red-hot needles being inserted." The thumbs may be spared as they are less exposed to light but were affected in one patient. Both recovered completely and in the autumn one resumed tetracycline therapy without mishap.

HARVEY BAKER

Department of Dermatology,
The London Hospital,
London E1

Barium meal in uncomplicated dyspepsia

SIR,—We read with interest the paper by Dr G M Mead and his colleagues (4 June, p 1460). We have carried out a smaller study with very similar findings. Twenty-three consecutive patients referred to an emergency medical clinic (by their general practitioners) with ulcer-type dyspepsia of at least one month's duration were studied. We excluded patients with significant weight loss or a history suggestive of gastrointestinal blood loss. Alternate patients were referred for barium meal examination and all patients were given the same advice with regard to regular meals, antacids, and stopping smoking. Of the 11 patients referred for barium meal, radiological findings were normal in eight and abnormal in two (one duodenal ulcer and one hiatus hernia); one patient failed to attend for examination.

All patients were asked to attend for follow-up one month after their initial consultation. Symptoms had resolved in all 10 patients who had attended for barium meal examination. Only nine out of the 12 not referred for barium meal attended for follow-up and symptoms had resolved in all of these. There was no significant difference between the two groups in terms of either duration of symptoms (44 ± 14 (SEM) months for barium meal group *v* 42 ± 10 months for no barium group) or age (51 ± 2 years *v* 47 ± 3 years).

Our conclusion is that routine barium meal examination is of no value in the initial management of uncomplicated dyspepsia.

J R W LYALL
T E T WEST

Department of Medicine,
St Thomas's Hospital Medical
School,
London SE1

Chloroquine-induced involuntary movements

SIR,—Drs E M Umez-Eronini and Elspeth A Eronini (9 April, p 945) report involuntary movements following chloroquine administration in five adult patients. They refer to the report of amodiaquine-induced involuntary movements by Akindele and Odejide,¹ but their claim that involuntary movements in association with chloroquine treatment have not been reported previously needs reappraisal.

Bhargava *et al.*² from India, reported six instances of an extrapyramidal syndrome following medication with antimalarials, including five cases in which chloroquine alone had been given. They also referred to 28 additional cases in which extrapyramidal symptoms supervened after the administration of antimalarials along with small doses of phenothiazines. Common clinical manifestations included upward rolling of the eyeballs, retraction of the neck and back, trismus with speech difficulty, and coarse tremors.

During the past year we have encountered four instances of this syndrome in children aged 2½–12 years following chloroquine therapy for malaria. All the patients developed typical self-limited extrapyramidal manifestations 2–96 h after the administration of chloroquine. The clinical picture was similar to that of phenothiazine toxicity, but none of the patients had received any phenothiazines. The cerebrospinal fluid was examined in two cases and was normal. There was no clinical evidence of cerebral malaria in any patient.

The exact mechanism of production of these symptoms by chloroquine is uncertain because there is no structural similarity between chloroquine and other chemotherapeutic agents, such as phenothiazines and reserpine, which are known to produce extrapyramidal side effects. In view of the fact that two patients had received rather excessive amounts of chloroquine it would appear that the side effects reported are dose-related rather than idiosyncratic.

To the best of our knowledge extrapyramidal side effects of antimalarials have not previously been reported in children below 12 years of age. It needs to be emphasised that drowsiness and involuntary movements may follow the administration of chloroquine and may not be of uncommon occurrence in countries where malaria is endemic. Though the clinical picture is alarming and creates anxiety in the family, spontaneous recovery invariably occurs within 48 h.

SUNIT SINGH
P SINGH
M SINGH

Department of Paediatrics,
All India Institute of Medical
Sciences,
New Delhi, India

¹ Akindele, M O, and Odejide, A O, *British Medical Journal*, 1976, 2, 214.

² Bhargava, R K, *et al.*, *Journal of the Association of Physicians of India*, 1973, 21, 969.

Strawberry pickers' foot drop

SIR,—Two young patients presented with foot drop to the accident and emergency department of this hospital in July 1977. Both had noticed weakness and tingling in the feet following a prolonged period of strawberry picking five days previously.

One patient, a girl aged 21, had complete foot drop in one leg and a positive Tinel's sign over the lateral popliteal nerve below the head of the fibula. The other patient, a man aged 29, had bilateral weakness of the dorsiflexors of the ankle and toes with no sensory loss but with a positive Tinel's sign over the lateral popliteal nerve.

These two young people had thus developed lateral popliteal nerve palsies while picking strawberries. This appears to have resulted from local pressure on the nerve while squatting for several hours with the knee flexed and the outer part of the leg pressed on the ground with the foot extended and inverted. The girl was evidently in the same position with pressure on the affected leg for two to three hours, and recovery of her complete palsy is likely to be slow and possibly incomplete. With the man the pressure was maintained first on one side and then on the other and, as the palsies were incomplete, recovery may be more rapid in this case.

It would be interesting to know if similar cases of lateral popliteal nerve palsy due to this

cause have been encountered elsewhere among strawberry pickers. It would seem important to warn strawberry pickers of the danger of this pressure palsy and advise them to avoid squatting for long periods in the same position.

P M WESTON
MICHAEL L E ESPIR

General Hospital,
Nottingham

Paternity testing

SIR,—A passage from your otherwise excellent leading article on the above subject (16 July, p 145) might be taken to mean that any practitioner who has a problem involving paternity in his practice is at liberty to sample the individuals concerned and post off the samples to an approved laboratory for testing. This is certainly not so in my own laboratory and, I would suspect, in the majority of laboratories approved for this purpose. No samples should ever be sent without arrangements being made, preferably in writing, with the approved tester beforehand. The point is all the more important because of the biological nature of the materials involved, which means that samples arriving via the post on a day when preparations have not been made to receive and test them are invariably spoiled and the parties required to be re-sampled, which is a considerable nuisance for all concerned.

ALAN USHER
Approved tester to
the Home Office

The Medico-Legal Centre,
Sheffield

Drug metabolism in the elderly

SIR,—Dr Fatima Farah and others (16 July, p 155), using acetanilide, have demonstrated an age-related decline in microsomal oxidation rates in man. The same has been found in a number of other studies^{1–3} in which antipyrine has been administered. Although this decline could be due to aging per se, other factors that alter with age should also be taken into account.

Nutritional state is one such factor. For instance, vitamin C deficiency is known to occur in about half of the old people admitted to geriatric wards⁴ and has been shown to impair drug metabolism in man.⁵ Low folate levels are often found in the elderly in the absence of anaemia,⁶ and clinical studies suggest that folate depletion may limit microsomal metabolism of phenytoin.⁷ Finally, vitamin A deficiency impairs hepatic oxidative metabolism in rats,⁸ although there is no information about the effects of this deficiency in man.

We have measured the antipyrine half life in 36 elderly people with normal liver and renal function as judged by routine biochemical tests. None were anaemic or took drugs known to influence hepatic microsomal enzymes. The mean half life of the group was 14.6 ± 5.5 h, very similar to the 14.8 ± 5.9 h reported in a group of 84 old people in Baltimore.² However, 16 of our patients had low blood levels of ascorbic acid, folic acid, vitamin A, and vitamin B₁₂, either singly or in combination. The mean half life of these was 18.6 ± 5.8 h, significantly longer than the