

a route largely ignored by both surgeons and radiotherapists until the work of Handley and Thackray. They appear to assume that irradiation of the internal mammary chain is efficient, and surgical attack unjustified. Have they any evidence of the efficiency of irradiation, or, in the case of these glands too, is the presence of apparently viable carcinoma after irradiation of no significance? It is obvious that if irradiation can deal efficiently with the invaded mammary chain it is preferable to surgery. But is it justifiable to condemn out of hand a trial of surgical methods without such evidence? At the very least, surgery has a place in the histological control of irradiation treatment. And, if surgery were as successful in dealing with the local disease in the internal mammary chain as it is in dealing with the local disease in the axilla, can we yet say for certain that this would have no influence on the course of the disease? Incidentally, Dr. Williams and his colleagues perhaps underestimate the extent of interest in the development of methods of surgical attack on the internal mammary chain. Apart from work in the United States to which they refer, it has for some time been the subject of wide inquiry and experiment, particularly in France and Scandinavia, and is also being explored in this country.

Finally, Dr. Williams and his colleagues note that in the procedures they advocate the "local recurrence index" is higher, but they do not appear to attach much importance to this. It may be that when general methods of control of malignant disease become more efficient the question of sterilization of its local manifestations will become unimportant. But at the present time, and with the bulk of the evidence suggesting that both surgery and irradiation are purely local in their effect, is not local persistence or recurrence of the disease the fairest test of the efficacy of such local forms of treatment?—I am, etc.,

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### Cough Syncope

SIR,—The use of some neutral and descriptive term such as "cough syncope" in place of "laryngeal epilepsy" is to be welcomed; and all those interested in this syndrome are indebted to Professor E. P. Sharpey-Schafer (*Journal*, October 17, p. 860) for demonstrating so clearly a physiological mechanism for its production. However, in implying that this explains all cases of loss of consciousness or fits following coughing, he claims too much. There are cases in the literature, and within my own experience, where the preceding cough was neither violent nor prolonged. In some it was subjectively different from the patient's "ordinary" cough and was recognized as heralding an attack. These cases should probably be regarded as primarily epileptic with coughing as an aura.

His further far-reaching statement that a "fit can occur in any normal subject if the blood flow to the brain is sufficiently reduced" may be true. However, some procedures, such as venepuncture, used to reduce cerebral blood flow experimentally, will also affect cerebral mechanisms directly *via* more than one sensory pathway. The range of sensory stimuli which may on occasion produce reflex epilepsy is wide; and the evidence for some cerebral abnormality in these cases is considerable. If such stimuli occur together with reduced blood flow, and a fit ensues, it seems arbitrary to assign the role of "cause" to the vascular change, and to assume cerebral normality.—I am, etc.,

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### Milk and Protozoal Infections

SIR,—We have read with great interest the communication on the effect of various diets on *Trypanosoma congolense* infections by Miss Audrey A. N. Keppie (*Journal*, October 17, p. 853), and we should like to add some details to the general story of milk and protozoal infections. We had not intended to publish the results below, but we feel now that the bare facts should be made known in order to prevent duplication of effort.

We have found that a pure milk diet has no suppressive activity against *T. rhodesiense* infections in rats when milk is offered three days before infection until death. The *T. rhodesiense* infestation was in fact somewhat more intense in milk-fed rats than in control normal diet-fed rats. This variance from the results obtained with *T. congolense* may be due to the swift multiplication of the human parasite in rats when compared with the more chronic strain of *T. congolense* used by Miss Keppie. The same diet effectively controlled normal and pyrimethamine-resistant strains of *P. berghei* under the same conditions.

Further, we have found that a diet of 2% casein hydrolysate in 50% glucose solution effectively suppresses *P. berghei* malaria in mice when offered in the same manner as milk. However, the effect is not so abrupt as with pure milk diets. Multiplication is permitted for six days before suppression becomes effective, but, once active, the diet reduces the parasites to negligible numbers. This is in contrast to the immediate results obtained with milk diets. The effect of casein hydrolysate diet is partially reversed by addition of *p*-amino-benzoic acid and almost completely reversed by addition of a mixture of most of the B vitamins.

May we say in conclusion that on examining all the facts we are not convinced that the inhibition of protozoal infections by milk or milk products is due wholly to a lack of any essential metabolite or of *p*-amino-benzoic acid in particular? The activity of milk as a whole is greater than such of its parts as have been tested in malarial infections, and the reversal of the effect by the addition of *p*-amino-benzoic acid is not always complete. We feel that this question is likely to be much more complex than at first it may seem.—We are, etc.,

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SIR,—I was very interested to read the communication of Miss Audrey A. N. Keppie (*Journal*, October 17, p. 853) concerning the effect of various diets on *T. congolense* infections in mice. You may be interested to learn that we have unfortunately failed to observe any such suppressive effect of a milk diet on *T. rhodesiense* infections in mice. On the contrary, we have found that animals placed on an exclusive diet of milk for a week before infection, and subsequently left on the diet, die at about the same time and with the same degree of blood infection as do animals on the ordinary laboratory diet of cubes and water. Moreover, parasites appear in the blood on the same day after infection in both groups.

So far, in fact, we have not been able to demonstrate any effect of milk on parasites other than those of malaria. For instance, whereas a milk diet gives rise to suppression of *P. berghei*, *P. cynomolgi*, and *P. knowlesi*, it has no effect on *Babesia canis* in puppies, although the latter exists within the erythrocytes.

Recent experiments have led us to believe that the suppressive effect of milk on malaria parasites may be a good deal more complicated than has been suggested. We have, for instance, noted that at about this time of the year or a little earlier the local milk may fail completely to protect rats against a strain of *P. berghei* which on other occasions has proved to be highly sensitive. It may be that this represents some change in the cow's diet reflected in the milk and arising possibly from change in pasture.

When we first reported the suppressive effect of milk in *P. berghei* malaria<sup>1</sup> we suggested that a similar phenomenon might explain the commonly reported low incidence of malaria in very young (breast-fed) infants. Since the publication of our paper I have been informed that in certain areas of Africa infection of breast-fed infants is sometimes quite common. Perhaps this may also be a reflection of the mother's diet at the time of breast-feeding. It would be possible to explain this variation in the suppressive power of milk in terms of variation in the amount of some essential factor such as *p*-aminobenzoic acid, but it seems to me too early on the evidence available to interpret the action of milk exclusively in terms of deficiency of *p*-aminobenzoic acid or any other metabolites. We have broadly confirmed