Thirdly, as stated in our paper, it is possible that some of the first dose remains in the upper airway before resuscitation. We think this unlikely as the babies had not breathed and were 'sucked out' during the resuscitation period. The differences in the initial compliances were more likely due to the fact that the control group had more very immature babies.

Fourthly, the babies were randomised as stated in the paper, certainly to a greater extent than in Dr Morley's previous publication.[3]

Finally, we consider that the studies were not anecdotal and that investigating the physiological response to forms of treatment are valid, even if they do not measure whether a particular form of treatment alters the long term outcome.

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Diet and behaviour

Sir,

The recent annotation 'Diet and behaviour' by Eric Taylor[1] is timely in view of the current public interest in this country in hyperactivity. But I would take issue with him on several points. 'There is no smoke without fire' is an old saying and the very consistent reports from many sources, here, in Australia, and the United States on the stimulant effect of some common foods and food colours cannot be so lightly dismissed. Although open trials 'no longer contribute to the debate', few have been carried out in this country; but having been involved in one myself I believe they point the way. Hyperactivity is probably multifactorial but, although so difficult to define in precise terms, is readily recognised by the usual criteria of inappropriate attention span, impulse control, restlessness, and rule governed behaviour developing in late infancy and not associated with gross neurological, sensory, or motor impairment or severe emotional disturbance. It is prevalent in two to three per cent of the child population, predominantly in boys.[2 3] Our open trial showed initial improvement in 30 of 35 (86%) children after exclusion diet and challenge testing. As most of this was due to the removal of food colouring, the eventual diet was neither harmful not irksome, but a good dietician is needed to work out the details.

I fully accept the placebo effect but do not think this explains all the benefits of the diet. Some children continued to react to the additives over an 18 month period while many others did not (the 'diet' responders), but the mechanism is not clear. Psychometric testing, using elements of the Stanford Binet test under 4 years of age and the Wechsler intelligence scale for school aged children, serially at six month intervals over a year (three assessments) showed significant improvement in two of four preschool and six of eight school aged children on diet alone—two of the latter increased their overall IQ by 15 and 20 points respectively over a six month period and this was maintained. A comparison may be made with two recent papers in *Clinical Allergy* on the effect of house dust mite hyposensitisation, where the beneficial effect was clearly seen clinically but no in vitro immunological change could be found to support the effect scientifically.[4–5]

Again I take issue on the subject of allergy. Several writers agree (as I do myself) that hyperactive children and their families show more signs of allergy to a wide range of foods than normal children; but that does not make hyperactivity an allergic condition.[6] Even the late Ben Feingold denied that this was an allergic response,[7] and Dr Collins-Williams in Toronto was unable to find a significant number of hyperactive children with positive skin prick tests to foods (Collins-Williams C. Fourth Charles Blackley Symposium, Nottingham 1981). In our trial none of the 35 children had a positive radio-allergosorbent test for dairy foods, wheat, or nuts but five were positive for grass pollen and three for domestic animals and house dust mite.

Thirdly, I think Dr Taylor falls into the trap that often leads psychiatrists to disappoint paediatricians, in that he looks at the problem situationally and not aetiologically. If, as has been claimed, 10% of the population will react atypically to almost any drug, then some hyperactive behaviour may be caused by one of these atypical reactions to food chemicals. It is worth investigating. Dietary treatment does need careful supervision but it is not too difficult and is well worthwhile for the 'responders'. Psychologically troubled children do need psychological help but the behaviour one sees in the children may be the result of misunderstanding and be induced by inappropriate adult behaviour resulting from failure to recognise the primary cause. If we accept that in many cases the condition persists into adult life (although the problem may change in form or intensity)[8 9] then the primary cause of hyperactivity is developmental (genetic?) as suggested by Barkley[2] and the American Psychiatric Association. But environmental factors may make it worse and be-

havioural therapy, special education, psychotherapy, dietary treatment, and even, on occasion, psychostimulant drugs may all be complementary rather than rival forms of treatment for this undoubtedly handicapping condition.

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A J Franklin
St John's Hospital,
Chelmsford,
Essex CM2 9BG

Dr Taylor comments:

I am grateful for Dr Franklin's interest and before replying to his arguments I should emphasise the agreements between our views. We both think that substances in the diet are sometimes capable of altering behaviour; so far as I can tell, we both think that they are not a major cause of hyperactive behaviour and that multiple treatments are needed by those with hyperkinesis. We disagree about the *frequency* of behavioural reactions to food.

Firstly, Dr Franklin appeals to the weighty authority of Professor Barkley and the American Psychiatric Association, in support of a wide—in my view, an overextended—concept of hyperactivity. This does not seriously affect the argument over the effects of diets; but authority is a dangerously two edged weapon. If one reads further in these cited texts, one will discover that neither has much time for the dietary theories. If Dr Franklin wishes us to accept their authority in the one matter, why not in the other?

Secondly, the evidence of his open trial does not rule out 'placebo' and other non-specific effects. Indeed, no uncontrolled trial in this area could plausibly do so. The psychologists who administered serial IQ tests (apparently only to 12 of the 35 children) should have warned him that practice effects, placebo effects, chance fluctuations, and regression to the mean on repeated testing should all make him very hesitant to conclude that individuals' IQ scores were significantly improved by diet. I should be more interested to know about the clinical features which predicted a good and continuing response to the diet. This might be a clue to the major current puzzle of knowing for whom to recommend a trial.

The other issues seem to be based on misunderstandings rather than substantive disagreements. I am very far from wishing to suggest that hyperactivity is an allergic condition. Dr Franklin may have interpreted my reference to an 'idiosyncratic' response to the Feingold diet as if I had meant 'allergic': I did not. Diets can contain psychotropic agents (such as caffeine and possibly erythrosine), allergens (such as tartrazine) and substances that are toxic only to the genetically predisposed (as in Feingold's theory). The annotation referred to all three. Finally, I do indeed share the wish to find the causes of hyperactive behaviour. The search will be better served by critical than by wishful thinking.

Pancuronium bromide induced joint contractures in the newborn

Sir,

We thank Drs Perlman[1] and Greenough[2] for their interest in our paper.[3] We apologise for indicating that maternal paralysis for status epilepticus was associated with joint contractures. Although Older and Harris showed the transplacental passage of maternal d-tubocurarine,[4] the infant had no joint abnormalities. This was an unfortunate oversight.

Dr Perlman should draw no more conclusions from our paper than the association between neuromuscular blockade with pancuronium and joint contractures. We accept (and state in our paper) that the one infant born with mild joint abnormalities who developed more noticeable contractures after pancuronium may have been unusually sensitive to immobilisation. In the other two cases contractures were not present at birth and developed during or shortly after paralysis. As stated in the text, we suggest that the action of pancuronium bromide may be potentiated by phenobarbitone and aminoglycosides, thus prolonging reduction of spontaneous movement or the duration of paralysis.

Dr Greenough states that no infant paralysed with pancuronium bromide in Cambridge over the past three years developed contractures but we suspect that what she meant to say was contractures were *not diagnosed* in any infants. The history of neonatal medicine is littered with iatrogenic complications, some of which are subtle and unnoticed for a considerable time until attention has been drawn to them. In our three patients the joint contractures limited full extension by 30° at the most; a small but important disability. Having recognised this condition in one infant we prospectively assessed passive joint movements in subsequent infants and detected contractures that we believe would be missed by less careful examination. It is unwise to assume contractures do not occur in Cam-