

Lymphocytes from children on the second day of the rash showed typical measles nucleoprotein tubules (fig. 1). These tubules were not seen in leukaemic lymphocytes. On the other hand, leukaemic lymphocytes showed membrane blebs on the surface (fig. 2) and besides these blebs particles were

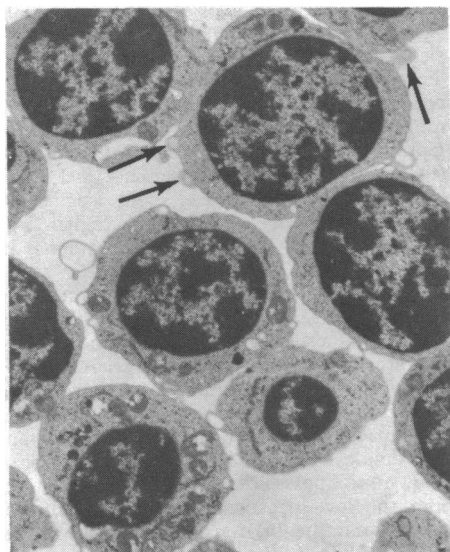


FIG. 2—Lymphocytes from a leukaemic patient. Note blebs on the surface of lymphocytes and three particles budding (arrowed) (x 4,625).

seen in various stages of budding from the surface of the lymphocytes (fig. 3A). These particles had an internal core of about 320 nm. The particles had a typical virus morphology with three concentric layers (fig. 3B). The size of the particles found in

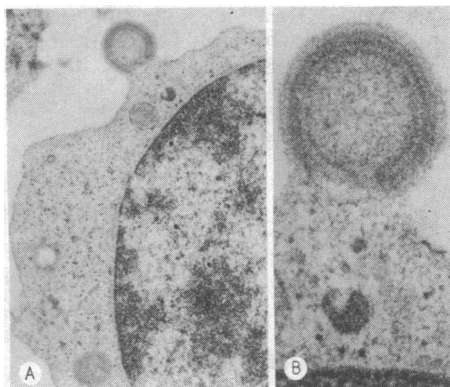


FIG. 3—A. Lymphocyte from another leukaemic patient showing virus-like particles budding from the surface (x 14,690). B. High-power view of budding particle. Note three relatively electron-dense coats with radial striation (x 48,970).

leukaemic patients was similar to that of the paramyxovirus group, but there was no evidence of internal nucleoprotein tubules.

According to the size, these particles do not appear to be virus and, on the ground of morphology, they do not appear to be mycoplasma. Though these results are repeatable their relationship to disease cannot be inferred at this stage.—I am, etc.,

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1 Hughes, D., and Caspary, E. A., *International Archives of Allergy*, 1970, 37, 506.

Idiopathic Gangrene in African Adults

SIR,—In recent times an explanation for the idiopathic gangrene of African adults has been sought in a disturbance of fibrinolysis, as exemplified by the paper by Dr. R. D. Barr and others (4 November, p. 273). These cases are as plentiful in Pretoria as they appear to be elsewhere in Africa. Over the past few years, however, we have become convinced that the mystery could be partly cleared up if the skin rash which accompanies the gangrene in a proportion of cases were correctly interpreted. The skin eruption in question is a papulonecrotic tuberculid. The acute arterial episode coincides with attacks of the skin eruption in a sufficient number of cases to indicate an important association. The skin lesion is also of a vascular, infarcting type, though the changes are on a small scale.

Papulonecrotic tuberculids have become rare in Europe, and many may feel diffident in making the diagnosis. However, in Africa one should take particular notice of rather unimpressive-looking eruptive follicular papules and pustules with a tendency to necrosis and pock-like scar formation. These may appear anywhere on the limbs, but are often gathered somewhat more closely together over the extensor surfaces of joints. The face, pinnae, and eyes (phlyctenulae) are also liable to show the lesions. A strongly positive tuberculin reaction, a lymph node focus, sometimes containing the human strain of *Mycobacterium tuberculosis*, and a prompt response to antituberculosis treatment will support the relationship. Histological examination of the papulonecrotic skin lesions, when performed by the general pathologist, yields little help.

Not much has been written about allergic vascular lesions in the subcutaneous arteries in tuberculosis. Nevertheless, there seems to be enough certainty about it for us to recommend strongly that cases elsewhere in Africa be examined with this possibility in mind. It will be interesting to learn if the unquestionably tuberculous cases also show prolonged lysis times, and how the skin and subcutaneous sites of vasculitis may compare with one another in the same case.—We are, etc.,

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Disseminating Cysticercosis in England

SIR,—We would like to bring to your notice the following case of cysticercosis cellulosa which occurred recently in this country.

On 31 August 1972 a male Hindu, aged 33 years, born in India but resident in Great Britain for the past 8½ years, was seen in the outpatient department. He had not returned to India since first arriving here and had not been abroad. He gave a history that he had had a dry cough six months previously lasting six weeks. As the cough improved he became aware of frontal headaches associated with pyrexia; investigations at the local chest clinic failed to reveal the cause of these symptoms. He was symptom-free for about two months, when his dry cough recurred and this was followed by a bout of sweating, rigors, headache, and pyrexia of 101–102°F lasting three days. A week later he was referred to the outpatient department.

On examination nothing was found abnormal clinically. The following investigations were carried out: Hb 94%, E.S.R. 20 mm in 1 hr,

W.B.C. 4,600/mm³, neutrophils 66%, lymphocytes 33%, monocytes 1%, serum albumin 4.4 g/100 ml, globulin 4.9 g/100 ml, total proteins 9.3 g/100 ml, Bilirubin <1mg/100ml, alkaline phosphatase 15 K.A. units/100 ml, thymol turbidity 3 units. Electrophoresis showed a slight diffuse increase in the gamma band and a slight increase of alpha-2 globulin, suggestive of a collagen disease.

On his second attendance at outpatients two weeks later a small, subcutaneous, freely movable, non-tender lump about the size of a hazel nut was discovered just below the right clavicle. On biopsy this was reported by Dr. W. R. Richards to be *Cysticercus cellulosae*, the intermediate stage of *Taenia solium*. The finding was confirmed by Professor G. S. Nelson of the London School of Hygiene and Tropical Medicine. X-rays of the patient's whole body failed to show evidence of calcification.

On his third outpatient attendance, after a further two weeks, six further subcutaneous nodules had appeared. He was admitted to hospital and starved for 72 hours; a Rehffuss tube was then passed to the duodenum and he was given 1 g of mepacrine dissolved in 40 ml of water, followed half an hour later by a saline purge. All his stools were collected, but no tapeworms or segments were passed.

The history of recent episodes of headache, severe sweating, shivering, and pyrexia would correspond to the generalized disseminating invasive stage of the infestation and this was confirmed by biopsy. Infestation may have resulted from (1) autoinfection in a subject already infested with *T. solium*, or (2) infection from some other person carrying *T. solium*, excreting the eggs, and contaminating food. The recent onset of the symptoms suggests that the infection was not acquired in India. As a Hindu, he denies ever eating pork in India. We failed to find evidence of intestinal infestation with *T. solium* now and thus of autoinfection. The evidence points to the infection having been acquired in this country, either from a carrier of *T. solium* or by eating infected pork. The patient admits to eating pork in the canteen at his place of work, but nowhere else. He lives alone with his wife, who is well and symptomless.

This case is reported from the point of view of the rareness of the condition in this country at the present time and of the exceptional rarity of observing a patient during the dissemination of the cysticercus in the body.

We are grateful to Professor G. S. Nelson for confirming the biopsy reports.—We are, etc.,

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Children's Wheelchair Clinic

SIR,—The experiences of Dr. K. S. Holt and others (16 December, p. 651) in running a handicapped children's wheelchair clinic at the Wolfson Centre are very similar to those encountered in other centres. Many children are failing to progress or are regressing because the expensive apparatus provided is inappropriate, improperly adjusted, or used incorrectly. The co-operation between the disciplines which Dr. Holt has achieved is a step towards surmounting some of these problems, but the training of doctors, physiotherapists, and other workers and the wider dissemination of information on available appliances will go only part of the way to improving the situation.