associated with an increased incidence of ventricular extrasystoles. The study was not designed to establish a causal link and could not have done so. Such a link rests on a large body of evidence, referred to by the authors, establishing an electrophysiological basis for the known initiation of arrhythmias by hypokalaemia. The study showed that the serum potassium concentration was related to the ventricular extrasystolic counts when all patients were considered, that extrasystoles were commoner in those taking thiazides in the long term study, and that complex rhythm abnormalities were commoner in patients receiving bendrofluazide. Correlations were not found when decremental potassium changes were analysed. Nor were correlations found in some of the subgroups studied, but the authors themselves point out that the numbers of patients in these subgroups were small (eight to 19). A further confounding factor is that the serum potassium concentration was measured only once before the 24 hour tape recordings were obtained.

The authors comment that in skeletal muscle only a weak relation exists between a low serum potassium concentration and reduced intracellular potassium concentration. Cardiac muscle is different. A net potassium loss from the body caused by diuretics or diet does not change intracellular potassium concentration in the heart despite a large fall in serum potassium concentration and a loss of potassium from skeletal muscle (for references see refs 1 and 2). Diuretics in therapeutic concentrations do not have a direct effect on potassium exchange.[1] If conducting tissues in the heart are similar to myocardial cells then it is the serum and not the intracellular potassium concentration that is more important in the genesis of ventricular extrasystoles in the presence of hypokalaemia.

No doubt many factors contribute to the initiation of extrasystoles, including serum potassium and magnesium concentrations, pre-existing but undiagnosed coronary artery disease, and neurogenic factors. It is impressive that in a relatively small group of patients (155 in the long term study, 228 in all) the MRC study has shown an association between ventricular extrasystoles and hypokalaemia. I do not know what the authors expected from their study and how tight the correlations were expected to be, but the negative interpretation of the results seems curious and inappropriate. The results would be equally compatible with, and provide evidence for, the view that hypokalaemia induced by thiazides was an important causative factor in the initiation of ventricular extrasystoles.[3]

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[1] Poole-Wilson PA, Cobbe SM, Fry CH. Acute effects of diuretics on potassium exchange, mechanical function and action potential in rabbit myocardium. *Clin Sci* 1978;**55**:555-9.
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**We sent a copy of this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—The main aim of the substudy was to compare the prevalence of ventricular extrasystoles in people treated with bendrofluazide with that in groups taking placebo tablets, rather than to test the hypothesis that Professor Poole-Wilson gives. The substudy did include parts which, though they could not have proved a causative relation between the concentrations of serum potassium and the ventricular extrasystolic count, could have strengthened the evidence for causality by showing, in longitudinal data, changes in these two factors occurring in the expected directions in groups of patients or in individual patients. We found no such evidence. As we said, however, and as Professor Poole-Wilson emphasises, the experimental design was such that our failure to find such evidence is far from being conclusive proof that there is no causative relation.

What impressed us about the correlations between serum potassium concentration and ventricular extrasystolic count and between serum urate concentration and ventricular extrasystolic count, was neither their strength nor their weakness but their pronounced similarity. This led us to suggest that these biochemical changes might be acting merely as markers of thiazide intake. Of course, neither this similarity nor any other aspect of our results refute the hypothesis that hypokalaemia induced by thiazides is associated with an increased incidence of ventricular extrasystoles, but we would not claim that our results strengthen the evidence that there is a causative relationship between hypokalaemia and ventricular extrasystoles.

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Epileptic seizures in general practice

SIR,—We would like to reply to Dr James Edeh's letter (5 November, p 1378) concerning our paper on epilepsy in general practice (3 September, p 641). In fact, our definition of active epilepsy, which was "a seizure in the previous 24 months in those with epilepsy (defined as two or more seizures)," does largely "conform to the generally accepted," and furthermore our finding of a prevalence rate for active epilepsy of 5·3/1000 is similar to those which both we and Dr Edeh quote. The separate analysis of those with single seizures, inactive epilepsy, and active epilepsy is given in our second paper.

Furthermore, we do not understand to what "controversy concerning the prevalence of epilepsy" Dr Edeh refers: the point of our article was to show that long term (usually permanent) remission occurs in most patients in whom epilepsy is diagnosed, and thus the lifetime prevalence rate (the number of patients who have ever had epilepsy) is much greater than the rate for active epilepsy, and about this there is general agreement.

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An avoidable recurrence of cri du chat syndrome in the next generation

SIR,—There are some lessons to be learnt from the cases described by Dr J Burn and others (29 October, p 1287) and there are possible solutions.

Medical students are taught a model of the consultation that includes taking a complete history of the present illness and a complete personal, psychiatric, family, social, and geographical history followed by a full physical examination. This is usually an inefficient and ineffective method in outpatient departments and in general practice. Doctors develop either intuitively or consciously a style of solving problems in which hypotheses are created early in the consultation and are checked by further history taking, observation of non-verbal communication, and examination and investigations as necessary. Perhaps we tend to throw out the baby with the bath water.

In general practice it is appropriate that either at registration or at the first consultation with a new patient a more formal history is taken. The length of the consultation may be shortened by asking the patient to complete a health questionnaire on registration. The answers can be confirmed and augmented at the first consultation. Murray *et al* have shown that patients fill in the cards with reasonable accuracy and completeness.[1] At the end of the first consultation the doctor can draw up a list of problems, which would, of course, include genetic problems and which should remain on the front of the patient's records for his lifetime and be available for updating. Questionnaires for patients and summary cards are available in both MRE and A4 sizes. (The central information service of the Royal College of General Practitioners would be pleased to advise.)

In our practice we are in the process of designing a family record card for every family on our list. This summarises the major problems of the family and includes a simple three generational family tree. This card is available at each consultation. An alternative solution is for patients to keep their own records or at least copies of their problem sheet.

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[1] Murray M, Sydenham D, Westlake R. A questionnaire as a data base in problem orientated records. *J R Coll Gen Pract* 1974;**24**:571-5.

Rectal indomethacin for control of postoperative pain

SIR,—We should like to add a word of caution to the article by Mr D J M Keenan and others (5 November, p 1335) concerning the control of post-thoracotomy pain by rectal indomethacin.

Stress ulcers can occur after thoracotomy,[1] and indomethacin treatment is associated with peptic ulceration.[2] The action of indomethacin, by inhibiting the production of prostaglandins, which have cytoprotective and antisecretory effects in the upper gastrointestinal tract, is mediated systemically, so no advantage in this respect is conferred by rectal administration of the drug. During the past 12 years 320 patients have been admitted to this hospital with perforated peptic ulcers. Of these 55 patients were currently taking indomethacin, eight by suppository alone. When compared with 320 age-sex matched controls (eight were taking indomethacin and in only one

was this by suppository) a significant difference was found (χ^2 (with Yates's correction)=37·2, df=1, p<0·001).

Mr Keenan and his colleagues concluded that indomethacin suppositories were "safe" because of the "absence of noted side effects." These conclusions, however, were based on the results of their use in only 30 patients. A recent study reported that three of 44 patients given indomethacin suppositories after surgery developed dyspepsia in the postoperative period, and another patient had a haematemesis.[3] Recently, a patient in this hospital started treatment with phenylbutazone for a painful bone metastasis seven days before a transurethral prostatic resection. He had no history of dyspepsia, but four days after the operation an acute duodenal ulcer perforated. The administration of non-steroidal anti-inflammatory agents at a time when stress ulceration can occur may be hazardous. This is further supported by the fact that stress ulcers in animals can be prevented by the administration of prostaglandins.[4]

Reasbeck *et al* found a significant increase in postoperative haemorrhage in patients receiving indomethacin suppositories.[3] In the study of Mr Keenan and others persistent postoperative bleeding occurred when subcutaneous heparin was administered concurrently, therefore heparin was not used routinely in their trial. If they assume that subcutaneous heparin can be replaced by indomethacin as prophylaxis against pulmonary embolus they provide no evidence to support this assumption.

We believe that postoperative indomethacin suppositories may have serious side effects and its widespread use cannot be recommended.

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[1] Goodman AA, Frey CF. Massive upper gastrointestinal haemorrhage following surgical operations. *Ann Surg* 1968;**167**:181-4.
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Control and prevention of tuberculosis

Sir,—The report by the Joint Tuberculosis Committee gives much needed guidance. Although I accept that fomites and the environment play a minimal part in the spread of tuberculosis, I wonder if we can ignore them altogether.

Some patients in general hospitals are particularly susceptible to infection, and, unless we have good evidence to the contrary, we should take reasonable measures to prevent the spread of a communicable disease. It seems rational to disinfect crockery and cutlery from untreated tuberculous patients or those receiving early treatment—for example, the first two or three weeks. Heat disinfection in a washing up machine is the preferred method, but, alternatively, disposables may be used at minimal cost. It is also rational that linen should be treated as potentially infectious and sealed in a hot water soluble or alginate stitched plastic bag for transport and should not be handled in the laundry before disinfection.[1] Waste from secretions should similarly be contained in a plastic bag and incinerated. Isolation of the patient in a single room is important, but to state that barrier nursing is unnecessary could be misleading as washing hands after handling a patient is the most important component of the barrier nursing technique.

Unfortunately, it is difficult to obtain good evidence on the necessity of some of these procedures, and the distinction between ritual and rational methods is not always clear. I suggest that with tuberculosis we should err on the side of safety. Advice on management of patient contacts would also be useful to staff controlling infection.

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[1] Lowbury EJL, Ayliffe GAJ, Geddes AM, Williams JD. *Control of hospital infection: a practical handbook.* 2nd ed. London: Chapman and Hall, 1981.

New drugs: antithrombotic treatment

Sir,—We wish to draw attention to a misleading statement in the article on antithrombotic treatment by Dr P Buckler and Professor A S Douglas (16 July, p 196). They state that calcium heparin consists of a "mixture" of sodium and calcium salts of heparin and give Minihep Calcium as an example. As the pharmacopoeial standards for the manufacture of calcium heparin allow not more than 0·1% sodium in the drug substance calcium heparin can hardly be said to be a mixture.

Furthermore, subcutaneous calcium heparin is said to give lower plasma concentrations than sodium heparin and thus a reduced risk of haemorrhagic complications, a point of view also advocated by other authors.[1-3] Comparative studies, however, have failed to find any differences between the two salts with regard to serum heparin concentrations (figure), haematoma formation, or clinical efficacy.[4-7] This is in accordance with a review made by Segesser and Gruber[8] that covered 20 studies with sodium heparin and 14 studies with calcium heparin totalling just under 7000 patients. The authors concluded that there was no difference between the two salts with regard to side effects and that one form of heparin had no clinically relevant advantages over the other (table).

These conclusions were confirmed by Kakkar, who, in an extensive double blind multicentre trial of postoperative low dose heparin prophylaxis, found wound hematoma in 3·6% of the group treated with sodium heparin and 4·5% of the group treated with calcium heparin.[9] It was concluded that doses of 5000 IU of sodium or calcium heparin given every 12 hours seem to be equally safe and effective in preventing postoperative venous thromboembolism.

It seems reasonable, therefore, to say that it makes no difference whether calcium or sodium salts of heparin are used.

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[1] Thomas DP, Sagar S, Stamatakis JD, Maffei FHA, Erdi A, Kakkar VV. Plasma heparin levels after administration of calcium and sodium salts of heparin. *Thromb Res* 1976;**9**:241-8.
[2] Kakkar VV. Low dose heparin present status and future trends. *Scand J Haematol* 1980;**25**, suppl 26: 167.
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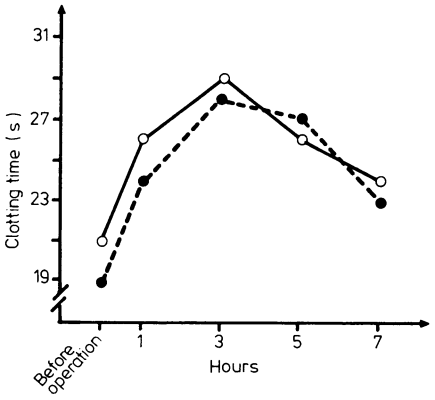

Determination of heparin concentrations in blood after administration of 5000 U sodium (○—○) and calcium (●- - -●) heparin. (Fig 4 from Lahnborg and Bergström.[4])

A simple system for references and reprints

Sir,—It is with some sympathy for Dr A S Henderson and Mr R Bosly-Craft (12 November, p 1448) that I write to bring to their attention, and to that of your readers, a more advanced system for references and reprints that has been in existence for many years. The Famulus system, based on a Fortran program originally designed by T B Yerke in 1967, is implemented on the ICL 2980 at Queen Mary College, London, on the Euclid system at University College, London, and also at the University of London Computer Centre.

Famulus allows each reference to be

Prevalence of deep vein thrombosis and pulmonary embolism in patients treated with sodium or calcium compared with untreated controls from several studies. Data obtained from tables 3 and 6 of review by Segesser and Gruber[8]

	Deep vein thrombosis			Pulmonary embolism		
	Total No studied	% Controls	% Treated patients	Total No studied	% Controls	% Treated patients
Sodium	1740	27·6	9·3	1681	1·6	0·2
Calcium	2429	32·7	12·8	4568	0·9	0·3