of gas gangrene.³ In Sweden Dornbusch *et al*⁴ found 247 of 252 strains were inhibited by 1 mg (or less)/l; the remaining five were less sensitive, MICs ranging from 2 to 64 mg/l.

Garrod *et al*¹ quote 2 mg/l as the usual MIC. Sapico *et al*⁵ found the MIC of most strains to be 3.1 mg/l and regarded them as of intermediate sensitivity. Whether the MIC of erythromycin for normal strains of *C welchii* is 1, 2, or 3.1 mg/l, a consideration of the likely blood levels of erythromycin given orally does not inspire much confidence in this antibiotic for the prophylaxis of *C welchii* infection, and for strains with unequivocal resistance, such as the one reported here, erythromycin would clearly be ineffective.

Eykyn and Phillips⁶ have recently suggested metronidazole as a possible alternative to penicillin for the treatment and prevention of gas gangrene; it certainly seems preferable to erythromycin when the blood levels of the two drugs are compared with their MICs for *C welchii*.

For prophylactic treatment of the patient undergoing a mid-thigh amputation or the insertion of a hip prosthesis penicillin will be the best-guess antibiotic. Should the patient be hypersensitive to penicillin it might be advisable to choose an alternative antibiotic not by guesswork but by preoperative sensitivity testing of the *C welchii* isolated from his faeces, since these are the organisms from which he is at risk.

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¹ Garrod, L P, Lambert, H P, and O'Grady, F, *Antibiotic and Chemotherapy*. Edinburgh and London, Churchill Livingstone, 1973.

² Brumfit, W, and Hamilton-Miller, J M T, *Journal of Antibiotic and Chemotherapy*, 1975, 1, 163.

³ Parker, M T, *British Medical Journal*, 1969, 3, 671.

⁴ Dornbusch, K, Nord, C-E, and Dahlbäck, A, *Scandinavian Journal of Infectious Diseases*, 1975, 7, 121.

⁵ Sapico, F L, *et al*, *Antimicrobial Agents and Chemotherapy*, 1972, 2, 320.

⁶ Eykyn, S J, and Phillips, I, *British Medical Journal*, 1976, 1, 1418.

Safety of children in cars

SIR,—Your leading article on this subject (1 January, p 2) surprised me greatly by making no mention of the desirability of placing young children in the rear seat of a car during all journeys whether they are restrained by a seat, by a harness, or not at all. Has the Transport and Road Research Laboratory nothing to say about the special hazards for children of sitting in the "death seat" next to the driver?

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*A leading article a year ago (24 January 1976, p 180) was entirely devoted to the dangers of front-seat riding for children.—Ed, *BMJ*.

Dysphagia

SIR,—Dr Michael Atkinson (8 January, p 91) endeavours to cover the whole of this vast subject and as a result there is obviously a lack of completeness. However, I think I must comment on two of the statements that he makes.

Dealing with peptic oesophageal stricture the author states that surgical measures

to control reflux should be considered in younger patients. In fact it is often the very elderly patient who presents the greatest difficulty and in whom dilatation is most unsatisfactory. Surgery on the hiatus hernia with an attack on the stricture or bypass operation often offers the only hope of a reasonable existence. A rather optimistic approach to this subject may be justified to the patient as a form of encouragement, but is certainly not justified in dealing with a lesion which may be extremely difficult to treat.

Secondly, in dealing with neoplastic dysphagia Dr Atkinson writes that if resection is contraindicated palliative intubation offers the only means of relief. This is quite untrue. A bypass operation, bringing the stomach under the skin on the chest wall and anastomosing it to the divided oesophagus in the region of the clavicle, avoids opening the chest and the risk of mediastinitis should it leak. This gives infinitely better swallowing than an intra-oesophageal tube and, even in the very elderly, has no greater mortality. Even in a case of broncho-oesophageal fistula due to a growth this operation with the modification of drainage of the lower oesophageal stump, otherwise unnecessary, will allow the unfortunate patient comfort in his last days.

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Effect of Intralipid on colour of blood

SIR,—A chance observation on the colour of a blood sample led to the conclusion that infusion of Intralipid 20% (an emulsion of fats for parenteral feeding) may cause blood to appear much better oxygenated than is the case.

A child of 8 years, having had major cardiac surgery, was undergoing a prolonged and complicated recovery period during which artificial ventilation and intravenous feeding were required. A blood sample drawn from an indwelling radial artery cannula was noted to be of a gratifying bright red colour, but on analysis by a Radiometer ABL 1 automatic blood gas measuring instrument the partial pressure of oxygen was only 8.2 kPa (62 mm Hg) and the oxygen saturation of the haemoglobin was only in the high 80s. Since the tendency to believe one's eyes and disbelieve the machine (especially if rather complicated) is a strong one we assumed that the Intralipid 20% infusion which was running into the child at the time of sampling had in some way deranged the ABL 1—we knew that such infusions disabled certain of the estimations performed in our main hospital laboratory.

The other explanation was that the machine was right and that the colour range was due to the Intralipid 20%. This was easily tested by mixing a normal venous blood specimen with small amounts

of Intralipid 20%, and noting the colour change in successively stronger mixtures as compared with the results from the ABL 1. We measured colour (as % oxygen saturation) with an American Optical reflectance oscimeter. The colour change was obvious to the naked eye, although mixing in a glass syringe was difficult since the fat globules tended to rise and separate. The results are shown in the accompanying table.

The child was not being infused at an excessive rate, but it is possible that the normal mechanisms for handling Intralipid infusion were not intact in view of the severity of the child's condition.

We have no knowledge of the rate of onset of this phenomenon at different infusion rates or of its half lives when infusions of different rates and durations are stopped. It is possible that the in-vivo observation reflected more than mere mixing (perhaps plasma protein-microglobule complex formation) and that the in-vitro demonstration was purely fortuitous: the patient's blood showed little tendency toward globule separation whereas the in-vitro experiment was plagued by it. Perhaps globules of different size are removed from the blood at different rates.

Various possibilities present themselves for further study, but in view of the hazard which may arise from such misleading appearances it seemed appropriate to point out that a bright red blood sample from a patient with an Intralipid infusion running is not necessarily of arterial origin (where doubt exists), that it is probably not as well saturated with oxygen as it appears to be, and that the difference may be large.

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The reprint game

SIR,—Referring to Dr John W Todd's letter (22 January, p 231), my experiences are exactly the same and I am equally exasperated. One correspondent even requested a whole chapter from a textbook. Most of them write from institutes which are certainly equipped with photocopiers. Their only excuse—which, however, none of them has voiced so far—might be that photocopies of illustrations are always extremely bad and indeed quite useless. A recent paper, written with a co-author, produced about 200 requests, mostly from the USA. Not one of them offered to refund postage or the not inconsiderable cost of the reprints themselves. The "reprint game" has developed into a "reprint racket."

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Effect of Intralipid on apparent oxygen saturation of blood as measured by reflective oscimetry (ABL 1)

	Percentage by volume of Intralipid 20% mixed with venous blood			
	0	5	10	15
Haemoglobin (g/dl) (by ABL 1)	12.9	13.7	15.1	16.3*
pH	7.277	7.295	7.291	7.288
P _{CO} ₂ (kPa)	7.26	7.39	7.22	7.22
P _O ₂ (kPa)	2.93	2.89	3.02	2.97
Base excess (mmol/l)	-2.4	-0.8	-1.8	-2.0
Standard bicarbonate (mmol/l)	20.9	22.1	21.2	20.9
Oxygen saturation (%) (derived by ABL 1)	29.7	29.7	31.9	31
Oxygen saturation (%) (measured by AO)	23†	28	37	45

Conversion: SI to traditional units—P_{CO}₂ and P_O₂: 1 kPa ≈ 7.5 mm Hg.
*Note rising haemoglobin values due to increasing turbidity of sample.
†This particular AO machine is known to read low at low saturations.