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

References


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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 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 'idiopathic' 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 and Greenough for their interest in our paper. 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, 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 he 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-
bridge if a prospective assessment of joint movement has not been performed in paralysed infants.

We agree with the Cambridge experience that ‘fighting the ventilator’ is likely to predispose towards pneumothorax.\(^5\) We do not recommend withholding pancuronium for fear of joint contractures but merely wish to report that joint contractures may occur in association with its use, and attention to regular limb physiotherapy may prevent this occurrence. In addition, we would support the suggestion that other methods of inhibiting unwanted respiratory activity in ventilated infants should be investigated further before assuming that pancuronium is the drug of choice in this context.\(^6\)

References


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Sir,

Drs Sinha and Levene in their report\(^1\) have concluded that the use of pancuronium bromide was responsible for joint contractures seen in three neonates in their series. It is quite conceivable that long term muscle paralysis may lead to contracture formation if passive movements are not carried out. It is difficult, however, to imagine how one dose of pancuronium bromide in their second patient and 24 hours’ treatment in their first could have caused contractures which did not improve even after two to four months of regular physiotherapy. These cases would suggest that pancuronium bromide somehow accelerates the ‘normal’ process of contracture formation.

All three affected infants had limitation of extension of joints. Paralysed babies lose the normal attitude of flexion and generally lie flat with their limbs extended at elbow and knee joints. This should freeze the knee joint in extension and lead to difficulty in flexing rather than extending the joints. The problem was observed in three of 13 babies given pancuronium bromide over an 8 month period. In spite of early detection and regular physiotherapy all of them had appreciable residual contractures at 2 to 4 months of age.

Pancuronium bromide is being used in my neonatal unit as an adjunct to mechanical ventilation. If we had missed this diagnosis and the babies had not been given physio-

therapy, I would have expected more babies to have been seen in the follow up clinics with joint contractures.

For these reasons it seems unlikely that temporary paralysis induced by pancuronium bromide was responsible for joint contractures in the first two cases.

I would, however, agree with the authors in recommending passive exercises for babies who are paralysed long term.

Reference


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Drs Sinha and Levene comment:

We thank Dr Singh for his letter. Unfortunately he is mistaken in his belief that paralysed infants lie in an extended posture. They adopt the classical ‘frog position’ seen in severely hypotonic infants. Their arms are semiflexed, wrists pronated, hips abducted, and knees partially flexed—certainly not fully extended. The knee contractures we noted fitted closely the position of partial knee flexion seen in paralysed infants. We feel that Dr Singh’s other points have already been discussed by us.

Monitoring of intracranial pressure

Sir,

In their short report, Levene and Evans\(^1\) suggest a ‘new’ method for continuous measurement of intracranial pressure. We would question, unless a theoretical or clinical reason is given, their statement that ‘subarachnoid catheters may be better suited to long term monitoring’, especially since these catheters are usually kept in place for short periods (76 hours was the longest duration in their study). Furthermore, insertion of an extremely large needle (16 G) into the fontanelle of a neonate seems unnecessarily invasive and might greatly increase the risks of trauma to brain tissue and infection. In 1982 we described a method of intracranial pressure monitoring using a much smaller (22 G) catheter.\(^2\) This method has been proved to be highly reliable and safe.

Intracranial hypertension, per se is of little clinical importance. Maintenance of cerebral perfusion pressure, adequate to ensure a cerebral blood flow and therefore sufficient substrate supply for cerebral metabolism, may be an important factor in the mortality and morbidity of childhood central nervous system diseases.\(^3\) We have previously shown\(^4\) that in cerebral ischaemia, the late development of increased intracranial pressure and its treatment does not significantly affect outcome in these patients. It is the severity of the ischaemic insult that