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 Peto 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

A A Dunk
Gastrointestinal Research Unit,
Royal Infirmary,
Forsterhill,
Aberdeen AB9 2ZB

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 are not certain.

References

Correspondence 681


1 Shakespeare W. Measure for Measure. Act III, scene 1.

C A Sobkowiak
Darlington Memorial Hospital,
Darlington,
Co Durham DL3 6HX

Hyperc当地laemia, 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
of only 125/minute which was interpreted incorrectly as a tachycardia. In fact their patient showed sinus rhythm throughout with an interventricular conduction delay on one occasion and 2:1 atrioventricular block on another.

If we are to accept this report of supraventricular tachycardia caused by hyperkalaemia, the authors should, therefore:

1. Define ‘supraventricular tachycardia’ and ‘bradytachycardia.’
2. Detail the rate and the duration of tachycardia, and their relationship to plasma potassium concentration.
3. Describe the electrocardiographic characteristics of the tachycardias and the bradycardias. These may give a valuable insight into the mechanisms of the arrhythmias.
4. Provide information about any observed electrocardiographic abnormalities which are known to occur in association with hyperkalaemia (such as T wave changes, QRS widening, prolongation and flattening of the P waves, etc).

It is important that the findings reported in this paper should be well substantiated because tachycardia secondary to hyperkalaemia in the neonate has not been reported previously. Just as one would not consider a report on hyperkalaemia which did not define hyperkalaemia and give details of potassium concentrations recorded, so it is difficult to take seriously a report of ‘supraventricular tachycardia’ and ‘bradytachycardia’ which fails to define either arrhythmia and gives no details or documentary evidence.

References

C WREN
Freeman Hospital,
Freeman Road, High Heaton,
Newcastle upon Tyne NE7 7DN

Drs Shortland and Levene comment:
We read with interest the comments of Dr Wren. We define supraventricular tachycardia as a heart rate of greater than 200 bpm with completely regular P waves on the cardiac monitor. It is extremely difficult to perform full electrocardiography in a sick preterm infant and we do not have facilities for permanent paper recording. In the infants that we have reported, the cardiac rhythm converted over a matter of seconds from sinus rhythm to a supraventricular tachycardia and remained in that rhythm for some time before suddenly reverting to a slower rate. This does not occur with sinus tachycardia due to systemic hypotension and we think that this is an unlikely cause for our findings. We have defined a bradycardia induced by hyperkalaemia as a heart below 60 bpm lasting for more than 60 seconds in the absence of other clinically apparent causes (for example, hypoxia or endotracheal suction.) The arrhythmias we have described all occurred when the serum potassium concentrations were raised, although in four infants the arrhythmia preceded the diagnosis of hyperkalaemia and in fact had led the medical staff to measure the serum electrolytes. We are surprised that Dr Wren states that supraventricular tachycardia cannot occur during hyperkalaemia, although we agree that bradyarrhythmias are most commonly described. We encounter supraventricular tachycardia only rarely within the first 48 hours of life in the preterm infant but we have found that most occur at a time when the serum potassium concentrations are raised. Although the relationship between hyperkalaemia and supraventricular tachycardia is poorly described, we suggest that they are causally linked.

Selective medical examinations on starting school
Sir,
We read with interest the article by O’Callaghan and Colver. The authors describe a class review after the first term, but it is apparent that the children have already undergone a four to four and a half year check. From the review, 20% of children are selected to be seen, which may be a repetition of earlier work.

It is not clear whether the four to four and a half year check is a population screen and whether the same doctor is responsible for the school review. No data on the numbers of problems identified at this check are given, or whether any educational liaison is undertaken.

In the Southampton area with a school age population of approximately 63 000 a selective system for school medicals has been in operation since 1970. All children are seen at six weeks and four and a half years, the latter representing a preschool medical examination usually performed by a clinical medical officer who is responsible for school follow up. Problems from health visitor assessments at seven to nine months and two and a half years are selected for clinical medical officer attention. Most defects are, therefore, identified before a child enters school.

The preschool examination permits an appraisal of the ‘whole child’ and all children seen are discussed with the headteacher and nurse. The doctor will select out those few children requiring school follow up. Selection visits each term with the head/class teachers and school nurse follow, allowing continuity.

The approach to selective screening adopted by the authors appeared to be rather disjointed involving a wide range of professionals. We should like to be reassured that the doctor providing the service looks at the whole child and having done so, provides continuity from preschool to school years.

In conclusion, the article gives the impression that selectivity is a new concept and suggests that blanket examinations are not necessary. Two important factors...
Hyperkalaemia, cardiac arrhythmias, and cerebral lesions in high risk neonates.

C Wren

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