appearance of this rash was considered compatible with erythema marginatum. On the seventh day she noticed painful, swollen ankles and wrists. Investigations on the seventh day showed a haemoglobin concentration of 117 g/l, a white cell count of $14.1 \times 10^9/l$ (with a neutrophilia), and an antistreptolysin O titre of 1200 IU/l. She received erythromycin 500 mg four times daily and aspirin with symptomatic improvement.

A recrudescence of her arthropathy, however, required hospital admission on the 14th day of the illness. On admission she was afebrile with swollen and tender wrists and ankles. A mid-systolic cardiac murmur was heard on auscultation. Investigations showed a haemoglobin concentration of 112 g/l, white cell count of $12.9 \times 10^9/l$ (with continued neutrophilia and reactive lymphocytes), antistreptolysin O titre of 800 IU/l, anti-DNAase B titre of 1200 IU/l, and erythrocyte sedimentation rate (Westergren) of 123 mm in the first hour. A throat swab did not grow any pathogens, and an electrocardiogram and echocardiogram were normal. Rheumatoid factor, antinuclear factor, and autoantibodies were not detected. She was treated with penicillin 500 mg four times daily, bedrest, and aspirin and improved over the following four weeks. The erythrocyte sedimentation rate fell to 45 mm in the first hour by the time of her discharge. Repeated electrocardiograms and echocardiograms were normal. Further history taking elicited the fact that one grandparent and an aunt had died of cardiological sequelae of rheumatic fever.

This woman's illness meets the revised criteria for acute rheumatic fever.¹ As in many of the recent cases in the United States this patient was middle class.² Many of the United States cases also occurred in adults.³ The family history of relatives affected by rheumatic fever suggests that she may have been predisposed to the disease. She had four young children, who may have been a source of rheumatogenic streptococci. It is not only paediatricians who should be alert to the recrudescence of rheumatic fever. General practitioners and doctors practising adult medicine should also be diligent in their approach to streptococcal pharyngitis and tonsillitis.

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SIR,—Dr J G Bissenden (30 April, p 1215) thinks that we have "gone soft" on the streptococcus. The evidence is the other way round, that the streptococcus has gone soft on us. It is not substantiated that antibiotics helped the decline of acute rheumatic fever,¹ and social factors seem to have been more important.

The streptococcus can be isolated from 30% of throat swabs from patients with sore throats but also from 10% of healthy controls. It certainly is unusual to treat sore throats with intramuscular penicillin followed by 10 days of oral penicillin. Such treatment makes little difference to the duration of the illness whether or not it is caused by the streptococcus, and, more importantly, it

does not seem to influence the development of rheumatic fever² or acute glomerulonephritis.³

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SIR,—Dr J G Bissenden (30 April, p 1215) deals with a future possibility rather than the present situation in Britain. Studies in general practice in Britain provide little support for the treatment of sore throats with antibiotics. The speed of recovery¹ or risk of rheumatic fever² seems to be uninfluenced by this, and, while I accept that disease patterns change, mucoid strains of group A streptococci, which are possibly "rheumatogenic," do not seem to be important pathogens here.

In the Salt Lake City cases discussed by Dr Bissenden fewer than one third of patients complained of a preceding sore throat.³ Throat swabs do not exclude the carrier state, which may not be associated with a systemic immunological reaction. In 1987 and 1988 between 24 and 37 patients attended this practice each week complaining of an illness in which sore throat was a major feature.⁴ This is equivalent to a full morning's surgery list for a general practitioner in a partnership of seven, and we think that education of patients to deal with minor symptoms without recourse to a doctor is important. No major complications were seen, and the return rate of patients was uninfluenced by whether antibiotics were given.

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Acute appendicitis and social class

SIR,—Professor D J P Barker and colleagues (2 April, p 953 and 956) reported a correlation between the incidence of appendicitis, as measured by appendicectomies, and the availability of certain household amenities and consumption of vegetables. Their conclusions are taken to support the hypothesis that "appendicitis is primarily caused by Western housing rather than by Western diet."

Their indicator of the incidence of appendicitis is actually the rate of appendicectomy, and it may be worth considering other possible explanations. Some years ago we reported a strong familial predisposition to appendicectomy, which we later interpreted in terms of a social class bias in classes I and II towards appendicectomies.¹ We drew attention to a paradoxical bias of "chronic appendicitis" in classes IV and V as suggestive evidence that it was the operation and not the inflammation that was social class related.² The same factor could explain the familial clustering of appendicectomies and the housing and dietary correlations reported by Professor Barker and colleagues.

In the absence of detailed understanding of the social class selection to appendicectomy or the housing and dietary characteristics of children who

suffer appendicitis but do not present for surgery it would be useful to know the social class composition of children whose appendicectomies confirmed histological appendicitis compared with those with negative histology. Community rather than hospital based studies may help to take into account potential selection to medical attention by social class.

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Malaria prophylaxis

SIR,—Dr C J Ellis states that thrombocytopenia is an argument for empirical treatment of febrile patients from areas where malaria is endemic (2 April, p 952.) He also states that all patients who have taken a prophylactic regimen that includes chloroquine must be treated with quinine, however mild the illness. This opinion is shared by Drs E L C Ong and E M Dunbar (30 April, p 1259).

It is unwise to treat a patient with falciparum malaria with any drug that has been used for prophylaxis. The mere fact that the patient has developed malaria despite prophylaxis indicates that the efficacy of the drug used is uncertain.

Although thrombocytopenia is present in 80% of all patients with malaria,¹ its predictive value is not established as 15% of febrile patients who are not suffering from malaria but come from endemic areas also have thrombocytopenia (unpublished observation). Empirical treatment for malaria would sometimes lead to non-treatment of serious bacterial diseases. Thrombocytopenia is present in 35% of patients with septicæmia and endocarditis.² Empirical treatment might also lead to less vigilance in the search for a correct diagnosis and could be justified only in the rare cases when microscopic examination of a thick blood film is impossible and the symptoms are suggestive of malaria.

We do not agree with Dr Ellis, who would give quinine to all febrile and thrombocytopenic patients taking chloroquine prophylaxis, or with Drs Ong and Dunbar, who advocate that quinine should be used to treat falciparum malaria without discussing other alternatives available outside the United Kingdom.

Treatment with quinine at the recommended dose of 10 mg/kg three times daily for 10 days is almost invariably associated with tinnitus, vertigo, and dizziness. The patient can rarely return to a normal life before the treatment ends. On the other hand, treatment with sulfadoxine and pyrimethamine is given as a single dose. This drug is still largely effective against African *Plasmodium falciparum* and can be given to patients who are not seriously ill. Side effects are few but might be serious, and all patients who are treated with this combination should, therefore, be asked about previous side effects from sulphonamides. When treatment with sulfadoxine and pyrimethamine is not acceptable patients with mild disease should be given a single dose of mefloquine rather than quinine. Nausea and vomiting often occur after mefloquine³ but these wear off within 24 hours, after which the patient can look forward to an uneventful convalescence.

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SIR,—The points raised by Drs E L C Ong and E M Dunbar (30 April, p 1259) have a wider application as illustrated by the following case.

A 39 year old woman from the Indian state of Gujarat presented with a four day history of myalgia, anorexia, sweating, vomiting, and bilateral frontal headache two weeks after entering Britain. There were no respiratory or abdominal symptoms, and her bowel habit was normal. Her husband and three children were all well.

A week previously an abscess on her left index finger had been treated successfully by drainage, and flucloxacillin had been given after a sensitive *Staphylococcus aureus* was isolated from the site. Two months previously falciparum malaria had been diagnosed and treated successfully with a five day course of chloroquine followed by primaquine, which she had taken until shortly before leaving India. Her symptoms then were identical with those she now presented with.

She was dehydrated, with a tachycardia of 120 beats/minute, and a fever of 38.8°C. There was no rash, and examination of her abdomen showed no abnormality. A clinical diagnosis of malaria was made, and a blood film was examined to confirm the diagnosis. No parasites were visible, however, and there was no thrombocytopenia. In contrast, a blood culture grew type A *Salmonella typhi*. Her condition improved on treatment with intravenous chloramphenicol followed by a 15 day course of oral treatment.

This case shows the importance of establishing a clear diagnosis on objective grounds before starting treatment. An unsubstantiated and presumptive diagnosis ignores the way in which infectious illnesses often mimic each other.

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Complications of nasogastric feeding

SIR,—Dr N C Hickey's and Miss D C T Watson's article (9 April, p 1038) prompts us to report two cases illustrating unusual complications of the use of nasogastric tubes.

A 19 year old man was admitted with a severe head injury after a road traffic accident. He was unconscious, and a computed tomogram of his brain showed a right sided intracerebral haemorrhage. Enteral nutrition was started five days after admission. A 12 gauge nasogastric tube was inserted and its position confirmed radiographically. Ten days later complete blockage necessitated removal of the tube, which was found to be knotted 3.5 cm from the tip. A 7 gauge tube was passed and correct placement confirmed. Fourteen days later this tube was also found to be knotted distally. A third tube presented no further complications.

A 20 year old girl with severe cerebral damage after an insulin overdose was admitted unconscious to the intensive care unit. She required artificial ventilation. Nasogastric feeding was started and proceeded uneventfully for three months using a series of 9 gauge tubes. Drugs given through the tube included phenytoin, baclofen, dantrolene, and methylcellulose. During

the fourth month a tube that had been in situ for 32 days fell out, and the distal end was noted to be missing. An abdominal radiograph showed the broken piece in the right iliac fossa (figure), and it was passed in the stool two days later. Four months later a blockage occurred and the tube was withdrawn. No obstruction was found, but there must have been a functional obstruction as the proximal tube had ballooned to three times its original diameter owing to pressure from the irrigating syringe.

Radiograph of pelvis showing broken end of nasogastric tube

Serious complications from malplacement of nasogastric tubes have been reported,^{1,2} but complications such as we describe do not appear in published reports, though anecdotes from colleagues abound.

Knottting of the tube is likely to be related to the length of tube in the stomach, and care should be taken that the tube is not so long that it forms coils which may precipitate knot formation. Ballooning and spontaneous breakage are more serious complications and imply structural damage to the tube itself, possibly caused by preparations given through the tube or by the acid environment of the stomach. Most manufacturers recommend that tubes are changed every two to four weeks, and in our second case this upper limit had been exceeded by four days. Enteral products and drug additives may block nasogastric tubes because of their viscosity or precipitation.^{3,4} Little is known, however, of the structural damage caused to the tube by such products. It is important that the manufacturers' instructions about regular replacement and flushing are adhered to.

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Controlling symptoms in advanced cancer

SIR,—Dr P L C Xavier's view (23 April, p 1196) that four hourly administration of oral liquid morphine for severe cancer pain is passé would not, I believe, find general agreement among doctors working in hospices. Titration of dose against pain is easier and quicker with liquid

preparations, and my experience as doctor to a domiciliary hospice team is that patients readily learn to do this for themselves if given initial clear and simple advice. Once stable pain control is achieved a change to slow release tablets is made easily if the patient finds this more convenient. There is a place for both types of treatment.

Similarly, one should not be too dogmatic about the choice of antiemetic. The cause of the vomiting should be diagnosed wherever possible and the most appropriate drug selected. Intractable vomiting often requires the use of a subcutaneous infusion pump, and in this case phenothiazines are best avoided because of the troublesome skin reactions they cause at the site of the infusion.

It was surprising that neither Drs T D Walsh and T S West (13 February, p 477) nor Dr Xavier mentioned the problem of anxiety in patients with cancer. This seems to be a universal problem, whatever the stage of the disease, although it is rarely expressed overtly. Treatment with anxiolytic drugs and counselling are important and often bring dramatic relief of other symptoms, most notably pain and dyspnoea.

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Prevalence of *Gardnerella vaginalis*

SIR,—Dr R R West and colleagues (23 April, p 1163) do not state whether vaginal swabs were submitted for examination or culture for organisms such as *Trichomonas vaginalis* or *Candida albicans*. An unspecified number of women was investigated for chlamydial disease, but no results for these investigations were given.

The role of *Gardnerella vaginalis* or the possible contribution of anaerobic organisms in the causation of bacterial vaginosis remains controversial.¹ Indeed, the authors report that anaerobes were more likely to be cultured from symptomatic women with *G vaginalis* infection than from those without symptoms. They recommend the use of side room tests such as the amine volatilisation test or microscopic examination for clue cells, yet these investigations have been shown to yield significant numbers of false positive and false negative results.²

An association between vaginal *G vaginalis* and serious long term sequelae such as infertility has not been shown. Suggesting that patients may require treatment for a "covert vaginal discharge" when assessment of vaginal discharge by patients (or doctors) is unreliable^{3,4} and prescribing antibiotics after the results of non-specific side room tests for an unproved pathogen are difficult to justify.

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AUTHORS' REPLY—The aim of our study was to establish the prevalence of *Gardnerella vaginalis* in