LETTERS TO THE EDITOR

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The editors will decide, as before, whether to also publish it in a future paper issue.

Protective role of cerebrospinal fluid in brain injuries

EDITOR,—We would like to offer a simple model of brain injury which explains many features of the so-called “skull injuries”—that is, those where damage results from the action of inertial forces only.

The model is easily constructed as follows. Fill a jam jar to the brim with water. Glue two threads to an egg, suspend the egg in the water, and screw the lid on tightly. If the jar is shaken horizontally as vigorously as possible, the egg will not usually touch the sides of the jar, let alone break. If, however, the jar is suddenly and impulsively rotated, one of the threads will normally break or pull away a small portion of the shell at the point of attachment.

Standard fluid mechanics explains why the egg is not damaged by linear motion. The acceleration of the jar gives rise to three fluid forces opposing the motion of the egg: a force due to the horizontal pressure gradient, the “acceleration reaction”, and the viscous drag. Together, these three forces can be shown to reduce the acceleration of the egg relative to the jar by a factor of 40–50, compared with what it would have been in the absence of the water. When the jar is rotated, inertia tends to keep the egg fixed in space and, as the water is incapable of exerting any significant moment on the egg, the thread breaks.

If we identify the egg with the brain, the water with the cerebrospinal fluid, the jar with the skull, and a broken thread with a bleeding bridging vein, we have a ready explanation for the generally accepted fact that brain injury is more easily caused by rotational than by linear acceleration.

As the argument is based on known fluid mechanical principles, the important question is the extent to which the model represents a real head. The model ignores the presence of brain movements, because the neurovascular structures in the brain stem permit small linear and rotational movements of the brain before any significant forces come into play. Apart from the published animal studies, verification of this model would require experiments with an instrumented cadaver head or an advance in imaging technology to permit real time tracking of the brain’s movement.

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Oral steroids and inflammatory markers in asthma

EDITOR,—Although the recent paper by El-Radhi and colleagues presents interesting data about decreases in inflammatory markers during the resolution of acute asthma, some of their conclusions are not valid. Firstly, acute asthma has a tendency to resolve without corticosteroid treatment. As all of the children with acute asthma (quite rightly) received steroids, the observed effect may equally reflect processes associated with spontaneous resolution. Indeed, corticosteroids do not inhibit the release of eosinophil cationic protein (ECP) from eosinophils. Secondly, the normal controls are inadequate. Atopy per se is associated with increased serum levels of ECP, and it is therefore to be expected that the asymptomatic atopic asthmatics will have higher ECP levels than the mostly non-atopic controls.

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CORRECTION

An error occurred in table 2 of Wisborg and colleagues’ recent paper (Arch Dis Child 2000;83:203–6). The correct figures are given in the table printed below:

Table 2 Crude and adjusted OR of SIDS according to different categories of smoking habits during pregnancy

<table>
<thead>
<tr>
<th>Smoking Status</th>
<th>Total no.</th>
<th>Total no. with SIDS</th>
<th>% SIDS (95% CI)</th>
<th>Adjusted* OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers from 16 weeks’ gestation</td>
<td>17536</td>
<td>8</td>
<td>0.5</td>
<td>Reference</td>
</tr>
<tr>
<td>Smokers</td>
<td>7450</td>
<td>12</td>
<td>1.6</td>
<td>3.5 (1.4–8.7)</td>
</tr>
<tr>
<td>1–9 cigarettes/day</td>
<td>3249</td>
<td>5</td>
<td>1.2</td>
<td>3.4 (1.1–10.3)</td>
</tr>
<tr>
<td>10+ cigarettes/day</td>
<td>4201</td>
<td>7</td>
<td>1.7</td>
<td>3.7 (1.3–10.1)</td>
</tr>
</tbody>
</table>

*Adjusted for maternal age

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