Our findings would suggest that it is most important that doctors record which phase they are using, whether in clinical practice or in the reporting of therapeutic trials.

M F D'SOUZA

Department of Clinical Epidemiology
in General Practice,
Brompton Hospital, and
Department of Community Medicine,
St Thomas's Hospital Medical School,
London

L M IRWIG

National Research Institute for
Occupational Diseases,
Johannesburg,
South Africa

¹ D'Souza, M F, Swan, A V, and Shannon, D J, *Lancet*, 1976, **1**, 1228.

² Reid, D D, *et al*, *Lancet*, 1966, **1**, 614.

³ Veterans Administration Cooperative Study Group on Hypotensive Agents, *Journal of the American Medical Association*, 1970, **213**, 1143.

Out-of-hours calls in general practice

SIR,—It never ceases to amaze me that a general practitioner is expected to be on call 24 hours a day, 365 days a year, and yet be able to come up bright-eyed and bushy-tailed to cope with any emergency. Legal restrictions are laid down on the number of hours that airline pilots, lorry-drivers, etc may work. Yet GPs who equally hold human lives in their hands are expected to carry on regardless—possibly paying the penalty of a mistake through fatigue before a service committee or in the law courts.

Now that the tendency is towards larger group practices and duty-rosters surely it is time that a "shift system" is introduced, with financial encouragement from the Department. It would not be impossible to work a system in which one man in a group was on duty from 6 pm to 8 am for, say, a week, leaving his colleagues to cope with the day work.

Personalised continuity of care is all very commendable, but a fresh, alert doctor is safer and more use to the ill patient.

D HOOKER

Truro

Vinyl chloride: the carcinogenic risk

SIR,—We were pleased to read your well-balanced leading article on the carcinogenicity of vinyl chloride (17 July, p 134). In one respect, however, your discussion of ceiling and threshold limits is misleading and likely to confuse those amongst your readers who are not pollution specialists. We have previously discussed the problems caused by using parts per million (ppm) in an unspecified manner in connection with the pollution of operating theatre atmospheres.¹

Vinyl chloride behaves as a non-ideal gas at room temperature. The ppm unit to which you refer, we presume, is by volume: ppm (v/v). This unit is generally rather loosely used but frequently this ratio is meant to be ppm (ideal volume/ideal volume) without actually being defined. Because of the non-ideality of vinyl chloride vapour a given mass occupies less volume at any particular temperature and partial pressure than predicted by the ideal gas laws. Although the resultant error arising from the use of ppm (ideal v/v) may be small, precision is essential in establishing the threshold and ceiling limits if defined safety levels are to be meaningful.

Later in your article confusion is introduced by the mention of an upper limit (in undefined ppm) of vinyl chloride monomer (VCM) concentration in polyvinyl chloride (PVC) used in fabrication. In this case we must presume that ppm by weight (ppm (w/w)) is meant, but to many readers this may not be obvious.

It is far simpler to use units such as $\mu\text{g/l}$ (equivalent to mg/m^3) for air pollution thresholds because the instruments used to monitor pollution are most easily and reliably calibrated in such mass/volume units. If such a convention were generally adopted comparisons between work from different laboratories would be less subject to confusion because uncertainties in the definition of the concentration limits would be eliminated. Similarly it would be less confusing to discuss VCM concentrations in PVC in terms such as $\mu\text{g/g}$ or mg/kg .

JOHN S ROBINSON
JOHN M THOMPSON

Department of Anaesthetics,

RONALD BELCHER
WILLIAM I STEPHEN

Department of Chemistry,
University of Birmingham

¹ Robinson, J S, *et al*, *British Journal of Anaesthesia*, 1976, **48**, 167.

Hypophosphataemic osteomalacia in patients receiving haemodialysis

SIR,—Dr J F Mahony and his colleagues (17 July, p 142) reported four uraemic patients with depletion hypophosphataemia and osteomalacia. Three of them had used aluminium hydroxide as antacid while the fourth had not. Dr K Y Ahmed and others (28 August, p 526) reported four patients with persistent pre-dialysis hypophosphataemia in whom osteomalacia developed without their having taken oral phosphate binders. Dr M Cochran and his colleagues (14 August, p 396) examined the action of calcitonin in uraemia and pointed out the role of elevated plasma phosphorus in the observed increase in sensitivity to the hormone in nine of 17 patients. In connection with these publications we would like to report the conclusions reached from our studies of the role of calcitonin in the development of uraemic bone disease.¹⁻³

(1) In chronically uraemic rats four weeks after subtotal nephrectomy calcitonin sensitivity seems to be decreased if the animal is nearly normophosphataemic, calcitonin sensitivity being measured by examining the decrease in serum calcium concentration after the intraperitoneal administration of calcitonin.

(2) In these animals the calcaemic reaction (that is, the duration of hypercalcaemia after an intraperitoneal calcium load, which seems to be a measure of endogenous calcitonin activity) lasts longer than in intact, pair-fed, age-matched controls.

(3) In patients with histologically established chronic renal glomerular disease with proteinuria and normocalcaemia but without elevated serum creatinine, phosphorus, or blood urea nitrogen values the elevation of serum calcium after an intravenous calcium load⁴ lasts longer than in healthy individuals. This may point to the early beginning of diminished calcitonin sensitivity in the course of chronic renal failure.

(4) The anephric, uraemic rat with highly elevated plasma phosphorus seems to be more sensitive to calcitonin than the intact one.

(5) Normal rats with hyperphosphataemia and hypocalcaemia due to repeated oral administration of phosphate are more sensitive to calcitonin than untreated rats.

It appears that our observations are in agreement with the findings of the authors mentioned above. We found that uraemia causes decreased calcitonin sensitivity, which may be counteracted by hyperphosphataemia. It is suggested, therefore, that careful consideration should be given to any reduction of plasma phosphorus in uraemic patients treated with chronic haemodialysis.

J SZÜCS

First Department of Medicine,
Semmelweis University Medical
School,
Budapest, Hungary

¹ Szűcs, J, Horváth, T, and Steczek, K, *Clinical Nephrology*, 1974, **2**, 161.

² Szűcs, J, *Lancet*, 1974, **1**, 1108.

³ Szűcs, J, Horváth, T, and Steczek, K, *Magyar Belorvosi Archivum*, 1975, **28**, 240.

⁴ Holló, I, Szűcs, J, and Steczek, K, *Endocrinologie*, 1971, **58**, 326.

Postinfluenzal depression

SIR,—This is a syndrome which everyone knows about (leading article, 21 August, p 440) but on which it seems impossible to get any worthwhile information. Having an interest in cerebral complications of influenza I have tried to find evidence for it. I wrote several years ago to a number of Birmingham practitioners at a time when influenza was prevalent but was unable to find any convincing case of post-influenzal depression.

Excellent records of epidemics since 1880 have been kept by the Birmingham Public Health Department and for more recent years the virus type is known. With the valuable help of a psychiatrist, Dr A Orwin, I went through the records of a major psychiatric hospital and could find no correlation between numbers of admissions for depression and occurrence of influenza outbreaks; such admissions were not more frequent at the time of epidemics, nor were waves of influenza followed by detectable waves of depressive illness at any interval afterwards.

Surely one ought to be able to get precise information, something better than anecdotes on single cases, on a subject everyone knows about? Can anyone help me?

T H FLEWETT

Regional Virus Laboratory,
East Birmingham Hospital,
Birmingham

Ingrowing toenail

SIR,—The operation for ingrowing toenail advocated by Mr B V Palmer and Mr D L Stevenson (7 August, p 367) is the classic wedge excision operation described by Watson-Cheyne over sixty years ago¹ and repeatedly described with various modifications since then.²⁻⁴

However, as I pointed out in 1958,⁵ this operation is both inefficient and illogical. It is inefficient because the deeply placed germinal matrix, which it is most important to excise, is the least well exposed. It is illogical because embedding is caused by pressure of the nail against the nail wall and it is unnecessary to remove both the factors in a disorder due to two incompatible factors; if the nail edge is eliminated then the sepsis in the nail wall will always subside.