Short reports

Atropine treatment of reflex anoxic seizures

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SUMMARY In 7 children with unusually severe or frequent reflex anoxic seizures atropine treatment, which was well tolerated, reduced seizure frequency by a mean value of 98%. Treatment withdrawal (five patients) was followed by an increase in seizure frequency and reintroduction (three patients) by restoration of control.

A reflex anoxic seizure is the outward manifestation of the cerebral ischaemia resulting from vagally mediated cardiac arrest provoked by a minor noxious stimulus (not always witnessed or recognised) and lasting for up to 58 seconds. These non-epileptic seizures are common in childhood, and frequently acquire the pejorative label 'epileptic'. Typically they are tonic, jerking is not prolonged, and recovery of awareness is prompt. The diagnosis can usually be made from the history and careful explanation and reassurance will allay parental anxieties. Where doubt persists or anxieties remain the ocular compression test will often clarify and, if positive, consolidate verbal reassurance. Treatment is seldom required but when necessary atropine has been recommended, although a clinical study of its effectiveness has not been reported. We investigated the use of atropine in preventing reflex anoxic seizures.

Patients

Seven patients were selected because prophylactic treatment had been requested by their parents. All were having severe or frequent attacks and three had received anti-epileptic medication—up to three different drugs—without effect. The clinical diagnosis was confirmed with the ocular compression test as previously described except that for this study the test was considered positive only if an asystole mediated anoxic seizure was induced and confirmed by the parent witness as identical to the child's natural attacks.

Method

Seizure frequency was recorded for a minimum period of one month before atropine treatment, for the duration of treatment and, where applicable, after treatment withdrawal and reintroduction. Initially a limited treatment period had been intended but in practice the parents determined the duration of treatment and in two cases insisted on its continuation. The remainder agreed to treatment withdrawal but in three cases requested reintroduction when seizures recurred.

Two preparations were used: atropine sulphate and methonitrate. Atropine methonitrate is supplied as a 0.6% alcoholic solution (Eumydrin) in a dropper bottle, one drop containing 200 μg. Early in the study we used atropine sulphate but latterly preferred Eumydrin because its quaternary ammonium structure is alleged to keep its distribution extracerebral, eliminating potential adverse effects on cognitive function from central antimuscarinic activity. Initially a low dose was given twice daily and increased until satisfactory seizure control was achieved, the frequency being increased if breakthrough occurred towards the end of a dose interval.

Results

After the introduction of atropine, seizure frequency fell by between 93% and 100% of the level before treatment (Table). In the two patients on continuous treatment, seizure control was maintained throughout the period of study whereas in the five remaining patients seizures became more frequent when treatment was withdrawn. Three regained control on atropine and two have remained off treatment with continuing frequent attacks (Figure). Two patients experienced anticholinergic symptoms, drying of nasal and buccal secretions in one and blurred vision in the other. Both problems responded to a reduced dose given at more frequent intervals.
Table: Age, atropine treatment, and frequency of reflex anoxic seizures in 7 patients

<table>
<thead>
<tr>
<th>Case No</th>
<th>Sex</th>
<th>Age (years)</th>
<th>At onset of seizures</th>
<th>Atropine treatment</th>
<th>Seizure frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Preparation</td>
<td>Dosage (ug/kg/day)</td>
</tr>
<tr>
<td>1</td>
<td>F</td>
<td>0-7</td>
<td>2-4</td>
<td>s</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>0-3</td>
<td>3-3</td>
<td>m</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>0-1</td>
<td>1-1</td>
<td>s</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
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<td>2-3</td>
<td>m</td>
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<td>1-1</td>
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<td>M</td>
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<tr>
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<td>F</td>
<td>0-3</td>
<td>0-4</td>
<td>m</td>
<td>80</td>
</tr>
</tbody>
</table>

s = atropine sulphate; m = atropine methonitrate; *Refers to the initial treatment period only.

Discussion

It may be argued that this study was unnecessary, that the pharmacology of the cardiac vagus is so well understood that the result was entirely predictable. Admittedly full atropinisation must abolish seizures if the physiological basis is as stated above, but we have shown that seizures may be controlled without the penalty of other atropine effects. That this is unlikely to be a placebo effect is seen from the ineffectiveness of prior anti-epileptic treatment in three patients.

Parents witnessing their child’s first febrile seizure believe the child to be dead or dying and it would be surprising if parents of children with reflex anoxic seizures reacted differently. Parental anxiety is understandable as is the high frequency with which resuscitative measures are applied, occasionally with harmful or even fatal results. One patient (case 5) was treated after a potentially harmful seizure during which her mother, thinking her daughter had died, gave mouth to mouth respiration. The child probably aspirated and did not regain consciousness for one hour. This reaction is not confined to parents. We have attended a two year old boy whose family doctor witnessed one of his seizures and, believing him to be dead, applied mouth to mouth respiration. The child vomited and aspirated, requiring intensive care. Atropine was offered but his parents declined. It is not clear in these situations which is more harmful, the seizure or its management, but in either case treatment should be offered.

Most children will be offered treatment because of their parents’ anxiety in which case treatment should be seen as supplementary to the explanation and reassurance already given and should be of limited duration. A third indication arises if seizures persist beyond the age of school entry. Treatment
was continued in two patients (cases 1 and 2) for this reason.

Poisoning may result if the method of administering Eumydrin (as a specific number of drops) is not understood and this should be stressed whenever it is prescribed.

References

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Fat loss during feeding of human milk

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SUMMARY A rise in the fat concentration of human milk within the syringe was noted towards the end of continuous infusion but not with intermittent bolus gastric feeding. The rise in the former was reduced most simply and effectively by using an eccentric nozzle syringe and tilting the pump up at an angle of between 25° and 40°.

Certain methods of feeding of human milk such as continuous infusion with a syringe are associated with a rise in fat concentration towards the end of the feed. We have also observed that some pumps retain this energy rich terminal milk in the syringe. We compared the fat concentration at the end of two types of enteral feeding and evaluated some methods of avoiding an excessive rise in this during infusion of human milk.

Material and methods

The fat in milk samples was estimated by the creamatocrit method at the beginning and at the end of two types of feeding—continuous infusion using a syringe pump and intermittent bolus gastric feeding. It was observed that while some pumps emptied completely at the end of a feed there was retention of about 2 ml milk in the barrel of others. To estimate the fat concentration towards the end of the feed the pumps were stopped when 2 ml milk remained in the syringe. Infusion feeding was studied using central and eccentric nozzle syringes and by alternative techniques detailed in the first column in the Table. Preliminary investigations showed that with the eccentric nozzle syringe fat retention was greater with the pump sloped up at angles less than 17° and more than 45°. Further investigations were therefore limited to positions within that range. In intermittent bolus gastric feeding the syringe was detached from the feeding tube when 2 ml milk remained in the barrel. All continuous infusions lasted between 3 and 4 hours and intermittent bolus feeding between 5 and 15 minutes. The results were subjected to standard statistical tests.

Results

The fat concentrations at the beginning and end of the feed and the difference between these values (mean (SD)) for the various methods are shown in the Table. There was no rise with intermittent bolus feeding (method (a)) and even when a sample of milk was aspirated from the feeding tube at the end of a feed fat concentration was not increased.

With the central nozzle syringe the rise in fat concentration was highest in the conventional method with the pump kept horizontal (method (b)), increasing to a maximum of 40%. When some milk was aspirated from the tube the concentration...
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