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ORAL FAT EMULSIONS COMBINED WITH PROTEIN SUPPLEMENTS IN THE MANAGEMENT OF ACUTE POLIOMYELITIS*

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Recent developments in food supplements are contributing to improved clinical results in the treatment of poliomyelitis, which, by our concept, is a systemic disease of virus etiology. The various poliomyelitis or poliomyelitis-like viruses thus far identified have a special predilection for the central nervous system and for the voluntary muscles.

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

The older therapeutic approach emphasizes immobilization and prolonged bed rest with no special nutritional care. Characteristic of the results are a high incidence of ankylosis, marked permanent muscle atrophy, prolonged malnutrition and a high incidence of orthopedic operations. The approach we use[1] combines (1) early ambulation; (2) muscle relaxation by curarization[2] and active exercise program,[3] (3) extended medical supervision; and (4) special nutrition.[4] In our opinion, this has given us superior clinical results, such as reduced severity of the acute phase, minimal weight loss, minimal disabling atrophy and ankylosis, accelerated convalescence, shorter hospitalization, reduced incidence of corrective surgery,[5] and favorable results in functional restoration.

In this presentation, I shall attempt to outline the special nutrition—one of the four important measures upon which our treatment of polio is based.

During the past three years, we have used hyperproteinization[6] with a high caloric intake to enhance utilization and to spare the body protein. At first, this high caloric intake was supplied by carbohydrates, but, during the last year, the extra calories have been supplied by oral fat emulsions[7] which have the additional advantages of rapid assimilation and weight gain.

Each patient's physical status was evaluated, and a diet was ordered consisting of easily assimilated protein preparations, protected by adequate caloric coverage, in quantities to attain a level of at least four grams of protein per kilo body weight, fortified with fat emulsion. In this way, from 100 grams or more of protein, and 500 or more calories in fat were added to the diet. To do this effectively, it was necessary to give between-meal feedings. Vitamins and minerals were ordered as needed. High-caloric protein drinks were also utilized for supplementation, since the optimum

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high intake of protein advisable cannot always be attained on the usual diet. The limiting factors are the bulk required, and the lack of stomach capacity for the extra-large amount of food. Other limiting factors may be anorexia, and the lack of sufficient strength to chew the food, under which circumstances the patient's only desire may be for liquid foods and beverages.

The oral fat was added to the milk together with the protein supplement, and, to make the preparation more palatable, sugar and vanilla or chocolate syrup (depending on the patient's preference) were added. There were some complaints of fullness of the stomach, but this was not a serious problem. The majority of patients preferred the drink after a meal or at bedtime. The cooperation of the nursing staff[8] and dietetic department is essential, as every tray is checked after it leaves the patient, any food not eaten being made up in an extra meal at night.

In most other regimens, during the acute fever stage, the patients are placed on a liquid diet or on parenteral fluids, both of which are always inadequate nutritionally. With our program, we supply all the necessary nutrients parenterally by giving glucose, protein, blood, and sometimes plasma, and orally by giving protein and fat emulsions.

After the acute phase, the patients are fed the general hospital diets, which are calorically adequate, but which, according to "Therapeutic Nutrition",[9] are inadequate in proteins. In checking the food trays after the patients had eaten, it was noted that at no time was all the food eaten, especially in the children's wards.[10] Meat, chicken, vegetables, broths and soups, cottage cheese, and salads were seldom touched. Scrambled eggs were usually scrambled a little more extensively, and left on the plate, while mashed potatoes were eaten partly. Jelly sandwiches were preferred. As a rule, cereals, desserts, bread and butter, and milk were consumed first. Fruit juices were usually taken well, but soft drinks, when available, had a high priority in upsetting the stomachs of the patients.

One of the main problems in poliomyelitis is wasting of muscle tissue. This wasting may arise from several factors: (1) dietary inadequacy; (2) fever, anorexia, nausea, and vomiting in the course of the acute phase; (3) the catabolic period attributable to the oversecretion of catabolic hormones by the adrenals as part of the stress reaction; (4) immobilization of the patient; and (5) the destruction of tissue cells by the infecting virus. The first three have been shown to be reversible through raising the level of feeding. Whether (4) and (5) are reversible has yet to be determined.

In the past, it was generally accepted that loss of weight was inevitable, the disease destroying the anterior horn cells, and thus producing muscle atrophy by denervation. In following the patients from the onset to complete functional restoration in our clinic, we have noted that they lost a great deal of weight, those with the most extensive paralysis suffering the greatest loss (averaging 10 to 40 pounds during the first three to six weeks). Because of this rapid early loss of weight, it is imperative to start the 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 on 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 bulk and muscle strength without the deposit of excess flabby fat. Excess fat in the presence of weak or paralyzed muscles is a liability which we always try to avoid by diet and exercise.

Total urinary nitrogen, urinary creatine excretions,[11] and blood chemistry were done on a number of patients during this study. Daily weights were taken at the same time each day.

Too often one does not realize that increased nutritional intake is very essential for the acute and convalescent poliomyelitis patient. In addition to the factors already mentioned, we should not forget that the muscle metabolism is seriously disturbed. There is the possibility that the muscle tissue may be directly attacked by the virus.[12] The extensive muscle spasm,[13, 14] which is always present, interferes with the circulation, and thus nutrition of the muscles. The energy-producing or controlling factors, such as adenosinetriphosphate, phosphocreatine, acetylcholine, and hemoglobin are probably interfered with. Blood sludging[15, 16] and increased capillary permeability further disturb the muscle nutrition. CoTui and others have emphasized the value of hyperproteinization in maintaining positive nitrogen equilibrium in muscle-depleting diseases. Cannon,[17, 18, 19] demonstrated the relationship of kind and amount of protein diet to antibody synthesis and to resistance to infection. Mills and Cottingham[20] (1943) reported that, for rats, the optimal phagocytic activity is definitely influenced by the temperature and by the amount of protein consumed. Kornberg[21] (1946) found that rats fed upon a protein deficient diet develop granulocytopenia, while Guggenheim and Buechler[22] (1946–1948) 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 the antibacterial defense corresponds roughly to the growth promoting quality of the respective proteins.

Sherwood[23] emphasizes the importance of protein nutrition in improving one's resistance to infection by pointing out that:

(a) Antibodies are protein in nature.

(b) It takes 4 to 8 weeks to deplete the protein reserves of the rat.

(c) Immune globulin is dependent upon the protein reserves and protein intake for its formation.

Rodriquez, *et al*[24]state that, in a case of avulsion of the brachial plexus, recovery of the muscles of the arm should be expected in approximately 24 months. If this does not occur, the assumption would be justified that, regardless of the physical therapy program (three times a week), the mor-

phologic, histologic, chemical, and other necessary properties of the muscle were not maintained in a sufficiently optimal state long enough for function to return, even though regeneration of the nerve might very likely have taken place. These authors very properly bring out a point we have been trying to emphasize,[25] namely, the tremendous importance we must place in the maintenance of optimal muscle physiology by correcting the underlying biochemical and physiologic disturbances. This we have been attempting to do[26] by directing our attention to early ambulation, muscle relaxation by curarization and an active exercise program, extended medical supervision, and special nutrition, the latter being fortified by oral fat emulsions[27] combined with protein supplements.

Summary

With our nutritional regimen, we supply the protein depleted poliomyelitis patients[28, 29] all of the needed protein and caloric requirements in easily assimilated food of high nutritional value. This is started immediately upon admission to the hospital.

It is generally accepted that hypoproteinemia in humans and animals reduces their ability to produce antibodies.[30, 31] The proteins fed replenish the body with its nitrogenous-tissue-building metabolites, which are so rapidly lost by the patients. Another important consideration is that the proteins themselves are a rich source of globulinantibody-building material, which the patient needs to overcome his virus infection.

The oral fat emulsions generate heat and energy with very little demand on the digestive system. The new chocolate flavored Lipomul is superior in tolerance and acceptability to the plain fat emulsions. This insures a palatable supplement of high caloric value, which makes possible full utilization by the body of every gram of protein given. Oral fat emulsions are proving to be very useful food supplements in high-caloric diets.

References

1. Boines, G. J. 1950. Treatment of acute poliomyelitis with emphasis on muscle relaxation and nutrition. Delaware State Med. J. **22:** 270–271.
2. Boines, G. J. 1952. The use of curare in poliomyelitis. M D. 9.
3. Ransohoff, P. F. 1951. Curare and physical therapy. M D. 407.
4. Boines, G. J. 1951. Nutrition in poliomyelitis. M D. 415.
5. De Palma, A. F. 1951. The orthopedic phase. M D. 418.
6. Co Tui. 1951. Clinical proteinology. M D. 540–541.
7. Shafiroff, B. G. P. 1951. The administration of fat emulsions in man. Trans. N. Y. Acad. Sci. **14**(2): 80–83.
8. McDonald, A. 1951. Nursing care in poliomyelitis. M D. 430.
9. Pollack, H., & S. L. Halpern. 1951. Therapeutic Nutrition. Nat. Acad. Sci. and Nat. Research Council.
10. Lynch, H. D., & W. D. Snively, Jr. 1951. Hypoproteinosis of childhood. J. Am. Med. Assoc. 115–119.
11. Boines, G. J., & J. C. Kakavas. 1950. Creatine excretion in acute poliomyelitis, preliminary report. Delaware State Med. J. **22:** 304–307.
12. Denst, J., & K. T. A. Neubuerger. 1950. Histologic study of muscles and nerves in poliomyelitis. Am. J. Path. **26**:863–881.
13. Boines, G. J. 1950. Muscle spasm and paralysis in the diagnosis of poliomyelitis. Med. Record. **163**:204.

14. CAUGHEY, J. E., & B. S. MALCOLM. 1950. Muscle spasm in poliomyelitis. Arch. Disease Childhood. **25**:15–21.
15. KNISELY, M. H. 1951. An annotated bibliography on sludged blood. Postgrad. Medicine. **10**:15–24, 80–93.
16. BOINES, G. J. 1952. Sludged blood in poliomyelitis. Delaware State Med. J. **24**:43–44.
17. CANNON, P. R. 1942. Antibodies and protein reserves. J. Immunol. **44**:107.
18. CANNON, P. R., W. E. CHASE, & R. W. WISSLER. The effects of a low protein diet and of plasmapheresis upon the formation of agglutinins. J. Immunol. **47**: 133.
19. CANNON, P. R., E. M. HUMPHREYS, R. W. WISSLER, & L. E. FRAZIER. 1946. Chemical, clinical and immunological studies on the products of human plasma fractionation. II. Serum protein regeneration and weight recovery in the hypoproteinemic rat. J. Clin. Invest. **23**:601.
20. MILLS, C. A., & E. COTTINGHAM. 1943. Phagocytic activity as affected by protein intake in heat and cold. J. Immunol. **47**:493.
21. KORNBERG, A. 1946. Amino acids in the production of granulocytes in rats. J. Biol. Chem. **164**:203.
22. GUGGENHEIM, K., & E. BUECHLER. Nutrition and resistance to infection. Bactericidal properties and phagocytic activity of peritoneal fluid in various states of nutritional deficiency. J. Immunol. **54**:349.
23. SHERWOOD, N. P. Immunology. C. V. Mosby. St. Louis.
24. RODRIQUEZ, A. A., H. W. KENDELL, & A. C. IVY. 1951. Strength frequency curves in polio and peripheral nerve injuries. Arch. Phys. Med. **32**:334–342.
25. BOINES, G. J. 1951. Muscles and physiology of exercise. M D. 363–365.
26. BOINES, G. J. 1951. Routine hospital care of poliomyelitis. M D. 426.
27. SHOSHKES, M., T. B. VAN ITALLIE, R. P. GEYER, & F. J. STARE. 1951. Fat emulsions for oral nutrition. J. Am. Dietet. Assoc. **27**:197–208.
28. BOWERS, A. G., R. M. EATON, J. S. CHUDNOFF, J. E. AFFELD, & A. L. CHANEY. 1950. Patterns of plasma protein changes in acute poliomyelitis patients: Treatment with blood plasma. Am. J. Med. Sci. **220**:46–54.
29. BOWER, A. G., J. S. CHUDNOFF, & A. L. CHANEY. 1950. Biochemical alterations in acute human poliomyelitis; Electrolyte patterns and trends. Calif. Med. **73**:406–412.
30. WOHL, M. G., J. G. REINHOLD, & S. B. ROSE. 1949. Antibody response in patients with hypoproteinemia. Arch. Internal Med. **83**:402–415.
31. WAIFE, S. O., M. G. WOHL, & J. G. REINHOLD. 1950. Protein metabolism in chronic illness: Effect of protein supplementation on nitrogen balance, hemoglobin, serum proteins, and weight in the malnourished and the effect of the nutritional status on nitrogen storage. J. Lab. Clin. Med. **36**:604–616.