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."

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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

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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, emepronium bromide, potassium, and Clinitest 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 fibreoptic 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 betweem 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.

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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

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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

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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