febrile convulsion. In the light of our own and other data⁷⁻⁹ it would be surprising if the overall incidence of bacteraemia in children with febrile convulsions is as low as that found by Lewis *et al.*² We would argue that blood culture should be more frequently done in all young febrile children and not only those who have presented with a seizure. Optimum management of those children found to be bacteraemic must await further study.

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Erythrapheresis in polycythaemia

SIR,—I was interested to read the report of erythrapheresis in patients with polycythaemia secondary to hypoxic lung disease (12 February, p 511). I noticed that no mention was actually made of blood volume estimation, which leaves the question of whether the individual patients were suffering from true or relative polycythaemia unanswered. In a sense the distinction may be unimportant since a raised erythrocyte sedimentation rate (or haemoglobin) from whatever cause is associated with an increased morbidity and mortality.¹⁻³ The authors compare the merits of their techniques with other methods for reducing red cell mass—for example, by venesection—"which may have fatal consequences" and take a long time to achieve a similar result,⁴ as does continuous oxygen administration.⁵ They kindly quote my original paper on exchange transfusion⁶ but state, "Isovolaemic exchange transfusion with the plasma expander dextran 40 allows more rapid lowering of packed cell volume, but it must be infused over about 12 hours and causes fluid shifts between vascular compartments." In fact no mention was made of having to infuse dextran 40 over about 12 hours. The only reference to time was in an individual case where 2000 ml of blood was replaced safely by a similar volume of dextran 40 within 75 minutes. I think the authors are confusing the details with a paper describing exchange transfusion with dextran 40 in polycythaemia secondary to hypoxic lung disease⁷ (to which no actual reference is made). Here the beneficial results accord remarkably similarly to those found by using erythrapheresis. Although a coauthor in that trial, I have never specified that a dextran

infusion needs to be kept running after the procedure unless it is indicated in its own right irrespective of the procedure.

There are, of course, technical problems in venesection thick sticky blood. Our present practice therefore is to insert a green "medicut" catheter into a radial artery and to lead blood away via a transfusion giving set.⁸ By using the thumbscrew clamp one can precisely regulate the rate at which blood is taken to the rate of infusion. Dr Wedzicha and his colleagues are correct in saying that dextran 40 causes fluid shift. It is hyperoncotic and when transfused rapidly will increase the circulating blood volume by about two "units" for each unit transfused. For this reason we now transfuse the patient with equal quantities of Hartmann's solution and dextran 40 to a volume similar to the blood taken and at the same rate. The infusion is iso-oncotic and abrupt changes in blood volume are avoided. The procedure is simple, safe, fast, cheap, and effective and can be performed anywhere, including in the home, where, for example, a patient with an acute thromboembolic stroke can be treated. Erythrapheresis requires expensive complicated equipment, takes longer, is available in few centres, and cannot be easily transported. It does, however, have the merit that red cells alone are discarded and so as a research tool it can help clarify the issue, "to what can we ascribe the change in the patient's condition following exchange transfusion?" If this "space age leech" does nothing else but draw people's attention to the ill effects of too many red cells² it will justify itself.

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Personality and socks

SIR,—I hope that Professor Philip Rhodes's advice on non-verbal communication (26 February, p 706) will linger after his illustrations have faded for life would be drab if every young doctor destined for interview was attired in charcoal or dark grey suit, plain shirt, plain tie, and black shoes. The only discretion allowed seemed to be in the choice of socks, which are not mentioned. This is where personality could show through, subtly indicating an appreciation of those things in life held to be important. Red is likely to be the in colour in industrial towns, while true blue may gain favour in the shires. Socks are generally expected to match, and it would be unwise to hedge bets by wearing one blue and one red. Yellow is becoming fashionable, but,

if in doubt, a pattern reflecting the colours of the local football team may be the safer ploy and could put the candidate one up. No special measures are necessary to display the hosiery—indeed, overtightening the braces is contraindicated. Should you be asked to sit behind a table which might obscure the committee's view of the socks bear this with equanimity and rely on the sharp eyes of the assessors to pick up the message (albeit subliminally) as you enter and leave the room.

In seaside resorts absence of socks might go unnoticed during the summer, but holy socks would not be forgiven even in a cathedral city. Unless you can let slip that you own a Rolls silk socks may be the best way of suggesting you are a high flyer. On the other hand (or rather foot), hand knitted socks may appeal to the homely lay member and would be ideal for concealing an electronic device for recording what was promised at interview or, if unsuccessful, what went wrong.

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Computed tomography in the investigation of dementia

SIR,—Dr J R Bradshaw and others (22 January, p 277) have made an important contribution to the practical approach to the patient with dementia of recent onset by presenting the results of computed tomography of the brain used as part of the diagnostic investigations in 500 consecutive cases. While acknowledging that: "It is clearly impracticable to scan all patients with dementia," the authors identified the presence of headache, focal signs, papilloedema, and speech disorder as helpful diagnostic pointers towards the presence of brain tumours, subdural hematomas, and hydrocephalus—all structural lesions of the brain which are potentially curable and easily recognised on the computed tomogram. I believe, however, that the authors' conclusion is rather timid because their paper contains factual evidence suggesting that a total of 10 clinical features have a positive correlation with the subsequent finding on the computed tomogram of a potentially treatable brain lesion.

The demented patients studied can be divided into two subgroups: 52 individuals with evidence on the computed tomogram of brain tumours, hydrocephalus, or subdural hematomas and 448 individuals without such structural lesions. The 20 clinical features analysed can be construed as testing the presence of the structural lesions mentioned, and their sensitivity and specificity can be calculated by changing the format of the data. For example, focal neurological signs were present in 17 patients with potentially treatable structural lesions and 52 patients without evidence on the computed tomogram of such lesions. The sensitivity of this clinical feature—that is, the proportion of patients correctly identified as having structural lesions—is 44%; its specificity—that is, the proportion of patients without structural lesions who did not have focal neurological signs—is 88%.

After calculating the sensitivity and specificity for each clinical feature one has to determine if these parameters are good enough for the original purpose—that is, the separation, however imperfect, of the two subgroups. This determination is possible by

calculating the receiver operating characteristic for each sensitivity-specificity pair.^{1,2} When analysed in this way 10 clinical features emerged as having testing ability; they are listed in the table in order of their value.

Receiver operating characteristics of clinical indicators for brain computed tomography in patients with recent onset dementia

Clinical indicator	Sensitivity (%)	Specificity (%)	d'
Focal neurological signs	42	88	0.97
Papilloedema	6	100	0.87
Headache	17	95	0.69
Mental confusion	52	65	0.43
Speech disorder	19	90	0.41
Age group 65-74	50	61	0.27
Ataxia	11	93	0.25
Female sex	54	56	0.25
Seizures	6	96	0.20
Disorientation	15	89	0.19

Sensitivity = Number of patients with the clinical indicator present in the group of patients with potentially reversible structural brain lesions.
Specificity = Number of patients with the clinical indicator absent in the group of patients without potentially reversible structural brain lesions.
d' = Discrimination index for each clinical indicator's receiver operating characteristic curve.

In a patient with recent onset dementia the presence of focal neurological signs, papilloedema, headache, mental confusion, and speech disorder are relatively strong indicators that a structural brain lesion may be present; women, those in the 65-74 age group, and patients with ataxia, seizures, or disorientation should also be considered for computed tomography of the brain.

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¹ Weinstein MC, Fineberg HV, Elstein AS, *et al.*, eds. *Clinical decision analysis*. Philadelphia: WB Saunders, 1980:118-21.

² Swets JA. The relative operating characteristic in psychology. *Science* 1973;182:990-1000.

Calcium antagonist withdrawal syndrome

SIR,—The report by Dr V Bala Subramanian and others (12 February, p 520) purports to demonstrate a calcium antagonist withdrawal syndrome similar to the withdrawal phenomenon which may follow abrupt cessation of beta-adrenergic blocking drugs. They observed the effects of withdrawal of calcium ion antagonists in 143 patients using ambulatory ST segment electrocardiographic monitoring with a frequency modulated tape recorder. They found: "clear deterioration in the signs and symptoms of myocardial ischaemia in five patients." It is not clear from their report, however, whether a true "rebound phenomenon" occurred or whether worsening signs and symptoms, effectively suppressed by treatment, simply reappeared following discontinuation of treatment. The three case reports presented do not establish this.

Rebound phenomena have not been encountered in the many previous studies in which calcium ion antagonists have been suddenly withdrawn. Although Dr Subramanian and others used a particularly sensitive method, which other investigators have not always used, their cases were obviously sufficiently symptomatic following drug withdrawal to attract medical attention. The study involved a large number of patients treated (in the three instances reported) for several months. The relevance of the variety of calcium ion antagonist, dose and duration of treatment, or with-

drawal protocol cannot be assessed from the information available.

It is obviously important to establish the incidence of a calcium ion antagonist withdrawal syndrome, and the best evidence of its existence will derive from double blind placebo controlled trials. Recently Frishman *et al.*¹ compared the effects of abrupt withdrawal from long term treatment with propranolol or verapamil in a placebo controlled double blind randomised cross over study of 20 patients. They found no evidence of deterioration from placebo baseline levels following abrupt withdrawal of verapamil, whereas two patients had a severe exacerbation of angina following abrupt withdrawal of propranolol.

At present, in contrast to beta-blockers, there is no conclusive evidence that the calcium ion antagonist withdrawal syndrome exists.

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¹ Frishman H, Klein N, Strom J, *et al.* Comparative effects of abrupt withdrawal of propranolol and verapamil in angina pectoris. *Am J Cardiol* 1982; 50:1191-5.

Writer's cramp

SIR,—Dr P Hudgson (19 February, p 585) rejects our suggestion¹ that writer's cramp is a form of focal dystonia for the wrong reasons.

He writes: "The painful muscle spasms characteristic of writer's cramp and related disorders are induced only by the movements with which they are associated, whereas dystonic posturing in torsion dystonia often develops spontaneously and is not necessarily painful." Dr Hudgson assumes that writer's cramp is painful, but this is not usually the case. Only 11 of our present series of 72 patients with this condition have complained of pain. The key feature of the illness is the abnormal muscle spasm that occurs on writing. Even those who compose our dictionaries are confused over this matter. The fifth edition of the *Concise Oxford Dictionary* (1964) defines a cramp only as a "contraction of muscles," but the latest seventh edition now includes the word pain. Because of these problems some authors prefer to call writer's cramp "graphospasm." The second point raised in this particular criticism is also open to question. While the muscle spasms of full blown generalised torsion dystonia may appear spontaneously, the illness typically begins with an "action dystonia" of the kind seen in writer's cramp and related disorders. Indeed, in a series of 42 patients with idiopathic torsion dystonia (dystonia musculorum deformans) the first symptom was an action dystonia of the arm or leg in no less than 28 cases.²

Dr Hudgson continues: "There is no published evidence that writer's cramp ever evolves into generalised torsion dystonia. . . ." In that same series of 42 patients with dystonia musculorum deformans reported in 1974² no less than eight patients first complained of a writer's cramp. In a subsequent publication³ we reported that writer's cramp was the first symptom of classical idiopathic torsion dystonia in 13% of cases and occurred in the course of the disease in as many as 77% of patients.

"Torsion dystonia is an inherited disorder . . . there is no clear cut evidence implicating genetic factor[s] in the aetiology of writer's cramp," he also writes. We agree, but this is true of all other isolated focal dystonias with onset in adult life, including spasmodic torticollis and cranial dystonia (blepharospasm/oromandibular dystonia). These latter conditions, like writer's cramp, may be the presenting feature or may occur in the course of inherited dystonia musculorum deformans. When they appear for the first time in previously healthy adults, however, there is rarely a family history and the condition is unlikely to progress. The

background and prognosis for adult onset focal dystonia is very different from that in childhood, despite the similarity of the movement disorder.⁴ Occasional adults with any of these focal dystonias may, however, have inherited the disease from their forebears and may pass on classical generalised torsion dystonia to their children, although this is rare.

Of course, all this does not provide any answers to the aetiology and pathophysiology of writer's cramp and the other "occupational neuroses." One of the main points of our paper¹ was to show that writer's cramp was not due to some psychological disturbance, which has been widely assumed from the unfortunate use of the term "neurosis" in this context. Gowers originally employed the word to refer to a physical illness with no discernible cause rather than implicating what we now mean by a neurotic illness. Sadly, however, many practitioners still seek some psychological cause for writer's cramp, much to the distress of those afflicted.

The cause of writer's cramp is unknown. As to its pathophysiology we speculated that: "The brain must contain a mechanism capable of generating the engram of our script independent of which muscles are required, and of sensory feedback, so we would look to some breakdown of this mechanism to explain writer's cramp."² This suggestion did not imply the existence of any "writing centre," so was not dependent on the concepts of Gowers which Dr Hudgson considered a "less than entirely reliable pillar." It was based on the straightforward observation that we all have our characteristic signature, which is executed independent of the muscles involved and with the eyes shut or the hand anaesthetised. Some programme for that sequence of movement must be contained within the nervous system. We suggest that a defect of that programme or of its execution may be responsible for writer's cramp.

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² Marsden CD, Harrison MJG. Idiopathic torsion dystonia (dystonia musculorum deformans). A review of forty-two patients. *Brain* 1974;97: 793-810.

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How will the new genetics work?

SIR,—The recent summary by Dr Tony Smith on the new genetics (1 January, p 1) will help to expose doctors to some of the recent developments in molecular genetics which will assume clinical relevance over the next few years. I would like to note three other recent findings which bear on his discussion.

Dr Smith mentioned the increasingly common use of restriction enzyme polymorphisms and linkage studies to detect genetic disorders either prenatally or pre-symptomatically. These studies are presently limited by the need to have common polymorphisms coupled with appropriate family studies to be of use, and these are not always available. Two groups have recently shown that specific short (less than 20 bases)