matches. Contact tracing has been possible with the help of a fixture list and the medical student health officer of the "opposing" hospital. The condition is known as scum pox locally.

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Why don't venereologists like prophylactic antibiotics?

SIR,—Minerva asks, "Why don't venereologists like prophylactic antibiotics?" (16 August, p 524).

My reasons for not issuing prophylactic antibiotics are: (1) Reasonable precautions against acquiring venereal infections are possible. (2) The patient may not be infected. Better a short wait to know this than an eternity of doubt because antibiotic prophylaxis was attempted. (3) Venereal diseases are not life threatening. Therefore to my mind there is no reason for what could be the indiscriminate and injudicious use of antibiotic agents to protect a patient. (4) There are a variety of venereal infections, with varying incubation periods, which may coexist and which are sensitive to different agents. There is no acceptable blunderbuss therapy which would eliminate all possible infections. (5) Until one is aware of the type or types of infection, one important function of a venereal disease clinic—contact tracing—is hardly possible. (6) The use of certain agents, while eliminating particular infections, may obscure the development of others so that, in the long term, to offer prophylactic treatment might not be in the best interests of the patient. (7) If I did ever agree to give prophylactic antibiotics I would be submerged with requests.

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Protein synthesis and breakdown after vaccination

SIR,—The attempt of Dr Peter J Garlick and others (26 July, p 263) to measure by ethical means the rates of human protein synthesis and degradation is a laudable one, but a bit puzzling. Theirs is an inferential method, as tissue sampling would be required for direct measurement of the rate of incorporation of label into protein, and of the specific activity of the labelled amino acid in the precursor pool. Any inferential method, to be valid, should produce results in agreement with those obtained by direct methods.

Dr Garlick and his colleagues write, "Rates of protein synthesis are acutely responsive to the rate of dietary protein intake . . . the rates of turnover shown . . . applied to fed subjects. Protein synthesis is *therefore* greater than breakdown . . ." (our italics). The conclusion derives only from the use of their own formula and its assumptions. In animal studies, using direct measurements of the rates of protein synthesis, it is found that there is a rhythm in the rate of protein synthesis over the 24 hours, with the highest rates at times when food intake is least, during the sleeping and resting period. Examples are protein synthesis in brain,¹ skin,² bone,³ and heart muscle.⁴ The only exception is the liver,

where the synthesis of short half-life proteins associated with the metabolism of incoming nutrients is very sensitive to food intake.

The discrepancy between the results obtained by direct methods and what Dr Garlick and others wrote of whole-body protein synthesis leaves an uneasy feeling that their indirect method may not be valid. One of their assumptions is that there is a constant metabolic pool of amino nitrogen from which amino-acids are removed for protein synthesis or are deaminated and excreted. This does not reflect the true picture—levels of plasma amino-acid vary considerably over the 24 hours and do so independently of protein intake.⁵ Amino-acids are actively transported into tissues against a concentration gradient. Plasma levels do not correlate with tissue levels, let alone with the intracellular pool of amino-acids that are charged with transfer RNA and that are the actual precursors for protein synthesis. In reality, intracellular amino-acids are again highest while food intake is least.⁶

We wonder whether the method of Dr Garlick and his colleagues is really reflecting glycine metabolism in the liver and not the whole-body protein turnover of which they wrote in their article.

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Porphyria cutanea tarda and beta-thalassaemia minor with iron overload

SIR,—We wish to comment on the recent report by Dr R W G Chapman (24 May, p 1255) concerning the simultaneous occurrence of familial porphyria cutanea tarda and beta-thalassaemia in a Turkish mother and daughter. Dr Chapman suggested that iron overload, a rare result of the thalassaemia trait, may have precipitated porphyria cutanea tarda in these patients, who were presumably genetically predisposed to the condition.

The biochemical evidence supplied to support the diagnosis of porphyria cutanea tarda in the mother is a urinary uroporphyrin level of 218 nmol (182 µg)/day, which is above normal but is very low for overt porphyria cutanea tarda in our experience.¹ Furthermore, a full porphyrin analysis of the patient is necessary to confirm the diagnosis since elevated urinary uroporphyrin levels can occur in several other conditions, including some stages of variegated porphyria,¹ where the cutaneous symptoms are identical.² In fact, if this criterion alone is used false-positives for porphyria cutanea tarda are at least as common as the disease itself; and a diagnosis of overt or subclinical biochemically positive porphyria cutanea tarda must be verified by demonstrating a marked isocoporphyrin fraction in the faeces,³ using thin layer chromatographic separation of the porphyrin methyl esters.⁴

In the daughter no excess urinary porphyrins

were detected and the diagnosis of subclinical porphyria cutanea tarda is biochemically unsubstantiated. Latent porphyria cutanea tarda is a possibility, however, but this can be diagnosed only with thin-layer chromatographic analysis of urinary porphyrins, where an unusual profile is found including uroporphyrin, 7C00H, 5C00H, and sometimes 6C00H porphyrins alongside the normal coproporphyrin, while the total porphyrin levels are not raised.^{4,5} Alternatively, some reports describe a decreased activity of the enzyme uroporphyrinogen decarboxylase in the red cells of patients with latent familial porphyria cutanea tarda.^{6,7}

We feel, therefore, that the suggestion of an association between familial porphyria cutanea tarda and beta-thalassaemia trait in these two patients is extremely interesting but cannot be justified until the above sophisticated analyses have been carried out and their results fully reported.

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The anaesthetist and the pain clinic

SIR,—I was most surprised to read in the article by Dr J W Lloyd (9 August, p 432) on the anaesthetist and the pain clinic that "tolerance to drugs, particularly the opiates, develops rapidly, and not uncommonly these patients need doses such as 120 mg of morphine every two hours. . . ." Has Dr Lloyd never read the many, many articles from the various hospices over the past few years on this very subject, repeatedly showing it to be a myth that tolerance and habituation occur if pain is properly managed? I would point him to Saunders¹ and Twycross,² to say nothing of my own modest experience³ with 71 patients suffering from terminal malignant disease. Of these, 46 required diamorphine at some stage in their illness, but 35 never needed more than 10 mg every four hours and only one needed 80 mg three hourly for a short time. None became habituated and many went home on another, much milder analgesic, if any.

I was also most amazed to read that Dr Lloyd classed morphine and diamorphine among drugs used only for non-ambulant patients. Almost all our patients were ambulant until a few days before death (and if they were not it was often for reasons other than their malignant disease) and we certainly did not withhold the powerful analgesics from patients who were ambulant. One man had been hospitalised for some time (and in bed) with severe pain from spinal metastases from a colonic carcinoma. We stabilised him on 80 mg of diamorphine every three hours and on that dose he was able to get up and dressed for