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.5

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.

References

Diet and behaviour

Sir.

The recent annotation ‘Diet and behaviour’ by Eric Taylor1 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 nor 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 Barkley8 and the American Psychiatric Association. But environmental factors may make it worse and be-
havioural therapy, special education, psychotherapy, diet-
ary 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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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 Associa-
tion, 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 uncon-
trolled 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 condi-
tion. 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 Perlman1 and Greenough2 for their interest
in our paper.3 We apologise for indicating that maternal
paralysis for status epilepticus was associated with joint
contractures. Although Olden and Harris showed the
transplacental passage of maternal d-tubocurarine,4 the
infant had no joint abnormalities. This was an unfortu-
nate oversight.

Dr Perlman should draw no more conclusions from our
paper than the association between neuromuscular block-
ade 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 con-
tractures after pancuronium may have been unusually
sensitive to immobilisation. In the other two cases contract-
ures 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 poten-
tiated 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 move-
ments 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-

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