Formulation of a larger-strength methotrexate tablet would permit larger oral doses to be given as we have found that there is a physical limit to the number of tablets which can be taken during one day.

R BELL
J R SULLIVAN
W J MOON
J MARTY
J SHAW

Haemato-oncology Group,
Royal Melbourne Hospital,
Melbourne, Victoria

The battered fetus

SIR,—May I please inquire through you why it is that the battered fetus seems never to be a subject of concern comparable to the battered baby, the maltreated child, or the battered wife or granny. If the screaming of a young infant is considered provocative to a violent man, then surely so is the gravid uterus, and many battered wives must have had their abdomens attacked during pregnancy by frustrated or suspicious men.

I would like to know if bruising of the abdominal wall is observed during antenatal visits to maternity hospitals as frequently as black eyes are in mothers attending with child outpatients and how often trauma is considered as a contributory cause of antepartum haemorrhage and premature labour. Bizarre congenital deformities of the skeleton such as acute angulation of a tibia, genu recurvatum, or congenital dislocation of a wrist ought to prompt us to inquire about "accidents" such as falls down stairs or motoring injuries, and alternative explanations may be disclosed. It was certainly the opinion of the mothers of two babies whom I have attended that their grotesque tibial deformity was directly associated with extreme violence to the abdomen in mid-pregnancy, and a third mother (with whom I am still in touch) is equally convinced that her daughter's genu recurvatum hip dislocation was associated with an opacity of the sclera and anterior chamber of the eye on the same side as a result of repeated kicks and blows directing the infant's own foot into the eyeball.

It may be that in compiling a comprehensive list of significant "life events" which could have some bearing on the pregnancy we have been too squeamish and should include direct assault with boot or fist.

R J PUGH

Department of Paediatrics,
Hull Royal Infirmary,
Hull, Humberside

Otosclerosis and the operating microscope

SIR,—I agree with Dr I S Hall (11 February, p 367) that Julius Lempert did not use an operating microscope as I doubt if one existed in the early 'forties. In 1947, in association with Messrs Hutchisons of Edinburgh, I developed an operating microscope, and a description of this was published in the *BMJ*.¹ So far as I am aware this was the first operating microscope used in clinical practice.

The fenestration operation was developed by Maurice Sourdis of France and by Professor Gunnar Holmgren of Stockholm, from whom and his wife I have received great hospitality during visits to Sweden both before and after the war. He must certainly be regarded as the

main pioneer of the operation, but I have the impression, more than 30 years later, that, while he used a microscope, this was not an operating microscope but was used for examination of the fenestra after it was made. I do not think that the working distance of his microscope was long enough to allow operative procedures or if there was sufficient illumination. I think that this impression is probably confirmed by the fact that after the publication of my description of the microscope I had a letter from Professor Holmgren's successor, the late Dr Paul Frenckner, asking me where he could obtain an operating microscope. At this time two operating microscopes were being produced in Britain, one by Messrs Beck and the other by Messrs Cook, Troughton, and Simms, of York.

The prototype of the microscope which was developed here has been presented to the museum of the Royal College of Surgeons of Edinburgh.

A BROWNIE SMITH

Edinburgh

¹ Smith, A B, *British Medical Journal*, 1947, 1, 776.

"Chickenpox oesophagitis"

SIR,—The letter from Dr K D Bardhan (11 February, p 370) on oesophageal involvement during chickenpox provided me with some (admittedly) macabre amusement, as well as a sense of relief. Dr Bardhan was unable to find any references to oesophageal involvement in chickenpox, even with the assistance of a computer. Involvement of the oesophagus by varicella was reported in 1940 by Johnson,¹ and the lesions varied from (microscopic) early bullous lesions to massive ulcers. I am relieved that Dr Bardhan, an old friend, recovered since Johnson's report deals with a fatal case and I have had the opportunity, in recent years, to study an almost identical case.

The moral of the story, if one exists, is that computers are *not* infallible and what you don't know won't hurt.

D J DESA

Department of Pathology,
McMaster University,
Hamilton, Ontario

¹ Johnson, H N, *Archives of Pathology*, 1940, 30, 292.

Effect of penicillamine after gold treatment

SIR,—We have long suspected that patients with rheumatoid arthritis (RA) who have previously received gold are under increased risk with side effects from penicillamine.¹ This patient's case adds weight to our hypothesis.

A 36-year-old woman with definite RA was seen in 1964. She was treated with gold, which produced a remission. The gold had to be stopped after 950 mg because of an itchy maculopapular rash affecting her loins and her lower abdomen anteriorly.

She remained well until 1975, when she again developed pain in her fingers and feet, the fingers starting to be deformed. The fingers and hands showed changes of typical RA, the erythrocyte sedimentation rate being raised at 26 mm in 1 h. The symptoms remained well controlled with a simple analgesic and anti-inflammatory drugs until June 1976, when she became worse and alclofenac was started. This almost immediately improved her until November 1977, when she became very much worse, with gross swelling of her fingers and ankles. At this stage penicillamine in the small dose of 50 mg daily was substituted for alclofenac.

After 12 days—that is, after a total dose of only 600 mg of penicillamine—she again developed a rash on her trunk which was described as being identical in character and site with the rash which she had experienced on taking gold. The rash took a month to clear; it is of great interest that over that month the RA apparently completely remitted and she returned to full function and was completely free of stiffness when she awakened in the morning.

This clinical situation, although anecdotal, seems to us to be more than a coincidence and we feel it likely that the very small amount of penicillamine that she received had mobilised gold from her tissues which led to the remission.

JOHN GOLDING
ALBERT T DAY
M A WILSON

Harrogate Royal Bath Hospital,
Harrogate, Yorks

¹ Golding, J R, and Day, A T, *British Medical Journal*, 1973, 2, 593.

Perhexiline-induced neuropathy

SIR,—Your leading article on "Drug treatment of chronic stable angina pectoris" (25 February, p 462) rightly emphasises that neurological changes occurring with perhexiline maleate therapy are reversible but that "the ratio of benefit to risk has to be calculated carefully before this effective antianginal drug is prescribed for long-term use."

Our experience of 10 patients with perhexiline-induced neuropathy supports the view that this complication is reversible. The first three patients have previously been described in detail.¹ The diagnosis was made in each case on the basis of clinical features confirmed by objective tests (nerve conduction velocity and autonomic function tests), with improvement when the drug was withdrawn. Of these 10 patients, eight were men and two women; their ages ranged from 49 to 68 (mean 57.8 ± 5.4 years). Perhexiline was prescribed if they were unable to tolerate conventional treatment with beta-blockers or if their anginal symptoms were uncontrolled by this treatment alone. All 10 patients had peripheral neuropathy and five patients in addition had evidence of autonomic neuropathy (postural hypotension, abnormal autonomic function tests). Two patients were taking perhexiline in a dose of 200 mg/day and the remaining eight taking higher doses (300–400 mg/day). The duration of therapy ranged from 3 to 18 (mean 10.3 ± 5.0) months before presentation with neurological impairment.

In all cases the drug was withdrawn and complete recovery occurred over a period of 3–8 months. Clinical recovery was fairly rapid, but return to the normal range of nerve conduction velocity was more prolonged. Only one patient had another condition (diabetes mellitus) which may have contributed to the development of neuropathy, and the features in this patient also reversed on withdrawal of the drug. Two patients have subsequently died from ischaemic heart disease but the other eight remain well. The use of perhexiline in these patients had been successful in controlling severe anginal symptoms initially and there was marked deterioration in symptoms when the drug was withdrawn.

We have used perhexiline now in many cases in similar doses and for prolonged periods without the development of neuropathy. We feel, therefore, that this drug has an important place in the management of patients