredient of the diet and is, moreover, a commodity that offers far more yield of food energy per acre than any other agricultural product. In a perpetually hungry world, this aspect must not be minimized. In addition, people like sugar and sugar-containing foods. Relatively few are willing to curtail their use of it to any significant degree even if they were convinced that to do so would benefit their teeth. They literally prefer to eat cake with false teeth than to abandon cake for any appreciable period of time. Hence the program designed to control tooth decay by widespread or long-sustained sugar restriction is foredoomed to failure as a method of *mass* control of this most widely prevalent of diseases. Such measures are likely to be utilized fully as recommended only by scattered super-conscientious parents and exceptionally self-disciplined or even hypochondriac individuals.

*Italicized by Dr. Hockett.

Indeed, these are startling statements. To come out and flatly state that the purpose of the Foundation's Dental Caries Research program is to find out, not what part sugar plays in the production of dental caries, but how tooth decay may be controlled effectively without restricting the sugar intake, is a new low in the annals of science. By implication, the Doctor *tacitly admits that sugar does play a part in the causation of dental caries.* This sort of philosophy reminds me of an example cited by a professor of therapeutics in respect to the control of pain. He said: "You are called to see a man who is sitting on a tack and refuses to get off the tack. He complains of pain and pleads for medicine to relieve it. You do not attempt to solve his problem by prescribing opium for the relief of pain. First, get the man off the tack. Remove the cause and the effect will take care of itself."

Doctor Hockett's resort to name-calling is a reflection on the calibre of the intelligence of people whom he labels super-conscientious parents, exceptionally self-disciplined or even hypochondriac individuals. It must be a source of great satisfaction to the Doctor to be convinced that he alone is right in perspective and fact regarding dental caries.

Apparently, despite Doctor Hockett's many attainments in the academic field, he seems disinclined to sponsor an eye to eye correlation between cause and effect.* In the article he critically reviews the various theories concerning the genesis of dental caries. Paradoxically enough, each theory presents only partial aspects of the total problem. Unfortunately, space does not permit a more extensive discussion of the fact that if all of these theories were intelligently correlated, then some worthwhile information could be adduced.

*There is no premeditated intent on the part of the writer to unduly criticize Dr. Hockett personally. But since he is the major directional personality in the Sugar Research Foundation, and apparently its authorized spokesman, then the criticisms must necessarily involve him as a prestige symbol.

His statement, "chemists know very well that fresh and dried fruits contain the same kind of sugar that is produced from beets and cane" is a statement that would be refreshing if it were not so obviously a bit of special pleading.

Sugar is as much a problem for economists, statesmen and "scientific" directors of research foundations, financed by processing units of the food industry, as it is for clinical nutritionists. In effect, this nutritionally emasculated substance has starved or malnourished its producers, processors and consumers. On the other hand, it has also given us the sugar-saturated children of our urban and rural slums, anemic victims of our advertising-fostered Sweet Tooth.

Between 1850 and 1900 the world's sugar consumption increased tenfold. It trebled between 1900 and 1940. In the United States the 1926 annual *per capita* consumption was 109 pounds, probably accounting largely for the alarming incidence of dental caries found in our young men by selective service board examiners in World War II. With the exception of a brief spurt in 1941, the annual *per capita* consumption was 100 pounds, representing about 17% of the calories in the average American dietary. This does not reflect true *per capita* consumption. Many intelligent people have decreased their sugar consumption and I dare say consume no more than 15 to 20 pounds yearly. On the other hand, many people do consume far more than 100 pounds a year.

Whether you like it or not, you are going to eat sugar. Over the years the drive has been for a universal extension of sugar as an ingredient in almost every dish concocted in a factory or a kitchen. It has found its way into numerous beverages, salad dressings, gravies and sauces; into all kinds of cakes, pastries, pies and other bakery products; and in the candy industry all the way from lollipops to the high-priced bonbons that Lotharios give their ladies. Increasingly, it has been combined with meats—sugar-cured hams, Ready-meat, Treet, Spam, corned beef, corned beef hash and other

proteinaceous concoctions. It is a common ingredient in packaged, cellophane-wrapped, tinned and glass-container foods. Therefore, the average person, although he does not use sugar as sugar, is almost certain to consume generous quantities of it in staples if he buys tinned, packaged or any other commercially prepared foods.

The propensity for the combination of cocoa or chocolate with sugar or syrups in the production of an allergy has been cited. The relationship of sugar and sugar-rich foods to ulcerative colitis is that they supplant nutritious foods, thus simultaneously creating an absolute and relative deficiency of necessitious nutrients. The excessive use of sugar should be considered a positive factor in laying the groundwork for the onset of chronic idiopathic ulcerative colitis. Its consumption should be sharply limited. In my experience, the ulcerative colitis patient reacts much more favorably when he does not consume sugar as sugar, in the form of sweet beverages or in various food concoctions which contain large quantities of sugar. Indeed, I have observed several recurrences which were traceable to the consumption of large quantities of sugar-rich foods.

Chemical Adulterants:

During the last war more than 500 new chemicals were introduced into our food supply without sufficient knowledge of their effect on public health or welfare.

Producers and manufacturers were quick to adopt the use of many chemicals and so seriously has this endangered the health of the nation that Doctor Paul B. Dunbar,[54] Commissioner of the Pure Food and Drug Administration said:

> What new disease may grow out of the use of synthetic foods, no man can tell, but when man starts competing with nature in the blending of food elements he should be sure that his formula does not bear the skull and crossbones.

In addition, several thousand new insecticides, fungicides and herbicides (weed killers) have been placed on the market in the last few years. In many instances, little is known

concerning the acute or chronic pathologic effects of these on man. Unfortunately, it is difficult and sometimes impossible to remove the residue of some of these new poisons from food products. Still more unfortunate for the consumer, a number of these poisons are absorbed into the plant tissues and cannot be removed. Since they do not alter the appearance of the plant tissues, the consumer does not know that they are ingredients of the foods he buys. Therefore, you may buy "chemicalized" toadstools instead of wholesome foods. The problem of preventing potentially toxic quantities of these substances from being distributed as components of foods faces the food processor as well as the farmer. Oftentimes they are handicapped because of inadequate methods for determining the presence of a contaminating pesticide.

No doubt, there are certain wise chemists who are employed by the chemical suppliers to the food processing industry who will expect us to evolve the same kind of a biologic expedient which the house-fly developed to make himself immune or resistant to DDT. It must not be forgotten that as soon as a living organism fails to be destroyed by some of the miracle chemicals, then they are supposed to be resistant to this or that. This is getting to be the great alibi of this era despite its innate ridiculousness.

To date, no one knows what effect these chemical adulterants may exert upon the intensification or the production of what we now consider allergic states. This problem remains to be investigated adequately. The probable effects of agene as a factor in producing a disturbance of encephalographic tracings in allergic youngsters has been cited. These are generalized observations and are open to question. We need eye to eye correlations concerning the effect of these new chemicals upon public health and welfare.

Food, Soil and Water:

Food values go back to the soil. Some soils, even in their virgin state, are inadequately supplied with the miner-

als needed to grow healthy crops. Many soils have been depleted by overcropping and erosion. This disturbance of the ecological balance coupled with the discharge of silt, municipal garbage, and industrial wastes into the streams, soon adversely affects the foodstuffs obtained from fresh-water or marine life. The water supply of municipalities is also impaired — an effect which nutritionists have ignored.

From coast to coast the contamination of municipal water supplies is so great that an ever-increasing amount of chlorination is required. This is nutritionally important. In my experience, excessively chlorinated water will produce gastro-intestinal disturbances in apparently healthy people. It requires little imagination to envision the effect of this "processed" water supply on individuals with disorders or diseases of the gastro-intestinal tract.

Recently extensive experiments have been initiated on the effect of fluorine-treated drinking water upon the development of teeth and the prevention of dental caries in youngsters. Fluorination of drinking water is urged despite the knowledge that if drinking water contains one part or more of fluorine per million, it is likely to produce a condition known as chronic endemic dental fluorosis — in other words, mottled enamel.

The results of this induced disturbance of the ecologic balance of nature — in effect the application of the homeopathic (antidotal) philosophy of therapeutics to the environment — are as yet inconclusive.

In general it may be said that nutritionists can no longer afford to ignore the biocoenetic soil disturbances that attend improper agricultural methods, the injudicious use of artificial fertilizers and the flood of new and highly-toxic pesticides and insecticides. All these things are bound to affect the biologic quality of the food crops consumed by both domestic animals and man.

What Plant and Animal Breeders Don't Yet Know:

Corn yields have been greatly increased through the use of the new self-pollinated hybrids, and equal gains are expected as plant breeders extend their experiments to the other cereals and to food crops in general. Whether or not the qualitative nutritive content can be maintained or increased in quantitative increases obtained by hybridization, remains to be seen.

Artificial insemmination has a number of demonstrated economic advantages for the cattle breeder, especially the owners of prize males. To date, no one knows what biologic effect this disconcerting innovation will have upon the female.

Perverting Food Habits for Profit:

Observations of the dietary habits of primitives and animals have shown that the food intake of primitives and practically all animals tends to be controlled by the self-regulating mechanisms of the natural appetite. How does it happen that this automatic mechanism of food selection has become atrophied in civilized human beings? How does a food addiction become institutionalized in culture? Which comes first? Is a whole pattern of food production, processing, and distribution built up to satisfy the perverted appetites of individuals in whom the self-regulating mechanism of food selection has become atrophied? Or does the individual eat what the social or economic environment in one way or another persuades or forces him to eat — until eventually he develops physiologic perversions and addictions which support and extend these pressures? The reader will find a more extended discussion of this in TOMORROW'S FOOD[55] and in an article entitled OUR CIVILIZED FOOD HABITS.[56]

To a considerable extent the perverted food habits encountered by clinical nutritionists are the creation of the food advertisers. Huge vested interests have been established in such seriously damaging addictions as alcoholic

beverages, the cola drinks, candy bars, soda fountain concoctions, pastry-factory products, chewing gum, etc. In addition to this, producers, processors, and distributors are all interested in increasing the sale of their food products. Many devices have been worked out for doing this. Today we find the food processor, his hired huckster and lobbyist effectively entrenched in almost every aspect of our way of life. Organized medicine and dentistry, the hospital system, custodial institutions, schools of dietetics and household arts, domestic science organizations, women's clubs, the industrial lunch room, the publishing business, the public school system and other organizations have been effectively influenced by the food industry. The net result of this greed motivated drive has been to root the traditional mixed meal into our food culture and economy. This method of meal planning has no scientific basis in fact or principle.

It has been and still is the result of co-active circumstances induced by food scarcity, and paradoxically enough, by the gluttonous tendency of man during periods of food abundance. It is an *inurement* practice originally based upon these two factors. Corporate units of the food industry were the first to recognize that this unsound practice, dictating as it does that every meal must be basically composed of concentrated starches and sugars, proteins and fats, *was their most potent tool for promoting the increased consumption of every product sold in the food market.* It is for this reason that the food industry as a whole will so impetuously attack anyone who questions the scientific propriety of the mixed-meal method of meal planning. And since the press and the radio share in these vested interests, it is almost impossible to combat them effectively.

Natural Foods Are Best — if you can get them:

I am aware that it is unusual, to say the least, to suggest as the keystone of treatment for a specific disease, like idiopathic ulcerative colitis, what amounts to a social

and economic reconstruction of the patient's food environment. Yet I submit that this prescription follows naturally from the writer's experience with the disease. My experience has shown conclusively that a diet of well-grown, unprocessed, un-chemicalized, natural foods is not only best but necessary in most cases to bring about a cure of the disease. The ulcerative colitis patient should permanently renounce the mixed meal.

Pavlov, The Mixed Meal, and Ulcerative Colitis:

In my experience an unhealthy condition of the intestinal flora is usually revealed by the examination of stools of mucous colitis patients. In attempting to correct this condition one is again confronted by the stubborn *status quo* of established food production and consumption patterns, including the mixed meals that have come to be considered normal and necessary by the peoples of Western civilization, although they are not at all characteristic of the diets of many outstandingly healthy primitive peoples. Nor does the adoption of the mixed meal, combining carbohydrates, fats and proteins accord with the best research we have had to date on the digestive results of such mixtures, namely that of Pavlov.

Many years ago the great Russian scientist proved that a mixed meal containing excessive quantities of the ternary elements predisposed a faulty digestion of carbohydrates or proteins. If proteins are well digested, the carbohydrates would be only partially digested and the residue would promote a fermentative type of intestinal flora, and vice versa.

Pavlov's findings have never been disproved. Rather, they have been improved by subsequent research. Today physiologists agree that each food excites a specific, precise and purposive activity of the digestive glands and processes. The peculiarities in the secretion are not limited to the properties of the digestive juices, but are also characteristic

in respect to the rate of flow, the duration of the flow, and also to total quantity. See figures 1, 2 and 3.

Pavlov[57] has tabulated some interesting results concerning the digestive juices. On page 37 of his book, THE WORK OF THE DIGESTIVE GLANDS, he states in respect to gastric (stomach) digestion:

> Not alone the digestive power, but also the total acidity*, varied with the nature of the diet. The acidity is, however, greatest with flesh (0.56%) and lowest with bread (0.46%). In a similar way the total quantity of juice poured out and the duration of its secretion are seen to be dependent upon the kind of food. This relationship is equally clear whether, in estimating the food, one takes its total weight, or its amount of dried substance, or, lastly, its content of nitrogen (since the gastric juice acts only on the protein constituents).
>
> If the quantity of juice secreted during a given period be divided by the number of hours in the period, the mean hourly rate of secretion is obtained. Even this number, which represents the mean degree of gland activity, is different for the different sorts of food. Comparing equivalent weights, flesh requires the most, and milk the least gastric juice; but taking equivalents of nitrogen, bread needs the most and flesh the least. The gland work per hour is almost the same with milk and flesh diets, but far less with bread. The last, however, exceed all the others in the time required for its digestion, and the flow of juice is correspondingly prolonged.
>
> The special features of gland work, dependent on the nature of the food, are not limited to the distinctions given. They likewise prominently appear as qualitative variations in the juice secreted hour by hour. This time I furnish only one example for each kind of food, and beg you to believe that it repeats itself with the same beautiful precision we have already seen.
>
> These facts are highly interesting and of the greatest importance. Each separate kind of food determines a definite hourly rate of secretion, and produces characteristic alterations in the properties of the juice. Thus, with a flesh diet, the *maximum rate of secretion occurs*** during the first or second hour, the quantity of the juice furnished in each being approximately the same. With bread diet we have invariably a pronounced

*The acid was estimated titrimetrically, and is expressed in percentages of HCl.
**Italicized by Pavlov.

maximum in the first hour, and with milk a similar one during the second, or the third hour.

On the other hand, *the most active juice** occurs with flesh in the first hour, with bread in the second and the third, and with milk in the last hour of secretion. Thus the period of the maximum outflow, as well as the whole curve of secretion, is characteristic for each diet.

It appears to me that the facts here given lend strong support to our previous conclusion, that the variations seen in gland activity during the course of a digestion period have some essential meaning. When, for example, a special curve of secretion is determined by every single kind of food, surely this must argue a definite purpose and be assigned a special significance.

We have now learned something of the many fluctuations in the work of secretion under different conditions. Their conformity to laws is a guarantee that they are important.

On page 40 Pavlov continues:

The work of the gastric glands in providing juice for the different foodstuffs, must be recognized to be also purposive in another sense. The vegetable protein of bread requires for its digestion much ferment. This demand is supplied less by an increase in the volume of the juice than by an extraordinary concentration of the fluid poured out. One may infer from this that it is only the ferment of the gastric juice that is here in great requisition and that *large quantities of hydrochloric acid would be useless or possibly injurious.*** We see, from the following, *that during gastric digestion of bread an excess*

QUANTITIES AND PROPERTIES OF GASTRIC JUICE POURED OUT ON DIFFERENT DIETS: 200 GRMS. FLESH, 200 GRMS. BREAD, 600 C. C. MILK

	QUANTITIES OF JUICE IN C.C.			DIGESTIVE POWER IN M.M.		
Hour	Flesh	Bread	Milk	Flesh	Bread	Milk
1st	11.2	10.6	4.0	4.95	6.10	4.21
2nd.	11.3	5.4	8.6	3.03	7.97	2.35
3rd.	7.6	4.0	9.2	3.01	7.51	2.35
4th.	5.1	3.4	7.7	2.87	6.19	2.65
5th.	2.8	3.3	4.0	3.20	5.29	4.63
6th.	2.2	2.2	0.5	3.58	5.72	6.12
7th.	1.2	2.6	..	2.25	5.48	...
8th.	0.6	2.6	..	3.87	5.50	...
9th.	..	2.9	..	...	5.75	...
10th.	..	0.4	..	...	...	...

*Italicized by Pavlov.
**Italicized by Author.

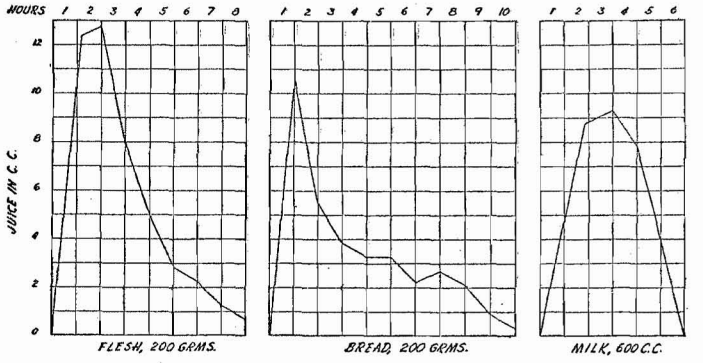

Fig. 1. (Fig. 10 from Pavlov) — Curves representing the rate of secretion of gastric juice with diets of flesh, bread, and milk.

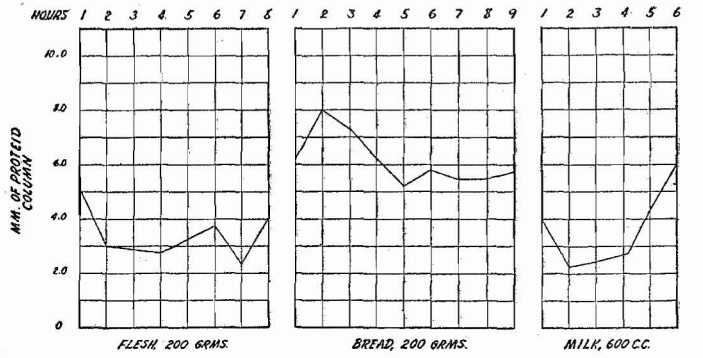

Fig. 2. (Fig. 11 from Pavlov) — Curves representing the digestive power of gastric juice, hour by hour, with diets of flesh, bread, and milk.

*of hydrochloric acid is actually avoided.** The total quantity of juice secreted on bread is only a little larger than that secreted on milk. It is distributed, however, over a much longer time, so that the mean hourly quantity of juice with a bread diet is one and one half times less than after taking milk or flesh. Consequently, *in the digestion of bread, but little hydrochloric acid is present in the stomach during the period of secretion.** This harmonizes well with the facts of physiological chemistry, namely, *that the digestion of starch, which is con-*

*Italicized by author. See Figures 1, 2 and 3.

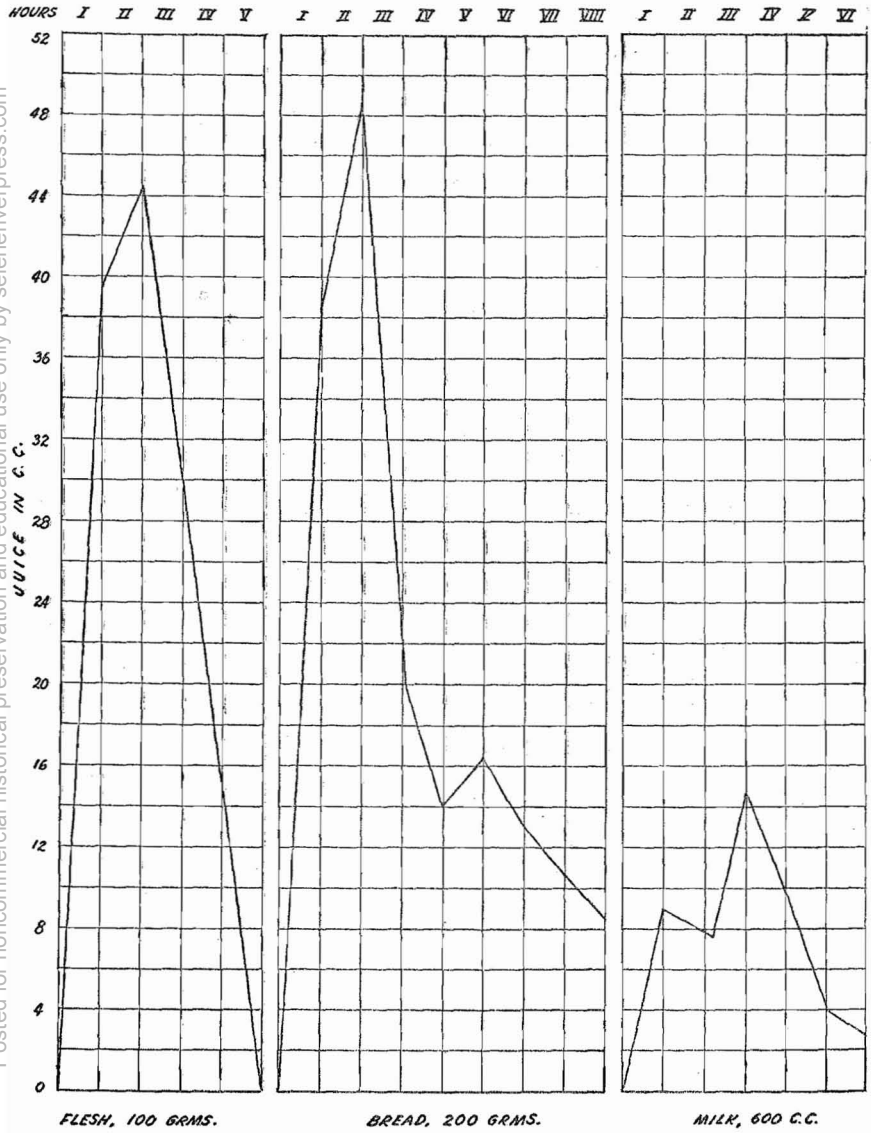

Fig. 3. (Fig. 12 from Pavlov) — Curves of secretion of pancreatic juice with different diets.

*tained in large quantities in bread, is impeded by an excess of acid.** From clinical observations we know further that in cases of *hyperacidity, a large part of the starch of bread escapes unused from the gastro-intestinal canal* while *flesh is excellently digested.**

As an aid towards the digestion of starch, or at all events in relation thereto, another phenomenon plays a part which has already been several times mentioned, but not yet explained. I mean the long pause of at least five minutes which always intervenes between the taking of food and the beginning of secretion. This interval invariably occurs, whether the observation be made on the large stomach of a sham fed animal, or on the miniature pouch of a dog normally fed.

This latent period, as it may be termed, is never less than four and one-half to five minutes, but may often be as long as ten minutes. What is its significance? We have no reason for assuming that it depends on conditions, such as the time necessary for the glands to fill up to their mouths, or till the juice moistens the inner wall of the stomach and runs in streamlets towards the fistular orifice. This cannot be the explanation since the latent period occurs when the glands are already filled with juice. Further, it would be singular if the gastric glands, *per se*, were incapable of responding to a stimulus before the lapse of five minutes after its application. Nothing, therefore, remains but to recognize in the occurrence, a definite aim. Perhaps these five to ten minutes are provided to allow the action of the amylolytic (starch splitting) ferment of the saliva to proceed. But this explanation cannot, of course, be regarded as very convincing, so long as the question has not been systematically investigated.

Moreover, Pavlov demonstrated the inhibitory effect of fat upon the flow of the digestive juices on flesh and milk. These experiments have shown that if fat is introduced immediately before, during, or after a flesh meal, it will inhibit the flow of the gastric juice for a period of one to two hours. Thereafter, a rapid outpouring of the digestive juice occurs at a time when the flesh should have been half digested. The sudden increase in the acid titre explains why flesh combined with butter and other fats, and/or why proteins containing excessive amounts of fats

*Italicized by Author.

(forced-fed livestock) will frequently produce hyperacidity one or two hours after meals. Therefore, the premise that concentrated fats should not be combined with concentrated proteins is based upon sound physiology — not upon faddism.

Protagonists of the mixed meal have cited milk as nature's classic example to disprove the scheme of "concordant" meal planning. Cow's milk contains 5, 3.3, and 4 per cent respectively of carbohydrate, protein and fat. This contrasts strikingly with the ternary elements present in a typical mixed meal — protein soup, steak, bread, butter, potatoes, fat, and protein-rich desserts.

A study of the characteristic "milk juice" graph will emphasize the part which fats play in depressing the ferment and acid titre which are so clearly evident in the "flesh juice" and "bread juice" graphs. Pavlov demonstrated that by increasing the fat content of milk, a depression of the ferment and acid titre would follow, thereby confirming the evidence that fat acts as an inhibitor of the digestive process.

I have quoted at length from Pavlov, both because of the brilliance and contemporary pertinance of his observations, and because so many nutritionists and other professionals are unfamiliar with his work — vaguely they recall that it "had something to do with dogs and conditioned reflexes." Pavlov's observations and researches concerning the limitations of the digestive function of the alimentary tract, and Sir Robert McCarrison's[58] sane perspective regarding the fundamental aspects of a sound food culture and economy, have laid the floor for the meal planning scheme used in this work.

Proper Nutrition Necessitates Abandoning the Mixed Meal:

The mixed meal, *as planned by servants, cooks, housewives, and so-called dieticians,* is an indiscriminate jumbling together of generous and oftentimes excessive quantities

of *concentrated proteins, concentrated starches and granular sugar products, and concentrated fats,* to form the nucleus of a meal. This meal is then garnished with desserts, ice cream, pastries, overly-cooked vegetables, sauces, dressings, gravies, jellies, and a plate of salad dressing in which may be found a leaf or two of lettuce.

It requires real effort to emancipate the average person from the obsession that he must eat all of these foods at the same meal.

The mixed meal, when appraised in the light of the newer knowledge of nutrition, is non-specific, non-purposive and totally unscientific, and the adherence to its formularism intensifies the nutritional shortcomings of the average dietary. Mixed-meal habitués, even though their appetites may be sub-normal, consume excessive amounts of the ternary elements.

Does the mixed meal fit into Nature's scheme of digestion? Or does it exceed the limitations of the digestive organs? I have cited the classic research of Pavlov, which medical students are made to read and told to respect since Pavlov's findings have never been refuted. Pavlov is "orthodox" when taught to medical students. But let some physician attempt to apply Pavlov's findings to clinical dietetics, and he is at once denounced by the mouthpieces of medical orthodoxy.

I am at a loss to explain this attitude on the part of some orthodox medical editors. They will admit that the mixed meal too often exhibits serious shortcomings, yet, they champion furiously the practice of mixed-meal formulation. How can they reconcile their defense of the mixed-meal with accepted scientific facts that prove the mixed-meal to be a digestive mess?

I fear the answer to this question is more likely to come from the social psychologist than from the pundits of my own profession. The average physician is in fact as much a creature of his institutional environment — includ-

ing his food environment — as the patients he treats. This same observation includes most nutritionists, especially the academic type. And the mixed-meal of today is richly institutionalized and rooted in our chaotic food culture and economy. It has no warrant in the past or current findings of the physiologist. *It needs none.* Enough that cooks, chefs and dieticians habitually combine concentrated carbohydrates, sugars, proteins and fats at each meal and into the composition of their salad and dessert masterpieces. Enough that the food industry and its allies have geared their technology and their economics to the perpetuation of this and other nutritional anarchisms. Unhappily nobody has a vested interest in *sound* nutrition except the victims of *bad* nutrition — including the sufferers from ulcerative colitis.

Neither science nor the nutritional welfare of the consumer dictated the war-time flour and bread enrichment program, since carried over into peacetime by the state-by-state pressure of the milling and pharmaceutical lobbies. Admittedly a bad compromise that set a dangerous precedent, "enrichment" as a policy and a program has been made to look more and more questionable by the subsequent discoveries and advances of nutritional science.

Today nothing is more clear to the student of nutrition than that there is no such thing as a one-to-one relationship between any specific deficiency disease and corresponding deficiency of a specific food accessory factor. Unhappily, however, nothing has been more clear to the food processors than that the adoption of the "enrichment" program gave them the green light to process whatever they liked out of natural foods — and then make advertising capital out of putting a little of it back in the form of added synthetic vitamins and/or inorganic minerals. As a result the debasement of staple foods has hit a new low. The traditional mixed meal composed of these debased staple foods perpetuates and intensifies these nutritional

shortcomings. Today's mixed meal is composed preponderantly of foods that have been refined, pasteurized, sterilized, homogenized, fortified, enriched, restored, chemicalized, and otherwise sophisticated by food technologists who seldom question or investigate the far-reaching effect of all forms of food processing upon public health.

Can Science Better Nature:

Tampering with food is a pastime not exclusively reserved for food processors. Two other groups are interested in "improving" processed and non-processed foods. One of them seeks to do this by combining a number of nutritionally reputable foods to make a super-duper food product. In their zeal to offset the evils of processed and adulterated foods, they have unwittingly out-done themselves. Their products are not as harmful as the embalmed and debased products of the processors; but they cannot be considered the ideal solution for the ills besetting a sick food culture and economy. For example, one "health expert" has combined twelve different grains and cereals to make what he considers a complete cereal food. We are all familiar with the "health" breads which may be composed of wheat flour, dates, figs, nuts, raisins, carrot meal, soy bean meal, molasses, honey, etc. This sort of a bread is not as well tolerated as plain whole wheat bread. For many years, the dignified scientists with academic degrees have considered these food "improvers" and self-appointed "health" experts, as crack-pots, quacks, faddists and sometimes plain lunatics.

The second group of food reformers is composed chiefly of non-medical biochemists and nutritionists connected with the land grant colleges. Until recently, this group displayed more interest in animal nutrition than in human nutrition. Paradoxically enough, nutritional research has been largely absorbed into the priesthoods of agriculture and agronomy. Instead of physicians and dentists taking the lead in this subject, we now see that it is firmly entrenched in the hands

of the professors who staff the various Federal, State and privately-operated educational institutions. Unfortunately for the human animal, these nutritionists translate the results of animal experimentation into human equivalents. It is disconcerting to realize that these gentlemen are now copying the "crack-pots" in concocting super-duper foods. The most recent example of this has been the development of a "health" bread by the Department of Nutrition at Cornell University and its adoption by the mental hospitals of the State of New York. This bread is composed of unbleached flour, soybean meal, milk solids, and a generous supply of wheat germ and wheat germ oil. It has been tested on rats and the bio-assay has been satisfactory. The ideal "health" bread would have been a whole grain or cereal bread — nothing added, nothing removed. But this ideal bread is not pleasing to the millers, the bakers and the editor of the NORTHWESTERN MILLER. One wonders whether or not the development of this bread was an expedient device for circumventing the potential objections of the millers to serving the inmates of state institutions a whole grain bread which is nutritionally superior to their commercial breads.

In my opinion, none of these "improved health foods" should be prescribed for ulcerative colitis patients — their digestibility is complicated by the combinative over-concentration of many kinds of good food.

Constructive Meal Planning:

The most important therapeutic requisite is to teach the patient to plan each meal constructively, the object being to offset the shortcomings and minimize the excesses of which he is a victim. Moreover, the patient must be made to realize the nutritional shortcomings of all processed foods as outlined in the sketchy consideration of the agencies and factors which may alter or impair natural foodstuffs. In addition to this, he should be taught in a collective sense the indispensability of such nutritive factors as minerals,

vitamins, enzymes and hormones contained in natural foods, natural roughage, etc., all of which may be obtained more economically from natural foods than from drugstores.

The physician must be familiar with the fact that natural foods are relatively and needlessly expensive and hard to get. The poorer the patient the more dependent he is upon food production patterns which from the nutritionist's point of view can only be regarded as perverted and pathological. Moreover, the poorer the patient, the more faithfully he reads the current newspaper propaganda (including "plugging" food columns), the more reverentially he considers food advertisements in the magazines and listens to the goateed quackery of the radio huckster. To ask the individual patient to repudiate the starched and sugared perversions, the cellophaned, "enriched" and officially sanctified debasement of the prevailing American diet, is to ask a good deal; more in fact than many patients can achieve unaided. The data appended to this report document this observation, all too frequently. Certainly my percentage of failures would have been at least halved if I had been able to supply my patients with inexpensive and convenient means of getting the natural foods that their welfare required, or, if they had co-operated intelligently and wholeheartedly in following the specific and precise dietary instructions prescribed for them.

If a patient's nutrition is to be improved, then the physician should supply him with precise instructions for planing meals. The proper nutrition of the patient will depend upon two factors:

1) a comprehensive knowledge on the part of the physician of what constitutes proper and improper nutrition, and
2) upon the co-operation of the patient.

The instructions should be specific and precise in respect to the kind of food which may be consumed at each meal. In APPENDIX No. I will appear master menus for break-

fast, lunch and dinner; also, for eating between meals and at bedtime. The general arrangement of this plan is graphic. The foods which are to be included in each meal are arranged in groups; for example, all of the fruits are listed in a group, as are the vegetables, salads, proteins, carbohydrates, fats, etc. The groups are designated as OBLIGATORY or OPTIONAL. If the patient has a tendency to put on weight, or if a diabetic, the OPTIONAL groups are omitted. If underweight, the OPTIONAL groups are included.

If an allergic state is present the offending foods are deleted from the master menus and the illustrative menus. Thus, in the active phase of ulcerative colitis, milk, cocoa, chocolate, eggs, fish, shellfish and other allergens are omitted.

The physician should also keep in mind the physiologic fundamentals previously outlined relating to the digestive processes which Pavlov has proven so well. If these are understood, it is logical to formulate three definite rules regarding the planning of meals.[59, 60]

First, concentrated carbohydrates should not be combined with concentrated proteins.

Second, concentrated fats should not be combined with concentrated proteins.

Third, fruits, raw or cooked, should not be combined with concentrated carbohydrates.

The last rule regarding the combination of fruit and starch is based upon the fact that the digestion of starch is primarily a reduction process and this must take place in an alkaline or neutral medium. The acid contained in the fruit prevents the proper hydrolization of the starch and delays its digestion.

Briefly stated, the meals are planned as follows:

BREAKFAST: To be made up solely of fresh, uncooked fruits, berries, melons, unsulphured dried fruits, milk

or buttermilk (if allowed) and unsweetened coffee or tea or coffee and tea substitutes.

LUNCHEON: This is the starch meal and consists of vegetable soup without meat stock (optional), an unprocessed starch, a raw vegetable salad, cooked fresh vegetables (optional), butter, and a milk beverage, (if prescribed).

DINNER: This is the protein meal. It is composed of a vegetable soup which may contain meat stock, but no starch; a flesh protein, or flesh protein substitute (as cheese, or nuts, or legumes); two or more properly cooked vegetables (one should be of the leafy variety); a large green salad; fresh fruits, berries or melon for dessert; and unsweetened coffee or tea, if desired. No starch is allowed at this meal.

Much of these data concerning nutrition may seem superfluous. In my opinion, a large percentage of the failures in the management of this disease is predicated upon the unfamiliarity of the physician and the patient with the multiplicity of factors which collusively operate to debase the biologic quality of the prescribed dietary. It is for this reason that the foregoing discussion is so *apropos.*

Part VII
ANAMNESTIC COMMENTS

VII

ANAMNESTIC COMMENTS

Clinical investigation and study of this enigmatic disease first aroused my interest some 30 years ago following the death of three patients suffering from ulcerative colitis. The writer concluded that the stumbling block in treating this disease was the nutritional shortcomings of the orthodox bland dietary prescribed or recommended, not alone for ulcerative colitis, but for other diseases as well.[61] We should recognize that the average dietary at that time was not as debased and degraded by processing as it is today. This may have been a factor in the treatment success of 24 co-operative patients with a precise, specific dietary.

To discard the orthodox colitis dietary required courage. Twenty-four patients co-operated wholeheartedly in adhering to a dietary composed of natural foods. The prompt and satisfactory results more than justified the hypothesis that chronic idiopathic non-specific ulcerative colitis is a nutritionally induced hemorrhagic disease. Simultaneously, it involves multiple nutritional deficiencies and excesses (imbalances of the nutriture) in individuals with psychoneurotic backgrounds. My experience with an additional group of 18 co-operative patients between 1938 and 1943 strengthened my conviction that ulcerative colitis was not idiopathic or non-specific in origin or nature. The integration of the laboratory findings in a group of 13 co-operative patients between 1943 and 1950 with specific nutritional factors (excesses and deficiencies), and the better therapeutic response of these patients to a balancing of their nutriture supplied more evidence to support this postulation.

To presume that these simultaneous excesses and deficiencies were the chief etiologic factors concerned with this disease is not illogical. Conversely, the presumption that

the correction of these nutritional imbalances would effect a cure, is logical.

Despite an encouraging percentage of success with a substantial number of cases that had not yielded to conventional ulcerative colitis therapy, the writer is convinced that any truly effective large scale attack upon the disease must take the form of public health measures designed to break the control of the commercial food processor over our food economy and culture. Only a positive national food policy, designed to grow healthy food on healthy soils and to deliver these foods to the consumer with a minimum of destructive processing and dangerous adulteration, *in meals constructively planned,* will meet the challenge of the ulcerative colitis problem.

Six years ago, intensive clinical and laboratory investigations were applied to 13 proven cases of idiopathic ulcerative colitis. When the results of these investigations were correlated with current data regarding the hemorrhagic diseases, it was possible to postulate a new concept of this disease in respect to its etiology, pathology and treatment. These data confirm the hypothetic premise arrived at 30 years previously that chronic idiopathic ulcerative colitis was a nutritionally induced disease.

This etiological concept has necessitated a discussion of all dimensions of what is wrong with our food culture and economy and unless the reader evolves a working perspective concerning the many factors which have contributed to the genesis of our planless food culture and economy, then, the therapeutic approach cannot be regarded as proper or adequate.

Part VIII
CONCLUSIONS

VIII

CONCLUSIONS

1. The data presented justify the postulation of a new etiologic, pathologic and therapeutic concept of what has hitherto been known as chronic non-specific idiopathic ulcerative colitis. The syndrome should no longer be considered non-specific or idiopathic because it is a nutritionally induced hemorrhagic disease that frequently becomes chronic. The effect of the general and specific nutritional imbalances (simultaneous excesses and deficiencies) have been discussed in relation to the nutriture of emotionally unstable individuals.

2. The ternary elements, chiefly in the form of processed carbohydrates, sugars, fats and proteins, are consumed in amounts exceeding and inconsonant with the physiologic requirements. The major nutrient deficiencies are vitamins A and B, vitamin C (especially the citrin fraction), vitamin K, the vitamin P flavonols, minerals (chiefly calcium, iron and the trace elements) and proteins of good biologic quality.

3. When the nutriture becomes unbalanced as a result of these simultaneous nutrient excesses and deficiencies, accessory phenomena occur — psychoneurotic, psychosomatic, allergic and bacteriologic (pyogenic infections of the colon). In individuals with a psychoneurologic diathesis, the underlying etiologic (nutriture) factor is frequently over-sloughed by psychosomatic and allergic symptoms to the extent that the chief etiologic factor is too frequently considered subordinate to these accessory phenomena. This accounts for much of the confusion in the literature of what has been mislabeled chronic non-specific idiopathic ulcerative colitis. This new concept eliminates the confusion because it designates the role which the ternary excesses and

deficiencies of the accessory food factors play in the etiologic and pathologic phenomena.

4. This concept has necessitated an all too brief discussion of what is wrong with our husbandries; agronomic and agricultural practices; artificial soil fertilization; soil, forest and water conservation; the injudicious use of insecticides, herbicides and pesticides; nutrient losses which occur as a result of improper harvesting, transportation, distribution and marketing practices; chemical adulteration of food; as well as some damaging processing practices in food factories and/or in kitchens.

5. It has also necessitated *a critique of the scientific validity of the average mixed meal.* It is the result of many non-scientific factors which influenced the food culture and economy of early peoples. These influences were largely concerned with food scarcities, lack of transportation and preservation facilities and the gluttonous propensity of some people during periods of food abundance. In the last analysis, *it is mainly an inurement practice now utilized by the processing units of the food industry, food distributors and their advertisers as a tool for enlarging the sale of their sophisticated food products.*

The average mixed meal is composed largely of processed food products, rich in ternary elements but practically devoid of what has unfortunately been misnamed "accessory food factors." It not only serves as a tool for promoting the increased sale of sophisticated food products but also as a tool which has unwittingly been employed by the processing units of the food industry to further debase and degrade a sick food culture and economy.

6. It was necessary to enumerate and discuss all of these factors to formulate a working perspective as to what constitutes proper and/or adequate, and improper and/or inadequate nutrition.

7. When a comprehensive nutritional perspective is formulated, the therapy of this disease becomes specific and

constructive. This perspective orients the physician and the patient regarding the enormity of the nutritive and economic impact which has been imposed upon them by a distorted, perverted and debased food culture and economy. It is my hope that the data presented in this article will initiate a revision of all former concepts of this enigmatic disease.

BIBLIOGRAPHY

1. Cruveilhier, Jean: *ANATOMIE PATHOLOGIQUE du corps humain ou descriptions avec figures lithographiees et coloriees, des diverses alterations morbides dont le corps humain est susceptible.* Paris, J. B. Bailliere, 1829-1842.
2. Wieks, S. and Moxan, W.: *Lectures on Pathological Anatomy.* U. Pharmakol. *16:* 188, 1882.
3. Kruse, Prof. W.; Rittershaus, Dr.; Kemp, Dr.; und Metz, Dr.: *Dysenterie und Pseudodysenterie.* Ztschr. f. Hyg. u. Infektionsk. *57:* 417, 1907.
4. Logan, A. H.: *Chronic Ulcerative Colitis,* a Review of 117 Cases. Northwest Medicine *18:* 1, 1919.
5. Bargen, J. A.: *Experimental Studies on the Etiology of Chronic Ulcerative Colitis.* J. A. M. A. *83:* 332, 1924.
6. Daniels, Geo. E.: *Psychiatric Aspects of Ulcerative Colitis.* New Eng. J. Med. *226:* 178, 1942.
7. Mackie, Thos. T.: *The Medical Management of Chronic Ulcerative Colitis.* J. A. M. A. *111:* 2071, 1938.
8. Fleming, Alexander: *On a Remarkable Bacteriolytic Element Found in Tissues and Secretions.* Proc. Royal Soc., London, Ser. B. *93:* 306, 1922.
9. ———: *Lysozyme.* Proc. Royal Soc. Med. *26:* 71, 1932.
10. Meyer, Karl; Gellhorn, A.; Prudden, John F.; Lehman, William L.; Steinberg, Anita: *Lysozyme in Chronic Ulcerative Colitis.* Proc. Soc. Exptl. Biol. & Med. *65:* 221, 1947.
11. Meyer, Karl; Prudden, John F.; Lehman, William L.; Steinberg Anita: *Lysozyme Content of the Stomach and its Possible Relationship to Peptic Ulcer.* Proc. Soc. Exptl. Biol. & Med. *65:* 220, 1947.
12. Thompson, R.: *Lysozyme and its Relation to the Antibacterial Properties of Various Tissues and Secretions.* Arch. Path. *30:* 1096, 1940.
13. Grace, W. J.; Seton, P. H.; Wolf, S.; and Wolff. H. G.: *Changes in Lysozyme Secretion in Various Emotional States.* Am. J. M. Sc. *217:* 241, 1949.
14. Grace, W. J.; Wolf, S.; and Wolff, H. G.; *Life Situations, Emotions and Chronic Ulcerative Colitis.* J. A. M. A. *142:* 1044, 1950.
15. Andresen, Albert F. R.: *Gastrointestinal Manifestations of Food Allergy.* M. J. & Rec. *122:* 271, 1925.
16. Lium, R. and Porter, J. E.: *Observations on the Etiology of Ulcerative Colitis; Distribution of Lesions and its Possible Significance.* Am. J. Path. 15: 73, 1939.
17. Groen, J.: *Psychogenesis and Psychotherapy of Ulcerative Colitis.* Psychosomatic Medicine *9:* 151, 1947.
18. Cornell, Beaumont S.: Ed. — *Is Ulcerative Colitis a Psychogenic Disease?* Am. J. Dig. Dis. *14:* 241, 1947.
19. Andresen, Albert F. R.: *Ulcerative Colitis — An Allergic Phenomenon.* Am. J. Dig. Dis. *9:* 91, 1942.
20. Boyd, William: *A Textbook of Pathology.* 5th Ed. Philadelphia, Lea & Febiger, 1949, pp. 122, 128.
21. Norman, N. Philip: *Corrective Treatment of the Commoner Forms of Enterocolitis.* Intern. Clinics 1: Ser. 33, 50, 1923.
22. Levitan, B.: *Action of Vitamin P on the Stability of Connective Tissue Ground Substance.* Am. J. Phys. *157:* 422, 1949.
23. Levitan, B. A.: *The Biochemistry and Clinical Application of Vitamin P.* New Eng. J. Med. *241:* 780, 1949.
24. Howell, W. A.: *Theories of Blood Coagulation.* Phys. Rev. *15:* 435, 1935.
25. Seegers, Walter S.: *Blood Coagulation and the Practical Significance of Recent Advances in Knowledge of Prothrombin and Ac-Globulin.* Circulation *1:* 2, 1950.
26. Peck, Samuel M. and Copley, Alfred L.: *Medical Progress: Diagnosis and Treatment of Skin Manifestations of Capillary Fragility.* New Eng. J. Med. *235:* 900, 1946.
27. Burrows, Harold: *Some Factors in the Localization of Diseases in the Body.* London, Bailliere, Tindall and Cox, 1932.
28. Boyd, W.: Op. cit. p. 326.
29. Cella, Louis J., Jr. and Means, James A.: *Capillary Fragility and the Intercellular Cement Substances.* Marquette Med. Rev. *13:* 67, 1948.
30. Willard, J. H.; Pessel, J. F.; Hundley, J. W.; and Bockus, H. L.: *Prognosis of Ulcerative Colitis.* J. A. M. A. *111:* 2078, 1938.

31. Bassler, Anthony: *The Etiology and Treatment of Ulcerative Colitis (non-specific).* Am. J. Dig. Dis. *16:* 275, 1949.
32. Pennington, Geoffrey A.: *Ulcerative Colitis: Clinical Features, Diagnosis and Treatment.* Med. J. Australia *1:* 405, 1949.
33. Bercovitz, Z.: *Recent Advances in the Treatment of Chronic Ulcerative Colitis.* Med. Clin. of N. Am. *24:* 683 (May) 1940.
34. Armentano, L.; Bentsath, A.; Beres, T.; Rusznyak, I.; and Szent Gyorgyi, A.: *Ueber den Einflusz von Substanzen der Flavongruppe auf die Permeabilitat der Kapillaren-Vitamin P.* Deut. med. Wochschr. *62:* 1326, 1936.
35. Maratka, Z. and Ivy, A. C.: *The Inhibition of the Shwartzman Phenomenon by Antihistamine and Vitamin P-like Substances.* Gastroenterology 11: 357, 1948
36. Manwaring, Wilfred H.; Chilcote, R. C.; and Hosepian, V. M.: *Capillary Permeability in Anaphylaxis.* J. A. M. A. *80:* 303, 1923.
37. Beiler, J. M. and Martin, G. J.: *Inhibitory Action of Vitamin P Compounds on Hyaluronidase.* J. Biol. Chem. *171:* 507, 1947.
38. Zacho, C. E.: Cited from Zacho, C. E. *The Influence of Ascorbic Acid and of Citrin on the Capillary Resistance of Guinea Pigs.* Acta Path. & Microbiol. Scand. *16:* 144, 1939.
39. Vacek, Vladimir: *Neuere Erkenntnisse ueber Vitamin P.* Schweiz. Med. Wochschr. *71:* 155, 1941.
40. Kushlan, S. D.: *Gastrointestinal Bleeding in Hereditary Hemorrhagic Telangiectasia.* Gastroenterology *7:* 199, 1946.
41. Maratka, Z.: *Traitement de la Colite Ulcereuse par la Citrine.* Gastroenterologia *72:* 372, 1947.
42. Norman, N. Philip: *A Lactose or Dextrine Laden Diet, Bacillus Acidophilus Implantation and Clinical Results.* Med. Times *49:* 126, 1921.
43. —— and Eggston, Andrew A.: *Intestinal Infections and Toxemias and their Biological Treatment.* N. Y. Med. J. *116:* 623, 1922.
44. —— and Slicher, Anna M.: *A Practical and Inexpensive "Screentest" for Cancer.* Am. J. Dig. Dis. *17:* 31, 1950.
45. Lerner, Henry H. and Rapaport, Howard G.: *Bio-photometric Studies in 30 Cases of Chronic Ulcerative Colitis.* Am. J. Dig. Dis. *4:* 239, 1939.
46. Kimble, Marian S. and Corden, Edgar S.: *The Importance of Riboflavin and Ascorbic Acid in the Utilization of Vitamin A.* J. Biol. Chem. *128:* 52, 1939.
47. Mellanby, Sir Edward: *Diet and Canine Hysteria.* Brit. Med. J. *2:* 885, 1946.
48. Dees, Susan C. and Lowenbach, Hans: *The Electroencephalograms of Allergic Children.* Am. J. Allergy *6:* 99, 1948.
49. New Gould Medical Dictionary: First Edition. Philadelphia, Blakiston Co., 1949, p. 945.
50. Pottenger, Francis M., Jr.: *The Effect of Heat Processed Foods and Metabolized Vitamin D Milk on the Dentofacial Structures of Experimental Animals.* Am. J. Orthodontics and Oral Surgery *32:* 467, 1946.
51. Melnick, D. and Oser, B. L.: *The Influence of Heat Processing on the Function and Nutritive Property of Proteins.* Food Technology *3:* 57, 1949.
52. Consumer's Guide: *Help Yourself to Cheese.* U. S. Dept. Agr. Washington, D. C. VIII: No. 7, 15, 1942.
53. Andresen, Albert F. R.: *Ulcerative Colitis — An Allergic Phenomenon.* Am. J. Dig. Dis. *9:* 91, 1942.
54. Dunbar, Paul B.: The New York Times (U.P.) August 20, 1949.
55. Rorty, James and Norman, N. Philip: *Tomorrow's Food.* New York. Prentice-Hall, Inc., 1947.
56. Norman, N. Philip and Rorty, James: *Our "Civilized" Food Habits.* The Antioch Review *4:* 434, 1944.
57. Pavlov, I. P.: *The Work of the Digestive Glands.* 2d English Ed. London, Charles Griffin & Co., Ltd., 1910, pp. 23-47.
58. McCarrison, Sir Robert: *Studies in Deficiency Diseases.* London. Henry Frowde & Hodder & Stoughton. 1921. Reprinted by the Lee Foundation for Nutritional Research, Milwaukee, Wis. 1945.
59. Norman, N. Philip: *Food Combinations: An Original Scheme of Eating Based Upon the Newer Knowledge of Nutrition and Digestion.* J. Med. Soc. N. J. *21:* 375, 1924.
60. ———: *Food Combinations: The Practical Application of the Newer Knowledge of Nutrition to the Physiological Limitations of the Digestive Processes.* Inter. Clinics *31: Ser.* 35, 233, 1925.
61. ———: *Dietetics and Treatment of Mucous Colitis.* Am. J. Electrotherapeutics and Radiology *41:* 120, 1923.

APPENDIX 1

Constructive Meal Planning

Instructions for Each Meal

1. At the FRUIT AND MILK MEAL (Breakfast) omit all breakfast foods, breakfast cereals, all kinds of bread (toasted or untoasted), buns, muffins, rolls, doughnuts, coffee rings, griddle cakes, cakes, cookies, and other starches. Also omit table sugar and other food products containing a high percentage of sugar as jellies, jams, marmalades, and commercial syrups. Also omit eggs, ham, bacon, sausage, steaks, chipped meats, fowl, fish, cheese and other concentrated proteins.

2. At the STARCH MEAL (Lunch) omit meats, fish, shellfish, eggs, fowl, cheese, nuts, leguminous vegetables, fruits (except plantains), dried fruits, melons, berries; also ice cream, cake, pie, pastry, pudding, tarts, custard, gruel, candy, bottled sweet beverages and after dinner mints.

3. At the PROTEIN MEAL (Dinner) omit fats and starches; i.e.; butter, cream, milk, cocoa, chocolate, oily dressings, gravies, oils, also all kinds of breads and crackers, potatoes, waffles, rolls, pancakes, pies, tarts, all kinds of cakes or cookies, candies, honey, syrups, sugar, and after dinner mints.

4. If desired, these meals may be interchanged — the protein meal may be eaten at mid-day and the starch meal at six or seven P.M. The average person should adhere to the above order of meals.

BREAKFAST

BREAKFAST: This is the FRUIT AND MILK MEAL.

Choose from FOOD GROUPS 1, 2, 3, and 4.

Note if food group is OPTIONAL OR OBLIGATORY. If overweight, OMIT OPTIONAL GROUPS.

GROUP 1: OBLIGATORY — Choice of One or Several.

Unpeeled apples	Ripe bananas	Oranges	Grapefruit
Grapes, all kinds	Fresh cherries	Tangerines	Mangoes
Fresh pineapple	Fresh peaches	Persimmons	Prickly Pear
Fresh pears	Papayas	Nectarines	Limes, lemons
Fresh figs	Fresh apricots	Tangelos	Plums
Sapodilla	Sweetsop	Jujubes	Kumquats
Rose apple	Pitanga	Pomegranate	Litchi

MELONS
- Cantaloupe
- Cranshaw
- Casaba
- Honeyball
- Honeydew
- Montreal
- Musk
- Persian
- Rocky Ford
- Watermelon

BERRIES
- Blackberries
- Blueberries
- Elderberries
- Gooseberries
- Huckleberries
- Juneberries
- Loganberries
- Mulberries
- Raspberries
- Strawberries

American dates (California, Nevada, Etc.)

Unsulphured, sun-dried apples

Unsulphured, sun-dried raisins

Unsulphured, sun-dried peaches

Unsulphured, sun-dried apricots

Unsulphured, sun-dried domestic figs

Note: If an orange or grapefruit is selected, peel and eat pulp and all. If the juice is squeezed out of the fruit, do not strain; sip slowly when drinking. The best plan is to take small amounts of several fresh fruits, melons, berries and dried fruits and make a fruit cocktail. Melons, dried fruits, and ripe bananas supply the natural sugars for energy and sweetening. Cooked or canned fruits should not be used except in an emergency. Buy the "brand" of canned fruit which is unsweetened. It is unnecessary to cook dried fruits.

GROUP 2: OPTIONAL — CHOICE OF ONE.

Top milk — Cream — Unsweetened whipped cream — Sour cream.

GROUP 3: OBLIGATORY.

½ to one pint of milk or buttermilk, or fermented milk (Bulgarian or acidophilus).

GROUP 4: OPTIONAL.

1 cup *unsweetened* coffee, with or without cream or milk.

Note: OMIT prunes and cranberries.

Illustrative Menus — Breakfast

Note: For special instructions for breakfast regarding what to omit at this meal refer to page 100.

2 slices pineapple
1 sliced pear
2 sliced peaches
½ cup dates or raisins
Pint of milk

1 apple, sliced
1 ripe banana, sliced
8 dates
Top milk on fruit
Pint of milk

1 honeyball melon
1 or 2 oranges or apples
1 pint of milk or buttermilk

1 or 2 oranges
1 or 2 apples
1 pint of milk or buttermilk

1 orange
1 ripe banana
1 bunch grapes
Pint of milk or buttermilk

Slice 1 apple and 1 orange
Cup of raisins
Cream or top milk
Pint of milk or buttermilk

2 sliced oranges
1 cup blackberries
1 sliced apple
½ cup raisins, dates or figs
Make fruit cocktail; add cream
Pint of milk

1 sliced grapefruit
Cup strawberries
Cup of raisins or dates
Sour cream or whipped cream on fruit
Pint of milk or buttermilk

2 oranges
3 apricots
1 apple
Half cup raisins
Bottle of acidophilus milk

1 cup strawberries
1 or 2 ripe bananas
1 cup blueberries
Cream or sour cream
Glass milk or fermented milk

2 oranges or 1 grapefruit
Pint of milk

6 sliced peaches with cream
Pint of milk

2 slices fresh pineapple
⅛ honeydew melon
½ cup fresh figs
8 pitted dates
Make fruit cocktail; add cream
Top milk or sour cream

Cup of huckleberries
Slice of canteloupe
2 sliced peaches
½ cup raisins
Whipped cream on fruit
Pint of milk or acidophilus milk

2 ripe bananas
2 peaches
1 persimmon
1 pint of milk or buttermilk

1 or two apples
Cup of raisins or 10 dates
1 pint of milk or buttermilk

½ honeydew melon
1 or 2 apples
1 pint of milk or buttermilk

1 bunch of grapes
1 pear
Cup of raisins or 10 dates
1 pint of milk or buttermilk

2 apples
Pint of milk

1 orange
2 or 3 peaches
1 bunch grapes
1 pint of milk or buttermilk

1 or 2 raw apples
1 ripe banana
1 pint of milk or buttermilk

½ honeydew melon
Whipped cream
Pint of milk

Slice 3 peaches and 1 apple
2 slices of fresh pineapple
6 sun-dried figs
Cream or sour cream on fruit
Pint of milk or buttermilk

1 mango
1 pint of milk

Note: If desired, coffee or tea with cream or milk, without sugar, may be added to any breakfast.

LUNCH

LUNCH: This is the STARCH MEAL.

Choose from FOOD GROUPS 1, 2, 3, 4, 5, 6 and 7. Note if food group is OPTIONAL OR OBLIGATORY. If overweight, OMIT OPTIONAL GROUPS.

GROUP 1: OBLIGATORY — SERVE AS FIRST COURSE.

Choose one or several (preferably several) of the following salad foods: Alligator pear (avocado), bean sprouts, raw beets, borage, broccoli, raw carrots, cauliflower head, celery, Italian celery (finnochi), Chinese celery, chicory, chives, raw cabbage (all kinds), cucumbers, dandelion greens, endive, escarole, fennel, field salad, garden cress, green peppers, lettuce, mushrooms, young mustard greens, onions (all kinds), parsley, pepper grass, purslane, radishes and tops, romaine, scallions and tops, shallots, sorrel, sour grass, raw spinach, spring kale, tomatoes, watercress.

GROUP 2: OPTIONAL.

Vegetable soup made of fresh vegetables — select vegetables from Groups 1 and 5. At the starch meal, potatoes, whole corn meal, whole wheat cereals, whole barley, rye, corn or rice may be used to thicken soups. At this meal do not add proteins to soups — meat, meat stock, eggs, etc.

GROUP 3: OBLIGATORY — STARCHES: CHOICE OF ONE OR TWO.

Baked Irish or sweet potatoes, yams, yautias, plantains (red bananas).

Hashed brown potatoes (with or without onions).

Fry in deep fat Irish or sweet potatoes, yams, yautias or plantains.

Whole wheat bread, muffins, or biscuits.

Corn on the cob; whole corn meal bread, or breadsticks made without eggs; pumpernickel.

Whole cracked wheat, graham, or rye bread.

Wild rice, or unpolished (brown) rice (double boiler cooking).

Buckwheat, whole wheat, or whole corn meal cakes (no sausage, ham or eggs).

Whole grain wheat, barley, millet, rye, oats (soak and cook in double boiler).

Whole wheat or rye spaghetti (without meat sauce).

Whole corn grits or lye hominy.

GROUP 4: OBLIGATORY — FATS.

1 or 2 squares of sweet butter, or occasionally 3-4 slices of bacon.

GROUP 5: OPTIONAL.

Choice of one or several cooked vegetables: French artichokes, Jerusalem artichokes, asparagus, arucole, fresh green beans or peas, bean sprouts, string beans, beets and tops, broccoli and tops, Brussel sprouts, cabbage (all kinds), carrots and tops, cashaw, cauliflower leaves and head, celery, celery root, chicory, chick peas, chickweed, Chinese celery, celeriac, chives, collards, cowslip, cucumbers, dandelion greens, eggplant (do not peel), escarole, garden cress, kale, sea kale, kohlrabi, lettuce, leeks, mushrooms, mustard greens, okra, onions (all kinds), orach, parsnips, green field peas, fresh garden peas, green peppers, palmetto cabbage, pigweed, poke sprouts, pumpkin, quinoa, romaine, rutabaga and tops, salsify (oyster plant), black salsify, sauerkraut, squash (all kinds), scallions and tops, shallots and tops, sorrel, sour dock, spinach, Swiss chard, tomatoes, turnips and tops, vegetable marrow.

GROUP 6: OPTIONAL.

Honey, PURE maple syrup, or unadulterated cane or sorghum molasses.

GROUP 7: OPTIONAL — BEVERAGES.

Choice of milk, buttermilk, fermented milk (acidophilus, Bulgarian), cocoa or chocolate made of old fashioned cocoa or chocolate and milk and sweetened with honey or brown sugar.

Illustrative Menus — Lunch

Note: For special instructions for lunch regarding what to omit at this meal refer to page [illegible] 118.

Fresh vegetable soup
Whole wheat bread
Butter
Baked potato
Boiled cabbage
Lettuce and tomato salad
Glass of milk

Fresh vegetable soup
Graham bread
Butter
Steamed rice and bran (mushroom sauce)
Combination salad
Cocoa made with milk

Steamed rice, maranara sauce
Chow mein — no meat, fowl, noodles
Glass of milk

Boiled whole grain wheat
Butter
Combination salad
Milk or cocoa

Cream of asparagus soup
Whole corn muffins
Mixed vegetable salad
Butter
Pint of milk

2 or 3 slices whole rye bread
Butter
Honey
Combination salad
Milk or cocoa

Creamed potatoes
Butter
Spinach or garden cress
Celery and radish salad
Graham bread
Glass of milk

Celery, radishes and raw mushrooms
Baked Idaho potato
Butter
Escaloped egg plant
Glass of milk

Hashed brown potatoes
Butter
String beans or asparagus
Lettuce and tomato salad
1 slice whole wheat bread
Honey
Glass milk or acidophilus milk

Cream of tomato soup
Graham crackers
Butter
Steamed rice and bran
Stewed okra and tomatoes
Cole slaw salad
Glass milk or buttermilk

Whole wheat bread or muffins
Butter
Turnip tops
Broccoli
Cole slaw salad
Cocoa made with milk

2 or 3 whole corn muffins
Honey
Butter
Steamed spinach
Steamed turnips
Escarole salad
Glass of milk or cocoa

Cream of tomato soup
Whole wheat bread
Butter
Baked sweet potato
String beans
Cole slaw salad
Glass of milk

Fresh vegetable soup
Butter
Candied sweet potatoes
Broccoli or kale
Garden cress and field grass salad
Glass milk or buttermilk

Cream of asparagus
Baked sweet potato
Butter
Cauliflower or cabbage
Sliced tomatoes
Glass milk or buttermilk

Whole wheat bread or muffins
Steamed kale or cabbage
Steamed turnips
Sliced tomatoes and chicory salad
Iced or hot cocoa
Honey

Celery, ripe olives, cole slaw
Corn bread sticks
Butter
Endive and radish salad
Glass milk or buttermilk

Celery and scallions
Steamed rice and butter
Vegetable casserole
Romaine salad
Glass of milk or buttermilk

Whole wheat bread
Butter
Combination salad
Milk or cocoa

Steamed rice
Chop suey without meat or fowl
Glass of milk

DINNER

DINNER: This is the PROTEIN MEAL.

Choose from FOOD GROUPS 1, 2, 3, 4, 5 and 6. Note if Food Group is OPTIONAL OR OBLIGATORY. If overweight OMIT OPTIONAL GROUPS.

GROUP 1: OBLIGATORY — SERVE AS FIRST COURSE.

Large combination salad as served at lunch. See GROUP 1 on LUNCH MENU, page 108.

GROUP 2: OPTIONAL.

Fresh vegetable soup which may or may not contain fresh meat, meat stock, fish, shellfish or fowl. Use several kinds of fresh vegetables. Flavor with chives, onions, scallions and tops, mushrooms, tomato or tomato paste, garlic, bayleaf, basil, thyme, Savita, Vegex, leek, parsley, paprika, chervil, sage. No crackers, bread, rolls, breadsticks, or other starches with soups at the protein meal.

GROUP 3: OBLIGATORY — CHOICE OF ONE FROM EITHER A, B, or C.

(A) FLESH PROTEINS.

BROILED — Steaks, all chops (except pork), chicken, any fresh fish (no canned, smoked, or dried fish), kidneys, hearts, liver, venison, game birds, squabs, country sausage (occasionally), lobster, oysters, crabs, sweetbreads, fish roe, brains, shrimp.

FRIED IN DEEP FAT — Chicken, fish, frogs' legs, stuffed crabs, shrimps, oysters, scallops.

ROAST — Beef, lamb, mutton, veal, lean pork, venison, duck, goose, chicken, turkey, guinea hen, pheasant, rabbit. Pork and rabbit should be well cooked.

BAKED — Oysters, clams, fish, ham.

RAW — Oysters, clams.

BOILED — Shrimp, crabs, crawfish, lobster (no rich dressings), turtle, tongue, tripe.

STEAMED — Clams.

EN CASSEROLE — Any meat which lends itself to "stewing" with chopped vegetables. Flavor with scallion tops, mushrooms, garlic, chives, parsley, dill, okra, leek, tomato, tomato paste, bayleaf, thyme, basil, Savita, Vegex.

EGGS — Poached, boiled or scrambled — no bread or other starches. If desired, scramble eggs with chopped onions, mushrooms and parsley.

(B) NON-FLESH PROTEINS.

CHEESE — Cottage cheese, cream cheese, or American "store" cheese — excellent and economical substitutes for flesh proteins; or Swiss cheese, the hard Italian cheeses, liederkranz, camembert, roquefort, Swiss gruyere or brie.

NUTS — Pecans, walnuts, Brazil nuts, almonds, peanut butter.

Cheese or nuts may be served on salad.

(C) LEGUMES.

Lentils, soy beans, cranberry beans, navy beans, kidney beans, red beans, Mexican beans, Boston beans, frijoles, blackeyed peas, cow peas, dried wax beans, chiches, lima beans, pinto beans.

GROUP 4: OBLIGATORY.

Two or more cooked vegetables; see Group 5 on Lunch Menu, page 109.

GROUP 5: OPTIONAL — DESSERT.

Choice of one or several of the following fresh fruits: Grapes, orange, grapefruit, tangerine, nectarine, peach, persimmon, cherry, mango, papaya, apple, pomegranate, pineapple, tangelo; also any berry or melon. Do not add cream, milk, or sugar.

GROUP 6: OPTIONAL — BEVERAGES.

Tea, or coffee; without cream, milk, or sugar.

Illustrative Menus — Dinner

Note: For special instructions for dinner regarding what to omit at this meal refer to page 118.

Broiled sirloin steak, mushrooms or onions if desired
2 or 3 vegetables — carrots, string beans and grilled egg plant
Large salad of romaine and tomatoes
Large fruit cocktail without cream or sugar for dessert

Celery, radishes and ripe olives
Scrambled, poached or boiled eggs
Asparagus, cauliflower and carrots
Garden cress or pepper grass salad
Fresh fruit cocktail, without cream or sugar

Broiled chicken, broiled lobster or broiled lamb chops
2 or more fresh vegetables as spinach, cauliflower and garden cress
Salad of mixed greens
Sliced fresh pineapple without cream or sugar for dessert

Boston baked beans, baked lima beans or lentils
String beans, red cabbage and beets
Celery, radish, tomato and romaine salad
(No fruit with a legume)

Fresh vegetable soup
A large combination salad made up of onions, radishes, cucumbers, tomatoes, celery, raw carrots and upon which from ¼ to ½ pound of grated American cheese is sprinkled
2 raw apples with skins

Clam juice cocktail
1 dozen clams or oysters
Combination salad of celery, raw carrots, onions, tomato and lettuce
Fruit cocktail of fresh fruits without cream or sugar

Fresh fruit cocktail or casaba, or Persian melon
Broiled flounder, white fish or blue fish, tomato sauce
Grilled egg plant, cauliflower and spinach
Romaine and celery salad
Watermelon or honeydew melon

Celery, ripe olives and tomatoes
Broiled flounder, tomato sauce
String beans
Grilled egg plant
Baked squash
Salad of mixed greens
Raw apple

Celery and radishes
1 dozen walnuts, crushed and added to Combination Salad
Steamed kale
Combination salad
Fruit cocktail, no sugar or cream

Celery and radishes
Chicken broth (no crackers, breadsticks)
Chicken or beef chow mein, no rice or noodles
Sliced tomatoes
1 or 2 raw apples or oranges

Celery and coleslaw
Fresh fruit cocktail
Vegetable soup (no crackers or bread)
Broiled steak or lamb chops
Broccoli and asparagus
Romaine and tomato salad
Watermelon or honeydew melon

Vegetable soup
Combination salad of coleslaw, celery, onion, tomato, endive, and raw carrots with ¼ lb. of grated Swiss cheese
Fruit cocktail without cream or sugar

Celery, coleslaw or scallions
Fresh fruit cocktail
Roast duck without stuffing
String beans
Fresh green garden peas
Pepper grass salad
California grapes

Baked ham (not sugar cured)
Spinach
Broccoli
Beets
Salad of mixed greens
Fruit cocktail, no sugar or cream

Celery, ripe olives and scallions
Fresh vegetable soup (no breadsticks, etc.)
2 or 3 poached eggs, no toast
Asparagus and broccoli
Tomato and endive salad
Half grapefruit, no sugar

Fresh vegetable soup (no crackers or bread)
Celery and ripe olives
Combination salad with 15 pecans
Steamed kale
Steamed carrots and tops
Sliced oranges

Celery and radishes
Fresh fruit cocktail
Broiled squabs
Baked squash
Grilled or escaloped egg plant
Romaine and tomato salad
Sliced fresh pineapple, no sugar or cream

Finnochi and coleslaw
Tomato omelet (2 or 3 eggs)
Baked squash
Steamed onions
Romaine salad
Fresh fruit cocktail

Fresh vegetable soup (no crackers or bread)
Celery, coleslaw and pepper grass
Roast beef or lamb
Steamed spinach
Steamed okra
Sliced tomatoes
Concord grapes

Savita broth
Celery, coleslaw and ripe olives
Chicken or beef chow mein, no rice or noodles
Romaine salad
Fresh fruit cocktail, no cream or sugar

Eating Between Meals and at Bedtime

Eating Between Meals and at Bedtime

It is desirable for very thin persons to eat more than three meals a day. The underweight person can increase his food intake by including the *optional groups of foods* to be found on the breakfast, lunch and dinner menus. Additional meals of fruit and milk, say at 10:30 A.M., 4:30 P.M., and at bedtime are permissible. If one does not desire fruit, then any whole grain bread, or cereal with milk or cream, and a glass of milk or buttermilk is allowed.

Stout persons should omit all the optional groups of foods and also refrain from eating between meals and before retiring.

APPENDIX II

Case I. First consulted by this 35 year old female on September 23, 1920. The past history was insignificant. The psychosomatic factor involved the care of an aged parent who was a custodial problem. For the past six months chief complaints consisted of severe headaches, mental depression, loss of weight, dizziness, nausea, and eight or more bloody stools per day with tormina and tenesmus. X-ray diagnosis — chronic idiopathic ulcerative colitis. Noteworthy physical findings were a —30 per cent basal metabolic rate, abdominal distention, and a moderately severe secondary normocytic anemia. The sigmoidoscopic examination revealed a granular, hemotumescent rectal and sigmoidal mucous membrane with shallow ulcerations. The intestinal flora was of the putrefactive type with many *B. aerogenes capsulatus,* plaques of disintegrating colonic epithelium, red blood cells, pus cells, and a marked diminution of *B. coli.* Her diarrhea responded to Sun's cholera mixture and a dietary composed mainly of natural foods fortified with three oranges and one lemon daily. Thyroid extract, gr. ss b.i.d., was prescribed for the low BMR and hematinic medication for the anemia.

Within two weeks her stools were less frequent and free of macroscopic blood. Tormina and tenesmus had almost disappeared. Microscopic examination of the stool revealed that the mucous membrane was still bleeding. Sigmoidoscopic examination showed that the shallow ulcerations had almost completely disappeared but hemotumescent areas of mucous membrane were still present and oozing blood.

Six weeks later, re-examination of her stools revealed a marked reduction of red blood cells and pus cells; plaques of disintegrating epithelium had almost disappeared and the fecal flora was greatly improved. Her stools averaged four or five per day. Occasionally she suffered a transient exacerbation which responded promptly to the diarrhea mixture. Three months later the ulcerations had completely disappeared, and the colonic mucous membrane appeared

normal. The hematologic picture was normal. Colonic irrigations were administered every two weeks. Within a year she was free of complaints. The BMR remained slightly on the low side, averaging −12.

She has been employed constantly since January 1923, and recently celebrated her 66th birthday, hale and hearty.

Case II. First consulted by this 35 year old female on November 23, 1920. Briefly, the history was of recurrent attacks of bloody diarrhea over a six year period. Otherwise, the past history was negative. Patient was a high-strung, over-conscientious individual. When first seen, she was bedridden and marasmic (weighed 85 pounds); she was unable to stand; had a bed sore; repeated blood counts revealed a severe secondary anemia. She was moribund, slightly delirious, and for weeks her P.M. temperature had ranged between 100°-102° F. She was having twelve to eighteen spontaneous bloody stools per day. Upon examination the abdomen was distended and tympanitic; the left half of the colon was extremely tender and palpation revealed a thickened colon. Prolapsed, thrombotic hemorrhoids prevented rectal or sigmoidoscopic examination. The stool examination revealed large numbers of red blood cells, pus cells, plaques of disintegrating colonic epithelium, *streptococci, staphylococci, gas bacilli,* and a marked diminution of *B. coli* and mucous. For the first week she could not tolerate solid food and her meals consisted of acidophilous milk (fortified with lactose), three oranges and one lemon per day. She improved steadily and within two weeks was able to eat solid food. A month later on a dietary of natural foods, she was able to walk, feed herself, and help prepare her meals. Medication consisted of a combination of paregoric, albumin tannate, and bismuth subgallate; peroral anti-anemic therapy; and rectal suppositories. Six weeks later she was able to make the 75 mile trip to my office for an examination. She had gained twelve pounds and felt considerably improved. The physical ex-

amination was negative except for the colitis. The hematologic picture was improved (4,100,000 red blood cells, Hb 74 per cent). Urinalysis was negative. Stool examination revealed that the colonic mucous membrane was still oozing; there was a marked diminution of pyogenic microorganisms and a noteworthy decrease of exfoliating plaques of epithelium. Sigmoidoscopic examination showed the ulcerations and the hemotumescent areas of mucous membranes to be healing. Because of the extensive amount of tubulation, the number of stools averaged between six and eight per day. She again consulted me three months later. At this time she had gained 25 pounds and looked well. The hematologic status was within normal limits. No ulcers could be found upon sigmoidoscopic examination and the hemotumescent areas had disappeared. The colonic mucous membrane was granular and congested, and there were extensive areas of scarification. All medication, except the concordant dietary fortified with three oranges and one lemon per day, was discontinued.

She made a complete recovery. However, over seven years were required for the colon to compensate completely for the extensive amount of tubulation and scarification which attended the disease. For the past 20 years she has averaged three normal bowel movements per day. She has remained well since the latter part of 1922, and at the age of 64 holds a responsible full-time position.

Case III. First consulted by this 35 year old male on May 1, 1929. He was a baseball player who subsisted mainly on hotel and restaurant fare and the excessive consumption of hamburgers, frankfurters and the sweet rolls between which these delicacies are sandwiched; large quantities of sweet beverages and candy bars were a staple in his diet. In 1925, he began to have attacks of diarrhea, usually lasting a few days and recurring every two or three weeks. These attacks became more frequent, severe and prolonged. He became acutely ill on April 1, 1929, with eight or more

bloody stools per day and severe tormina and tenesmus. He consulted a competent gastro-enterologist who made a diagnosis of idiopathic ulcerative colitis; a few days later the diagnosis was changed because *B. tuberculosis* had been isolated from a stool culture. Because of this he was advised to go to a tuberculosis sanatorium for a complete rest and a "super" dietary. While on his way he visited a friend whom I was treating for duodenal ulcer. This friend insisted that he consult me. He did so — reluctantly. Physical examination was negative, except for a 20 pound weight loss, weakness, and his feeling of being nervous and jittery. The sigmoidoscopic examination revealed a typical picture of idiopathic ulcerative colitis. This picture was distinct and the morphological differentiation between idiopathic ulcerative colitis and tuberculous ulceration of the colon was clear-cut. Repeated stool examinations were negative for *B. tuberculosis.* The stools contained many red blood cells, pus cells, disintegrating plaques of colonic epithelium, a paucity of mucous, and excessive numbers of pyogenic microorganisms. The red blood cell count was 4,200,000, hemoglobin 79%, white blood cell count 9,800; a normal differential except for an acidophilia of 9%. He decided to undergo treatment for a trial period of three weeks.

After two weeks on a concordant dietary of natural foods, hematinic medication, and a diarrheal mixture containing paregoric, bismuth subgallate and albumin tannate, the number of stools decreased to four or five per day and macroscopic blood disappeared. One month later the stool examination was satisfactory; the sigmoidoscopic examination revealed a disappearance of ulcerations and an almost complete disappearance of the hemotumescent areas of mucous membrane. The hematology was normal — 2% acidophiles. Three months later he was considered cured and rejoined his baseball club. He had a recurrence of diarrhea (non-bloody) in 1930 and another in the latter part of 1931. This convinced him that he should perma-

nently renounce the mixed meal with its processed food content to achieve a complete cure. Since that time he has enjoyed excellent health and now owns and operates a large metropolitan restaurant.

Case IV. First consulted by this 33 year old male on July 19, 1929. His chief complaints were lassitude, a 15 pound weight loss, poor appetite, and a rectal fissure that failed to heal because of frequency of bowel movements (eight to ten bloody stools daily with tormina and tenesmus). He had a hemorrhoidectomy in 1924, an appendectomy in 1927, and had been suffering from recurrent attacks of bloody diarrhea for the past four years. The psychosomatic factor was related to an occupational maladjustment since he was dissatisfied with the brokerage business. A change in work, as soon as possible, was recommended. In 1931, he had an opportunity to effect a change, with subsequent good occupational adjustment.

Noteworthy findings consisted of a fissure in the posterior commissure of the anus; the sigmoidoscopic picture was typical of idiopathic ulcerative colitis; stool examination revealed a preponderance of *streptococci, staphylococci,* and *B. aerogenes capsulatus;* the quantity of mucous was decreased; exfoliative plaques of colonic epithelium and a profusion of red blood cells and pus cells, plus a secondary normocytic anemia were found. He was placed upon a constructive scheme of meal planning, as outlined in Appendix I, peroral hematinic therapy, and Sun's cholera mixture with opium. Within two weeks the macroscopic blood had disappeared from his stools. Stool examination revealed a marked decrease in the number of pyogenic bacteria, plaques of colonic epithelium, red blood cells, and pus cells. The amount of mucous was increased. Six weeks later the sigmoidoscopic picture was greatly improved, and the anal fissure had healed — the colonic ulcerations had healed and there was an almost complete disappearance of hemotumescent areas of mucous membrane. Stool examina-

tion also revealed that the number of red blood cells and pus cells had greatly diminished. The hematologic status was almost normal. Colonic irrigations were administered every two weeks for a few months and thereafter only once a month. Within 16 months he was considered cured. He has not had a bloody stool since that time, although upon occasion, in recent years, he has not been too careful of his dietary and beverage habits.

Case V. First consulted by this 53 year old male on July 10, 1941. Chief complaints were bloody diarrhea, 10 to 12 stools per day (2 to 3 spontaneous ones) with tormina and tenesmus; pain in the left lower quadrant; excessive amounts of intestinal gas; loss of 12 pounds in weight; weakness and lassitude. Seven years ago, after a picnic, he had an attack of diarrhea which lasted for two weeks. At that time he was hospitalized for four weeks. His illness was attributed to "food poisoning." He partially recovered, but continued to have attacks of diarrhea, sometimes blood tinged, and three months later he had an acute recurrence of bloody diarrhea. He was given large doses of a sulfa drug which temporarily checked the diarrhea. Shortly thereafter, he developed shingles which involved the left sixth, seventh, and eighth intercostal areas. After the onset of the shingles the bloody diarrhea recurred in an intensified form. He also began to lose his hair and was considerably disturbed about this circumstance.

Noteworthy findings were the following — weight was 188 pounds (his usual weight was 200 pounds); height 70¼", heart and lungs were negative; blood pressure 155/85; retinoscopic examination revealed a 1 plus arterial change in the retinal arteries; the abdomen was distended with tenderness and slight rigidity over the left lower quadrant. Gait, station, and reflexes were normal. Rectal examination revealed a four plus anal spasticity with hemorrhoids. Sigmoidoscopic examination revealed a granular, hemotumescent colonic mucous membrane with several

shallow ulcers; capillary fragility and permeability could be readily demonstrated. Stool examination showed a large number of red blood cells, pus cells, plaques of disintegrating colonic epithelium; numerous pyogenic organisms — *streptococci, staphylococci,* and *gas bacilli.* There also seemed to be a reduction in the number of *B. coli.* Hematologic examination — red blood cells 4,300,000; hemoglobin in grams 10.49 (76%); color index .88; volume index 1.1; saturation index .76; icterus index 4 units; reticulocyte count .6%; sedimentation rate 32 mm. in one hour; platelet count 275,000; prothrombin time 32 seconds; hematocrit 42%; percent cell volume 100; white blood cell count 5,090; differential was normal except for a 6% acidocyte count.

Treatment consisted of constructive meal planning; a prescription for liver concentrate, extract of bone marrow and the total B complex; vitamin K (100 mgs. daily); and a natural vitamin C concentrate.

On September 24, 1941, sigmoidoscopic examination revealed a disappearance of the hemotumescent areas and ulcerations, and only a moderate amount of congestion of the colonic mucosa. He felt greatly improved; the number of stools had been reduced to three or four per day and he had full control of bowel movements. The dose of vitamin K was reduced to 60 mg. per day. On November 4, 1941, he returned for re-examination, complaining of occasional discomfort in the lower left quadrant. The sigmoidoscopic was negative as was the stool examination. The hematologic picture was normal; the prothrombin time was 21 seconds; platelet count 385,000; sedimentation rate 12 mm. in one hour. On December 18, 1941, the sigmoidoscopic was normal; the number of stools averaged two or three per day; the hematologic picture was normal; reticulocyte count 1.4%; the sedimentation rate was 8 mm. in one hour; the prothrombin time was 18 seconds and the platelet count 395,000. Vitamin K was discontinued, as was the liver

concentrate and extract of bone marrow and the B complex. He was to continue the Vitamin C and to take a supplement of natural vitamins known as catalyns. Examinations on March 6, and March 20, 1942, revealed that the hematologic picture was normal in all respects. The three significant findings concerned prothrombin time (18 seconds), platelet count (390,000), and the sedimentation rate (8 mm. in one hour). Sigmoidoscopic examination revealed a normal colonic mucosa with evidence of slight scarring. No bleeding could be demonstrated. The stool examination corroborated this clinical evidence. He was considered cured. He had regained his normal weight and felt well enough to undertake a new business venture which necessitated his moving to a Western city. He was an intelligent and conscientious patient who became thoroughly convinced that the cause of his illness was the huge consumption of processed foods, containing excessive quantities of the ternary elements and marked deficiencies of such nutrient factors as vitamins, minerals, roughage, etc. He also realized that the mixed meal was the perfect device for encouraging people to eat the great variety of processed foods which have become staples in our food supply.

Case VI. First consulted by this 26 year old female on July 28, 1943. Chief complaints consisted of 10 to 14 bloody bowel movements per day; loss of appetite; nervous, jittery, apprehensive, depressed and suffered from frequent crying spells. She had been suffering from bloody diarrhea for the past 10 years. These attacks lasted for one or two weeks and were followed by remissive intervals of three to four weeks. They gradually became more frequent and were accompanied by nausea, severe tormina and tenesmus. The psychoneurotic factor was precipitated by the death of her father when she was 16. The ensuing reactive depression was intensified because it became necessary for her to go to work. Unfortunately, her employer was an exacting, disagreeable, and unsympathetic person. At the age of 20 she

married. Although her marriage eliminated the economic stress, she became progressively worse, and on two occasions had to be hospitalized. During her hospitalizations she received large doses of sulfa drugs, with a resulting adverse effect upon her colitis.

Noteworthy findings — weight 106 pounds; height 63"; heart, blood pressure and lungs negative. The abdomen was distended and tender, the transverse, descending and sigmoidal portions of the colon were tender and palpable; sigmoidoscopic examination revealed extensive hemotumescent areas of mucous membrane and the presence of several deep ulcers; capillary fragility and permeability were easily demonstrable; stool examination revealed large numbers of pyogenic bacteria, *B. aerogenes capsulatus,* and an apparent reduction of *B. coli;* there were plaques of disintegrating colonic epithelium and many red blood cells and pus cells. Hematologic status showed 4,410,000 red blood cells; hemoglobin in grams 11.04 (80%), color index .90, volume index 1.0, saturation index .73, icterus index 5.1 units, reticulocyte count .4%, hematocrit 38%, sedimentation rate 42 mm. in one hour, prothrombin time 29 seconds, 272,000 platelets and 4,600 white blood cells with a disturbed differential — 43% neutrocytes, 53% lymphocytes, 4% acidocytes. Urinalysis was essentially negative. Patient had been taking antianemic medication.

Treatment consisted of constructive meal planning, 120 mgs. of vitamin K per day, the total vitamin C complex, peroral and parenteral hematinic medication and a prescription for a powder containing:

Albumin tannate		
Bismuth subnitrate	aa	grs viiss
Powdered opium		grs ss

The number of doses per day depended on the number of bowel movements.

On October 4, 1943, she reported for examination. She felt stronger, and reported less mental depression and nerv-

ousness. Nausea was rare, and bowel movements had decreased to six or eight stools per day, some containing blood; tormina and tenesmus in the morning. Sigmoidoscopic examination revealed that the deep ulcers were healing and that the hemotumescent areas were less congested. Hematologic examination now showed 4,980,000 red blood cells, hemoglobin in grams 12.97 (94%), color index .95, volume index 1, saturation index .94, icterus index 4.1, reticulocyte count 2.4%, sedimentation rate 8 mm. in one hour, prothrombin time 21½ seconds, 365,400 platelets, and 5,750 white blood cells; differential — 52% neutrocytes, 44% lymphocytes, 2% acidocytes, 1% basocytes, 1% monocytes. Urinalysis was negative.

On November 18, 1943, she felt considerably improved, with the number of bowel movements decreased to four or five per day without macroscopic blood. Sigmoidoscopic examination disclosed that the ulcers had healed; the hemotumescent areas had almost disappeared; the oozing of blood from these areas slightly colored the swab stick. Stool examination showed a marked reduction in the number of red blood cells, pus cells, exfoliating colonic epithelium, and pyogenic microorganisms. Hematologic examination: 5,190,000 red blood cells, hemoglobin in grams 13.80 (100%), color index .98, volume index .98, saturation index 1, icterus index 4.6 units, reticulocyte count 2.3%, hematocrit 45%, sedimentation rate 8 mm. in one hour, prothrombin time 20 seconds; 435,000 platelets, 6,500 white blood cells, and a normal differential.

At this time she was advised to discontinue the hematinic medication. The anti-diarrheal powder was changed to Sun's cholera mixture; vitamin K was reduced to 40 mg. per day. Continuation of total vitamin C complex was recommended.

She returned on August 11, 1944, without any complaints. Sigmoidoscopic and stool examinations were negative; hematologic status was normal. The yardsticks — platelet count,

prothrombin time, and sedimentation rate were all normal. All medication was stopped.

Again, when she reported for examination on June 7, 1945, the sigmoidoscopic, stool and hematologic examinations were negative. The yardsticks — prothrombin time, platelet count, and sedimentation rate again were normal. She was then considered a cured case. The question arose as to whether or not she should become pregnant. She was advised to do so. She became pregnant, had a normal course and delivery, and is now the happy mother of a healthy son.

Case VII. First consulted by this 28 year old female on October 23, 1944. Chief complaints were six to ten bloody stools per day, with tormina and tenesmus, rapid pulse rate, loss of 26 pounds in weight, a feeling of exhaustion, extreme nervousness and depression. As an infant she had a chronic eczema of the face, head, and lower extremities which lasted for about a year. At the age of 13, she had an attack of bloody diarrhea subsequent to chronic constipation. She was then hospitalized for two weeks. During the following six years she had only occasional attacks of bloody diarrhea. Between 1935 and 1939, she had three severe attacks which required hospitalization. On first admission artificial fever therapy was tried with poor results; on the second she received two transfusions; during the third hospitalization she was treated in orthodox fashion. She married in 1940, became pregnant and had an acute exacerbation of symptoms lasting to the fifth month of her pregnancy. The illness suddenly subsided and she then went through a normal pregnancy. She again became pregnant in 1942, and this pregnancy followed the same pattern. She was then symptom free until May, 1944, when she began having bloody diarrhea, P.M. temperature, felt extremely nervous, had palpitation of the heart, was physically exhausted, and lost 26 pounds within four months.

Noteworthy findings: October 23, 1944 — weight 100 pounds, height 62½"; patient was physically exhausted,

nervous and highstrung. Her tongue was magenta colored, furred and fissured in the center. The thyroid was moderately enlarged and palpable. Blood pressure was 100/65, pulse rate 110, sinus rhythm; tenderness and some rigidity over the transverse, descending and sigmoid portions of the colon; internal hemorrhoids with a peri-anal dermatosis; sigmoidoscopic examination revealed a few shallow ulcers, extensive confluent hemotumescent areas of mucous membrane, bleeding actively. Stool examination — the stool was a mixture of fecal matter and blood with many red blood cells and pus cells. A large amount of exfoliating colonic epithelium was present as were numerous *streptococci, staphylococci,* and *B. aerogenous capsulatus.* Hematologic examination yielded the following results: 4,350,000 red blood cells, hemoglobin in grams 8.28 (60%), color index .70, volume index 1.1, saturation index .62, icterus index 4 units, reticulocyte count 1.2%, hematocrit 40%, 4,200 white blood cells; differential — 45% neutrocytes, 46% lymphocytes, 8% acidocytes, 1% basocytes; the prothrombin time was 28 seconds; 185,200 platelets, and a sedimentation rate of 62 mm. in one hour.

Treatment was outlined with constructive meal planning, 80 mg. of vitamin K per day, peroral hematinic medication with vitamin B, total vitamin C complex, Sun's cholera mixture — 1 teaspoonful in ¼ glass water every three hours. She returned for a routine check-up on November 14, 1944, greatly improved, partially able to resume her household duties, and complaining of feeling hungry between meals. Her bowel movements had decreased to three or four per day, with only an occasional bloody stool. She had regained complete control over her bowel movements, gained six pounds in weight, felt optimistic and acknowledged a marked decrease in her previous nervousness. Sigmoidoscopic examination revealed that the ulcerations and the hemotumescent mucous membrane had begun to heal, with much less bleeding. The hematologic examination also manifested

a steady improvement. The "yardsticks" showed considerable improvement — prothrombin time 23 seconds, 387,500 platelets, and a sedimentation rate of 38 mm. in one hour.

On December 19, 1944, when she reported for re-examination, her improvement was satisfactory to all concerned. She weighed 112 pounds, did not feel nervous, and was most optimistic about her condition. She now had two or three non-bloody movements per day. The hematologic picture also was greatly improved, with 4,520,000 red blood cells, hemoglobin in grams 11.59 (84%), color index .90, volume index 1, saturation index .90, icterus index 4.3 units, reticulocyte count 2.2%, hematocrit 40%, 5,200 white blood cells; differential — 50% neutrocytes, 43% lymphocytes, 3% acidocytes, 1% basocytes, and 2% monocytes. The "yardsticks" were satisfactory — prothrombin time 21 seconds, 380,000 platelets, and a sedimentation rate of 20 mm. in one hour. On February 5, 1945, the "yardstick" findings were — prothrombin time 19½ seconds, 380,000 platelets, and a sedimentation rate of 32 mm. in one hour. She confessed that she had been on a cake, pastry, and candy binge, but faithfully promised to resume her prescribed dietary. On April 20, 1945, she was symptomless, the hematologic picture was normal, as were the "yardsticks" — prothrombin time 18½ seconds, platelet count 370,000, sedimentation rate 13 mm. in one hour. Sigmoidoscopic and stool examinations were negative. Shortly thereafter she became pregnant and went to full term without any difficulties. On January 28, 1946, the hematologic picture was normal. The "yardsticks" were as follows: prothrombin time 18½ seconds, platelets 390,000, and sedimentation rate 10 mm. in one hour. She weighed 125 pounds and was symptom free. No signs of the disease could be detected sigmoidoscopically.

On October 18, 1949, she came in with her husband for a consultation. For several years he had suffered from a pyloric ulcer. X-ray studies revealed that a major surgical

operation was imperative for relief of his condition. Despite the anxiety and worry about his condition, she had no recurrence of symptoms and remained well — supporting the thesis that the well nourished individual can adjust better to life situations than those inadequately nourished.

Case VIII. First consulted by this 22 year old female on May 28, 1944. Chief complaints: weak, irritable; excitable; palpitation of heart on exertion with shortness of breath; morning nausea and vomiting; mental depression; crying spells; loss of 37½ pounds; rapid loss of hair; 5-10 bloody stools per day (some spontaneous); tormina and tenesmus and abdominal distention.

Past history: In August, 1943, weighed 120 pounds; became pregnant and spontaneously aborted during the tenth week. She was hospitalized for two days and curetted. Two weeks later she felt well enough to return to work. Four weeks later began having blood-tinged stools. This condition became progressively worse and on December 1, 1943, she had a severe attack of nausea and vomiting which lasted for six hours. Thereafter she felt weak and exhausted and her stools were more frequent and bloody. A diagnosis of "intestinal flu" was made (no sigmoidoscopic, proctoscopic or stool examination). The diarrhea could not be controlled and she was hospitalized in the latter part of January, 1944. A diagnosis of chronic idiopathic ulcerative colitis and inflamed hemorrhoids was made. She remained in the hospital for two weeks, took massive doses of a sulfa drug and was placed on the orthodox colitis dietary. She weighed 104 pounds. Her condition was most serious and she was transferred to a large Metropolitan hospital for better care. Treatment: three transfusions; intravenous glucose on four consecutive days; liver, iron and vitamin B orally; paregoric and large doses of sulfa drugs. Her condition became progressively worse and two weeks after admission her husband took her home. Nausea and vomiting continued; 8-10

bloody stools; progressive loss of weight, physical strength and rapid loss of hair.

Noteworthy findings of May 28, 1944: The patient had to be carried into the office; too weak to walk or stand; height 62″; weight 82½ pounds; pale, sallow color of skin; furred and fissured tongue of magenta color; almost completely bald; coarse tremor of the face, tongue and fingers; chest and lungs negative; heart had a systolic murmur over the mitral area; blood pressure 85/55; pulse rate 120; tender tympanitic abdomen; external, thrombotic hemorrhoids; exaggerated deep reflexes; impossible to do a sigmoidoscopic because of hemorrhoids and the patient's prostrated condition; temperature 100° F.

Hematologic examination: 3,800,000 red blood cells; hemoglobin in grams 9.10 (64%); color index .92; volume index 1.2; saturation index .77; icterus index 4.2 units; reticulocyte count .9%; hematocrit 39%; 7,600 white blood cells; normal differential except for a 4% acidophilia; sedimentation rate 100 mm. in one hour; prothrombin time of 28 seconds and 178,400 platelets. Treatment: Peroral liver, iron and vitamin B; constructive meal planning; vitamin C; vitamin K (80 Mg. per day) and the albumin tannate, bismuth subgallate and powdered opium prescription; anesthetic rectal ointment and suppositories. On June 13, 1944, was greatly improved; the hemorrhoidal condition was satisfactory; 4-6 bloody stools per day; sigmoidoscopic examination revealed shallow ulcerations; confluent hemotumescent areas of mucous membrane, and the stool examination was characteristic for chronic idiopathic ulcerative colitis. Medication continued.

On July 12, 1944: 3-4 non-bloody stools per day; hematologic and sigmoidoscopic picture greatly improved; same medication except to substitute Sun's cholera mixture for the original antidiarrheal prescription and to reduce vitamin K to 30 mg. per day. Prothrombin time 24 seconds; sedi-

mentation rate 80 mm. in one hour; 225,000 platelets; reticulocyte count 2.1%, hematocrit 41%.

September 20, 1944: weight 106½; new hair growing; hematologic and sigmoidoscopic examinations satisfactory. Discontinue vitamin K; reduce liver, iron and vitamin B; continue vitamin C.

On November 17, 1944: weight 110 pounds; heart normal; blood pressure 120/68; pulse rate 86; new growth of hair; felt well enough to return to work; 2-3 non-bloody bowel movements; hematologic examination; 4,985,000 red blood cells; hemoglobin in gms. 12.70 (92%); color index .93; volume index 1.0; saturation index .92; icterus index 4.4 units; reticulocyte count 2.2%; hematocrit 43%; prothrombin time 20½ seconds; sedimentation rate 35 mm. in one hour; 300,000 platelets; 6,300 white blood cells; normal differential; urinalysis negative. Stool examination negative; sigmoidoscopic negative except for occasional ulcer scar. Treatment: discontinue Sun's cholera mixture; continue vitamin C; reduce quantity of hematinic medication.

To make a long story short, her improvement continued and she became pregnant in the latter part of 1946, had a normal experience and is the happy mother of a healthy son. Her alopecia has been replaced by a thick growth of hair and she weighs 128 pounds. Her last examination was on September 24, 1948, and it was normal for the physical; basal metabolic rate; hematology; blood chemistry; E.K.G.; and sigmoidoscopic. She is considered a cured case as long as she adheres to a proper nutritional regimen.

Case IX. First consulted by this 29 year old female on May 25, 1946. Chief complaints: 6-10 bloody stools per day; morning nervousness; "shaky feeling in stomach"; headaches; mental depression; crying spells which usually preceded her irregular, painful menstrual periods; poor appetite; loss of 20 pounds in weight. Past history: At the age of 17 she had an acute attack of diarrhea while on a train bound for Chicago. The hotel physician told her that she

was suffering from amoebic dysentery, currently epidemic in Chicago. She was placed under treatment without results. One month later, upon her return to New York City, she consulted a specialist who attributed her condition to a rapid pulse rate (no basal metabolic rate, stool or sigmoidoscopic examination) and she was treated with sedatives for several months without benefit. She then consulted specialist No. 2. When he heard the history of the onset of her condition, he immediately diagnosed it as amoebic dysentery (again, no stool or sigmoidoscopic examinations) and she received an injection of a drug (perhaps emetine hydrochloride) for ten consecutive days which produced temporary blindness after the ninth injection, and she developed marked palpitation with cardiac irregularity (extra systoles). Following this unfavorable response iron injections were administered to build her up. She had 36 injections. In addition, castor oil and calomel were also prescribed. She endured this form of therapy for six months, became progressively worse and was finally referred to specialist No. 3 for diagnosis and treatment. He diagnosed her illness as idiopathic ulcerative colitis and advised hospitalization for three weeks. While hospitalized, she received massive doses of a sulfa drug in current vogue and was discharged feeling worse than when admitted. This specialist then referred her to an assistant for office treatment. This consisted of colonic irrigations and intraperitoneal injections of an anesthetic in oil to control hyperperistalsis. In addition she took a mixture of "atropine, codeine, bismuth, novatropin and calcium gluconate." Her diet was limited to strained vegetables, processed starches, stewed fruit and a limited amount of protein. These treatment measures were unsuccessful. She was then referred to specialist No. 4. After a superficial examination, he prescribed a dietary of ice cream, chocolate and cocoa sodas, malted milk drinks, and sugar-rich carbonated beverages to "fatten her." In addition "psychotherapy" was instituted. The net result of these psychotherapeutic

"sessions" was to advise either marriage or a love affair. She was advised to promote contacts with males and to enjoy the sex companionship which might ensue although she tried to explain that her social life was limited because of constant diarrhea, loss of strength and a debilitated physical state which necessitated that she obtain much rest. She soon rebelled against this pornographic "therapy" and consulted specialist No. 5 who told her that her idiopathic ulcerative colitis was caused by "nerves" and was probably incurable. She was advised to face facts and resign herself to suffering from this malady until death. His only comforting advice was that she might get some symptomatic relief from sedatives. It was at this time that she read of my success in treating a noted baseball player, who suffered from a severe form of the same disease, and decided to consult me.

The past history of this case is the interesting part of it. It is difficult to understand how these "specialists" could justify their diverse diagnostic opinions without stool and sigmoidoscopic examinations, and basal metabolism tests.

On May 25, 1946, the patient weighed 100¼ pounds; height 63½"; lungs negative; heart negative; blood pressure 109/64; pulse rate 92; basal metabolic rate as calculated from these findings plus 16½. The abdomen was distended and tender; the caecum was enlarged and readily palpable. The rectal examination revealed a four plus sphyncteric spasticity; sigmoidoscopic examination revealed extensive hemotumescent areas of mucous membrane with shallow and deep ulcers. The stool examination was typical for the condition — repeated examinations of freshly obtained stools were negative for amoeba. Hematologic status: 3,800,000 red blood cells; Hgb. 10 gm. (68%); hematocrit 37%; percent cell volume 86; MCV 96 cu. microns; MCH 26.3 micro-micrograms; MCHC 27.2%; color index .90; volume index 1.01; saturation index .79; icterus index 4.8 units; cell diameter 6.9 micra; reticulocyte count 1.9%; sedimentation rate 46 mm. in one hour; prothrombin time 26½ sec-

onds; 210,000 platelets; 6,100 white blood cells; differential: 43% neutrocytes, 48% lymphocytes, 7% acidocytes, 2% monocytes.

Urinalysis negative. Dark adaptation test unsatisfactory. Basal metabolic rate plus 14. Blood chemistry: 119 mg. glucose; 167 mg. cholesterol; 8.2 mg. calcium; 2.9 mg. phosphorus; 6.2 mg. total protein; 460 mg. chlorides as NaCl.

Treatment: constructive meal planning; restrict all sugar and processed starches; increase consumption of proteins and moderately increase salt intake; Lugol's solution of iodine — eight drops b.i.d.; 40 mg. vitamin K per day; one capsule of a liver and iron preparation with each meal. Vitamin C supplement — juice of one lemon or 2 limes in 4 ounces of unstrained orange juice q.d. Control diarrhea with following Rx:

Bismuthi subgallatis	60 gm.
Tinct. opii camph.	90 cc.
Elixiris lactopep qs. ad.	180 cc.

Sig: Shake label. Teaspoonful q. 3 or 4 h., as indicated. She improved with a better dietary and the above mentioned treatment regimen. However, there were several exacerbatory periods which were directly connected with the psychoneurotic factor — she was unable to obtain satisfactory employment. This continued until 1948 at which time she obtained a desirable position. With my better understanding of the nature of the disease, vitamin K, hesperidin with vitamin C, and rutin were prescribed. These supplements plus a stricter adherence to her dietary, normalized her condition within a couple of months and she has been free of exacerbations since Dec. 22, 1948. She now weighs 128 pounds, is employed in a full time position and I consider her cured. On last examination, May 24, 1950, the hematologic picture was normal; the yardsticks (sedimentation rate, prothrombin time and platelet count) were all normal; the stool examination was negative; sigmoidoscopic examination was negative for hemotumescent areas and ulcerations;

occasionally an ulcer scar was noted; all other physical findings were normal.

Case X. First consulted by this 15½ year old female on Dec. 9, 1949. Chief complaints: 6-8 bloody stools per day; nausea and vomiting, especially in the morning; dizzy spells; weak and tired; P.M. temperature ranging between 100°-102°F.; pain in left knee; rapid pulse and palpitation upon exertion or when excited; loss of weight (12 pounds); mental depression; lack of initiative; no interest in school or social activities; appeared to be schizoid.

Past history: pneumonia when an infant; acute rheumatic fever when 8 years old which required 4 months of active treatment; occasional mild exacerbations usually involving a knee or an elbow; at the age of 13 she had an acute attack of bloody diarrhea. Treated for two weeks with a soft, bland diet; large doses of a sulfa drug and an intestinal antispasmodic. No improvement. Penicillin was substituted for the sulfa drug. Her reaction to this antibiotic was unfavorable and another sulfa drug was prescribed. Her condition was worsened by this new drug and the family decided to discontinue the professional care for a while. She began eating most of the foods which had been tabooed — salads, cooked vegetables, red meats, raw fruit — and her general condition improved. The stools were less frequent and less bloody. She put on four pounds of weight. After a food and sweet beverage binge in September, 1949, she had an acute exacerbation of the rheumatic fever which involved both knees and she was confined to her bed for about ten days; P.M. temperature ranged between 100°-101°F.; lost five pounds in weight; ran a rapid pulse and had marked palpitation and dizzy spells upon exertion. She was morose and felt weak and tired. The diarrhea subsided and she developed a tendency to constipation. About October, 1949, she began to improve and gained strength and weight until the later part of November, 1949. On November 28, she had a severe dizzy spell, nausea and vomiting, and a recurrence

of the bloody diarrhea — 7-8 stools per day. The onset of this attack was apparently predicated upon the circumstance that her father was ordered to a hospital for a severe illness.

Cinical findings: height 65"; weight 96 pounds; temperature 99.6°F.; gums soft and bleeding; early dental caries; thyroid slightly palpable; chest negative; lungs — soft, moist rales over both apices; heart — a rough systolic murmur over the mitral area; pulse rate 124; blood pressure 100/57; distended and tender abdomen, especially over the left lower quadrant; spasticity of the anal sphincters; swollen and tender left knee joint. Sigmoidoscopic examination: the lower six inches of the terminal colon were involved — there were confluent, hemotumescent areas with demonstrable capillary fragility and permeability; also moderate numbers of shallow ulcers.

Hematologic status: 3,600,000 red blood cells; Hgb. in grams = 8.69 (63%); hematocrit 38%; percent cell volume .90; cell diameter 7.4 micra; M.C.V. 100 cu. micra; M.C.H. 24.1 micro-micrograms; M.C.H.C. 22.8%; color index .87; volume index 1.2; saturation index .70; icterus index 4.2 units; reticulocyte count .7%; sedimentation rate 35 mm. in ½ hr. and 80 mm. in 1 hr.; prothrombin time 28 seconds; 195,200 platelets; 14,000 white blood cells; differential — 2% bands; 38% segmenters; 52% lymphocytes; 5% acidocytes; 3% monocytes; approximately 30 and 34 percent of anisocytosis and poikilocytosis, respectively; some toxic granulation. A.M. and P.M. specimens of urine showed slight traces of albumin and the P.M. specimen had a moderate trace of sugar; microscopic — excess uric acid crystals in both specimens.

Blood chemistry: 124 mg. glucose; 150 mg. cholesterol; 64.5 mg. cholesterol esters; 5.9 mg. total protein; 7.9 mg. calcium; 2.7 mg. phosphorous; 478 mg. chlorides as NaCl. The clot retraction pattern (Bolen) was disturbed. The dark

adaptation test was approximately 50% below par. The vitamin C level was 1.2 mg. The B.M.R. was plus 18.

Diagnosis: 1. Improper nutrition, 2. subacute rheumatic fever, 3 chronic hemorrhagic ulcerative colitis, 4. a secondary anemia due to loss of blood and inadequate nutrition.

Treatment: 1. Eliminate processed starches and sugars from diet, 2. increase flesh proteins (had been advised to restrict flesh proteins because of her rheumatism), 3. rest in bed until temperature subsided, 4. constructive meal planning, 5. peroral liver and iron, 6. 40 mg. vitamin K daily, 7. hesperidin and vitamin C — 100 mg. each daily, 8. 1 tablet of dehydrated buckwheat juice (rutin) with meals, 9. control diarrhea with Sun's cholera mixture & opii.

On January 16, 1950, she weighed 97½ pounds. The temperature did not exceed 99°F. in the afternoons; sigmoidoscopic examination revealed a marked improvement. Her appetite was good; she was optimistic and felt strong enough to walk several blocks daily.

On February 13, 1950, she weighed 102½ pounds; the hematologic status was almost normal; prothrombin time 22 seconds; sedimentation rate 18 mm. in one hour; 310,000 platelets; clot retraction pattern was normal; the biophotometer test was good.

On February 15, her father's condition became much worse and unfortunately her mother allowed her to visit him in the hospital. She experienced a severe emotional reaction and fainted. It required 4 days of sedation to regain her composure. Despite this, the bloody stools did not recur although for a period of about one week she did have 3-4 loose stools per day.

On March 15, 1950, she weighed 106½ pounds; sigmoidoscopic examination revealed a disappearance of ulcers and the hemotumescent areas had faded into a moderate congestion of the mucous membrane. The P.M. temperature was normal and she evinced interest in resuming school and engaging in social activities. The dose of all of her medica-

tions was reduced 50%. The diarrhea and loose movements had disappeared.

On May 15, 1950, she weighed 110½ pounds; had gained ½ inch in height; her gums were firm and did not bleed; P.M. temperature normal; hematologic status normal; sedimentation rate 12 mm. in 1 hour; prothrombin time 20½ seconds; 390,000 platelets; Bolen test negative. No evidence of rheumatic fever. Sigmoidoscopic examination negative; stool examination negative. Instructed to discontinue vitamin K; to take one rutaplex tablet night and morning; to take a tablet containing 50 mg. each of hesperidin and vitamin C once a day; to continue taking a capsule of ferrous lextron with lunch and dinner.

The satisfactory response of this young girl to specific nutriture therapy supports the postulation that this clinical syndrome is basically a hemorrhagic disease. Not only the colitis, but the rheumatic fever responded promptly to nutritional therapy — there may be a clinical kinship between these two diseases.

Case XI. First consulted by this 25 year old female on February 5, 1947.

Chief complaint: 4-5 bloody stools a day with tormina and tenesmus; palpitation of the heart; tremor of fingers; loss of 15 pounds in weight.

Past history: Nothing significant except a maladjustment to the family circle. Her father was an extremely religious man and unsympathetic in his attitude. To escape this situation, she obtained employment in 1943. This position was not satisfactory because her employer insisted upon her working long hours and was disagreeable and hypercritical of her work. In 1944, she had a severe attack of diarrhea. Two weeks later she began to notice blood in her stools. She was advised to quit her job. She returned home and was placed under orthodox treatment for ulcerative colitis. She improved until October, 1946, when the bloody diarrhea recurred. Apparently this recurrence was precipitated by the

antagonistic attitude of her father. Orthodox treatment — sulfa drugs, bland diet, antispasmodics, etc., failed to improve her condition.

Clinical findings: weight 102; height 58½"; blood pressure 110/66; pulse rate 96; heart negative for murmurs; EKG normal; abdomen slightly distended and tender over the caecal and sigmoidal sections of the colon; 2 plus spasticity of the anal sphincters. Sigmoidoscopic examination revealed a typical picture of ulcerative colitis which was confirmed by the stool examination.

Hematologic status: 4,500,000 red blood cells; Hgb. 12.8 gm. (89%); hematocrit 36.3%; percent cell volume 84.4; cell diameter 7.4 micra; M.C.V. 79.8; M.C.H. 28.1; M.C.H.C. 35.2%; color index .96; volume index .92; saturation index 1.0; icterus index 7 units; reticulocyte count .5%; sedimentation rate 10 mm. in ½ hour, 32 mm. in 1 hour, and 62 mm. in 2 hours; prothrombin time 25¼ seconds; 290,000 platelets; 13,500 white blood cells; 53% neutrocytes; 39% lymphocytes; 6% acidocytes; 1% basocytes; 1% monocytes.

Blood chemistry: 100.5 mg. blood glucose; 150.5 mg. cholesterol; 96 mg. cholesterol esters; 6.7 mg. total protein; 12.4 mg. calcium; 5.2 mg. ionized calcium; 2.8 mg. inorganic phosphorous; Ca-P ratio 4:1; 603 mg. chlorides as NaCl.

The B.M.R. was plus 14; there was a typical disturbance of the Bolen test. The dark adaptation test was below par.

Treatment: 1. Control bowel movements with Sun's cholera mixture ć opii. 2. Constructive meal planning. 3. Increase consumption of flesh proteins. 4. Restrict salt. 5. Hematinic preparation containing liver extract, iron and the vitamin B complex. 6. 30 mg. vitamin K q.d. 7. Hesperidin and vitamin C, 50 mg. aa t.i.d. 8. 1 rutaplex tablet t.i.d. 9. Lugol's solution of iodine, 8 drops b.i.d. She responded to this treatment and in three months the hematologic picture was normal. The diarrhea ceased and there was a complete disappearance of blood from the stools.

Subsequent sigmoidoscopic examinations revealed that the ulcers and hemotumescent areas had healed.

She got married in 1948, became pregnant in 1949, had a normal experience, and is now the mother of a healthy baby. Her last visit was on July 16, 1950. She weighed 125 pounds and had no complaints. The blood chemistry and hematologic status were normal. The sigmoidoscopic examination was negative. The B.M.R. was −4. All medication was discontinued but she was advised to continue her nutritional scheme.

Case XII. First consulted by this 31 year old male on January 28, 1950.

Chief complaints: Morning headaches; 4 or 5 watery stools per day — one or two bloody in character; general feeling of jitteryness and a lack of energy.

Past history: As a youngster he had "growing pains" which were not definitely diagnosed as rheumatic fever. In 1940, he failed to pass his navy examination because of nervousness and a blood pressure of 190/128.

Six months ago he became extremely nervous and had a "fluttery feeling" in his stomach with 8-12 bowel movements per day, mostly after meals and during the night.

Clinical findings: height 67½"; weight 148 pounds; chest and lungs negative. The heart rate was 96; blood pressure 180/82; abdomen was tender over the left lower quadrant; the anal sphincters were spastic; sigmoidoscopic examination revealed a moderate number of hemotumescent areas which were friable and oozing; the ulcerations were shallow and extremely small; microscopic studies revealed the presence of many red and white blood cells with the exfoliation of colonic epithelium.

Hematologic status: 6,160,000 red blood cells (nutritional, hypertensive polycythemia); Hgb. 17.2 gm. (121%); hematocrit 47.6%; percent cell volume 116.1; cell diameter 7 micra; M.C.V. 77.2; M.C.H. 27.9; M.C.H.C. 36.1%;

color index .98; volume index .94; saturation index 1.0; icterus index 11.5 units; reticulocyte count 2.1%; sedimentation rate 6 mm. in ½ hour, and 18 mm. in one hour; prothrombin time 26¾ seconds; 230,000 platelets; 6,900 white blood cells; 1% bands; 57% segmenters; 31% lymphocytes; 5% acidocytes; 1% basocytes; 5% monocytes.

Blood chemistry: 112 mg. blood glucose; 296.5 mg. cholesterol; 224 mg. cholesterol esters; 7.2 mg. total protein; 16 mg. calcium; 6.9 mg. ionized calcium; 4.1 mg. inorganic phosphorous; Ca-P ratio 3.6:1; 515 mg. chlorides as NaCl; the Bolen test for malignancy revealed the characteristic disturbance of the clot retraction pattern so frequently observed in ulcerative colitis; the B.M.R. was plus 16; the Wassermann reaction was negative.

The dark adaptation test was not satisfactory being about 25% off base; both specimens of urine were negative throughout.

Treatment: 1. Constructive meal planning. 2. Restrict salt, sugar, sweet beverages, all bakery products made of white flour, and the cholesterol-rich fats. 3. Vitamin K 40 mg. qd. 4. One tablet of dehydrated buckwheat juice t.i.d. 5. Hesperidin and vitamin C, 50 mg. aa t.i.d.

His response to this therapy was prompt. Two weeks later his blood pressure was 150/70 and the pulse rate was 80. The diarrhea had ceased and there was no sign of blood in the stools. He was last examined on March 30, 1950. There was a complete disappearance of the hemotumescent areas and punctate ulcers. The hematologic status was greatly improved. The number of red blood cells had dropped to 5,350,000 with a corresponding improvement in all other values. The sedimentation rate was 12 mm. in 1 hour; the prothrombin time was 21½ seconds and the platelet count was 310,000. He was advised to continue taking rutin, hesperidin and vitamin C, and to discontinue the vitamin K.

The chronicity of the condition in this person had not been established, accounting for his prompt response to specific nutriture therapy. The psychoneurotic factors involved were related to the nature of his business and to a domestic situation which cannot be discussed.

The chief vitamin supplements used in this work were:

1. Synkayvite (vitamin K) (Roche's).
2. Hesperidin and vitamin C (ascorbic acid) (National Drug Company).

APPENDIX III

Bibliography of Collateral Reading

1. Bicknell, Franklin, and Prescott, Frederick: *The Vitamins in Medicine.* London, William Heinemann Medical Books, Ltd., 1948, 2d Ed.
2. Cummings, Richard Osborn: *The American and His Food.* Chicago, University of Chicago Press, 1941.
3. Drummond, Jack Cecil, and Willoraham, Anne: *The Englishman's Food: A History of Five Centuries of English Diet.* London, Jonathan Cape, Ltd., 1939.
4. Graham, Edward H.: *The Natural Principles of Land Use.* New York, Oxford University Press, 1944.
5. Hambidge, Gove: *Hunger Signs in Crops: A Symposium.* American Society of Agronomy and the National Fertilizer Association, 1941.
6. Hawkins, Harold F.: *Applied Nutrition.* Milwaukee, Wisconsin. Lee Foundation for Nutritional Research, 1947, 2d Ed.
7. Howard, Sir Albert: *An Agricultural Testament.* New York, Oxford University Press, 1940.
8. Interim Report of the Pioneer Health Center: Biologists in Search of Material. London, Faber & Faber, Ltd., 1938.
9. Kellogg, Charles E.: *The Soils that Support Us.* New York, The Macmillan Company, 1941.
10. Orr, Sir John Boyd: *Food for the World: A Symposium.* Chicago, University of Chicago Press, 1945.
11. Page, Melvin E.: *Young Minds with Old Bodies.* Boston, Bruce Humphries, Inc., 1944.
12. Picton, Lionel: *Nutrition and the Soil.* New York, The Devin-Adair Company, 1949.
13. Price, Weston A.: *Nutrition and Physical Degeneration.* New York, Paul B. Hoeber, Inc., 1939.
14. Saunderson, Mont. H.: *Western Land and Water Use.* Norman, Oklahoma, The University of Oklahoma Press, 1950.
15. Schultz, Theodore W.: *Food for the World: A Symposium.* Chicago, University of Chicago Press, 1945.
16. Stern, Bernard J.: *Social Factors in Medical Progress.* New York, Columbia University Press, 1927.
17. *The Frustration of Science: A Symposium by British Scientists.* New York, W. W. Norton & Company, Inc., 1935.
18. Wiley, Harvey W.: *The History of a Crime against the Food Law.* Privately printed, 1929.
19. Wrench, G. T.: *The Wheel of Health.* London, The C. W. Daniel Company, Ltd., 1938.
20. Yearbook of Agriculture: *Soils and Men,* 1938.
21. Yearbook of Agriculture: *Food and Life,* 1939.
22. Yearbook of Agriculture: *Farmers in a Changing World,* 1940.
23. Yearbook of Agriculture: *Climate and Man,* 1941.
24. Yearbook of Agriculture: *Keeping Livestock Healthy,* 1942.
25. Bakeless, John: *The Eyes of Discovery.* Philadelphia. J. B. Lippincott Co., 1950.

"Note: Lee Foundation for Nutritional Research is a non-profit, public-service institution, chartered to investigate and disseminate nutritional information. The attached publication is not literature or labeling for any product, nor shall it be employed as such by anyone. In accordance with the right of freedom of the press guaranteed to the Foundation by the First Amendment of the U. S. Constitution, the attached publication is issued and distributed for informational purposes."