publicity in recent years," and the implication that "dyslectics," like me, should be considered together with other children with reading backwardness.

As I understand it, "dyslexia" can only be identified by a disparate performance in reading (A) as against other intellectual modalities (B)—a low value of a ratio A:B. This is, of course, discernible only if A is low and B is high, so we cannot possibly have any idea of the number of children who have the positive attributes of dyslexia leading to a low value of A if it is not discernible because B is not high enough. The children who are identifiable by present techniques must be a small minority of the real problem, so it is unlikely to be uncommon.

The problem deserves special stress not only because of its size but because it is aggravated by arbitrarily imposed social policy. There are many ways of communicating, and the stress on the written word in education and above all in examination has led to a vicious spiral of selection for opportunities for those good at these functions. There is some reason to believe that they will become less relevant, and the deprivation of able dyslectics of opportunity purely on these grounds is surely morally unjustified, and possibly damaging, not only to us but also perhaps to the community if our different attitudes provide another perspective of things. We are not talking about a disease but about a difference.

Egalitarian approaches to education are clearly biologically unsound, and the principal problem for the dyslectic. These policies are readily reversible, and the dyslectic's potential may be so great that any implication of disability is obvious nonsense. Still the talk is of "remedial education" when what many of us need is "appropriate education," accentuating and making full use of our strengths. These are the reasons why both the size and the nature of the problem fully justify special consideration, and the publicity so far has been quite inadequate.

J F SOOTHILL

Department of Immunology,
Institute of Child Health,
London WC1

¹ Department of Education and Science, *Report of the Committee of Enquiry into the Education of Handicapped Children and Young People*. London, HMSO, 1978.

Contamination of sterile fluids

SIR,—In their letter on this subject (8 July, p 123) Dr G Ayliffe and his colleagues imply their acceptance of the concept that "if it's sterile it's clean." As a pharmacist I apply the reverse concept that "if it's clean enough it's also likely to be sterile."

Microbiological contamination of intravenous fluids has led to spectacular and unpleasant problems. General contamination of fluids manufactured in an unclean environment or used in an unclean way rarely leads to spectacular or "headline" problems. The damage¹ to patients in these circumstances is insidious and might well have long-term deleterious effects.

To produce fluids acceptably free from micro-organisms is easy—your correspondents admit that the likeliest contaminant organisms are easily killed by moist heat. To produce fluids sufficiently free from rubbish of clinical significance is not easy. A very clean working

environment in the fluid production area helps. Such fastidiousness is not simply designed to reduce hospital infection, as your correspondents imply, but to reduce morbidity of patients generally, be it from micro-organisms, cotton fibres, or plastic going down the cannula.

Therefore I believe that at least one high standard "manufacturing" centre is required per area health authority.

I W MARSHALL

St James's Hospital,
Leeds

¹ Dimmick, J E, *et al*, *New England Journal of Medicine*, 1975, **202**, 685.

Hazard of chemical sympathectomy

SIR,—It is a pity that enthusiasts for a technique cannot admit that there are other methods which are equally effective and safe if properly performed. We did not wish to decry the use of the image intensifier for chemical sympathectomies (29 April, p 1143), and where it is readily available some operators may well find its use of considerable assistance. However, where it is not easily available it was our intention to suggest that the procedure can be done safely without it. Our experience and that of many others in Britain (without scouring the world for support!) give credence to this contention.

Hoxton¹ stated that "the whole technique of inserting the needles with accuracy should be mastered by careful practice in many cases before any injections of phenol are undertaken." Reid *et al*² and Hoxton stressed the need for the needle to be deeply inserted, a point we also made in our original letter. Dr R A Boas and others (24 June, p 1700) doubtless noted too that the technique to which we referred³ included a test injection of lignocaine which would reveal inadvertent intrathecal injection. Incidentally, we also follow the injection of phenol in water with a small volume of radio-opaque dye and the patient is then sent to the x-ray department for posteroanterior and lateral views of the lumbar spine.

To suggest that the use of the image intensifier is a foolproof method of ensuring correct needle placement and is devoid of all risks is hardly true. Furthermore, it is surely a little presumptuous and certainly unfair, without further details of the case, of Drs A P Rubin and B R Master (25 March, p 790) to suggest that the complication described by Mr R C Smith and others (4 March, p 552) in their report "is entirely preventable" by its use. It may express the authors' convictions, but it has very serious medicolegal implications and its accuracy is open to question since the absence of reports of mishaps may not necessarily mean that there have not been any.

In summary, we believe that there is more than one way of performing a satisfactory chemical sympathectomy but that none is entirely free from hazard. Whatever technique is adopted considerable experience and care are necessary in order to reduce the risks to a minimum. We feel it is unreasonable that a patient should be denied the benefit of a chemical sympathectomy merely because the facility of an image intensifier is not available. In those cases in which complications have occurred it would be necessary to examine every detail of technique before one could

claim either that it was avoidable or that an image intensifier could have prevented it.

JAMES M B BURN
L LANGDON

Shackleton Department of Anaesthetics,
Southampton General Hospital,
Southampton

¹ Hoxton, H A, *British Medical Journal*, 1949, **1**, 1026.

² Reid, W, Watt, J K, and Gray, T G, *British Journal of Surgery*, 1970, **57**, 45.

³ Mehta, M, *Intractable Pain*, p 213. Philadelphia, Saunders, 1973.

Thrombocytopenia and subclavian cannulation

SIR,—The safe placement and efficient function of catheters for long-continued venous access are important practical considerations in clinical medicine. The problem arises when superficial veins are not available and here direct puncture of large vessels may be unavoidable. One site that is popular for central venous cannulation is the percutaneous introduction of the catheter into the subclavian vein, and to those experienced in the technique the subclavicular approach is useful, reliable, and quick. However, complications may arise, including pneumothorax and bleeding from the vessel wall. The latter may assume significance in the patient with disturbed haemostasis and we have recently encountered the development of large haemothoraces in two thrombocytopenic patients following apparently technically successful introduction of the catheter.

The first patient was a 14-year-old girl with severe acute aplastic anaemia. She had previously sustained a cerebral haemorrhage from which recovery had been complete and was referred for bone marrow transplantation because no haematological response had followed conservative therapy. Superficial veins were inaccessible and the subclavian vein was easily cannulated by a surgeon experienced in the procedure. The platelet count was $19 \times 10^9/l$ ($19\,000/mm^3$) and since neither bleeding nor purpura was present and the patient was scheduled for transplantation allogeneic platelets were not infused. Some hours later a large haemothorax developed requiring blood and platelet transfusion. The patient subsequently died from a second cerebrovascular accident before transplantation could be performed.

The second patient was a 42-year-old woman with stage IV lymphocytic lymphoma. She was admitted with uterine bleeding and a platelet count of $19 \times 10^9/l$ ($19\,000/mm^3$) following cytotoxic chemotherapy. Since no superficial veins were accessible and no other bleeding was evident subclavian cannulation was undertaken by one of us (SCM). The procedure was uneventful, but over the next few hours a large haemothorax appeared, requiring surgical drainage.

We have now come to consider thrombocytopenia to be a relative contraindication to percutaneous subclavian cannulation, particularly in patients who have already bled. When this technique offers the only route of venous access allogeneic platelet transfusion appears a sensible prophylactic procedure.

In the light of this experience we now introduce a large-bore cannula under direct vision, particularly when long-continued venous access will be required as in patients with acute leukaemia and those undergoing bone marrow transplantation. To date we have experienced no difficulties with the latter procedure and even when retained for periods over 30 days infection has not been

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