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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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