

## Correspondence.

"Audi alteram partem."

### PROTEIN-BALANCE IN INFANT FEEDING.

To the Editor of THE LANCET.

SIR,—In Dr. Eric Pritchard's lecture on artificial feeding which appeared in your issue of April 29th, he says that—

"No matter how accurately a diet may comply with the calorie requirements, nevertheless if the balance or ratio between the three main constituents—i.e., the protein, fats, and carbohydrates—is not reasonably correct, in the long run the infant's nutrition will be bad."

Later on he says:—

"Of the many anomalies which surprise and puzzle critics of our national methods of artificial feeding there is none so amazing and inexplicable as the practice of altering the balance of artificial substitutes for breast milk as the infant grows older."

In answer to Dr. Pritchard I should like to quote Dr. Emmet Holt's opinion on this point as it agrees with my own experience in a large number of cases where cow's milk has to be substituted for human. In an article<sup>1</sup> on food requirements of infants, he says:

"The protein intake of the normal nursing infant is very low. It ranges during the first nine months of life appreciably from 8.0–12.0 g. daily. This is equivalent to only about 1.5 g. per kilo. Up to nine months the proteins taken by nursing infants seldom exceed 12 g. daily. This amount must be sufficient to supply not only maintenance need but the need for growth during the most active period of growth in the life of the child, for we find our best examples of good nutrition and growth in breast-fed infants. The reason for the adequacy of this small protein intake is that the protein of woman's milk is certainly best adapted in its amino-acid composition for digestion and assimilation by the infant. When cow's is substituted for woman's milk experience has shown that the protein intake must be considerably increased, doubled, or trebled. Infants 1–9 months of age receive from 15–30 g. of protein daily when fed on the usual modification of cow's milk. This represents fully 3.8 g. and often 4.0 g. of protein per kilo. The increase of protein requirement when cow's is substituted for woman's milk is probably due to the difference between the two milks in the amino-acid content. Woman's milk contains about twice as much lactalbumin as cow's."

In another part of the same article Holt says:

"In the early days of milk modification by the percentage method, it was thought that cow's milk mixtures in which the proportions of fat, sugar, and protein were as nearly as possible those found in woman's milk would be the best substitute for it. The many failures from the use of such formulas have been attributed to the too high fat, but it now seems quite evident that the low protein was even more at fault. It has become increasingly clear with clinical experience that the majority of infants will not thrive normally on cow's milk unless they are given two or three times as much protein as is contained in woman's milk."

I cannot help feeling that Holt's explanation is the right one and that Dr. Pritchard makes a mistake in not differentiating sufficiently between the human and cow proteins. I maintain that a considerable number of infants do not thrive on Dr. Pritchard's formula because they do not absorb a sufficient amount of protein for their adequate nutrition. The results of increasing the protein are often quite remarkable. The increase must, of course, be made very gradually for fear of producing dyspepsia, but apart from dyspepsia, I know of no bad symptoms which can be attributed to excess of protein.

I am, Sir, yours faithfully,

Kensington, June 2nd, 1922.

RONALD CARTER.

### GONOCOCCAL MENINGITIS.

To the Editor of THE LANCET.

SIR,—I have taken great interest in the annotation on gonorrhoeal meningitis in THE LANCET of April 15th, as it has been my privilege to see and

<sup>1</sup> American Journal of Diseases of Children, vol. xxii., No. 4, October, 1921. L. Emmet Holt and Helen Forbes, New York.

investigate such a case. The case which occurred in Staffordshire when I was doing cerebro-spinal fever work in the Army, was briefly as follows:—

I was called to see a man suffering from the physical signs and symptoms of acute meningitis. The patient was under treatment for acute gonorrhoea at the time, and the onset of meningeal symptoms was sudden and typical. I saw him within 24 hours of the onset, and performed lumbar puncture, taking off 35 c.cm. of very purulent cerebro-spinal fluid, and giving intrathecally 30 c.cm. polyvalent anti-meningococcal serum, until a confirmation of the diagnosis was reached by laboratory methods. On examination of films prepared directly from the fluid an intracellular Gram-negative diplococcus was found, which did not look like meningococcus, but was very suggestive of gonococcus. The fact that the man was under treatment for gonorrhoea suggested to me the possibility of this being a case of acute gonococcal meningitis, and cultural confirmation was carried out. Cultures were made on trypsin legumen agar, and on blood-smear agar plates, and there was no growth after 24 hours; but after three days colonies of gonococcus appeared, the greater growth being on blood-smear agar. To further prove that these were gonococcal colonies they were tested against the type serum, and type cocci of the meningococcal group for agglutination with negative result. They were also tested for fermentation with glucose and maltose, and it was found that the glucose alone was fermented, thus further establishing their identity.

The patient died within 24 hours of lumbar puncture, and no post-mortem was done, but I think the cultural proofs sufficient to warrant the diagnosis of acute gonococcal meningitis. Without such proofs I do not consider such a diagnosis can be made, as the meningococcus can cause an acute secondary infection in the male genital organs, as proved by orchitis, and epididymitis occurring as complications of cerebro-spinal fever. To rely simply on the finding of a Gram-negative intracellular diplococcus in the cerebro-spinal fluid, and a similar organism in the seminal vesicles, without even a urethral discharge, does not seem to me to be conclusive. The organism in such a case might quite well be the meningococcus, as in my own experience I have isolated this organism from the cerebro-spinal fluid, sputum and rash in the same case, and I think it is not beyond the limits of possibility to isolate the organism from the seminal vesicles where the meninges are primarily affected, and especially if there was a complication present such as orchitis or epididymitis.

I am, Sir, yours faithfully,

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June 1st, 1922.

### WHY IS SEPTIC AURAL DISCHARGE SO COMMON?

To the Editor of THE LANCET.

SIR,—Whilst engaged on work in a clinic for school children for defects of the ear, throat, and nose I was struck with a few points which may be of interest in the present discussion in your journal.

A number of children of the early age of 7 to 10 years were referred to me by the school medical officer for marked deafness, in most cases without an associated otorrhoea and in the smaller number with the discharge beginning from the age of 2 or 3; on examination it was astonishing to find an advanced condition of catarrhal otitis media with the usual thickened and retracted drumhead which had been insidiously creeping on for years. In a large number of those with otorrhoea there was no history of any of the exanthemata, but merely following upon an ordinary cold, and regarded lightly at the time, and the parents were told that the condition would clear up by syringing, &c. In nearly all these cases there was definite evidence of chronic post-nasal catarrh associated with adenoids. But in many of the cases there was an absence of any marked hypertrophy of the adenoid mass and definite chronic hypertrophic rhinitis (with or without septal deformity), and, of course, hypertrophy of the turbinates, especially the middle. Also in cases of otorrhoea and of deafness children were listed for operation (before