standard that centres could embellish as they wished. This advanced life support course was piloted in 1992 and is now available to centres that conform with the requirements. Advertisements often state that courses conform to the guidelines of the European and British Resuscitation Councils. However, some centres fail to satisfy the standards laid down for the new advanced life support course.

The Resuscitation Council (UK) has therefore agreed to approve on request every course that satisfies the advanced life support requirements. These courses will be given a number that can be checked with the coordinator at our London office and successful participants on the courses will be entitled to receive a certificate from the Resuscitation Council.

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## Alcohol not a predictor of peripheral oedema in chronic bronchitis

EDITOR,—Ronald Jalleh and colleagues conclude that alcohol consumption is an important independent predictor of peripheral oedema in patients with chronic bronchitis and emphysema by an unknown pathological mechanism.[1] I wish to suggest two possible explanations, which I believe are inadequately touched on by the authors.

The patients with the higher alcohol intake in the study are more likely to develop cirrhosis and cardiomyopathy, both of which are associated with the development of oedema. The investigators state that patients had no evidence of liver or ischaemic heart disease. Confident exclusion of the presence of both conditions, however, relies on the correct choice of investigation performed.

Classical clinical signs associated with cirrhosis may be absent with little disturbance in biochemical tests of liver function despite established histological changes on biopsy.[2] Similarly, the absence of evidence of ischaemic heart disease is less important than a measure of the degree of left ventricular dysfunction present in both study groups.

Clarification by the authors of the type of assessment of liver and cardiac status is required before any conclusions can be made about the effect of alcohol on hypoxia.

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1 Jalleh R, Fitzpatrick MF, Jan MA, MacNee W, Douglas NJ. Alcohol and cor pulmonale in chronic bronchitis and emphysema. *BMJ* 1993;306:374. (6 February.)

### Authors' reply

Paul McKee suggests that our observations could be explained by cirrhosis and cardiomyopathy. Though this is possible, all of our patients had normal results of liver function tests (bilirubin, γ glutamyl transferase, and alkaline phosphatase) and normal serum albumin and total protein concentrations, and none had a history of either ischaemic chest pain or myocardial infarction. In addition, their mean alcohol consumption since the age of 15 was modest and was similar to that of the general population in Edinburgh. It is difficult to believe that the proportion with oedema (46 of 73) reflects a similar frequency of cirrhosis or cardiomyopathy in the general population in Edinburgh. Indeed, even the oedematous subjects had a relatively low alcohol consumption, with a mean consumption since the age of 15 of 12 units a week. Furthermore, in our study, women were at greater risk of oedema, whereas women are resistant to alcoholic cardiomyopathy.[1] Thus, though cardiomyopathy and cirrhosis could potentially explain our observation, we do not think that either is a likely mechanism.

Additionally, we wish to correct McKee's belief that we reported an "effect of alcohol on hypoxia"; this was not the case.

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1 Wu CF, Sudhakar M, Jaferi G, Ahmed SS, Regan TJ. Preclinical cardiomyopathy in chronic alcoholics: a sex difference. *Am Heart J* 1976;91:281-6.

## Non-medical use of ketamine

EDITOR,—In his article on ketamine abuse K L R Jansen makes two misleading statements: firstly, that ketamine "unlike most other anaesthetics is a cardiorespiratory stimulant rather than a depressant."[1] In relation to the respiratory system this is not always so. As stated in its data sheet, "Depression of respiration or apnoea may occur following high doses of ketamine."[2] This is particularly so if used with or after another central nervous depressant.[3] In the social setting ketamine is often taken with other substances which have a respiratory depressant effect such as alcohol and benzodiazepines.

Secondly, Jansen states that "people coming to medical attention under the influence of ketamine are best placed in a quiet, darkened room until they recover." This must be challenged as it is hazardous and potentially fatal advice. As well as respiratory depression ketamine may produce depression of laryngeal reflexes predisposing to aspiration and airway obstruction. Aspiration under ketamine anaesthesia is well documented.[4] The administration of diazepam for panic attacks as suggested in the article will heighten the risk of aspiration.

I therefore suggest that patients presenting after ketamine abuse should be admitted to an intensive care unit where vital signs can be closely monitored and respiratory measurements can be made regularly. If the patient is judged to be at risk of aspiration tracheal intubation should be performed to protect the airway; this is to be preferred to giving analeptics.[2] A full toxicology screen may be required to exclude other substances taken concomitantly.

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1 Jansen KLR. Non-medical use of ketamine. *BMJ* 1993;306:601-2. (6 March.)
2 Walker G, ed. *ABPI data sheet compendium 1993-4.* London: Datapharm Publications, 1993:1172-4.
3 Jones RM. Inhalational and intravenous anaesthetic agents. In: Nimmo WS, Smith G, eds. *Anaesthesia.* Oxford: Blackwell Scientific, 1989:34-59.
4 Taylor PA, Towey RM. Depression of laryngeal reflexes during ketamine anaesthesia. *BMJ* 1971;ii:688-9.

### Author's reply

As Dr Gill points out, ketamine is not always a cardiorespiratory stimulant. However, the advice in the datasheet concerns the rapid intravenous injection of high dose ketamine, as a result of which depression of respiration and apnoea may rarely occur. My editorial concerns the non-medical use of ketamine, in much lower doses than those required to produce apnoea, and taken by oral and nasal routes.

I strongly disagree that people who have used ketamine in a non-medical context require admission to an intensive care unit for regular monitoring, any more than those many patients who are given ketamine in outpatient settings, particularly in the Third World, or soldiers given ketamine as a battlefield anaesthetic, require such attention. Ketamine fatalities are extremely rare, even in cases of massive overdose. The interventions that Dr Gill suggests are excessive, unlikely to benefit the patient in the overwhelming majority of cases, and dramatically increase the probability of a frightening emergence reaction. A quiet, calm environment is an appropriate and recognised procedure for recovery from ketamine anaesthesia. A full screen for other substances is a good suggestion, and any other substances that may be present will require such treatment as is appropriate for those substances.

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## Hammering psychiatric practice

### Secure in our insecurity

EDITOR,—Ian Robertson's assertion that the psychiatric professions attract a bigger proportion of insecure people than normal[1] made this psychiatrist almost faint with insecurity, for was my immediate desire for evidence of this "fact" not obvious proof of it? A perfect spring Saturday was ruined—my erstwhile confidence a transparent defence. Elbow in the marmalade, deaf to reassurance from family, I gloomily picked over my career, every apparent success further evidence of bottomless insecurity.

And then I remembered. At the bear garden that passed for a lecture on Freud to Glaswegian medical students many years ago the beleagured lecturer suddenly pointed at a particularly offensive man and opined that Freud would regard him as an obviously repressed homosexual. The student's apoplectic denial and increasingly detailed account of his legendary success with women were taken, using selected quotes from the master, as yet further evidence of his homosexuality. He couldn't win. It was a perfect demonstration of pseudo-psychoanalytic logic—still in vogue, apparently, in Cambridge.

I leapt up, suddenly secure enough to write the following:

> Dear Mr Robertson,
> Where is the evidence of this fact?
> Yours securely.

A J D MACDONALD

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1 Robertson I. To a man with a hammer, everything looks like a nail. *BMJ* 1993;306:937. (3 April.)

### ECT gets a bad press

EDITOR,—What on earth has got into Ian Robertson?[1] After reading his gratuitous slur on psychiatry and electroconvulsive therapy I had to look twice to check that he was a scientist and not a scientologist. He works himself into a lather about ancient abuses yet advances no realistic remedy. Prejudice against our profession is an occupational hazard for psychiatrists, but we hardly look for it in the pages of the *BMJ*.

All health workers may sometimes seek a solution to personal problems through their work, but where is the evidence that psychiatrists and their colleagues are less secure than those in other specialties? Robertson seems to have been watching too many film and television shrinks.

Electroconvulsive therapy may have been abused in the past to control "misbehaviour," as everyone "knows" from *One Flew Over the Cuckoo's Nest,*