approach to ocular injury is most necessary. But that surely is a matter for the technicians, and in your description of the ideal modern armamentarium this is underlined. It is, however, appropriate to point out that very effective reparative surgery can be carried out without miniature motorised rotary vitrectomy instruments, ultrasonography and, except in very specialised cases, immediate penetrating corneal grafts. To make such things a sine qua non of reparative ocular surgery can only, in the broader sense, do disservice to the ultimate consumer, the patient. As always, "should" is an even better moderator than "can."

M J GILKES

Sussex Eye Hospital,
Brighton

SIR,—While in no way detracting from the general improvement advocated in the treatment of eye injuries in the leading article (14 May, p 1237), I do not like the impression that the consultant's main job is to deal with such cases. One expects a consultant to be busy enough with cold surgery and consultation on problem cases not to have this disrupted unnecessarily by traumatic emergencies. The training of one's juniors in the use of fine instruments is very largely achieved by their dealing with the traumatic case, and one would not like to deprive them of this or give them the impression that they were not capable of dealing with it. I have seen many perforated eyes heal up excellently without surgery and others go wrong in spite of, or even because of, meticulous surgery. With all the intricate apparatus at one's disposal these days there is a real danger of overtreatment.

JOHN PRIMROSE

Oldchurch Hospital,
Romford

Oesophageal ulceration due to clindamycin

SIR,—Delayed passage of tablets may cause oesophageal ulceration, especially in the presence of disordered motility.¹ This complication has been reported after the ingestion of aspirin, tetracycline, emeptronium bromide, potassium, and Clintest tablets.¹⁻³ We report oesophageal ulceration after oral clindamycin (Dalacin C).

A 22-year-old housewife, who had no previous dyspepsia, developed substernal pains after swallowing a 150-mg capsule of clindamycin given for a paronychia. A drink of water was subsequently taken for relief, but her symptoms increased, so that there was complete dysphagia for solids and continuous pain. At fiberoptic endoscopy one week from the onset of symptoms there were two necrotic ulcers with surrounding hyperaemia on the anterior and posterior oesophageal walls, 25 cm from the incisor teeth. Distally the mucosa appeared normal and there was no evidence of a stricture or hiatus hernia. After 10 days on a bland diet and Mucaine she was almost symptom-free. Oesophagoscopy after two weeks showed complete healing.

Upper gastrointestinal symptoms associated with clindamycin therapy are uncommon.⁴ Pharyngeal ulceration following clindamycin therapy has been reported,⁴ presumably associated with lodging of the capsule in the pharynx. Capsules would not normally be

expected to cause mucosal damage during oesophageal transit. However, barium sulphate tablets remained in the oesophagus for between five and 10 minutes in 57% of patients studied by Evans and Roberts,¹ many of whom had no oesophageal abnormality. Delayed passage through the oesophagus may have allowed the clindamycin capsule to dissolve and prolonged mucosal contact caused ulceration. This suggests that capsules as well as tablets should be taken with a meal or followed by a glass of water.

D R SUTTON
J K GOSNOLD

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¹ Evans, K. T. and Roberts, G. M. *Lancet*, 1976, 2, 1237.

² Habeshaw, T. and Bennett, J. R. *Lancet*, 1972, 2, 1422.

³ Kavin, H. *Lancet*, 1977, 1, 424.

⁴ Committee on Safety of Medicines, Adverse Reaction with Clindamycin. Total Reports 1964-75.

Premature baby statistics

SIR,—Dr R R Gordon's statistics on the survival of premature babies (21 May, p 1313) are interesting but can be no more. For it is impossible to know whether we should upbraid him or congratulate him for not reaching the survival figures from other hospitals without a long-term follow-up to indicate whether the survivors achieved a satisfactory life style.

Earlier this week I was concerned with the management in a residential school of a young epileptic girl who is now well on the way to a lifetime of residential care. Her birth weight was under 2 lb (0.9 kg), and about a third of that of her twin, who died. Was the survival of this tiny baby a triumph of neonatal paediatrics or a social and family disaster which medicine, having presided over the origin, is happy to pass on to others now that the problems are becoming serious?

So long as authors write and you, Sir, publish articles which mention survival while ignoring the quality of life, then so long will medicine deserve the strictures of, say, Ivan Illich. As Dr Gordon's statistics go back so far, would he consider investigating what happened to the children born in the early years of his survey period?

C P TREVES BROWN

Fairfield Hospital,
Hitchin

Severe thrombophlebitis with naftidrofuryl oxalate

SIR,—I refer to the paper by Mr C R J Woodhouse and Mr D G A Eadie (21 May, p 1320). In Manchester a trial of the use of naftidrofuryl oxalate (Praxilene) against placebo in rest pain is in progress at the Royal Infirmary and at Tameside General Hospital. At the Royal Infirmary a continuous drip is used until the site needs to be changed. Thrombophlebitis is frequent, and the housemen think they may be able to guess which patients are on the active drug by the incidence of this complication. At Tameside General Hospital I use Butterfly 21 (Abbott Laboratories) needles, giving the infusion (200 mg in 500 ml dextrose/saline) over two hours, keeping the line open with saline until the second infusion is due, and removing the cannula in the early evening. I have had no trouble with thrombophlebitis either in the trial patients or

those receiving Praxilene for other reasons. With care the same vein may be used on successive days. I would therefore recommend this method of administration to avoid thrombophlebitis and to allow the patient freedom from a drip or indwelling cannula during the night.

MICHAEL GANN

Tameside General Hospital,
Ashton-under-Lyne

Fibrinous peritonitis

SIR,—Fibrinous peritonitis occasionally follows treatment with practolol. Its occurrence after treatment with other beta-blocking agents which have not been preceded by practolol seems not to have been reported.

We describe a patient who had a fibrinous peritoneal reaction. She had had a number of drugs including oxprenolol (Trasicor), but not practolol. A 50-year-old woman was found to have a gastric ulcer in 1967 at another hospital. In 1971 she was first seen at this hospital and treated with carbenoxolone successfully. Associated anginal pain was managed with glyceryl trinitrate. In February 1973, when she attended for follow-up, she complained of chest pain on exertion and was given Trasicor 10 mg tds. Three months later the chest pain was better, but she is said to have thought that Trasicor did not suit her. The drug was stopped on 29 May 1973. In February 1976 the gastric ulcer again gave trouble and was treated with potassium citrate bismuthate (De-nol) 5 mg qds for four weeks. In March 1977 the patient was found to have a rectal neoplasm. At operation for this the entire contents of the abdomen were bound together with filmy adhesions such as are found after treatment with practolol. The operation was completed with some difficulty.

After extensive inquiries we are satisfied that this lady never received any other drugs than the ones mentioned and certainly never received practolol. Clearly the case is not proved that these adhesions were caused by the Trasicor, but there is at least quite a possibility.

S C KENNEDY
MARY DUCROW

East Birmingham Hospital,
Birmingham

Intrauterine fetal transfusion

SIR,—In advancing an unconvincing case for a controlled trial between plasmapheresis alone and plasmapheresis with intrauterine transfusion for the management of very severe Rh haemolytic disease the writer of your leading article (16 April, p 990) referred to a paper by my former colleagues and me.¹ He cited our report as evidence that "the neonatal mortality at 32 weeks in infants with untreated severe Rh haemolytic disease is 40%." We made no such statement, nor any other comment that might be misinterpreted in this way. Indeed, since we try to treat the treatable (although not always with success), we have no experience at all of untreated severe Rh disease.

The leading article quotes from a recent report by Palmer and Gordon,² who referred to an earlier statement by Fairweather *et al*³ that, before the introduction of intrauterine transfusion, patients with liquor bilirubin

ratios above 1:1 at about 32 weeks "would probably have been induced at 32 weeks' gestation; but combining such serious prematurity with severe haemolytic disease had resulted in almost a 40% neonatal mortality." It seems that you misread this very easily understood statement. It is a pity that your readers should be thus misinformed.

C R WHITFIELD

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¹ Whitfield, C R, *et al*, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1972, **79**, 931.

² Palmer, A, and Gordon, R R, *British Journal of Obstetrics and Gynaecology*, 1976, **83**, 688.

³ Fairweather, D V I, *et al*, *British Medical Journal*, 1967, **4**, 189.

*We regret an error in the reference numbers. The statement in our leading article that "most series treated by intrauterine transfusion alone record a 40% survival" should have referred to the paper by Professor Whitfield and his colleagues,¹ and the reference to the neonatal mortality at 32 weeks is, of course, derived from Palmer and Gordon's paper.² The report by Palmer and Gordon does raise serious doubts about the value of intrauterine transfusion, and we consider that a re-examination of its value in relation to the new development of plasmapheresis is indicated. This conclusion is independent of any discussion of the exact mortality figures of the untreated disease, which we agree are difficult to obtain.—ED, *BMJ*.

Overheated wards

SIR,—Why are hospital wards invariably overheated? It seems paradoxical that patients, cured of their life-threatening illnesses, are becoming endangered by that fiendish species—the NHS radiator. All too often I have witnessed prostrate patients perspiring away their water and electrolytes, which are enthusiastically replaced intravenously by harassed house officers who fail to understand why their patients are so dehydrated. Please, before this summer's heatwave, will someone in the heating department have a brainwave, and economise?

ANDREA HEMLOCK
Medical student

Birmingham

What to tell the employer

SIR,—Dr John M Goldman in his letter (7 May, p 1221) raises some very interesting points, and I feel the following personal case is probably worth reporting.

Some years ago an old student of my own, who now was an assistant medical officer of health in a certain county in England, came to see me when on leave. I was horrified to find that he had an advanced cancer with secondaries in his liver, and all that one could do to relieve his obstruction was to bypass it, but this of course did not in any way prevent the fatal onset, which was only going to be 2, 3, 6 months ahead. While the young man was in hospital I had a letter, in confidence, from the chief medical officer of the county asking *in confidence* for the medical details of the case. I replied very fully, again in strict *confidence*, pointing out that the man had less than some six months to live. I got no acknowledgment

of my letter, but one can guess the reaction when my patient some weeks later read in the *Journal* that his appointment was now being advertised. I don't think I have ever had such a case of bad faith, and it naturally has made me very careful of what to tell the employer.

IAN FRASER

Belfast

What to tell the occupational physician

SIR,—I was distressed to read Dr J M Goldman's letter (7 May, p 1221), in which he discussed communication with occupational physicians, such as me, and even more distressed to read the headline "What to tell the employer." Please let me emphasise that a consultant or a family doctor corresponding with an occupational physician is *not* corresponding with an employer any more than I am corresponding with the area health authority when I write to a consultant. Dr Goldman's first paragraph contains the offensive suggestion that occupational physicians are not primarily concerned with the welfare of their patients and do not preserve medical confidence. And what would Dr Goldman say if I were to write, "An inquiry from an oncologist can sometimes best be answered by a telephone call—one has then the opportunity to satisfy oneself about the doctor's essential good faith"?

I suggest that Dr Goldman refers to the BMA publication *Medical Ethics*, 1974, p 31: "The doctor in occupational medicine and the general practitioner have a common concern—the health and welfare of the individual workers coming under their care. Less often, this concern may be shared with the hospital doctor, the community physician or some other professional colleague. As in all cases where two or more doctors are so concerned together the greatest possible degree of consultation and co-operation between them is essential at all times—subject only to the consent of the individual concerned."

No person, neither employer nor doctor, is entitled to any medical information whatever from a doctor about an employee or a candidate for employment without the consent, explicit or implied, of the patient. No occupational doctor will tell an employer anything without such consent, and even with consent he will tell the employer only what he properly needs to know. So when an occupational doctor asks, with the consent of the patient, for detailed medical information it is entirely proper for him to be given it. It is his job, in which he is a specialist, to interpret that information honestly and in the interest of his patient.

Dr Goldman's second paragraph discussed what should be told an employer who asks for a patient's medical history. He says that a prospective employer is not entitled to full medical information but "he is (presumably) entitled to be forewarned against the risk of taking on an employee who will very soon be incapacitated. . . ." He goes on to say that the patient's own doctor may properly give a favourable report without the consent of the patient. Few doctors I hope would be prepared to give any information, favourable or unfavourable, without the consent of the patient. A moment's reflection would make it plain that, if it were permissible for a doctor to report favourably without consent, his refusal to report could immediately and rightly be

assumed to be equivalent to an unfavourable report. Dr Goldman also suggests that the patient's family doctor and not the occupational physician should decide in all cases whether a man will be accepted into employment as fit to do any particular job. I find this a very strange suggestion, and one unlikely to appeal to many prospective employers. Dr Goldman's suggestions are—shall we say?—ingenuous, but then if I were to address you on the chemotherapy of leukaemia I would hardly do better.

R E W FISHER

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Hot flushes and cold turkey

SIR,—The recently increased interest in the menopause encourages me to draw attention to the similarity between features associated with this state and those related to withdrawal of drugs of addiction or dependence. The characteristic features of the menopause are classified into three groups: autonomic instability, emotional and psychological instability, and biochemical changes. Of these the first two are also prominent characteristics of the addicted subject who is exposed to acute, or even phased, denial of his narcotic, nicotine, or alcohol. That the autonomic instability is in the one instance typified by "hot flushes" and in the other by "cold turkey syndrome" could be reflective merely of the different sites of activity of the agent of dependence. If relief of the emotional and autonomic symptomatology associated with the menopause is not completely provided by substitution therapy, this could indicate simply that the critical compound was an apparently minor metabolite of one of the female sex hormones.

A similar picture is surely to be seen in the puerperal subject. Indeed, I have for some years referred to the puerperium as the "mini-menopause," and would respectfully suggest that those intensively engaged in investigation of the menopause might benefit from a consideration of the postpartum patient as a "model" for study.

J SELWYN CRAWFORD

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Oral contraceptives and the uterine vessels

SIR,—Your leading article on pregnancy and oral contraceptives and the uterine vessels (30 April, p 119) asks if oral contraceptives cause disease of the uterine arteries.^{1 2} We have previously found that changes in small uterine vessels, those in the endometrium, are one of the earliest effects of taking oral contraceptives. Proliferation of endometrial arterioles is related to headache and migraine incidence,³ distended sinusoids to tiredness and dilated leg veins, and stromal condensation round sinusoids to leg cramps.⁴ Sinusoid changes and headaches usually precede development of thrombosis. One patient had particularly large sinusoids after 43 years of treatment. She had developed migraine for the first time in her life during cycle 40 and bilateral phlebitis in cycle 43.

There has seemed little doubt to me that oral contraceptives cause generalised vascular over-reactivity, probably by alteration of immune mechanisms. These changes are