domiciliary practice. We are currently taking part in a study analysing the use of our guidelines by general practitioners in the domiciliary use of nebulised salbutamol as it is only then that we will really know whether nebulised salbutamol is both safe and effective in reducing hospital admissions.

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1 Cockledge SH, Jackson MB, Williamson L, Cain ARR, Osborne JP. Acute asthma in childhood and the role of nebulisers. *Practitioner* (in press).

*.*The authors reply below.—ED, *BMJ*.

SIR,—Dr Jenkinson makes some valuable points. We agree that undertreatment of children with asthma is common. In this study, however, over half the children were regular outpatient attenders and many of them attended a specialised asthma clinic. The amount of medication, method of administration, and written instructions were given to a large proportion of the children. They were not inadequately treated by conventional means. They were not, however, treated by three to four Rotacaps or six to eight puffs of aerosol for an exacerbation. Dr Jenkinson's observations need further study.

We do not wish to imply that nebulised salbutamol is the only effective way to deliver bronchodilators, but published work suggests that nebulised salbutamol is more effective than dry powder (albeit in conventional doses).^{1,2} We believe that the child with considerably reduced air entry has difficulty in inhaling single or multiple short sharp doses of salbutamol. The sustained action of nebulised salbutamol allows the child about 10 to 15 minutes of constant inhalation. The dose of the drug may be less important. For this reason spacers and spacehalers have been shown to be as effective as nebulised salbutamol.³

We were interested in the points made by Drs Osborne and Cockledge in that they have added another clinical method of assessment. However, we would not have counted a child who needed nebulised salbutamol for 24 hours as a responder. We believe that such a child, particularly under the age of 3 years, without a highly motivated general practitioner would still need to be observed in hospital.

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1 Grimwood K, Johnson-Barratt JJ, Taylor B. Salbutamol: tablets, inhalational powder, nebuliser? *Br Med J* 1981;282:105-6.

2 Webber BA, Collins JV, Bramthwaite MA. Severe acute asthma: a comparison of three methods of inhaling salbutamol. *Br J Dis Chest* 1982;76:69-74.

3 Freeland M, Van Asperen PP. Nebuliser versus nebuliser in children with acute asthma. *Br Med J* 1984;288:1873-4.

Containing the use of diagnostic tests

SIR,—Dr F G R Fowkes is quite right to draw attention to the continuing worrying increase in requests for diagnostic tests in hospital clinical practice (16 February, p 488), many of which have little or no influence on patient management. Often these are not only a waseful drain on overstretched monetary resources but also a source of worry, discom-

fort, and even danger to the patient. In his many sensible recommendations to reverse the trend he makes one important omission, however—namely, the better instruction of medical students and young hospital doctors in this matter.

The seeds of the malpractice of uncritical requests for further diagnostic tests (whether laboratory, organ imaging, neurophysiological, etc) are often sown in the undergraduate clinical years. Dr Fowkes's recommendations for improvement—financial incentives, decision aids, audit feedback, and control by diagnostic departments—though sensible, are, as he implies, often difficult to implement, chiefly because the door has been locked after the horse (the embryo doctor) has bolted. What needs to be emphasised far more in both undergraduate and early postgraduate clinical training is the meaningful and judicious choice of further diagnostic tests: this is best taught through the problem oriented approach to clinical problems. What seems not to be appreciated by so many students (and also all too many senior doctors) is that diagnostic tests are simply non-bedside investigations which are sometimes required to complement the history and examination in order to make a diagnosis. They should be taught that conceptually there is no difference between asking a question of a patient, making a physical examination, and requesting diagnostic tests. They are all investigations at different levels which have as their primary intent the diagnosis of a clinical problem. All too often there is a strange assumption that further "diagnostic" investigations must always be done, the assessment of a clinical problem being incomplete without them. Indeed, admission to hospital in itself seems often to justify an uncritical approach to the use of investigations. This is in great contrast with much of primary health care. For example, a child with diarrhoea at home will in most instances be perfectly well managed by the family doctor without "urea and electrolytes" being requested, an almost inevitable investigation if hospital admission were to be arranged. Few children admitted to hospital with a chest infection will emerge without a radiograph. Yet in primary care chest infections are often managed quite satisfactorily with the aid of the simplest of investigations—the stethoscope. What concerns me especially, as a paediatrician who for the past 10-15 years has been teaching most of undergraduate paediatrics in the second of three clinical years, is that by the time students reach us all too many have already acquired the bad habit of an uncritical investigative approach. Perhaps the large and readily available veins of adults are too tempting.

Only if all clinical disciplines speak with one voice, supported by their laboratory and other colleagues, will the problem be resolved. It should be incumbent on those ultimately responsible for patient care to urge a far more critical use of further diagnostic tests: this includes the rampant continuing abuse of routine investigations beloved in so many wards and clinics—urines, throat swabs, etc. They should set an example to follow. Indeed, it is sad that many young house officers make requests for fear of rebuff by their older (but obviously not necessarily wiser) colleagues. It would also help if more attention were given in postgraduate examinations to the discriminatory use of further diagnostic tests. If candidates were aware that less time would be given to the rare and esoteric and more time to examining their deductive thought processes concerning the analysis of a clinical problem good habits would emerge at a very early stage.

In 1961 Richard Asher wrote the following catechism about medical investigations: "It is a salutary exercise in mental discipline to catechise oneself when ordering any medical investigation, saying, 'Why do I order the test? What am I going to look for in the result? If I find it will it affect my diagnosis? How

will this affect the management of the case? Will this ultimately benefit the patient?' " We would all do well to heed this advice.

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Cardiac arrhythmias: theory and practice

SIR,—The classification of antiarrhythmic drugs devised by Dr Vaughan Williams and heavily drawn on in the leading article by Dr J K Aronson (16 February, p 487) is not based entirely on the effects of drugs on the action potential morphology. For example, β adrenergic antagonists have variable and complex effects on action potential duration in whole animals but little effect on isolated cells. The effect of drugs on repolarisation is the basis of the original subdivision of class I. The subclass Ic was not in Vaughan Williams's original classification but was added by Harrison¹ on the basis of its effects on intraventricular conduction. The effect on action potential duration is reflected by changes in the measured QT interval² not the QTc interval, a major determinant of which is heart rate.

A clinical classification³ based on the effects of drugs on tissues must take account of both beneficial and potentially harmful effects. Thus, sinus node automaticity is depressed not only by drugs from classes II and IV, but also by most drugs from classes I and III.⁴ A potent effect of class Ic drugs is depression of conduction in accessory pathways. This is not indicated in the table. These agents are very effective in controlling the arrhythmias associated with such pathways.⁵

We accept that antiarrhythmic agents cannot be selected for treatment solely on the basis of a classification system. However, when combined with other important information, knowledge of the action of a particular drug is essential in deciding the mode of therapy. For this reason it is imperative to ascertain the nature of the rhythm disturbance before starting empirical treatment.⁶ Such information as the mode of initiation and termination of a tachycardia may provide valuable clinical information. For example, a tachycardia involving the atrioventricular node and an accessory pathway may tend to terminate spontaneously in the atrioventricular node. In this setting a drug which further prolongs refractoriness of this structure may be more effective than a drug which affects the accessory pathway. Documentation of spontaneous episodes by tape monitoring may be useful in this respect. Alternatively, the tachycardia may be studied by intracardiac stimulation.⁷ This approach is especially valuable in patients with recurrent ventricular arrhythmias, in whom empirical treatment may not only be ineffective but can be dangerous,⁸ and we are astonished that the technique should be so casually dismissed by Dr Aronson. Indeed, in patients with sustained ventricular tachycardias associated with serious symptoms it is imperative to consider the use of invasive methods of assessment. The method is not dangerous and direct current countershock in a laboratory environment can be safely applied to terminate deliberately induced arrhythmias. It is safer to adopt this approach to identify effective antiarrhythmic treatment than to take the risk of a spontaneous recurrence of the arrhythmia during treatment with untested drugs.

We accept that drug treatment of cardiac arrhythmias remains far short of "ideal." However, it should be pointed out that greater advances in the understanding and treatment of clinical arrhythmias have occurred in the last 15 years than in the previous 50 years.

There are few "controlled" studies com-

paring different drugs for the treatment of serious arrhythmias. It does not follow, however, that the data obtained from such "single drug" studies are of no value. For example, in many studies using intracardiac techniques, tape monitoring, or exercise testing the patient acts as his own control and drug effect (or lack of it) can be accurately documented. There is now very good evidence that the acute effect of a drug on a ventricular tachycardia correlates well with long term suppression of the arrhythmia and prolonged survival.⁹ Rigorously controlled comparisons of drugs in this patient group would be clinically inappropriate and beset with ethical difficulties.

We believe there is no place for an empirical approach to the treatment of tachycardias. Careful analysis of spontaneous recordings should allow correct diagnosis in most cases. Armed with this information and the clinical pattern of the arrhythmia, the physician should be able rationally to select a drug with known effects with a high chance of therapeutic success.

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- 1 Harrison DC, Winkle RA, Sami M, et al. Encainide: a new and potent antiarrhythmic agent. In: Harrison DC, ed. *Cardiac arrhythmias. A decade of progress*. Boston: G K Hall Medical Publishers, 1981:315.
- 2 Milne JR, Hellestrand KJ, Bexton RS, et al. Class I antiarrhythmic drugs—characteristic electrocardiographic differences when assessed by atrial and ventricular pacing. *Eur Heart J* 1984;5:99-107.
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- 4 Breithardt G, Seipel L. The influence of drugs on sino-atrial conduction time in man. In: Luderitz B, ed. *Cardiac pacing*. Berlin: Springer Verlag, 1976:58-72.
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- 9 Swerdlow CD, Winkle RA, Mason JW. Determinants of survival in patients with ventricular tachyarrhythmias. *N Engl J Med* 1983;308:1436-42.

SIR,—It was interesting to read Dr J K Aronsen's excellent leading article (16 February, p 487). However, there were some important omissions. Specifically, the principal effects of flecainide acetate (Tambacor)—the only class Ic agent licensed in the UK—were not mentioned at all. In the table of classification of antiarrhythmic drugs by site of action no heading for "His-Purkinje system" was included. The principal site of action of flecainide acetate is the His-Purkinje system.² In the same table, under the heading "Accessory pathways," class Ic drugs are not listed despite the numerous mentions of the dramatic and selective effects on them of flecainide acetate.¹⁻⁴

Given the known electrophysiological characteristics of the a, b, and c subgroups of class I antiarrhythmic agents, there would seem to be no justification for the statement that a class Ia agent would be a more preferable substitution than a class Ic agent in the event of a lignocaine (class Ib) failure. The limited

evidence available would suggest that flecainide acetate with its considerably greater efficacy and low negative inotropism would be the better substitute.^{5,6}

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- 1 Hellestrand KJ, Bexton RS, Nathan AW, Spurrell RAJ, Camm AJ. Acute electrophysiological effects of flecainide acetate on cardiac conduction and refractoriness in man. *Br Heart J* 1982;48:140-8.
- 2 Morley CA, et al. *Eur Heart J* 1984;5, suppl B:128.
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- 4 Estes NAM, Garan H, Ruskin JN. Electrophysiologic properties of flecainide acetate. *Am J Cardiol* 1984;53:26B-9B.
- 5 Kjekshus J, Bathen J, Orning OM, Storstein L. A double-blind crossover comparison of flecainide acetate and disopyramide phosphate in the treatment of ventricular premature complexes. *Am J Cardiol* 1984;53:72B-8B.
- 6 Hoff PI, et al. *Eur Heart J* 1984;5, suppl B:121.

Natural progesterone and antihypertensive action

SIR,—Mr J W W Studd and his colleagues (16 February, p 558) await my pen. I am grateful to them for providing useful support for my work on the hypotensive action of progesterone, which I first showed 30 years ago by daily observations, over two menstrual cycles, of blood pressure, weight, and urine analysis in women with premenstrual syndrome. There was an observation cycle and during the following cycle the women received 25 mg progesterone intramuscularly daily from mid-cycle to menstruation.¹ My trials on the prophylaxis of intramuscular progesterone in pre-eclampsia in 1957-60² were repeated 12 years later with remarkably similar results using progesterone suppositories.³ Further confirmation of the value of progesterone therapy in the prophylaxis of postnatal depression has been accepted, so Mr Studd must wait patiently for its publication. I have been asked to explain the role of progesterone in the health of women, but my writings have been limited to the use of progesterone in the treatment of ill patients.

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- 1 Dalton K. Discussion on the premenstrual syndrome. *Proceedings of the Royal Society of Medicine* 1955;50:337-47.
- 2 Dalton K. Controlled trials in the prophylactic value of progesterone in the treatment of pre-eclamptic toxemia. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1962;69:463-8.
- 3 Dalton K. *Premenstrual syndrome and progesterone therapy*. London: Heinemann, 1977:113-7.

Chemotherapy for small cell carcinoma

SIR,—We were interested to read Dr Stephen G Spiro's leading article on chemotherapy for small cell carcinoma (9 February, p 413). We should like to add to his review of studies the results of a prospective study carried out at the London Chest Hospital in 1966-78 on 500 men and 76 women under the care of one physician in whom lung cancer was diagnosed.

Histological or cytological diagnosis was obtained in 424 (85%) of the men and 66 (87%) of the women. Small cell carcinoma was found in 63 (15%) of the men and 15 (23%) of the women, making a total of 78 patients. This group carried the poorest prognosis compared with other cell types; 65 (83%) died within one year, 45 (58%) in less than six months (see figure). At the completion of the study in December 1978, 77 out of 78 had died,

Survival according to type of lung cancer.

giving a median survival of five months, maximum (one patient) 21 months. The sole survivor died three months after the close of the study, having survived three years after diagnosis. The mean age at diagnosis was 60 years (range 40-86 years). Surgery was carried out in only 14 patients and in eight the diagnosis was not made until after surgery. Nine of these 14 patients were found to have unresectable carcinomas at thoracotomy. The median survival of the group was seven months, which was not significantly different from that of the five patients who had had resection (median survival eight months).

Radiotherapy alone was carried out in 35 patients, including the sole survivor. The median survival in this group was six months. Eight other patients received combined but unplanned chemotherapy, plus (with the exception of one patient) radiotherapy. The median survival here was also six months, maximum 18 months (one patient). The remaining 21 patients were untreated; their median survival was only two months.

Irrespective of the form of treatment the median survival was only six or seven months. Dr Spiro's review has shown that, at best, for patients with limited disease combination chemotherapy has prolonged survival to a median of 12 to 16 months; at worst, for patients with extensive disease, the median survival is not much better than that of our own patients, whose results would therefore form a useful baseline for comparison with future studies.

We are grateful to Dr M Caplin for permission to study his patients.

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Reducing errors in the accident and emergency department: using radiographers

SIR,—May I reply to the comments of Dr I Hudson and Mr D V Skinner (16 March, p 857) on our article (9 February, p 421). I have not advocated the issuing of formal reports by radiographers. This is quite unacceptable medicolegally, and I hope we have