antigen is considered to have worse immune function than a patient who produces weak positive reactions to two antigens. We still think that this definition of relative anergy is difficult to reconcile with the accepted conceptual framework of cell-mediated immunity.

The statement that skin testing is widely used in North America was based on the considerable publicity it has received and the suggestion that failure to skin test patients before operation is malpractice (Aspen meeting, 1980). We apologise for misquoting Dr Meakins and others regarding the use of parenteral nutrition before operation; we should have referred to another of their papers which states that: "Aggressive total parenteral nutrition constitutes the best single proved method to manage the anergic patient."² This report also indicates that a single skin test is of limited value and that serial testing provides more information regarding the prognosis of an individual patient. We have also performed such a study,⁹ but we obtained little information which was not available from clinical examination and easily determined biochemical indices.

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Metabolic acidosis in paracetamol poisoning

SIR,—Dr A Zezulka and Dr N Wright (25 September, p 851) describe lactic acidosis occurring 11 hours after ingestion of 75 g of paracetamol, and attribute the acidosis to paracetamol toxicity. There is experimental evidence in animals which shows that paracetamol can inhibit glucose synthesis from lactate and can stimulate lactic acid production in the liver via glycolysis.¹ In our experience, however, gross abnormalities of lactic acid metabolism are unusual after paracetamol poisoning.

We have measured blood lactate concentrations in 20 samples from eight patients who had taken overdoses of paracetamol in the preceding 24 hours. At the time of sampling all patients had normal liver function tests and a normal anion gap. The amount of paracetamol ingested was 25 ± 14 g (mean $\pm$ SD) and the blood lactate concentration was 1.4 ± 0.6 mmol/l (12.6 ± 5.4 mg/100 ml), which is similar to the value of 1.1 ± 0.1 mmol/l (9.9 ± 0.9 mg/100 ml) reported in a group of inpatients with a variety of illnesses.² Average blood lactate concentration in individual

patients was not correlated with the quantity of paracetamol ingested ($r=0.36$, $p>0.15$). We question whether Dr Zezulka and Dr Wright are correct in attributing the lactic acidosis to paracetamol, although we recognise that their patient took a larger overdose than did any of ours. Many overdoses involve more than one drug, and we wonder whether another agent—for example, alcohol—might have caused the acidosis in this case.

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- ² Huckabee WE. *Am J Med* 1961;30:833-8.

SIR,—We were interested to read the report by Dr A Zezulka and Dr N Wright (25 September, p 851) which suggests that metabolic acidosis may occur early in severe paracetamol poisoning. The following case report supports this and also shows the value of charcoal haemoperfusion in those who present late with high plasma paracetamol levels.

A 43-year-old woman was admitted unconscious having been known to be fully conscious 12 hours previously. She was in grade IV coma (classified according to the Edinburgh criteria),¹ hypotensive (blood pressure 100/60 mm Hg), but breathing spontaneously. She also had persistent recurrent myoclonus with vertical nystagmus. Fluid from a lumbar puncture performed at the time was heavily blood stained. Biochemical investigations showed a severe metabolic acidosis (pH 7.1) with a low plasma bicarbonate concentration (6 mmol (mEq)/l). The remaining electrolytes and urea concentrations were normal. A full blood count and platelet count were also within normal limits. A coagulation profile showed a prothrombin time of 20 seconds (control 12 seconds) and a kaolin-cephalin time of 32 seconds (control 30 seconds). Computed tomography could not be performed at this time because the patient was too restless, and she was therefore transferred from a neurosurgery unit to Guy's hospital as a suspected case of salicylate poisoning. Toxicological analysis, however, showed a plasma paracetamol concentration of 1150 mg/l with no other drugs present. An infusion of acetylcysteine was started, but in view of the very high plasma concentration of paracetamol and the uncertain time of ingestion it was decided to remove the drug by haemoperfusion using a Kurary DHP-1 column (Medical Products Department, Kurary Co Ltd, Osaka, Japan). This was started 17 hours after admission (paracetamol level 810 mg/l) and continued for 10 hours (paracetamol level 47 mg/l). The patient subsequently developed convulsions and required ventilation. Biochemical analysis showed that the metabolic acidosis had been corrected and there were no other abnormalities, but a computed tomogram showed multiple cerebral infarcts and a left-frontal intracerebral haematoma. She remained unconscious for a further 48 hours, during which time liver transaminase concentrations became slightly raised (aspartate aminotransferase 372 IU/l), but these and the prothrombin time returned to normal. At the end of this period the patient died as a result of her neurological damage, and a postmortem examination confirmed the results of the computed tomogram. During haemoperfusion 26.5 g paracetamol was removed.

We have reported previously² that haemoperfusion may be of value in patients severely poisoned with paracetamol who present late. It is possible that this patient developed only mild hepatic damage because of the removal of a significant quantity of paracetamol by this technique. A series of four cases of

paracetamol poisoning whose early course was complicated by metabolic acidosis in the absence of hepatic failure will be reported in detail shortly.

We are grateful to Professor M H Lessof for permission to report details of this patient, who was under his care.

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- ¹ Matthew H, Lawson AAH. *Treatment of common acute poisonings*. 4th ed. Edinburgh: Churchill Livingstone, 1979.
- ² Winchester JF, Gelfand MC, Helliwell M, et al. *Arch Intern Med* 1981;141:370-4.

SIR,—Paracetamol has unfortunately become a popular choice of drug for deliberate overdose, which is often fatal unless early treatment can be instituted. The recent case report (25 September, p 851) prompts us to describe a similar experience.

A 15-year-old girl took 80 paracetamol and eight temazepam tablets after a family row. Sixteen hours later she arrived in the casualty department, where her paracetamol level was found to be 190 mg/l; she was given appropriate treatment with acetylcysteine, vitamin K 10 mg, and in view of a metabolic acidosis (pH 7.2) 200 ml of 4.2% sodium bicarbonate, and was referred to our unit for further treatment. She arrived about 20 hours after ingestion with a paracetamol level of 238 mg/l, a prothrombin time of 28 seconds, and a serum aspartate transaminase value of 171 IU/l. Charcoal haemoperfusion, a regimen for treating liver failure, and fresh plasma were given. At 36 hours, when haemoperfusion was discontinued her paracetamol value was <20 mg/l but her prothrombin time was 36 seconds and serum aspartate transaminase value 353 IU/l; she was again given fresh plasma. She remained conscious, but by 60 hours the prothrombin time was 60 seconds and serum aspartate transaminase value >3000 IU/l; she was treated again with fresh plasma. She gradually improved, and by the seventh day after admission her liver function had returned to normal. She was discharged to the ward, and later returned home.

In this case metabolic acidosis was not as severe as in the recent report, but we suggest this represents a deteriorating clinical situation associated with late presentation rather than a specific entity. Both these cases give credence to the growing evidence^{1,2} that late treatment of paracetamol poisoning should always be undertaken even when the predicted outcome appears grave.

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Aflatoxins and kwashiorkor

SIR,—The article by Professor R G Hendrickse and others (25 September, p 843) recalls research at the National Institute for Medical Research by A J P Martin and myself.¹ In two successive winters work had to be suspended because the biological tests in guinea-pigs gave inconsistent results owing to the animals developing a syndrome analogous to kwashiorkor. This disease occurred in winter in many laboratories in which, as in our case, Bruce and Parkes selected diet 18 was the

main source of food.² The addition of skimmed milk to the diet improved the condition even though an amino-acid analysis of the diet showed it to be satisfactory, although the methionine content was on the low side. Later work showed the syndrome to be due to aflatoxin in the peanut meal in diet 18.

Professor Brock of Cape Town, the world authority on kwashiorkor, visited the laboratory at that time and studied slides of liver etc produced by the late Dr Janet Niven. Among the liver changes Dr Niven found incidence of malignant change—all the more remarkable because the guinea-pig seldom develops cancer, man, monkey, and guinea-pig being cancer-resistant species.³

In brief, the guinea-pig fed on a diet adequate in protein produces a kwashiorkor-like syndrome which is improved by feeding additional protein, and this provides evidence to support the hypothesis that the disease in man may be due to aflatoxins rather than to malnutrition.

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¹ Long DA, Martin AJP. *Lancet* 1956;i:464.

² Paget GE. *J Pathol Bacteriol* 1954;67:393.

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Appointment of senior administrative staff

SIR,—The advisory appointments committees for consultant posts include a non-medical member, as do those for senior registrar posts. Yet for the appointment of senior administrative staff in the districts of the National Health Service for the 1982 reorganisation there is no stipulation that there should be a medical member of such an advisory committee. For efficient working of the National Health Service, however, both the medical staff and the administrators need to work in harmony. It does seem regrettable that there is no such medical input.

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Career intentions of senior house officers in paediatrics

SIR,—The Short Report recommended an increase in consultant posts and a reduction of junior posts to match training needs. The number of senior house officer posts has consequently been frozen. Local discussions make it clear that there is widespread misunderstanding among health service administrators about the career intentions of paediatric SHOs.

A recent survey of junior paediatric staff in the Yorkshire region (51 responses from 54 SHOs) showed the following. Of 41 British SHOs 28 plan to enter general practice (25 are in general practitioner vocational training schemes), nine wish to become paediatricians, two obstetricians, two are undecided. Twenty-two are female. Of 10 non-British SHOs eight wish to practice paediatrics, all overseas, and two wish to enter general practice. Realistically not more than 10% of paediatric SHOs will become consultant paediatricians.

The same inquiry showed that although most paediatric registrars wish to become paediatricians some are overseas doctors intending to practice overseas and others will not succeed in fulfilling their ambitions. Realistically about 50% of paediatric registrars will become registrars.

Broad agreement has recently been reached on a three-year training programme for clinical medical officers. For those proposing to work in the child health field (about 95% of the total) a six-month SHO post in paediatrics will be mandatory. In addition, paediatric experience at SHO level is often sought (very wisely) by doctors planning to practice obstetrics, child psychiatry, or paediatric surgery.

It is clear that most paediatric SHO posts are used for training general practitioners; many will be needed to train clinical medical officers; very few (perhaps 10%) are occupied by future consultant paediatricians.

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Pathology and university cuts

SIR,—The letter from the President of the Royal College of Pathologists (10 July, p 138) is the latest of several warnings on the staffing crisis in pathology which have appeared in your columns during the past two years. Judging by the most recent medical staffing tables published annually by the Department of Health and Social Security,¹ abstracts of which appeared in the *BMJ* (25 September, p 905), there has so far been no significant improvement, and it seems necessary to try once again to alert the profession to the impending crisis.

In 1980 attention was drawn to the fact that in contrast to the popular bedside specialties three of the pathology specialties (chemical pathology, histopathology, and medical microbiology) were understaffed at SHO and registrar levels,² and it was concluded that about 150 additional posts were required to provide a balanced staffing structure. This paper was accepted by the Central Manpower Committee, and the conclusion has not been disputed.

In its response³ to the fourth report (1981) of the Social Services Committee the Government stated its intention that the ratio of trainees to consultants, currently 1.8 to 1 in the major medical and surgical specialties, should be reversed by 1996. Already the ratio in the shortage pathological specialties is less than 1 to 1.8—that is, beyond the Government's target. It is difficult to consider the staffing structure for the individual pathology specialties at SHO and registrar levels because many posts are designated general pathology and are a general training in the pathological specialties before concentration on one particular specialty. The situation in histopathology, however, is particularly serious for the following reasons.

(1) The number of consultant histopathologists in post in 1972 was estimated by the Royal College of Pathologists to be about half what was required for a satisfactory service.⁴ In spite of a steady increase in work load, which has accelerated recently as a result of modern imaging techniques and the greater scope offered by endoscopy and fine-needle biopsy, the number of consultant histopathologists has since increased by only 1% annually.

(2) Each year many of the requests from regions for new consultant posts in histopathology are rejected by the DHSS because of insufficient numbers of senior registrars known to be completing their training during the year.

(3) The number of consultant vacancies in histopathology over the next 10 years will be unusually high because about 40% of consultant histopathologists are aged 55 or more (compared with 25% for all hospital specialties).

(4) The university staffing cuts are hitting histopathology particularly hard because a higher proportion of consultants are academics (29% compared with 12% for all hospital specialties), and the same applies to senior registrars (37% compared with 22%). Already 56 academic posts in histopathology have been abolished in the past two years, and this exceeds any increase in National Health Service posts. Clearly there are more academic losses to come.

The DHSS tables on medical fields of recruitment (1982) show that over the past six years the number of consultant histopathology posts advertised annually has increased from 30 in 1975/76 to 70 in 1980/81. Of those posts for which data were available for analysis, the percentage of posts remaining unfilled during the year they were advertised has increased from 3% in 1975/76 to 42% in 1980/81, while the average number of candidates per post has fallen to an average of 1.3 over the past three years. Similarly, over the past six years an average of 30% of senior registrar vacancies have remained unfilled compared with 14% for all hospital specialties.

Although some years ago recruitment into histopathology was unsatisfactory unprecedented numbers of applications (in some instances over 60) are now received in response to advertised SHO vacancies, and there is a large number of would-be recruits seeking posts in both teaching and non-teaching hospitals. Based on previous estimates² about 115 additional posts at SHO/registrar level are still urgently needed in the shortage pathology specialties to maintain present consultant staffing levels. These estimates do not allow for the increased level of consultant staffing advocated by the Royal College of Pathologists.⁴ Unfortunately, actions which are being considered and taken—for example, a freeze in the total number of SHO posts—are aimed at correcting the staffing structure in those clinical specialties with excessive numbers of trainees. There is a danger that the needs of shortage specialties will be swept aside by such measures.

A few regions have appreciated the staffing crisis in pathology and have created more training posts, but others have taken no effective steps to try to improve the future supply of consultants. The recent reorganisation of the NHS, with its emphasis on greater autonomy at hospital and district levels, may have many advantages, but the correction of staffing imbalances and particularly the total numbers of training posts in each specialty must surely be planned and executed centrally. This will require not just the imposition of a ceiling on the numbers of posts in congested specialties but also effective steps to increase the numbers of training posts in the shortage specialties.

Unless more training posts in histopathology can be established quickly some districts will find that it is too late to investigate why their histopathology service is ailing. The only form of investigation left will be an autopsy to find out why the service died.

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² Anderson JR, Tighe JR. *Br Med J* 1980;281:1370-2.

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