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 "closed 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.[1] 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.[2]

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 the brain stem. However, this approximation is justified, at least for small 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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1 Batchelor GK. *Fluid dynamics*. Cambridge: Cambridge University Press, 1967.
2 Gennarelli TA, Thibault LE. Biomechanics of acute subdural haematoma. *J Trauma* 1982;**22**: 680–6.

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,[1] some of their conclusions are not valid. Firstly, acute asthma has a tendency to resolve without corticosteroid treatment.[2] 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.[3] Secondly, the normal controls are inadequate. Atopy per se is associated with increased serum levels of ECP,[4] 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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1 El-Radhi AS, Hogg CL, Bungre JK, *et al*. Effect of oral glucocorticoid treatment on serum inflammatory markers in acute asthma. *Arch Dis Child* 2000;**83**:158–62.
2 Gleeson JG, Loftus BG, Price JF. Placebo controlled trial of systemic corticosteroids in acute childhood asthma. *Acta Paediatr Scand* 1990;**79**:1052–8.
3 Venge P, Bystrom J, Carlson M, *et al*. Eosinophil cationic protein (ECP): molecular and biological properties and the use of ECP as a marker of eosinophil activation in disease. *Clin Exp Allergy* 1999;**29**:1172–86.
4 Remes S, Korppi M, Remes K, *et al*. Serum eosinophil cationic protein (ECP) and eosinophil protein X (EPX) in childhood asthma: the influence at atopy. *Pediatr Pulmonol* 1998;**26**: 167–74.

Reliability of percentage ideal weight for height

EDITOR,—I write to point out an error in a recent paper by Poustie and colleagues.[1] The authors state that there is no computer package available in the United Kingdom for calculating percentage weight for height (%WFH). This is incorrect, and for many years there has been available just such a package entitled W4H, under the copyright of Great Ormond Street Hospital for Children NHS Trust. The programme can be used with any version of Windows from 3.1 upwards, Excel, and on Psion hand held computers. The programme was produced by the Eating Disorders Research Team at Great Ormond Street and can be purchased from me at the address given below.

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1 Poustie VJ, Watling RM, Ashby D, *et al*. Reliability of percentage ideal weight for height. *Arch Dis Child* 2000;**83**:183–4.

Answers to quiz on page 164.

1. Adult respiratory distress syndrome and sand aspiration. The spirometry findings suggest air trapping by grains of sand, causing blockage of inspiration and expiration via a ball valve mechanism.
2. A CT scan of the lungs and a bronchoscopy, with diagnostic and therapeutic lavage.
3. Drowning and near drowning account for a significant morbidity and mortality in children, especially in seawater areas. The incidence of aspiration of mud, sand, and aquatic vegetation is less well known. A high index of suspicion is required as management may include diagnostic and therapeutic endobronchial/alveolar lavage. Initial clues to significant aspiration include increased peak airway pressures during mechanical ventilation and the appearance of a sand bronchogram on the *x* ray.

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

	Total no.	*Total no. with SIDS*	*‰*	*OR (95% CI)*	*Adjusted* OR (95% CI)*
Non-smokers from 16 weeks' gestation	17536	8	0.5	Reference	Reference
Smokers	7450	12	1.6	3.5 (1.4–8.7)	3.0 (1.2–7.3)
1–9 cigarettes/day	3249	5	1.5	3.4 (1.1–10.3)	2.9 (0.9–8.9)
10+ cigarettes/day	4201	7	1.7	3.7 (1.3–10.1)	3.0 (1.1–8.5)

*Adjusted for maternal age