

NUTRITION *in* POLIOMYELITIS

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POLIOMYELITIS is a systemic disease of virus etiology involving primarily the central nervous system and the voluntary muscular system. It is essentially a complex medical problem which requires intelligent medical management and competent nursing care for best clinical results.

Clinically, poliomyelitis may be either abortive, nonparalytic or paralytic. The paralytic types may be spinal, bulbar, or encephalitic, in the order of their greatest frequency. Recovery is spontaneous and apparently complete in only the abortive type.

The prominent symptoms are common to the nonparalytic and paralytic types of poliomyelitis. These include fever, headache, gastrointestinal disorders such as nausea, vomiting, and diarrhea, symptoms of the common cold, sore throat, malaise, considerable apprehension, muscle pain and muscle spasm, and stiffness of the back and neck. There is no loss in muscle power in the nonparalytics, but there is muscle weakness or paralysis in the paralytic patients.

The previous clinical management of poliomyelitis consisted of complete bed rest, immobilization, and splinting of the affected parts which resulted in what Ransohoff¹ so aptly termed "physical stagnation." Treatment was usually directed mainly to the neuromuscular aspects of the disease, with a consequent neglect of the patient's nutritional status.

After many years of studying the problem and evaluating the conclusions clinically, we² recommend that poliomyelitis be treated by: (1) early ambulation; (2) muscle relaxation; (3) controlled prolonged medical observation in order to attain maximum recovery and

function of the neuromuscular power still retained by the patient; and (4) special nutrition program. Muscle relaxation is best obtained by the use of curare according to the technique originally described by Ransohoff³ and modified by us. When relaxation has been achieved so that there is a complete range of motion of all joints, the curare is discontinued and prostigmine is instituted⁴ in order to increase muscle strength and co-ordination. The physical therapy outlined by Ransohoff and the exercises described and illustrated by Billig and Loewendahl⁵ have proved effective in our therapeutic regimen. Personal assistance and walking aids are used to facilitate early ambulation.

The application of *nutritional knowledge* to support the body mechanisms in disease has not received adequate attention. The role of nutrition as an integral part of the therapeutic regimen in poliomyelitis unfortunately either is not fully appreciated or is not generally understood. McLester⁶ points out: "Nutrition is often a determining factor in recovery from disease. This has been slow of recognition and even today it warrants repeated emphasis." In poliomyelitis, specifically, the problem of disturbed muscle metabolism and protein depletion can be reversed in part with nutritional supplements. Increased protein intake is the crux of our nutritional program.

ROLE OF PROTEIN

We are all aware of the fact that one of the fundamental defenses of the body is dependent upon the presence of antibodies in the tissues and body fluids. Since antibodies are proteins, there seems to be no other mode of antibody synthesis but from dietary protein. According to Cannon,⁷ this leads to the obvious conclusion that consumption of an adequate diet is "sine quo non" for protein repletion if the

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body's acquired resistance is to be maintained.

The importance of protein in the defense against infection has been shown repeatedly.⁶⁻⁸ Cannon and his associates⁹⁻¹¹ demonstrated the relationship of kind and amount of protein diet to antibody synthesis and resistance to infection. According to Cannon,¹² one portion of the serum protein (the globulin) carries with it various antibodies against infectious agents. It is not surprising therefore that protein undernutrition may be associated with susceptibility to bacteria and viruses. According to Wohl *et al.*,^{13,14} "For effective production of antibodies it is necessary to restore protein reserves; if the capacity for protein synthesis is not defective, repletion and improved production of antibodies may be expected." Guggenheim and Buechler¹⁵ concluded from their studies that: (a) the humoral defense mechanism appears to be more sensitive to protein deficiency than the cellular defense mechanism; (b) different food proteins elicit different bactericidal and phagocytic powers (this quality of promoting antibacterial defenses corresponds to the growth-promoting quality of the respective proteins); and (c) immune globulin is dependent for its formation upon the protein reserves and the protein intake.

During the febrile stages of most acute infectious diseases, both total metabolism and nitrogen metabolism are considerably increased.^{16,17} Under ordinary conditions of feeding this results in a negative nitrogen balance with consequent waste of protein. This has been designated as the "toxic destruction of protein," and is apparently the result of combined effects of increased caloric expenditure and destruction of tissue. The destruction of protein appears to parallel the intensity or severity of the infection with which it is associated.

Metabolic disturbances caused by infection will lead to depletion of protein reserves which may be particularly hazardous in times of metabolic stress. That serum protein levels drop in various acute infections has been established. Eaton and Bower¹⁸ referred to this phenomenon as "the reaction of injury" or the "catabolic phase of injury." Bower

*et al.*¹⁹ concluded from a study of a group of acute poliomyelitis cases that: (1) the greater the clinical severity and involvement of the case, the more consistently will the serum albumin levels drop below normal. This drop occurs early, between the first and third day, and progresses unfavorably until the tenth day at least; (2) the deviation of the serum protein values from normal bears a linear relationship to clinical severity, i.e., the more severe the clinical involvement, the less the amount of serum albumin present. According to Whipple,²⁰ the production of plasma protein, which contributes freely to the formation of globin and hemoglobin, is depressed by infection.

The catabolic phase during the course of disease was thought to be irreversible; however, Co Tui,²¹ on the basis of his studies, holds that if such a catabolic factor is present, its effect can be counterbalanced by increasing the level of protein intake. He has even hypothesized that each disease entity has its own specific range of protein catabolism and, therefore, would require its own specific range of protein and caloric intake to maintain the protein status.

VITAMINS

McCormick²² offered a concept that "the paralytic phenomenon in poliomyelitis may be directly referable to acute B₁ avitaminosis precipitated by converging factors which increase the metabolic rate and accordingly the bodily requirements for this vitamin . . . and that the paralysis may be a secondary metabolic manifestation rather than a primary infectious disease."

An apparently increased resistance to the Lansing strain of poliomyelitis virus in experimental mice on vitamin B-deficient or restricted food intake was reported by Foster *et al.*;²³ however, they stated that their findings were difficult of interpretation and inconclusive, and intimated additional experimentation was needed to clarify the problem. In another experiment²⁴ they found no evidence that combined deficiency of phosphorus and vitamin D protected mice to any degree against poliomyelitis virus, although the ani-

mals were in a state of advanced deficiency; in fact, they were more susceptible than the controls on complete diets. Increase in susceptibility to the virus was greatest in the most deficient diets. This was particularly true in diets deficient in both phosphorus and calcium.

The influence of thiamine deficiency on the susceptibility of monkeys to experimental poliomyelitis was studied by Clark *et al.*,²⁵ who concluded that "this species when deficient in thiamin does not exhibit an increased resistance to poliomyelitis virus." The difference in response shown on the one hand by mice and on the other hand by cotton rats, even though they were subjected to similar nutritional stresses and inoculated with the same strains of viruses, led Weaver²⁶ to conclude that "this illustrates the danger of applying directly to human beings the results of animal experimentation."

Mathews²⁷ has called attention to the very close similarity of the neuropathological changes found in poliomyelitis and those produced by lack of vitamin B. He suggested that the destructive changes in poliomyelitis may be brought about in an indirect way by the intracellular vitamin B-depleting effect of the infectious agent.

We cannot subscribe to the concept that vitamin deficiency, be it thiamine or any other vitamin, represents a specific etiologic factor *per se* in poliomyelitis. We do believe, however, that thiamine and riboflavin are related to protein synthesis in the body, and any deficiency will interfere with this essential process.

MUSCLE WASTING

One of the essential problems in poliomyelitis is wasting of muscle tissue. Although the mechanism is uncertain, it has been suggested²⁸ that the muscle tissue may be directly invaded by the virus. Additional contributing factors may be dietary inadequacy, fever, anorexia, nausea and vomiting in the course of the acute phase, the catabolism attributed to the over-secretion of catabolic hormones by the adrenals as part of the stress reaction, and immobilization of the patient.

It was generally accepted that weight loss was inevitable in poliomyelitis as a result of

the destruction of the anterior horn cells, thus producing muscle atrophy by denervation. In following the patients in our clinic from the onset of the disease to functional restoration, we observed that those with the most extensive paralysis suffered the greatest weight loss—anywhere from ten to forty pounds—during the first three to six weeks. This may be ascribed to bed rest and inactivity, a decrease in appetite, and to destruction of tissue due to denervation. The muscle atrophy associated with poliomyelitis represents to us evidence of loss of plasma proteins as well as tissue proteins. With a depletion of these proteins the patient becomes weak, loses weight, and suffers a reduction in muscle mass. If this phenomenon is not attended to, further complications of protein deficiency will evolve, resulting in prolonged convalescence and additional muscle weakness and paralysis.

It is not often realized that an increased nutritional intake is essential in any infection, and, especially, in the acute and convalescent poliomyelitis patients. For effective tissue synthesis, two dietary essentials, proteins and calories, are mutually indispensable. Adequate nutritional intake is even more important for the convalescent patient than it is for the normal, healthy individual. It should be borne in mind that in poliomyelitis muscle metabolism is seriously disturbed. Considerable loss of muscle protein results from bed rest, and if we add to this the loss of plasma protein as a result of the acute infection, the protein requirements of the patient are greatly increased. This increased demand must be satisfied.

The extensive muscle spasm which is always present in poliomyelitis interferes with the circulation and, thus, nutrition of the muscle. Energy-producing substances, such as adenosine triphosphate and phosphocreatine, protein derivatives, are interfered with or reduced.²⁹ Blood sludging^{30,31} and increased capillary permeability further interfere with muscle nutrition.

PRESENT PROGRAM

We deem it imperative to institute a nutritional program as soon as the patient is ad-

mitted to the hospital in order to arrest, if possible, the early rapid weight loss. We are in agreement with Co Tui that the tendency to catabolism may be counterbalanced by increasing the level of protein intake. Our objective is to restore muscle mass and strength as quickly as possible without the deposit of excess fat. Such fat is a liability in the presence of weak or paralyzed muscles. When wasted muscles are to be reconstituted, as in poliomyelitis convalescence, both the quantities and proportions of all indispensable amino acids must be adequately supplied in the daily diet, together with calories, vitamins, and minerals.

We strive to make the patient ambulatory at an early date to enable him to participate intensively in exercise programs without premature fatigue, and to assume more quickly his place in school or work activities. Patient progress is measured by total urinary nitrogen, urinary creatine excretions, muscle testing, and weight readings taken at fixed times daily.

From a nutritional standpoint, our procedure with poliomyelitis patients includes an evaluation of the patient's physical status at the time of hospitalization, complemented with urine and blood studies. During the acute febrile stage, the patient is supplied with all necessary nutrients by the administration of glucose and protein hydrolysate parenterally, and, if the case is severe, blood plasma also. However, as in other types of nutritional therapy, good food by mouth, according to Stare and Thorn,³² is the most effective and satisfying way to administer protein. While temporary therapy with protein concentrates of various types is often valuable and even necessary, it is only a highly nutritional diet over a long period which will give satisfactory results. Increasing the amount of protein in a diet is one of the best general ways to improve a diet. This we accomplish by adding a daily supplement to the routine hospital diet (Table I).

Nutritional support should begin when the patient arrives in the hospital, at the very onset of the disease, when the patient's system is under the terrific stress of the infection. The protein intake should be supplemented

with fat and carbohydrates. The fat spares the protein by preventing its consumption for energy production and the carbohydrate spares the protein by relieving it of the necessity of providing carbohydrate. Hoesslin³³ found that the administration of a high caloric diet led to little increase of weight in protein-starved individuals if the high calories were given entirely as fat and carbohydrates. Only if the protein in the diet was also raised could they be made to put on flesh.

Hospital patients are frequently on a low intake not only of proteins but of total calories.³⁴ The general hospital diets are often calorically adequate, but according to Pollack and Halpern³⁵ are inadequate in proteins. This may be further complicated by the patient's failure to eat all of the food supplied at each meal, a situation particularly true in a pediatrics ward. Table II shows the nutrients in customary "routine" hospital diets. These are considered inadequate in protein and in calories.

Hospital diets do not furnish enough *protein* to supply the demand created by the disease process in poliomyelitis. We appreciate that no patient has the stomach capacity nor the gargantuan appetite to consume 150 to 300 Gm. of protein per day from kitchen-prepared food. Since Wohl *et al.*¹³ have shown that casein-type protein supplements aid measurably in antibody formation in hypo-proteinemic patients, we are able to depend on this type of supplement to obtain the thera-

TABLE I
Daily Supplement Added to Routine Hospital Diet

Supplement	Quantity used in daily mixture	Protein	Fat	Carbo-hydrate	Caloric value
		Gm.	Gm.	Gm.	
Fat emulsion	4 oz.	0	48	12	480
*Protein powder	2 oz.	42	0	13.2	220.8
Eggs	2	13.4	10.4	0	147.2
Corn syrup	1 oz.	0	0	22.2	88.8
Milk	1 qt.	31.6	38	48	660.4
Totals		87	96.4	95.4	1597.2

* Daily supplement drink mixture is best taken cold after meals and at bedtime. It may be fed by stomach tube in bulbar cases.

TABLE II
Routine Hospital Diets

Average weights		Average ages of patients		Protein	Fat	Carbo- hydrate	Protein	Fat	Carbo- hydrate	Total 24-hr. caloric intake
Lb.	Kg.	Years	Gm.	Gm.	Gm.	%	%	%		
29	13	1-2	45	70	125	15.5	36.3	48.2	1310	
55	25	3-9	60	75	150	20	40	40	1515	
85	39	10-13	75	100	200	15	44.5	40.5	2000	
110	50	15	85	120	200	15	49	36	2220	

TABLE III
Supplemented Diet used in Our Program
Compared with Special Hospital Diet

Diet for 15 yr. old 110 lb. (50 Kg.) of weight	Protein	Fat	Carbo- hydrate	Protein	Fat	Carbo- hydrate	Total 24-hour caloric intake
	Gm.	Gm.	Gm.	%	%	%	
Routine hospital diet (Table II)	85	120	200	15	49	36	2220
Our supplement added (Table I)	87	96.4	95.4	21.2	54.5	24.3	1597
Total supplemented diet used	172	216.4	295.4	18	51	31	3817
High protein high caloric hospital diet	144	135	486	16	32	52	3775

Note the *increase* in protein and fat and *decrease* in carbohydrate in our supplemented diet as compared with the hospital diet.

Patients find it difficult to consume all of the kitchen-prepared food in a high protein, high caloric hospital diet. There is less resistance in the feeding of the routine hospital diet plus four or more glasses of cold, flavored "milk shake" mixture.

peutic benefits of high protein intake. Waife *et al.*³⁶ found that patients whose protein intake was supplemented by an oral concentrate preparation stored large amounts of nitrogen. This avidity for protein reached high proportions in certain chronically ill individuals, even after months of presumably adequate protein intake.

A survey of protein supplements was made and two preparations have been selected because of their high biologic values and their palatability. To supply the additional calories deemed necessary to spare protein, we selected an oral fat emulsion* which was readily accepted by a majority of the patients.

On arrival at the hospital, each patient's physical status is evaluated, and the nutritional program is arranged. We supply all

necessary nutrients during the acute phase in the form of glucose, protein hydrolysate, blood, and sometimes plasma, parenterally, and protein and fat emulsions orally. After the acute phase, the patients are put on general hospital diets which are supplemented with the casein-type oral protein concentrates.† Each meal was carefully evaluated for its caloric value represented by protein, fat and carbohydrate. It has been our procedure to check each tray as it is taken from the patient for the calories not consumed, and this aggregate difference between calories supplied and calories consumed is made up in an extra meal at night. The protein-vitamin preparations are prescribed in quantities to attain a level of at least 4 Gm. of protein per Kg. of body weight,

* Lipomul®—Upjohn Company, Kalamazoo, Michigan.

† Vi-Protinal®—The National Drug Company, Philadelphia, Pa. Provimalt®—Humanic Brands, Summit, N. J.

TABLE IV
Caloric Value of Supplements
Used in Poliomyelitis

Supplement	Quantity	Protein	Fat	Carbo- hydrate	Caloric value
		Gm.	Gm.	Gm.	
Fat emulsions (Lipomul®) (Ediol®)	1 oz.	0 0	12 15	3 3.6	120 150
Protein powder (Vi-Protinal®) (Provimalt®)	1 oz.	21	0	6.6	110
Egg	1	6.7	5.2	0	74
Corn syrup	1 oz.	0	0	22.2	89
Milk	1 qt.	31.6	38	48	660

Nutmeg, vanilla, chocolate, or any other flavoring agent may be used. Shake thoroughly and serve cold.

fortified with oral fat emulsion to supply about 500 calories in addition to that obtained from the kitchen-prepared food (Table III).

The excellent cooperation of the nursing staff and the dietitians has made it possible for us to have a daily record of actual food consumption for each patient. We have achieved daily protein intakes up to 300 Gm. with very little or no patient resistance. The oral fat emulsion was added to milk with the protein-vitamin preparation. There were some complaints of fullness of the stomach, but this was not a serious problem.

Because of the early loss of weight (we have observed an average loss of 10 to 40 lb. during the first three to six weeks), it is imperative to start intensive feeding as soon as the patient is admitted to the hospital. As a result of our nutritional program, we have succeeded in having a large number of our patients maintain or gain weight during their hospitalization. This program is continued in ambulatory patients until maximum muscle strength and function have been attained. It must be emphasized here that our object is not to have the patient gain weight *per se*, but rather to maintain his muscle strength without the deposit of excess flabby fat.

It is important that the high protein intake program be carried over into the home care of the patient (Table IV). Not only should the family be instructed in the methods of preparing high protein foods and warned to withhold sweet drinks and candy from the

patient, but should also be instructed in the importance of continuing the supplemental protein feeding as part of the diet, especially with those patients who continue therapeutic exercises. It will definitely aid if the family fully understands the particulars of the patient's need for protein.

CAPILLARY PERMEABILITY

Capillary permeability may be increased in poliomyelitis as a result of the infection or the toxemia of the infection. According to Harrell and Aikawa,³⁷ intelligent management, with attention to alterations in the permeability of membranes, will lessen the severity of the patient's resistance to his infection, and will shorten convalescence.

There are a number of reports³⁸⁻⁴⁴ to indicate that Hesperidin, a flavonone derived from citrus fruits, combined with vitamin C, when used in adequate doses, corrects the increased capillary fragility and permeability found with various disease entities. On the basis of these reports, we have been using Hesperidin-C,[†] from 8 to 12 tablets or capsules daily, in all of our poliomyelitis patients. We intend to pursue this phase of our procedure further in a future study.

CONCLUSIONS

Since the institution of our nutritional program we have arrested the loss of body weight

† Hesperidin-C® (Hesperidin, 50 mg., Vitamin C, 50 mg.)—The National Drug Company, Philadelphia 44, Pa.

at a much earlier date in the process of the disease than has heretofore been observed, and have succeeded in having our patients retain their normal weight, yes, even gain weight, during their hospitalization. It is our impression that our therapeutic procedure emphasizing nutrition in the management of poliomyelitis has resulted in superior clinical results as evidenced by reduced severity of the acute phase, minimal weight loss, minimal disabling muscle atrophy and ankylosis, accelerated convalescence, reduced incidence for corrective surgery, and favorable results in functional restoration of affected parts.

We are in agreement with Elkins⁴⁵ that the responsibility for care of the poliomyelitis patient should be entirely in the hands of the physician. Treatment must be highly individualized and adapted to the patient's needs. The poliomyelitis patient is justified in feeling that his physician should be one who has studied the anatomic function of normal muscles, who has seen many forms of paralytic disabilities, and who can apply wisely his knowledge and experience.

The importance of nutrition is well expressed by Whipple and Madden,⁴⁶ who stated: "The circulating plasma protein is the medium of exchange and the body is solvent just so long as there are adequate proteins supplied for any emergency. When the body becomes insolvent, there may be foreclosure due to disease, infection, or injury." It is to keep the body solvent in poliomyelitis that we stress the importance of therapeutic nutrition.

The tremendous importance of maintaining optimal muscle metabolism by correcting the underlying biochemical and physiologic disturbance should be emphasized. Success stems from directing attention to early ambulation, to muscle relaxation by curarization and an active exercise program, to extended medical supervision—and particularly to therapeutic nutrition.

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RESUMEN

La nutrición en la poliomiéлитis

Con la institución precoz de su régimen nutritivo en el tratamiento de la poliomiéлитis

el autor ha conseguido suspender la pérdida de peso de sus pacientes a una fecha mucho más temprana en el curso de la enfermedad de lo que se había observado antes, e incluso ha logrado el mantenimiento del peso normal y aun un aumento ponderal en sus pacientes hospitalizados. Asimismo ha notado: reducida severidad de la fase aguda; mínimo de atrofia incapacitante de los músculos y de anquilosis; acelerada convalecencia; reducida incidencia de cirugía correctiva; y resultados favorables en la recuperación de los órganos afectados.

El autor insiste en la importancia de un óptimo aporte de proteína y el mantenimiento así del metabolismo por la corrección del dis-

turbio bioquímico y fisiológico subyacente. Ya en la fase aguda se administran todos los nutrimentos esenciales: glucosa, hidrolizados de proteína, sangre, y a veces plasma (por vía parenteral); proteína y emulsiones de grasas (por vía oral). Terminada la fase aguda, se suplementa a la dieta hospitalitaria con concentrados de proteína, tipo caseína, por vía oral. Los preparados de proteína y vitaminas se dan en cantidades capaces de lograr el nivel mínimo de 4 gramos de proteína por kilo de peso, y se les añaden emulsiones de grasas hasta proveer unas 500 calorías además de las provistas por las comidas. Con tal régimen se ha conseguido un consumo diario de hasta 300 gramos de proteína.