

cytofluorimetry to calculate the percentage of $\gamma\delta$ T cells in 3 newborn babies with serologically confirmed congenital *Toxoplasma gondii* infection. The proportions of $\gamma\delta$ T lymphocytes (CD45RO/CD45RA) were 9, 11, and 6%, whereas the mean in 19 uninfected children during the first day of life was 1.5% (SE 0.3). The overexpanded $\gamma\delta$ T-cell subset expressed the conventional V γ 9 and V δ 2 gene products, suggesting that the expansion had been driven by specific intrauterine antigenic pressure.¹

At least in the mouse, many $\gamma\delta$ T cells are localised in epithelial layers, which form a first barrier between host and pathogen. Hence, they could perform a critical surveillance function at this prominent port of entry for environmental microorganisms.¹ Activated circulating $\gamma\delta$ T lymphocytes with the V δ 2 phenotype express CD45RO,⁴ and this may, in part, explain why CD45RO+ T cells are increased in the blood of newborn infants with certain congenital infections.

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Is depletion of potassium in blood before transfusion essential?

SIR—Inaba and Shirahama (May 14, p 1223) report the results of a preliminary trial of sodium polystyrene sulphonate, in the filter of a blood giving set, to partly deplete potassium (K⁺) from irradiated red cell products.

Irradiated blood components are needed only by few transfusion recipients, including those who have received organ or bone marrow transplants and patients transfused with directed donations from relatives. In red cell units irradiated on day 1, the K⁺ concentration of the supernatant 28 days later can be as high as 66–70 mmol/L. The average volume of the supernatant in each unit of blood (plasma reduced/in optimum additive solution) is 100 mL. Therefore, the total K⁺ content of each unit would be about 7.0 mmol 28 days post-irradiation. The daily allowance of K⁺ for an adult is 0.7–1.0 mmol/kg per 24 h.¹ Therefore, an adult may be given nearly 10 units of red cells (irradiated 28 days previously) within a 24 h period before the maximum dose of K⁺ is exceeded.

Patients at most risk of hyperkalaemia are very small infants and patients in kidney failure. However, blood that is less than 7 days old is used for exchange and other large volume neonatal transfusions, to fulfill other requirements; patients with known renal failure are usually dialysed after blood transfusion. For patients who require rapid, large volume transfusions, a simple policy to avoid hyperkalaemia would be: (i) for patients who require irradiated blood products, use units irradiated less than 14 days previously; and (ii) for patients who do not require irradiated blood

products, include not more than 10 in-date irradiated units per 24 h.

We feel that a device to deplete K⁺ from red cell products would be necessary only on the very rare occasions when rapid transfusion is needed and there is no option but to use large volumes of irradiated red cells that have been stored for long periods. Large scale use of filters to remove K⁺ from all irradiated units is therefore unwarranted and a waste of valuable resources. Whether or not sodium polystyrene sulphonate releases leachable material, and if so, whether these are harmful also need to be established. Furthermore, the effects of this material on white blood cells and red cells also should be examined before such a device is made available for clinical use.

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Food intolerance

SIR—Young and colleagues' report of food intolerance (May 7, p 1127) from 20% of the 18 880 responders in two independent samples may surprise some readers but the findings were very similar in the two populations, both in the range of foods which triggered symptoms and in the individual symptoms triggered, and the proportions reported for comparable subcategories were too close to have occurred by chance. The study went on to estimate the prevalence of confirmed intolerance to eight foods by double-blind challenges, but there are serious deficiencies in this part of the investigation. Only 106 individuals (not a random sample) completed the challenge procedures, and these were not sensitive enough to exclude intolerance. The calculated prevalence for confirmed food intolerance is thus meaningless.

Doctors who regularly use effective exclusion diets know that recognised food intolerance is the tip of an iceberg, the remainder causing chronic symptoms apparently unrelated to food until "unmasked" by a break in exposure. Bronchospasm from foods has been demonstrated in over 50% of people with asthma when unselected individuals were tested with a wide range of foods^{1,2} compared with less than 4% when the patients and the foods were selected from the history and/or tests. In Young and colleagues' study, the few controls chosen because they were unaware of food intolerance showed a higher rate than the estimate for the study as a whole. Wheat, corn, and milk are among the most frequent causes of masked food allergy, so the choice of corn as a placebo was inappropriate. In another study,³ a reaction to corn was disregarded in a patient who also reacted to placebo (glucose); most glucose is made from corn. Young et al judged food intolerance solely by the difference between the symptom scores for test and placebo periods: genuine reactions would be obscured if the increased amount of corn (or rice) eaten during the placebo period caused symptoms. How many patients had symptoms with the placebo?

Young et al concede that the temperature of canning may have resulted in an underestimate but do not say how much of each test food was included in the cans or bars. Objective reactions may require a challenge dose of 100 g or more,⁴ and the amounts used by Young et al may not have been sufficient.

Some patients may already have been avoiding the challenge foods: avoidance often leads to an unstable tolerance³ which might not be broken by small quantities of the food on three successive days.

Of the 161 who undertook food exclusion and challenge fewer than 60% completed the study; "intercurrent illness" was one reason for the drop-outs. Wheat had been recognised as a trigger by only 5% of those reporting food intolerance and milk by 15%, although these foods have been implicated much more frequently in comprehensive investigations (eg, of patients with irritable bowel syndrome⁵). All challenged patients were asked to avoid the eight test foods for a week. When foods contributing to masked allergy are avoided most patients have a withdrawal reaction, which can be severe. Some of the "intercurrent illness" was probably withdrawal; even if only half the drop-outs were due to withdrawal reactions the positivity rate would increase from 24% to 34%.

Because fewer of the responders to the short than to the long questionnaire reported problems with food it was assumed that still fewer of the non-responders would have reported reactions, so Young et al arbitrarily reduced the estimate of the hypothetical number of positive challenges in non-responders. This is unjustifiable. Only 17% of initial non-responders returned the shortened questionnaire, and on such a low return the results are likely to be unreliable. There can be no basis for using these results to calculate an even lower rate for the remaining non-responders, without independent checks on the rate of food intolerance in either group.

47 patients were excluded from challenge because of the severity of their reported reactions but only 24 were accepted as showing food intolerance (those found to be RAST or prick-test positive and 25% of the rest). There is no justification for omitting severe food intolerance reactions from the calculations of prevalence (and adding them as an alternative afterthought). If Young et al were only interested in atopic food allergy, this should have been made explicit.

We suggest that the estimate of the prevalence of food intolerance presented in this paper is a serious underestimate.

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SIR—Young and colleagues provide firm evidence, based on double-blind studies, that about 1.6% of the population in a community survey have striking food intolerances. An approach using the prevalence of the most common conditions that readily respond to dietary modification showed a similar estimate of about 2% of the population;¹ this means that about one million people in the UK have important food intolerance. These estimates are probably low because some patients do not recognise that they have a

food intolerance. Investigation and treatment of these patients uses massive National Health Service resources often with little benefit. Many of these intractable patients can be successfully treated at low cost by often minor dietary modification. Recognition of food intolerance has been hampered by lack of understanding of the underlying biochemical and immunological mechanisms. Further research in this area is indicated, especially since the potential for symptom relief is considerable.

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SIR—Young and colleagues state that food intolerance seems to affect 1.4–1.8% of the general UK population whereas at least 19.9% of the Wycombe Health Authority population sample claimed food intolerance. However, we suspect that this prevalence rate, which was based on the results of a placebo-controlled study, is probably an underestimate.

We have done single or double-blind food challenges to evaluate the frequency of allergies. These tests have shown that the eight selected foods of Young's study represent only 60% of food allergies in children and 36.5% in adults.¹

Young and colleagues' controlled food challenges were done according to the results of a questionnaire (as shown in figure 1). Therefore, reactions to masked allergens were not taken into account since they would have escaped the participants' awareness. The facts that a controlled challenge was done with 13 patients having similar symptoms that were apparently unrelated to food, and that one test proved to be positive, tends to support the notion that masked allergies might be more frequent than thought.

Unfortunately, Young and colleagues do not provide precise information about the quantities of fresh food used in challenges tests. If less than 10 g of fresh food is used, it is not possible to detect non-severe allergies or to trigger pharmacological reactions such as headaches, digestive disturbances, &c.²

Lastly, Young and co-workers do not refer to severe anaphylaxis leading to death. Anaphylactic shock should be further studied since a link has been established lately between the high frequency of food allergies and anaphylactic shocks.²⁻⁵ We acknowledge the value of this well documented study, but we fear that the rate of 1.8% might be far from reality.

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Author's reply

SIR—With respect to Anthony and colleagues' comments, corn was carefully chosen as placebo on the basis of previous publications. Few patients had symptoms with placebo: each of the eight food stuffs tested was prepared in the challenge

material as a single dose of 8 g daily. This amount was chosen on the same basis of previous publications. The suggested withdrawal reactions were not within the scope of this study. We were extremely careful in our calculations of the final prevalence and included those who were excluded because of the severity of their reactions; the method of calculation of reactors among those excluded was selected so as not to overestimate the final prevalence figures. We calculated the difference by allowing for 100% and 50% of positive reactors. Again, we argued on our rationale for calculating prevalence on the assumption of a proportionally lesser perceived intolerance rate. Calculations based on 50% and 100% positive reaction rates did not substantially alter the final prevalence rate.

We agree with Finn's comments. We were aware that we needed to look at those who did not perceive a problem, and we tried very hard to recruit to this group, but the level of collaboration was poor since this group did not perceive a need to participate in the study. We are well aware of the comments made by many that we have underestimated the prevalence because we produced prevalence figures for only eight major foods—which accounted for only 49% of the foods reported to us on the questionnaire. We do agree that our prevalence figures are an underestimate and that much work should be done on the other foods that accounted for 51% of our reported reactions. Similar studies to ours need to examine these other foods, and should be done as carefully controlled, double-blind, placebo-controlled trials.

We agree with Moneret-Vautrin and Kanny that the anaphylactic problems should be looked at most carefully since we are seeing an increase in the UK of anaphylaxis due mainly to nut ingestion in young adults. Sadly, this nut ingestion is inadvertent in subjects who knew they were allergic to nuts and had made every effort to avoid them. We have seen an increase in anaphylaxis in these nut-allergic patients, and the problem is further compounded by the fact that in the UK, for the first time, we have seen an increase in latex type I allergy and the possibility that the impure allergen in poor quality latex may possibly crossreact with certain foods—particularly bananas, kiwi, avocado, chocolate, and melon. This cross-reaction should be a matter of great concern. It was not, however, our intention in this study to look at these anaphylactic problems. Their frequency was noted and allowed for in our calculations.

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Recovery after impaired cerebral perfusion in severe head injury

SIR—Davies and colleagues (May 28, p 1329) report 4 patients who had complete neurological recovery despite impaired cerebral perfusion in fulminant hepatic failure. We have recorded good recoveries or moderate disabilities after severe, coma-producing traumatic brain injury in 5 patients with persistent rise in intracranial pressure above 20 mm Hg, associated with reduced cerebral perfusion pressure below 60 mm Hg. In traumatic brain injury, sustained moderate intracranial hypertension (20–40 mm Hg) is associated with poor outcome.¹

Our patients represent a subgroup taken from a consecutive series of 55 comatose patients (age 1–80 years, mean 36·4) with head injury due to trauma, being studied as part of an investigation of the value of neurophysiological measures for predicting outcome. All had severe head injuries with a Glasgow Coma Score (GCS) of 8 or less

(mean 5·9) on admission and after cardiopulmonary resuscitation. Computed tomography (CT) scanning of the head was done. Patients were mechanically ventilated to maintain PaCO₂ at about 35 mm Hg and were sedated with continuous intravenous infusions of morphine (0·02–0·15 mg/kg per h) and propofol (1–3 mg/kg per h). In 30 selected patients the intracranial pressure was monitored by ventricular catheter. Pressure in excess of 25 mm Hg for 5 min or longer, despite satisfactory ventilation, was managed by osmotherapy (mannitol) or barbiturate coma (thiopentone at 1–3 mg/kg per h).

Details of the 5 patients (sex/age) are:

	1 (F/20)	2 (M/19)	3 (M/19)	4 (M/20)	5 (M/17)
Admission GCS	6	3	7	8	6
Craniotomy/ thiopentone	+/+	-/+	-/-	+/+	-/-
Days in coma	10	7	11	13	4
Peak ICP (mm Hg)	43	43	42	55	30
Minimum CCP (mm Hg)	45	56	51	54	52
Hours ICP >20 mm Hg	46	3	19	71	2·5
Hours CCP <60 mm Hg	48	2	1	24	4

+ = yes, - = no; ICP = intracranial pressure; CCP = cerebral perfusion pressure.

Patient 1 had a thin acute subdural haematoma. After 3 months she had made a full recovery, returned to work, and is able to ride her horse again. Patient 2 had diffuse swelling and a haemorrhagic contusion of the left occipital lobe associated with midline shift. After 6 months his only sequelae is hemianopia and he has returned to part time work. Patient 3 had multiple small haematomas suggestive of diffuse shearing injuries. At 6 months he has some visual loss but is otherwise independent at home. Patient 4 had an 80 mL occipital extradural haematoma and bifrontal haemorrhagic contusions. At 3 months he is completely recovered although his mother reports childish behaviour. Patient 5 had diffuse swelling and small haemorrhages. By 3 months he was fully recovered and has resumed his college course.

It should be noted that these patients were distinguished from our main series only by their youth and good recoveries. We intend to assess neuropsychological deficits at 1 year post-injury. Our and Davies and colleagues' patients are not the first cases of good recovery after sustained intracranial hypertension associated with compromised cerebral perfusion.² Cruz³ has documented a case of satisfactory recovery from severe traumatic brain injury with reduction in cerebral blood flow to a level indicative of impending brain death. We agree with Davies' opinions and are anxious that such patients should not be denied intensive care facilities, on the basis of measurement of intracranial or cerebral pressures alone. We endorse the recommendation that multimodality monitoring should be used to draw safer conclusions about outlook.⁴

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