about this operation is that it works, whereas less radical procedures, unless done with great care, do not; and its advocates can be excused on the grounds that much suffering, time and disappointment is saved.

Some years ago I was impressed by an article by Brearley,¹ which advised slicing off a large piece of the terminal pulp and lateral nail fold which had overgrown the corner nail "spike" and allowing the saucerised area to granulate. The resulting scar contracture extends the nail bed to the end of the toe, thus preventing recurrence. The procedure has the advantages that it can be done in the presence of acute sepsis and that nothing is lost.

With extensive excision followed by haemostasis the results are gratifying provided that it is done on the early age group before the side of the nail has started to curl under. It was this last observation which caused me to consider the aetiology more seriously and, although, boys being boys, one must hold them culpable, the evidence was that "picking" the nail was more a result than a cause. The granuloma which occurs at the corner of the nail has all the conditions for a pyogenic granuloma. With the overlap of the lateral nail fold and end pulp there is no gravity or natural drainage—or natural manicure—for the skin detritus and foreign material which collects there. As this accumulates under the corner of the nail it raises it off the nail bed, which then retracts proximally under the lateral nail fold, so worsening the condition.

It is possible to get these conditions in a finger, and ingrowing fingernails do occur. In the booted foot, however, the situation is aggravated by the pump-like action of the arched toenail against the toe cap. This has the effect of alternately raising and dropping the corners of the nail (I have tested this on a plaster-of-Paris analogue), so impacting the detritus even more firmly at the corner. It is this detritus which causes the foreign body granuloma and without this there is no ingrowing. The unbooted native does not suffer from ingrowing toenails. It is loose shoes, football boots, or Wellingtons, as well as holed socks, which cause the problem.

Of recent years I have concentrated on removing the lateral nail fold to secure adequate drainage and an automatic manicure. There is then usually no problem as the nail grows to the end of the toe and the terminal pulp retracts before it. Once this has occurred and the situation is maintained there does not seem to be any recurrence. I am intrigued by the account of gutter treatment because it establishes these conditions from the start and look forward to the kits becoming available. I doubt, however, whether it should be used in the presence of sepsis, and one difficulty I foresee is the bead-like swellings—possibly due to the incorporation of foreign material—which are often present on the sides of the nail.

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¹ Brearley, R, *Lancet*, 1958, 2, 122.

SIR,—Mr W R Murray and Dr J E Robb in their letter (11 August, p 391) are quite correct in stating there was no statistically significant difference in the results between the gutter treatment and avulsion of the toenail because of the small numbers studied, as we reported in our paper (21 July, p 168). They are, however, incorrect in using reoperation rate as the sole criterion of success, as is clearly shown in our results. Cure rate is the important factor from the patient's point of view. Our results indicate that a randomised study of 120 patients should show a good statistical

difference between the cure rate at one year for the gutter treatment (of around 55%) and the cure rate for avulsion (of around 30%), and such a study would indeed be valuable.

Comment was also made on the equipment required. The sterile kit is inexpensive and should be balanced against our recommended follow-up of 10 days with a final check at six months—a total of four visits for the gutter treatment, compared with a careful long-term follow-up for a period of six to eight months with multiple attendances as recommended by Lloyd-Davies and Brill¹ for simple avulsion treatment. Mr Murray and Dr Robb also stated that there is no theoretical reason why gutter treatment should result in better long-term results than avulsion. Fowler,² however, has described the theoretical reasons why this should be: after avulsion of the nail the pulp of the hallux is pushed dorsally during weight bearing and the distal nail grooves are thus obliterated. When the new nail advances to this area of obliteration recurrence of the ingrowing toenail readily occurs. The other common finding I have observed after avulsion of the toenail is that the new nail is increased in thickness and more brittle, and this increase in thickness may appear similar to onychogryphosis if avulsion has been carried out on more than two occasions.

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¹ Lloyd-Davies, R W, and Brill, G C, *British Journal of Surgery*, 1963, 50, 592.

² Fowler, A W, *British Journal of Surgery*, 1958, 45, 382.

SIR,—Recent correspondence (11 August, p 391) has suggested that avulsion of the toenail should be retained as a useful technique in any toenail management protocol. However, though I agree with Mr A W Fowler (25 August, p 500) that ingrowing toenails are multifactorial in their causation, a very frequent finding is drastic cutting of the toenail, which removes support from the pulp of the toe and allows it to prolapse around the edges of the nail. These cases are particularly suitable for treatment with the gutter procedure¹ and, together with instruction to avoid cutting the nails short, result in a high cure rate.

Where there is considerable embedding of the toenail or abnormal thickening of the nail, either a segmental excision (described by Mr Fowler)² or phenol cauterisation of lateral nail matrix is the treatment of choice. I prefer segmental excision in these cases (cure rate at one year 84%¹), but in a recent series of cases of phenol cauterisation of the lateral matrix³ there was a 94% cure rate of 50 patients reviewed at six months. Perhaps a prospective trial between these two methods is indicated. For onychogryphosis or where there is marked ingrowth and granuloma affecting both sides of the nail, phenol cauterisation of the whole nail bed is the treatment of choice.⁴

In my opinion, simple nail avulsion is rarely indicated and should be regarded only as a palliative operation. Many patients continue to suffer months or years of further discomfort following avulsion until their name reappears on the waiting list for definitive surgery. Although this may in part be due to poor selection and follow-up of patients, with the above plan of management there are very few failures of treatment, considerably fewer

hospital attendances for treatment, and satisfied patients.

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¹ Wallace, W A, Milne, O D, and Andrew, T, *British Medical Journal*, 1979, 2, 168.

² Fowler, A W, *British Journal of Surgery*, 1958, 45, 382.

³ Reed, L, personal communication.

⁴ Andrew, T, Wallace, W A, *British Medical Journal*, 1979, 1, 1539.

* * * This correspondence is now closed.—
Ed, *BMJ*.

Minor tranquillisers and road accidents

SIR,—In their interesting paper (7 April, p 917) Dr D C G Skegg and others claimed to have shown an association between minor tranquillisers and increased risk of serious road accidents. I am not sure that they have.

From the details of the five patients listed, one finds that the fourth patient was knocked off her bicycle when a car door opened. She was taking a tablet containing meprobamate and ethoheptazine, an analgesic known to cause dizziness in some patients. It seems unlikely that the meprobamate was responsible for an accident that may not have happened in the absence of the careless behaviour of a motorist, but whether the analgesic or the tranquilliser played any part cannot be decided.

The fifth patient, who fell off her bicycle, was taking two benzodiazepines, an antidepressant, and insulin for her diabetes. I share the admitting doctor's uncertainty: "Mechanism of fall unknown." The hypotensive effect of the antidepressant or hypoglycaemia from insulin could have been as much responsible as the benzodiazepines.

The third patient, a known heavy drinker, had taken alcohol and diazepam, a mixture that is well known to be potentially lethal, particularly for car drivers. In the absence of a blood test I would be disinclined to accept his story that he had drunk "a little alcohol," bearing in mind the penalties for driving under the influence of liquor; far safer to blame the tablets the doctor gave him.

This leaves two patients whose accidents are "unexplained." Why did the first case drive his car head on into a lorry? Was he asleep or suicidal? In short, was it the drug or his mental condition that caused his death? Possibly the second case is the only one in which one can reasonably infer that his use of chlorthalidopoxide was a cause of inattention resulting in his not looking where he was going.

Clayton¹ has reviewed the very real problems of relating the use of psychotropic drugs to the frequency of collisions on the road. In truth, apart from individual patients who admit to difficulties experienced when driving after taking their medication, we know very little about this important problem. That many psychotropic drugs affect psychomotor skills and judgments in the laboratory or test track situation is beyond dispute. However, most studies are carried out on young, healthy, male volunteers taking drugs for short periods of time, and one wonders what relevance these have to older patients on long-term medication who are driving daily on the roads. How many suffer serious accidents as a result is not, at present, known.

Hence I wholeheartedly support the authors' request for a large, randomised control trial to sort out the effects of drugs and underlying

diseases. Also, in the light of past investigations, I agree that all patients on psychotropic drugs should be warned that their driving might be adversely affected by their drugs and that this effect can be aggravated by alcohol. If all medical practitioners give this warning and all patients heed it, we are going to find it even more difficult to carry out the randomised control trial that is so badly needed.

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¹ Clayton, A B, *Human Factors*, 1976, **18**, 241.

Benzodiazepines and traffic accidents

SIR,—Dr A Landauer (21 July, p 207) raises several points relating to the role played by diazepam in road traffic accidents.

All 1,4-benzodiazepines have been shown to possess sedative activity, which can severely impair the regulation and performance of the sensory-motor tasks undertaken by patients during the course of their everyday behaviour. It is not just impairment of car driving ability attributable to diazepam which should give rise to concern but the effect of 1,4-benzodiazepines in general on the performance of the psychomotor tasks associated with work, home, and leisure pursuits.

Laboratory assessments of performance do relate to the real life situation if appropriate measures are used. Reaction time to visual stimuli has been shown¹ to be an appropriate analogue of real life performance where co-ordination of eye, hand, and brain is an important feature. The 1,4-benzodiazepine derivatives in general have been found to reduce the critical flicker fusion threshold, giving objective credence to the often-reported subjective drowsiness experienced by patients taking such drugs. We have been able to show consistent depression of critical flicker fusion thresholds and impairment of reaction time tasks following both acute and chronic administration of a range of 1,4-benzodiazepine derivatives.²⁻⁴

Laboratory information has been augmented by concurrently obtained data from tests of car driving ability administered independently by Advanced Driving Institutes. The impairment of performance shown in the laboratory tests following the administration of a 1,4-derivative, lorazepam, has been mirrored in the reduced performance on actual car driving tests of brake reaction, steering, width estimation, parking, and garaging undertaken by the same subjects in a placebo-controlled study.

Such findings lead us to believe⁵ that the administration of 1,4-benzodiazepines, not just diazepam, produces an increased risk of accident in situations where the integrity of the sensory and motor systems is an essential prerequisite for the safe performance of the task. The pathologically anxious or insomniac patient might be a better performer following treatment with a benzodiazepine but a consideration of the vast number of these derivatives prescribed would suggest that many non-pathological individuals are receiving these drugs and suffering performance decrements.

Others have concluded⁶ that the administration of a benzodiazepine derivative does not necessarily cause a death or road traffic

accident. However, this is missing the salient point of the recently published epidemiological survey by Dr D C G Skegg and others (7 April, p 917), which is that there is sufficient cause for concern that the administration of a 1,4-benzodiazepine derivative increases the risk of accident in any situation where safety is dependent on unimpaired psychomotor performance.

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¹ Linnoila, M, and Mattila, M J, *Pharmacopsychiatry*, 1973, **6**, 128.

² Hindmarch, I, *Arzneimittel-Forschung*, 1975, **25**, 1836.

³ Hindmarch, I, *Arzneimittel-Forschung*, 1976, **26**, 2113.

⁴ Hindmarch, I, *British Journal of Clinical Pharmacology*, 1979, **7**, suppl p 77.

⁵ Snaith, R P, *et al*, *British Medical Journal*, 1977, **2**, 263.

⁶ Finkle, B S, *Journal of the American Medical Association*, 1979, **242**, 429.

Rupture of the uterus during prostaglandin-induced abortion

SIR,—We read with interest the letter by Mr A I Traub and Mr J W K Ritchie (25 August, p 496), in which they quite rightly point out the hazards of combining oxytocin with prostaglandin in the induction of a mid-trimester abortion; and we agree with their advice to delay the infusion of intravenous oxytocin. We think that it should be stressed, however, that the patient whom we reported (7 July, p 51) ruptured her uterus following the insertion of intra-amniotic prostaglandin and hypertonic saline. At no stage was oxytocin used.

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Induction of labour

SIR,—Following the article by Mr J H Shepherd and others (14 July, p 108) advocating the use of prostaglandin pessaries for induction of labour, we would like to illustrate the points raised in the letters of Mr A W Banks and Mr C J Hutchins (4 August, p 332) with two case histories and comment on our experience of the use of prostaglandin E₂ pessaries in a busy district general hospital.

The first case illustrates the dangers of unexpected delivery of an at-risk fetus in an inappropriate environment. A 24-year-old secundigravida (gestation confirmed by ultrasound scan at 17 weeks) was noted to be clinically small for dates from 27 weeks and so was monitored with plasma oestriols, serial ultrasound, and cardiotocography

(CTG). These were normal, but she developed mild pregnancy hypertension with proteinuria at 39 weeks. It was decided to induce labour, but as the Bishop score of the cervix was only 3, a 2-mg Prostin pessary was administered at 1300. She complained of irregular contractions soon after, and a CTG showed frequent but apparently small contractions. She did not require analgesia. She was judged not to be in labour, and monitoring was discontinued. At 1830 she developed the desire to bear down, and unexpectedly delivered on the antenatal ward. The baby weighed 2080 g but was fortunately in excellent condition, and needed no resuscitation.

The second case illustrates the real need for continuous CTG monitoring after use of prostaglandin pessaries in any at-risk pregnancy. A 27-year-old primigravida was clinically small for dates from 34 weeks. Plasma oestriol and human placental lactogen levels were always low, and from 37 weeks fell to below the 10th percentile. Antenatal CTG was normal, but because of the placental function tests, it was decided to induce labour at term. The Bishop score of the cervix was only 4, so a 2 mg prostaglandin pessary was inserted at 1120. Continuous CTG monitoring was performed (see figure). Because of large variable decelerations, which appeared over the next one-and-a-half hours despite keeping the patient on her left side, abnormal uterine action, and a cervix still only 3 cm dilated, it was decided to perform a caesarean section. A 3080-g infant was delivered in good condition.

We feel there are limitations to the use of prostaglandin pessaries for the routine induction of labour in a busy unit because: (a) the onset of labour cannot be predicted accurately; (b) patients may need cardiotocography for an indeterminate length of time; (c) when the cervix is favourable prostaglandins do not have any definite advantages over the tried and "midwife trusted" methods of amniotomy and oxytocin infusion; (d) they are expensive; (e) the potential advantage of mobility is frustrated by the need to monitor the patient and provide analgesia.

Prostaglandins do, however, have a valuable role in ripening the cervix and rendering it more favourable for induction by amniotomy and oxytocin infusion.

We are grateful to Messrs D Fairbairn and D P L May for permission to report on patients under their care.

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SIR,—I was pleased to see your leading article (18 August, p 407) stating that interest in methods of induction of labour is passing increasingly to non-invasive techniques which are more comfortable for the patients, as this statement is undoubtedly true. The simplest of these techniques is the intravaginal appli-

Cardiotocographic tracing in case 2.