Our interpretation of the practice in the two hospitals, shown by retrospective analysis, may not reflect future practice. A second opinion may not be required for every patient on a section 25 order for instance. Subsection (6) of clause 41 permits treatment without a second opinion:

(a) which is immediately necessary to save the patient's life, or
(b) which (not being irreversible) is immediately necessary to prevent deterioration of his condition, or

(c) which (not being irreversible or hazardous) represents the minimum interference necessary to prevent the patient from behaving violently or being a danger to himself or to others.

Treatment of a patient on section 25 may, therefore, be started and the patient's ability to give informed consent himself (without the need for a second opinion) may improve. The need for a second opinion (or not) is, however, predominantly in relation to section 25 as may be seen from

TABLE VI—Compulsory admissions to mental illness and mental handicap hospitals (England and Wales) in 1979

Section 25	6042
Section 26	1172
Section 29	8398
Section 30	299
Sections 60 and 65 ..	975
Section 72	89
Section 73	14
Section 135	10
Section 136	1623
Other compulsory powers ..	99
Total	18721

the statistics for England and Wales for 1979 (table VI). These figures include 191 patients admitted to the special hospitals.

It is also suggested that second opinions would mainly have to be found in large city NHS psychiatric hospitals which will ease their organisation. There are obvious manpower implications in providing this extra service to patients that will particularly affect city hospitals (and possibly the special hospitals).

The patients included in this study are mentally ill, and the need for second opinions in mental handicap hospitals is not known.

This analysis suggests that there will be difficulties in operating the proposed arrangements. Although they may not be insurmountable, the Government should consider restricting the range of treatment and procedures for which a second opinion is a statutory requirement.

We acknowledge with thanks the co-operation of the consultants at All Saints' Hospital and Highcroft Hospital, Birmingham.

This paper was accepted in evidence by the Special Standing Committee of the House of Commons on the Mental Health (Amendment) Bill on 27 April 1982.

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Lesson of the Week

Delayed diagnosis of malaria

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In May and June 1981, 14 patients were admitted to the infectious diseases unit in Glasgow with malaria. We report on two patients in whom the diagnosis was delayed. Both had taken co-trimoxazole shortly before admission.

Case reports

Case 1—A 56-year-old construction engineer was admitted two weeks after returning from a 10-day stay in Madagascar. He had worked in the bush and had frequently been bitten by mosquitoes. For antimalarial prophylaxis he had taken only three tablets of unknown type given to him irregularly by a workmate. For three days he had been shivering and sweating and had increasing lassitude, headache, and cough. He had vomited twice and complained of dysuria and frequency. Co-trimoxazole

Treatment with co-trimoxazole may delay the diagnosis of malaria

at the standard dosage was started on the day before admission. On examination he was restless but not drowsy, his temperature was 38.5°C, pulse 88 beats/min, and blood pressure 110/80 mm Hg. Coarse crepitations were heard at both lung bases, being more definite on the left. The edge of the liver was palpable 2 cm below the right costal margin but the spleen could not be felt. The marks of hundreds of insect bites were visible on his legs.

The results of initial haematological investigations were: haemoglobin 14.3 g/dl; white cell count $2.8 \times 10^9/l$ (2800/mm³) (87% neutrophils, 13% lymphocytes); platelets $25 \times 10^9/l$ (25 000/mm³). No malarial parasites were seen on a thin blood film. No thick blood film was examined because investigations are restricted in patients who may have viral haemorrhagic fever. A chest x-ray examination showed bilateral inflammatory changes and pleural thickening on the left. (Asbestosis had been diagnosed 12 years earlier.)

Because there had been some clinical improvement in his condition before admission and enteric fever was a possible

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diagnosis, treatment with co-trimoxazole was continued. But since blood cultures remained sterile and Widal titres were of a range and level consistent with TAB immunisation, tetracycline was added to cover rickettsial infection. Thin blood films made on day 3 and day 5 showed no malarial parasites on initial scrutiny, but macrophages containing pigment were seen. The film made on day 3 was then re-examined for over two hours on day 5 and a few altered gametocytes of *Plasmodium falciparum* were seen. The identification of the parasite was confirmed by the Malaria Reference Laboratory, London. No parasites were seen on a retrospective search of the blood film taken on admission though pigment in macrophages was noted. Serum from day 6 gave a strong reaction by indirect fluorescence to *P falciparum* antigens and a moderate reaction against *P feldi* antigens. Over this time his temperature had resolved by lysis (figure), but a standard course of chloroquine was still given.

Temperature chart for case 1 during treatment.

His recovery was uneventful and on day 11 platelet and white cell counts were normal. The haemoglobin concentration had fallen to 11.9 g/dl. There was no rise of Widal titres during convalescence.

Case 2—A 12-year-old schoolgirl had returned from a fortnight's holiday in Mombasa, Kenya, three weeks before admission to hospital. She had not taken antimalarial prophylaxis, though she had received TAB and cholera immunisation. She had felt hot and shivery, had had a pounding headache, lower abdominal pain, and urinary frequency, especially at night, for one week and repeated nosebleeds. She received ampicillin for four days, followed by two days of co-trimoxazole treatment before admission. She looked unwell and toxic. Her temperature was 39°C, her pulse 130 beats/min, and she had hepatosplenomegaly and mild stiffness in her neck.

The results of initial haematological tests were: haemoglobin 6.7 g/dl; white cell count $3.4 \times 10^9/l$ ($3400/mm^3$) (66% neutrophils, 32% lymphocytes, 2% monocytes); platelets $13 \times 10^9/l$ ($13\,000/mm^3$); and no malarial parasites were seen on thin blood films. The red cells showed moderate polychromasia. The cerebrospinal fluid was normal.

Two units of packed cells were transfused and chloroquine was given empirically. Her symptoms improved rapidly and her recovery was marred only by an episode of diarrhoea. The standard course of 1500 mg chloroquine base was given, followed by a single dose of pyrimethamine and sulfadoxine (Fansidar), as chloroquine-resistant strains of *P falciparum* have been reported from the Mombasa area. The diagnosis of malaria was confirmed by a rise in titre from 256 to 1024 in the indirect fluorescence test with *P falciparum* antigens. *P feldi* antigens cross-reacted strongly. After the serological results were obtained prolonged search (again over two hours) of thick and thin blood films made on admission did show a few deformed gametocytes, one trophozoite, and some macrophages containing pigment. Antibody titres to the H antigens of *Salmonella typhi* and *S paratyphi A* rose appreciably during her con-

valescence, but no O antibodies were detected. This was thought to be an anamnestic response.

Comment

We believe that the earlier treatment with co-trimoxazole delayed the diagnosis of falciparum malaria in both of these patients. Even though subsequent extended examination of blood films showed a scanty parasitaemia, this was not detected by our routine examination in a laboratory that was experienced in the diagnosis of malaria. An initial thick blood film was not made on the patient in case 1 as it was felt a viral haemorrhagic fever could not be excluded, and only limited tests were performed in a safety cabinet. We have subsequently modified this routine. It is acknowledged that even in severe falciparum malaria the parasitaemia may be light or intermittent.

Co-trimoxazole has long been known to modify and even cure falciparum malaria, whether caused by chloroquine-sensitive¹ or resistant² strains. It acts against the asexual forms in the red blood cell with little effect on other stages³ and, as happened in the patient in case 1, the fever resolves over a few days. Its role as an antimalarial drug, however, remains controversial and it is not routinely recommended for prophylaxis.⁴ Other protozoa are known to be affected by co-trimoxazole, including *Pneumocystis carinii*,⁵ *Leishmania* spp.,⁶ *Isospora belli*,⁷ non-virulent *Naegleria fowleri*,⁸ and *Toxoplasma gondii*.⁹

Doctors should therefore be aware of the possibility of modifying a large range of bacterial and protozoal illnesses when prescribing co-trimoxazole for an undiagnosed fever. Yet again the failure to communicate to travellers the need for regular malaria prophylaxis is illustrated.

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What is the action of powdered charcoal in the gut and what, if any, are the long-term side effects of taking this substance?

Charcoal powder is an adsorbent for many drugs, toxins, inorganic compounds, and gases. It has been used to adsorb gases when treating flatulence and intestinal distension; and activated charcoal is used in the emergency treatment of poisoning to prevent the absorption of poisons from the alimentary tract. Charcoal is rarely used long term, but I would expect it to interfere with the absorption of dietary factors such as vitamins and minerals, and some drugs administered by mouth. The preparations of activated charcoal currently used to treat poisoning have no reported side effects.—LINDA BEELEY, consultant clinical pharmacologist, Birmingham.