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
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

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 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 block-
ade with pancuronium and joint contractures. We accept
(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 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 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-
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. 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."

References

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Sir,
Drs Sinha and Levene in their report 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 physiotherapy, 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 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. 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. We have previously shown 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
Pancuronium bromide induced joint contractures in the newborn

S K Sinha and M I Levene

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