community survey of Distalgesic poisoning and thereby illustrating its true morbidity, mortality, and prevalence, we hope to have contributed to this end. Our results clearly indicate that in our area Distalgesic is now the principal cause of self-poisoning fatalities. This finding must surely be viewed in conjunction with the fact that there remains no conclusive evidence that this drug has any superior analgesic efficacy when compared with other manifestly less dangerous alternatives.

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Addiction to Distalgesic (dextropropoxyphene)

SIR,—I am frankly astounded to read that a patient with addiction to dextropropoxyphene was given methadone during the withdrawal phase, as reported by Dr Robert Wall and others (17 May, p 1213). Why could the patient not have been allowed to withdraw from the dextropropoxyphene without complicating matters by using another, even more highly addictive, substance?

Our "detox" unit in this city treats many cases of withdrawal from diazepam, heroin, dextropropoxyphene, etc; and it has rarely been shown necessary to complicate the picture or postpone the suffering by adding other drugs.

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* *We sent this letter to Dr Wall, whose reply is printed below.—Ed, *BMJ*.

SIR,—Dr Mellor raises an interesting point concerning the use of substitute narcotics in the treatment of opiate-type dependency. Methadone is now widely used to deal with the abstinence symptoms of opiate withdrawal. Dextropropoxyphene produces dependency of this type whose severity is reflected in the amount consumed and length of time of abuse. Our patient had attempted many times to withdraw from Distalgesic. She had tried sudden and gradual withdrawal without success. There was some concern about the abnormal findings in liver function tests and subsequent liver biopsy. These were no doubt due to paracetamol toxicity and made it unwise to use Distalgesic on a reducing scale to effect withdrawal.

This patient had undoubted dependency and proved inability to curtail her abuse. In order to ensure her compliance in a treatment programme she was placed on low and diminishing doses of methadone for a limited period of time and under strict clinical supervision. As was to be expected, the small dose of methadone ameliorated her withdrawal symptoms and ensured her co-operation in the early stages of treatment, which are often distressing to the patient. During several months of outpatient follow-up she did not resort to abusing Distalgesic or any other drug and she was discharged from this unit.

The point here is that sudden withdrawal may be effective in treating true addiction in highly motivated patients with mild-to-moderate dependency. However, there is a risk that treatment will be stopped and abuse resumed if the abstinence symptoms experienced are deemed by the patient to be too distressing. Methadone is of lower addictive potential than most other narcotic drugs; hence its use in treating opiate-type dependency. The dose used in this case was at least half that commonly used in effecting opiate withdrawal, was given on a rapidly reducing schedule, and was given for a very short period of time. The risks of developing dependency on methadone were therefore negligible.

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Adverse reactions to D-penicillamine after gold toxicity

SIR,—We were interested to read the article by Dr M J Dodd and his colleagues on adverse reactions to D-penicillamine after gold toxicity (21 June, p 1498).

We have looked at our own figures for patients developing gold toxicity and then starting treatment with penicillamine. Out of a total of 26 patients, 13 started penicillamine within six months of stopping gold therapy. Only one of these patients developed a reaction while on penicillamine (rash with gold and thrombocytopenia with penicillamine). The other 13 patients started penicillamine six months or more after stopping gold and four developed an adverse reaction.

The total incidence of adverse reactions (5/26) compares with an incidence of 8/44 for our patients on penicillamine without prior gold therapy. We conclude from our figures, firstly, that there is no increased incidence of adverse reactions to penicillamine in patients who have had an adverse reaction to gold and, secondly, that there is no increased risk if the penicillamine is started within six months of stopping gold therapy.

A detailed table is available on request.

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Prescribing psychoactive drugs to addicts

SIR,—Dr Thomas Bewley in his article (16 August, p 497) draws attention to the need for doctors in private practice to be particularly aware of the pitfalls which may occur in respect of prescribing psychoactive drugs to addicts. However, I believe that I speak for the main body of private physicians when I say that we are intensely aware of these problems—to such an extent that for most of us the situation does not arise in the context of agents which are controlled under the Misuse of Drugs Act.

What presents far greater difficulty in everyday practice is the problem of patients who request prescriptions of certain other drugs, including appetite suppressants such as

diethylpropion and other types of sedatives quite apart from the barbiturate group. Indeed, after some three years in full-time private practice I have become so concerned about this that I have made some early moves to develop a private doctor's forum whereby such problems could be mutually discussed; thus I could envisage the establishment of some form of register whereby that all-too-familiar patient could be confidentially registered and thus identified early on presentation. All of us must have had the suspicion that there is a nucleus of such drug abusers who will foist themselves on one physician after another, procuring psychoactive drugs and changing doctors as soon as the going gets rough.

Certainly more open discussion and widespread education of doctors concerning the best ways of handling patients at this particular interface must result in the profession acting with united policy to serve the best interests of the patient who behaves in such a manner; and, while there unfortunately will always be those who will compromise the professional ethic for fiscal reasons, a forum such as I have proposed should also serve to minimise any aspersions that private doctors in general are a soft touch for whatever drug a patient may ordain for himself.

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Disturbed behaviour induced by high-dose antipsychotic drugs

SIR,—We read with interest the report by Dr T R E Barnes and P K Bridges on disturbed behaviour induced by high-dose antipsychotic drugs (26 July, p 274). We would like to make the following comments.

(1) The disturbed behaviour described in the three reported cases was characterised by excitement, restlessness, and aggression. The behaviour is more likely to be due to akathisia, which is known to occur in patients on high doses of antipsychotics, than to a relapse of these patients' schizophrenic illness. The authors themselves stress that the symptoms were "atypical for the patient."

(2) Two of the patients reported improved on reduction of the dose of antipsychotics and one of them needed big doses of anti-Parkinsonian medication throughout. The third patient settled down slowly as the blood level of flupenthixol gradually decreased and the drug was replaced with haloperidol, which probably acts on a different site. These observations support our claim that the behaviour described is consistent with a known side effect, akathisia, and not with a relapse of the schizophrenic illness.

(3) The first explanation suggested by Drs Barnes and Bridges, enhancement of the presynaptic release of dopamine, may explain the development of akathisia on high doses of antipsychotics rather than the recurrence of schizophrenia.

(4) As to the second explanation, anticholinergic toxic psychosis, the fact that in none of the three cases reported was there any mention of impairment of consciousness makes this explanation very unlikely.

(5) The development of restlessness, excitement, and aggression on high doses of antipsychotics is by no means a rare condition. We can recall at least two patients in the past six months in whom similar symptoms were

noticed and one of them became aggressive to another patient. When asked, the patients gave clear reports of subjective feeling of mental and muscular unrest and inability to sit still. They both responded to an intramuscular test dose of 10 mg procyclidine hydrochloride, which produced marked improvement after half an hour. The symptoms recurred four to five hours later. Both patients settled when the dose of their antipsychotics was reduced and did not need routine anti-Parkinsonian medication.

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Rapid poisoning with slow-release potassium

SIR,—I would like to take issue with the emergency management of hyperkalaemia as suggested by Drs R N Illingworth and A J Proudfoot (16 August, p 485).

The most important and urgent requirement for patients with very high serum potassium concentrations is the protection of the myocardium, and this is achieved by the administration of often large amounts of intravenous 10% calcium gluconate (up to 50 ml is often necessary). This should result in a prompt reversal of electrocardiographic signs and the danger of arrhythmias is rapidly diminished. The other measures which are commonly used (intravenous 5% dextrose with insulin and intravenous sodium bicarbonate) in the management of high serum potassium concentrations will, of course, lower the serum potassium and hence reduce the risk of arrhythmias, but should not be given until the calcium has been given in large enough doses. Following these emergency measures, of course, steps to correct the situation giving rise to hyperkalaemia and to relieve the body of its total excess potassium will be needed.

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Spironolactone prophylaxis in mountain sickness

SIR,—Squadron Leader G J Turnbull (14 June, p 1453) wrote about his experience with spironolactone on Mount Kenya. In 1976 my wife and I trekked in Nepal from 600 to 4400 metres spending four days above 3000 m. We both experienced extreme lassitude and fluid imbalance but as the level of activity was low we were able to manage—and in any case it was a fairly simple matter to transport us below 3000 m and await the return of the rest of the party as we were retracing our steps on the return journey.

This year we decided to go on a 16-day trek in the Andes never going below 3000 m from start to finish, climbing a total of over 7000 m and reaching a height of about 5000 m. As it was a round trek we had no wish to be left on route so after a survey of the literature we decided to take spironolactone 25 mg three times a day from the day of our arrival in Peru for one week and then 25 mg twice daily for the rest of the trek.¹⁻³ Neither of us suffered any altitude effects, unlike several in the party.

I am not putting this forward as a clinical trial but for anyone going on a trekking holiday at this altitude to take spironolactone in this way seems to be without risk and to have great potential benefit. Trekking while suffering from altitude effects is hard work for anyone but especially for a pensioner and his wife.

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Percutaneous central venous cannulation

SIR,—We read with interest the article by Dr M Rosen and others (2 August, p 372). We endorse their view that the percutaneous insertion of central catheters via the infraclavicular subclavian vein should be performed by those trained in the art and house officers should be closely supervised during such an operation. The creation of new problems for patients already in jeopardy from their original pathology or surgery should be avoided at all costs. A prospective study in 1971 demonstrated that non-infectious complications correlated with clinical experience in the technique.¹ In addition to the points made by the authors concerning this route of insertion, we feel that it should not be used for patients who are restless and uncooperative; have a diminished pulmonary reserve or, for example, are developing a contralateral haemopneumothorax after trauma; or possess abnormal anatomy because of previous clavicular fractures, a thoracoplasty, or local radiotherapy.

The most significant complications associated with the procedure are air embolism, pneumothorax, and injury to the subclavian artery or brachial plexus. Significant variations in the anatomical relationship of the subclavian vein to the clavicle and first rib have been well demonstrated by necropsy studies.² Contrast radiography studies of the veins have also confirmed these findings.^{3,4} The course of the subclavian vein on the right and left sides differs slightly, and changes with posture have been emphasised. We have found that, with the patient supine and prior to the draping and preparation procedure, a short time must be spent studying the topographical anatomy.

The subclavian venous pulse may be seen by inspecting the infraclavicular fossa while the abdomen is palpated. In addition using a Sonicaid ultrasound probe the characteristic venous hum may be detected by trial and error. This is made easier by getting the ipsilateral arm a firm and rapid squeeze. The point at which the subclavian vein passes below the clavicle may be found and marked with indelible ink. Similarly, the pulsatile flow sounds from the artery are easy to recognise and this vessel may also be marked on the skin surface (figure). This simple technique effectively localises the vessels and thus arterial and brachial plexus injury are avoided. The catheterisation procedure must still be performed carefully and gently to avoid creating a pneumothorax. We believe this manoeuvre gives the novice a more precise localisation of the subclavian vein and it is of special value in obese and heavily built patients.

Finally, the problem of catheter embolism will continue to occur if through-needle catheters are used (that is, Bard-I-Cath and Abbott Drum-catheter). Such complications and indeed tragedies are still being reported to the medical defence organisations. Even if meticulous care is taken, any sudden unex-

Doppler probe detecting the subclavian artery (A) and vein (V).

pected movement by the patient can cause the catheter to shear on the needle bevel. Furthermore, after the insertion is completed and the flimsy plastic sheath fitted, the patient has been provided with a central venous line passing through a "sword of Damocles" and protected only by a plastic scabbard. This serious risk should not be accepted by the clinician on behalf of the patient when safer alternative catheter designs are available.

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- ² Borja AR, Hinshaw JR. *Surg Gynaecol Obstet* 1970;130:267.
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Emphysema: beginning of an understanding

SIR,—I read with interest your leading article on emphysema (5 April, p 961) and also the letter by Dr M C S Kennedy (2 August, p 388). I agree that little attention has been paid to nitrous fumes in the causation of emphysema, especially in mining.

The prevalence of emphysema in coal miners is high and it is also high in metal-liferous miners. I have had experience of underground conditions in the West Cumberland iron ore mines over a period of nearly 50 years. Explosives are required to fragment and bring down the ore and large quantities of nitrous fumes are generated by the blast. Concentrations of nitrogen dioxide near the working face can rise to 300 parts per million and remain high for up to two hours; thus miners in former years spent a large part of their working life in concentrations of nitrogen dioxide 50-60 times the permissible level. Men in those days claimed that the fumes rather than the dust were the cause of their shortness