encountered. We conclude that this offers a practical alternative to percutaneous cannulation of the subclavian vessels, particularly in patients with haemostatic abnormality.

STEVEN C MORRISON
PETER JACOBS

University of Cape Town Leukaemia
Centre and Department of Haematology,
Groote Schuur Hospital,
Observatory, Cape, S Africa

Spurious polycythaemia resolving during observation

SIR,—You recently published an interesting account by Dr L E Ramsay (13 May, p 1251) of spurious polycythaemia developing during a period of clinical observation. Very little has been documented about the natural history of this interesting condition. Follow-up observations that have been made indicate an increase in morbidity and mortality, generally from thromboembolic disease.^{1,2} We were interested to observe the resolution of this condition over several months in a patient of ours who presented having suffered a cerebrovascular accident. The patient also had horseshoe kidneys.

A 69-year-old printer presented with loss of consciousness followed by left-sided weakness. There was no history of previous syncopal episodes, visual disturbances, hypertension, gout, renal, cardiac, or lung disease, pruritus, headaches, or haematuria. He had noted two episodes of transient weakness of the left side over the preceding year and described himself as being prone to anxiety. He drank about 4 pints (2.4 l) of beer daily and smoked about 10 small cigars. There was no significant family history of disease. He had been taking digoxin, frusemide (40 mg daily), and potassium supplements for 12 months for ankle oedema but had no other symptoms of cardiac failure.

He was obese and plethoric; weight 98 kg, blood pressure 140/70 mm Hg, pulse 80/min, multiple ectopics. Cardiac, respiratory, and abdominal examinations were normal. He had a flaccid left-sided hemiparesis with upgoing left plantar response and left homonymous hemianopia. Fundi were normal and there was no nystagmus.

Investigations revealed haemoglobin concentration 20.9 g/dl, erythrocyte count $6.4 \times 10^{12}/l$ ($6\,400\,000/mm^3$), packed cell volume 0.625 (62.5%), leucocyte count $19.8 \times 10^9/l$ ($19\,800/mm^3$) with normal differential, platelet count $293 \times 10^9/l$ ($293\,000/mm^3$), erythrocyte sedimentation rate 10 mm in 1st hour. The blood urea concentration on admission was 18 mmol/l (108 mg/100 ml) but returned to normal within 48 h. Serum electrolyte concentrations were normal, as were liver function tests, serum cholesterol, triglyceride, urate, glucose, acid phosphatase, and arterial blood gas analysis. Leucocyte alkaline phosphatase score was 100 (normal range 35–100). Red cell mass was 29 ml/kg (normal range 25–35 ml/kg), plasma volume (labelled albumin technique) 26 ml/kg (normal range approximately 40 ml/kg). Tests for rheumatoid factor were negative. Intravenous pyelography showed horseshoe kidneys with normal excretion, creatinine clearance 70 ml/min.

The raised haemoglobin concentration on admission was assumed to be secondary to dehydration but persisted despite rapid restoration of the blood urea to normal with rehydration. The normal red cell mass with reduced plasma volume indicated "stress" polycythaemia (syn spurious, relative, or pseudo polycythaemia, Gaisböck's syndrome), which was supported by the history of smoking, obesity, and anxiety and the lack of evidence of primary or secondary polycythaemia. Diuretic therapy was not con-

tinued. Over the ensuing four months the haemoglobin concentration fell to 17.7 g/dl and the packed cell volume to 0.548 (54.8%), these levels having been maintained since that time (now seven months).

The alcohol intake³ and cigar smoking⁴ may have contributed to the reduction in plasma volume in this patient. Diuretic therapy was probably not contributory as it was of greater than one month's duration. The association with horseshoe kidneys is interesting but probably incidental. The resolution remains unexplained, but cessation of smoking, reduction in weight and alcohol intake, and reduced activity following hemiparesis are potential factors. The patient has made a good functional recovery from his stroke.

B W S ROBINSON
D CORLESS

Department of Geriatric Medicine,
Guy's Hospital,
London SE1

- ¹ Burge, P S, *et al*, *Lancet*, 1975, **1**, 1266.
- ² Weinreb, N J, *et al*, *Seminars in Haematology*, 1975, **12**, 397.
- ³ Smith, J F B, *et al*, *Lancet*, 1973, **1**, 637.
- ⁴ Smith, R J, *et al*, *New England Journal of Medicine*, 1978, **278**, 6.

Comparison of the tine and Mantoux tuberculin tests

SIR,—Despite the care taken by Drs J A Lunn and A J Johnson in the design and collection of data forming the basis of the report of the Tuberculin Subcommittee of the Research Committee of the British Thoracic Association (3 June, p 1451) the conclusion that the tine test is unsuitable for epidemiological use because of the high proportion of negative and doubtful results in Mantoux-positive examinees is at variance with experience in the west of Scotland.

Apart from the basic and perhaps insuperable consideration of whether the simple physical effects of puncturing the skin with a tine rather than splitting it with a hypodermic needle are comparable, the measurement of the diameter of the single largest indurated papule of the tine test reaction, excluding the increments of the other times, might bias observations in favour of the single reaction of the tuberculin needle. A study group comprising student nurses is neither demographically nor immunologically characteristic of the general population of this country.

Our epidemiological studies in the west of Scotland¹ have shown an appropriate degree of correlation between the presence or absence of radiological evidence of tuberculosis and tine test reactivity. In a contact survey following the identification of 11 active cases of pulmonary tuberculosis in the Lanarkshire village of Harthill in 1976² 3000 residents over the age of 14 were offered 70-mm mass miniature chest x-ray and a tine test, which was read by the same observer recording reactions 4 and 5 only on the tine test reading card as positive. The results for 282 men and 291 women were compared with those for 6857 men and 8206 women in age-matched total samples of the populations of Renfrew and Paisley.^{3,4} The same decline in tuberculin sensitivity, more marked in women after the age of 45, was observed as that noted in using the conventional Mantoux test.¹ The high-risk Harthill population exhibited the same higher reactivity levels as Renfrew compared

with the low-risk population in Paisley, and tine test positivity conformed satisfactorily with radiological evidence of tuberculosis.

The epidemiological value of the test was further demonstrated by the identification of a cohort of 50- to 54-year-old men and women with an apparent common excess exposure experience to tuberculosis in Harthill compared with the other two communities and who would require prolonged surveillance.

Larger studies of properly drawn samples of the general population would seem indicated before discrediting a widely used, safe, and convenient form of tuberculin testing which seems perfectly adequate for epidemiological purposes.

V M HAWTHORNE

Mass Health Examination Unit,
Glasgow

- ¹ Hawthorne, V M, *Scottish Medical Journal*, 1969, **14**, 222.
- ² Thomson, W O, Young, W C, and Campbell, A T, *Health Bulletin (Edinburgh)*, 1978, **36**, 57.
- ³ Hawthorne, V M, Greaves, D A, and Beevers, D G, *British Medical Journal*, 1974, **3**, 600.
- ⁴ Hawthorne, V M, *Current Medical Research and Opinion*, 1977, **5**, suppl 1, p 109.

Schizophrenic neurasthenic defect

SIR,—With reference to your leading article (8 July, p 76) stimulated by the paper of Cheadle *et al*,¹ surely it is no new finding that schizophrenics may show a deterioration succeeding the productive symptoms of the acute psychotic episode—indeed this was an integral part of Kraepelin's description.² Since this first description Germanic psychiatrists have recognised the deterioration also called schizophrenic dementia or defect. Huber,³ in a comprehensive review, describes a neurasthenic syndrome which is often revealed with the resolution of the more dramatic symptoms of schizophrenia. Johnstone *et al*⁴ have recently resurrected the suggestions of several workers, including Huber, that schizophrenic defect can be associated with demonstrable cortical atrophy. Rather than be surprised at the ongoing difficulties of remitted schizophrenics we should perhaps take heart that many can lead apparently normal symptom-free lives.

In a pilot study involving 31 schizophrenics from a depot phenothiazine clinic, in all of whom the diagnosis was confirmed according strictly to the criteria of Schneider⁵ or Spitzer and Endicott,⁶ I found that nearly half appeared to be leading unimpaired lives. Fifteen patients were either employed full time (8) or fulfilling a complete domestic role. A further four were employed part time. One-third were free from any defect of drive, sociability, or the capacity for emotional expression. Seventeen rated themselves as back to or better than their premorbid level. The "distinct signs of disease" which Bleuler⁷ saw in all his discharged schizophrenics were often quite absent. Thirty-nine per cent of patients had been free of productive psychotic symptoms for more than two years.

It is clear that many schizophrenics today have a prognosis no different from that of manic depressive psychosis—that is, to recover from the acute psychotic episode without demonstrable defect. What is less clear is whether this represents a real change from the past or whether the "mild deterioration" only which Bleuler saw in 60% of his patients with the ability to pursue an occupation preserved was a notional rather

than an objectively demonstrable entity. Research has been rightly directed to demonstrating the variables which influence the relapse of acute schizophrenia with productive symptoms. What is also needed is an objective assessment of the variables which may influence the development or resolution of schizophrenic defect. Have changes in institutional management changed the development of defect or merely where a schizophrenic lives? Do long-term phenothiazines influence the postacute course or merely productive symptoms? Is defect inevitably associated with an organic brain lesion?

G J LODGE

Department of Psychiatry,
Withington Hospital,
Manchester

¹ Cheadle, A J, Freeman, H L, and Korer, J R, *British Journal of Psychiatry*, 1978, **132**, 221.

² Kraepelin, E, *Dementia Praecox*. Edinburgh, Livingstone, 1919.

³ Huber, G, *Fortschritte der Neurologie, Psychiatrie und ihrer Grenzgebiete*, 1966, **34**, 409.

⁴ Johnstone, E C, et al, *Acta Psychiatrica Scandinavica*, 1978, **57**, 305.

⁵ Mellor, C, *British Journal of Psychiatry*, 1970, **117**, 15.

⁶ Spitzer, R L, Endicott, J, and Robins, E, *Research Diagnostic Criteria*. New York, New York State Department of Mental Hygiene, New York State Psychiatric Institute, Biometrics Research, 1975.

⁷ Bleuler, E, *Dementia Praecox or the Group of Schizophrenias*. New York, International University Press, 1950.

Patients' responses to barium x-ray studies

SIR,—Maureen Reynolds's first-rate paper about communication in hospital (24 June, p 1673) explored ground similar to the paper by Dr Jenifer Wilson-Barnett on patients' responses to barium x-ray studies published a few weeks earlier (20 May, p 1324). I have been astonished that this earlier paper did not provoke a flood of correspondence.

This trial showed that patients who received explanation and information about a barium meal or enema were less anxious than those who did not. Why on earth was such a trial necessary? Can there really be anyone anywhere who is surprised that explanation reduced anxiety? For those who cannot believe their common sense without a scientific paper to quote in their support there are already the equally unnecessary papers quoted in the introduction to the later paper.

However, what is most disturbing is the use of a control group. Can it be ethically and humanly acceptable to give procedures like barium studies to a group of patients and deliberately withhold explanation? Perhaps the simple and cheap innovation of subjecting each doctor to the common investigations that he or she uses would make us kinder and more humane doctors.

D HASLAM

Huntingdon, Cambs

* * * We sent a copy of this letter to Dr Wilson-Barnett, whose reply is printed below.—ED, BMJ.

SIR,—Far from there being a flood of criticism over this short research report the correspondence, both personal and published (24 June, p 1699), has been wholly complimentary.

This experimental study assessed the degree of anxiety evoked by barium x-rays and examined the effect of giving patients a

full explanation of the procedures and sensations involved well before screening. The fact that this explanation would alleviate anxiety was not accepted as common sense by staff, many feeling that explanation might have an adverse effect on patients.

The suggestion by Dr Haslam that information was deliberately withheld from control subjects was not the case. Control patients were subject to the routine and normal preparation for barium x-rays by staff. However, in other studies,¹ including that praised by Dr Haslam, patients were found to receive very little information about what to expect during investigations. Many staff members excuse themselves by saying, "Patients would rather not know." It may well be that they also do not have detailed knowledge about the ever-increasing number of diagnostic tests. Is there a better way to convince them that patients benefit from such explanation than to produce objective evidence?

If, as seems to be the case, information-giving to patients is neither common sense nor common practice in hospital, and if Dr Haslam believes that withholding information is unethical and unacceptable, is he suggesting that much of what goes on in hospital care is therefore unethical and unacceptable?

JENIFER WILSON-BARNETT

Department of Nursing Studies,
Chelsea College,
London SW3

¹ Wilson-Barnett, J, and Carrigy, A, *Journal of Advanced Nursing*, 1978, **3**, 221.

Economising on drugs

SIR,—Your leading article "Freedom to prescribe—in ignorance" (17 June, p 1573) comments on the various activities of hospital therapeutic committees.

It is not only in hospitals that the attempt to produce a basic list of drugs has been made. I am one of a group of six dispensing doctors and the need for ever-increasing economy in maintaining our drug stock led us to review the drugs we use with the aim of producing a basic stock list. This does take relatively as much time as you describe in hospital, and what started as an exercise to achieve financial savings has become a valuable educational exercise. Justifying a particular drug choice to one's colleagues, including trainees, concentrates the mind wonderfully, and we have even managed to produce a practice policy for the use of some categories of drug.

I am sure other people are working along similar lines and I would be interested to hear of any conclusions as an aid to our own efforts.

I R WALLACE

Newmarket, Suffolk

SIR,—In attempting to deal with this problem (leading article, 17 June, p 1573), I hope that both doctors and the Department of Health and Social Security will attempt to limit our ignorance and not our freedom. In an attempt to control prescribing habits Australia has four categories of drug. The result is more paperwork for everyone and the need to get special permission from the Department of Health to prescribe a drug such as flucloxacillin; and at the same time the prescribing

in some practices is as haphazard and irrational as any I have heard of in Britain. No amount of regulation will guarantee good prescribing.

By all means update the *British National Formulary*. By all means persuade the Association of the British Pharmaceutical industry to put page numbers in the index of approved names in their *Data Sheet Compendium*, which, despite its limitations and your doubts (3 June, p 1435), is a most useful handbook. By all means bombard us with advice in the *Prescribers' Journal* and the *Drug and Therapeutics Bulletin*. But let us resist the apparently simple solution of regulations and restrictions.

CHARLES A WEST

Church Stretton, Salop

Surgical approaches and drug treatment in the carcinoid syndrome

SIR,—Your leading article (17 June, p 1572) on the treatment of the carcinoid syndrome suggested that before hepatic artery ligation a course of cytotoxic drugs should be given. This would cause some release of vasoactive substances from the tumour and prevent a "carcinoid crisis" occurring with the more major necrosis that would follow ligation of the artery.

We have recently studied the 24-h urinary excretion of 5-hydroxyindole acetic acid in a patient with carcinoid syndrome and liver metastases (reported to the Royal Society of Medicine, Section of Endocrinology, Cambridge, May 1978) and found that after a bolus injection of cytotoxic drugs (cyclophosphamide, methotrexate, and doxorubicin (Adriamycin)) the excretion increased at least 25-fold. This presumably reflects a similar (or greater) release of pharmacological mediators from the tumour into the circulation, with the possibility of precipitating a crisis. Before any medical or surgical interference with a carcinoid tumour it is safer to try to inhibit the synthesis and effect of these substances by the administration of agents such as *p*-chlorophenylalanine and cyproheptadine.

The latter drug was very useful in controlling this patient's severe skin symptoms and it is surprising that no mention is made of its use in your article, as it appears to be relatively non-toxic and effective.

PAUL SIKLOS

Addenbrooke's Hospital,
Cambridge

Replacement therapy in Turner's syndrome

SIR,—The report by Drs J L Margo and K E Hawton of the association of anorexia nervosa with Turner's syndrome (1 July, p 15) highlights what is, in my view, regrettable but common practice. Here a girl was diagnosed as having Turner's syndrome when she was a child, but it was not until the age of 18 that she began treatment with oestrogens to induce female secondary sex characteristics. I do not suggest that this delay precipitated her anorexia nervosa, but it must have provoked considerable and avoidable anxiety.

There is no evidence that the time of starting oestrogen replacement therapy affects the final height of these patients.¹ Accordingly