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 report¹ 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 semi-flexed, 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 Evans¹ 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 shown⁴ 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