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NUTRITION AND INFECTION

JOSEPH A. JOHNSTON, M.D.*

Before the advent of chemotherapy and the antibiotics, the physician's efforts were directed largely toward supporting nature's own response to an infecting agent, and he came to realize early that the success of this reaction to disease was determined well in advance of his intervention by the composition of the tissue with which he had to deal. An analogous situation existed in the attempts to repair water and electrolyte losses in the diarrhoeal states. About thirty years ago, after Gamble's brilliant work had given us some confidence in the use of parenteral fluids, I analyzed the mortality figures in an infants' hospital for ten years that preceded and two that followed the introduction of his ideas. The patients came from the lowest economic circumstance and had obviously been malnourished before the acute episode that occasioned their admission. In spite of our seemingly more intelligent management of fluids, we had effected no change in mortality; we were still losing approximately 240 patients per thousand admissions. In studying the past histories of the fatal cases, it was obvious that the summer diarrhoeal episode to which we were attributing death was merely the final chapter in a chronic disturbance of nutrition that had produced what has been termed "metabolic bankruptcy." Something was missing which made impossible the reconstitution of normal tissue, and which could frequently be predicted from the determination of the serum proteins. The changed picture in this group has not been the result of either anti-biotics or of more refined calculations of electrolyte losses, but of greater appreciation of the nutritional requirements for growth, qualitatively and quantitatively. The diarrhoeal problem is still with us, but when it occurs in infants adequately fed before the episode, it no longer contributes prominently to mortality, save in sporadic outbreaks in the newborn.

In tuberculosis, the brilliant contribution of streptomycin, isoniazid, and PAS may have made us forget a few hard-won facts about the role of nutrition in the development and course of this disease. In a twenty-five year study of 1100 reactors removed from contact, we felt that we were able to demonstrate that endogenous reinfection does occur; that a literal "breaking-down" could be referred to a failure of nutrition; and that subsequent healing paralleled more than any one thing the adequacy of the nutritional state as reflected in the storage of nitrogen.¹ This was not the simple problem of providing a good diet, but involved rather a careful consideration of a host of factors influencing favorably or otherwise the retention of ingested materials—infectious, glandular and emotional.

Rheumatic fever is conceded to be a disease state occurring preponderantly in the lower economic strata. For its development there would seem to be three factors involved—the infectious agent, usually a streptococcus; an inherited predisposition, which probably describes a sensitivity state; and a conditioning factor, seemingly associated with something present in the lower economic strata and not so frequently found in the upper. The first two of these factors would be common to all economic levels. In considering the third, it is difficult to dissociate items such as housing, clothing, dampness and diet, though the intake of dairy products and animal protein will be found consistently to fall with income. The literature that would incriminate nutritional defects as playing a prominent role in the outcome of rheumatic fever is impossible to ignore.

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In Coburn and Moore's² thoughtfully evaluated study, the only question arising had to do with the particular items that were most significantly absent from the diets, since protein, calcium, iron and Vitamin D were all a suspected group. It was of particular interest that in fourteen children from wealthy families only one could be said to have had a good diet; in ten, eggs were absent from the diet, sometimes because the children disliked them, sometimes because they were considered to be allergic to them. In the study of Warner and Winterton,³ a low intake of milk seemed the most prominent item. As they reported, to quote them, "Perhaps the most striking fact suggesting that an increase in animal fat in the form of fresh milk and butter may be of prophylactic value is furnished by the figures from Christ's Hospital where the incidence of rheumatism was reduced to less than a third corresponding to a rise in the animal fat consumption from 50.6 grams to 103.6 grams, due largely to an increase in the rations of milk and butter." The increase in milk involved, of course, increase in protein and calcium which could hardly be ignored. Wallis⁴ has recently offered an interesting comment on the possible value of egg. In analyzing the diet habits of a significantly large number of rheumatics and controls, he found that 40 percent of the cardiacs ate few eggs in contrast to 16 percent of the controls. There was active dislike of eggs in 10 percent of cardiacs compared to 4.6 percent of the controls. He points out that the inhibitor of hemolytic activity of Streptolysis S-9 produced by group A streptococcus depends in part on serum phospholipid, one of whose ingredients is choline, a substance which can be neither synthesized nor stored in significant amounts. Eggs would provide an excellent source of it. I realize that there is constantly quoted the experience from the armed forces of the relatively high incidence of rheumatic fever among service men, though their diet was the same as those who remained free from the disease. On this, I would point out three things: (1) The composition of the tissue of these men was determined years in advance of their entrance into service, (2) the role of nutrition in rheumatic fever is less one of influencing incidence than of influencing final outcome, and (3) in our tuberculosis work we found that the perfect diet might be offered, but in the case of protein, only one adolescent out of three took this by choice if the preference for carbohydrate had been established by the dietary pattern at home.

In an attempt to evaluate the effect of cortisone in rheumatic fever during the past five years, I recently analyzed for control purposes, my own experience over a twenty-year period preceding the use of this hormone. The case material is derived from a clinic serving for the most part the middle economic stratum. My experience prior to this had been limited to three large teaching institutions serving the indigent, the type of patient dealt with in the best available follow-up study of rheumatic fever. Although my follow-up has not been nearly so long (a mean of 66.6 months per patient for those seen before 1938, with 27 followed for longer than 108 months, and a mean of 50.6 months for those seen between 1938 and 1949, when cortisone was started), the results are sufficiently contrasting to quote them:

OUTCOME IN RHEUMATIC FEVER

	Boston Study ⁵ . 1000	H.F.H Study 218
	20 years	8 years
No carditis	10.8%	27%
Carditis with no functional handicap	31.6%	44.4%
Carditis with functional handicap	17.5%	22.4%
Death	30.1%	6.4%

That rheumatic fever could and did occur in the better economic groups was evident, but the difference in the end results was impressive. In attempting to assess the relationship between nutrition and outcome, a plotting of the patients on the Wetzel grid was used as a rough appraisal of the former. I would accept the criticism that weight does not measure nutrition, but the correlation is high when extremes are considered. In the study, Wetzel's⁶ three central channels are used as "average;" his upper three as "above average;" and the lowest three as "below average." The first two categories are grouped as "good," the third as "poor." For purposes of a contingency table, a good outcome was considered to be the failure to develop carditis or signs of a carditis with no functional handicap; a poor result was considered to be a functionally handicapped child or a fatal outcome. A distribution curve skewed to the left would describe a group weighed as above average; to the right as below average. The correlation between outcome and nutrition as measured in this way proved highly significant.

It would be premature to place on record the outcome in the cortisone-treated cases, of whom we have had 82. The point is made here, however, that when that evaluation is made it must take into account the nutritional state of the patients studied as a factor conditioning outcome in advance.

In conclusion, I would stress my feeling that when a pediatrician is able to influence favorably the nutritional state, he is probably making a contribution to the entire life history of the response to infection. But I would point out further that this is not merely a problem of providing an adequate diet, but of carefully considering all of those factors which affect its utilization.

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