understand why the paediatricians and therapists who work in this field have put up with this for so long.

What then of conductive education? As a physician I suppose I should await the results of controlled clinical trials comparing this form of treatment with conventional treatment. As a parent, however, I cannot afford to wait, and I have therefore asked for my child to be assessed at the Petó Institute. If conductive education turns out to be of no benefit I will have lost comparatively little by taking him there. On the other hand, if conductive education proves to be far superior to our conventional approach to treatment, I feel I will have enhanced my son's quality of life by taking him to Hungary. The risk of not opting for conductive education at this time is, for me, too great to take.

I commend Mr Sutton and his colleagues at the Foundation for Conductive Education in their attempts to assess this form of treatment for motor disorders in a scientific way, and feel that this work is so important that it should be funded by central government and not left to the efforts of exasperated parents' groups and to charities. While these studies go on it is also important that funds be made available to improve existing services for children with cerebral palsy; they deserve more than we currently provide for them.

References

- ¹ Beach RC. Conductive education for motor disorders: new hope or false hope? *Arch Dis Child* 1988;**63**:211-3.
- ² Sutton A. Conductive education. *Arch Dis Child* 1988;**63**: 214-7.

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Conductive education

Sir,

I have read with great interest the articles on conductive education.^{1 2} It is widely accepted that there are variations in the availability of paediatric physiotherapy in this country and the methods of treatment. No far sighted physiotherapist would be dismissive of progress in any child nor would wish to diminish parents' hopes without being unrealistic. It remains to be seen whether it is possible to transpose totally a philosophy from one culture to another.

Mr Sutton ends his article with a quote from *Measure for Measure*: Claudio states 'The miserable hath no other medicine . . . ' I add the Duke's reply,³

Happy thou art not;

For what thou hast, forget'st. Thou art not certain.

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- ¹ Beach RC. Conductive education for motor disorders: new hope or false hope? *Arch Dis Child* 1988;**63**:211-3.

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- ³ Shakespeare W. *Measure for Measure*. Act III, scene 1.

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Hyperkalaemia, cardiac arrhythmias, and cerebral lesions in high risk neonates

Sir,

The recent report by Shortland *et al* on the association between hyperkalaemia and cardiac arrhythmias in premature neonates is interesting but the proposed observations it reports are not substantiated.¹ Bold statements about 'supraventricular tachycardia' and 'bradycardia' are unsupported and are contrary to experimental and previous clinical experience.

The authors have provided no evidence to support their diagnosis of 'supraventricular tachycardia.' They need to disprove an alternative interpretation that they were observing sinus tachycardia secondary to systemic hypotension and that intravenous calcium infusion raised the blood pressure, and only indirectly reduced the heart rate.

All cases of supraventricular tachycardia have an identifiable underlying mechanism. Although this can be detailed precisely by invasive electrophysiological study, it may also be predicted fairly accurately from a careful analysis of the electrocardiograms. Particular attention is paid to the atrial rate, the P wave morphology, the QRS morphology and the relationship between P waves and QRS complexes and valuable additional information may be obtained if the onset and termination of tachycardia are recorded. The response to therapeutic manoeuvres may also provide an insight to the type of tachycardia. All episodes of bradycardia also have an identifiable mechanism. This may be slowing of sinus rhythm, or failure of sinus rhythm with a nodal or ventricular escape rhythm, or atrioventricular block. All of these have been reported in association with hyperkalaemia but bradycardias are also very common in sick premature neonates and are then of great independent significance.

The experimental and clinical effects of hyperkalaemia on cardiac rhythm are well described. The raised serum potassium concentration reduces the resting transmembrane potential and thereby slows conduction. Although 'excitability' may theoretically be increased, in practice this is not observed but, rather, automaticity is decreased as a result of depression of spontaneous diastolic depolarisation. Hyperkalaemia may produce a variety of arrhythmias and conduction disturbances. Those which have been described are varying degrees of atrioventricular block and interventricular conduction delay producing a type of bundle branch block. Supraventricular tachycardia has not been reported and would not be expected. The neonate with tachycardia reported by Cohen *et al* had an atrial rate