LETTERS TO THE EDITOR

Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Obesity in Chinese children

I do not wish to add fuel to the fire by entering into a discussion on food advertising versus physical inactivity as the culprit for childhood obesity that was prompted by Dr Ashton's editorial (February 2004 *JRSM*[1]). I always tell my overweight or obese patients that body fat does not grow on the tree; it has to come from an external source, either overeating or underexertion. The best example comes from China.

China used to be known for her slender people; I never saw a fat person in China until very recently.[2] Now China is fighting obesity, especially childhood obesity, just like the rest of the world. According to the latest statistics from China, the proportion of obesity among children under the age of 15 increased from 15% in 1982 to 27% today.[3] Among the predisposing factors for increasing prevalence of childhood obesity in China, fast food and physical inactivity are the two most important. Because of the efficient advertisements of such fast food giants in the United States as McDonald's and Kentucky Fried Chicken, Chinese children are devouring the American fast food faster than ever.[2,4] All of the children in China recognize the image of Ronald McDonald, even though they might not be able to read English.[4]

That physical inactivity is the other important risk factor for obesity in Chinese children is illustrated by three observations. First, the effect of television on childhood obesity in China has been documented: each hourly increment of television viewing is associated with a 1–2% increase in the prevalence of obesity in urban China.[5] Second, childhood obesity is more prevalent in urban areas than in rural areas, not only because children in rural China are physically more active[6] but also because urban boys consume more fat than rural boys—23–30% versus 16–20%.[7] Third, urban children in China are engaged in more homework because they are under pressure to achieve scholastically, whereas rural Chinese children are engaged more in field work because of economic necessity.[8]

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Swallowed partial dentures

Mr Hashmi and his colleagues present an excellent discussion of the problems of diagnosing and managing swallowed partial dentures (February 2004 *JRSM*[1]). Management can be challenging even when the diagnosis is straightforward. We recently saw a man of 34 who had swallowed a set of partial upper dentures which contained a wire to fix the plate to the teeth. He had swallowed them accidentally while eating a meal. The dentures lodged in the upper oesophagus, at the level of the sternal notch, and the wire was easily visible on X-ray. Oesophagoscopy revealed the dental plate just below the level of the cricopharyngeus but multiple attempts at removal, with various instruments, were unsuccessful. Sometimes the answer is to split the plate and remove it in pieces, but even with heavy denture shears the material proved too hard to cut. Therefore an oesophagotomy was performed through a lateral pharyngeal approach. The wire was seen to penetrate the lateral oesophageal wall. The patient recovered without incident and was planning to discuss more suitable dentures with his dentist.

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Mr Hashmi and his colleagues (February 2004 *JRSM*[1]) do not cite a *BMJ* letter of mine in 1972 recording the retrieval, with difficulty, of a partial denture from the right main bronchus.[2] The prosthesis had been inhaled on diving into a swimming pool. My letter drew attention to a paper by Coman[3] reporting three cases, of which the most alarming concerned a man who had inhaled his partial denture when parachuting. Another illustrated the hazard of

metal clips to attach the prosthesis to neighbouring teeth: a hook caused a prosthesis to be stuck in the oesophagus for some 3 weeks, causing a para-oesophageal abscess. I suggested that it would be a wise precaution to remove these potentially lethal pieces of apparatus before engaging in violent exercise and probably also at night. I was, of course, thinking of lying on one's back mouth-breathing. However, a journalist clever in lateral thinking picked this up and, to my astonishment, I was telephoned by the *Today* programme to make a live comment about my recommendation of removing dentures before intercourse. Later I found myself surrounded by newspapers with headlines such as 'Love peril of your false teeth', 'The safe way of making love . . . by gum'.

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Swallowed toothbrush

A woman of 35 came to the accident and emergency department saying that she had accidentally swallowed an entire toothbrush 'whilst brushing the back of her throat'. She had no other symptoms other than slight odynophagia. A radiograph (Figure 1) shows the bristles in the fundus of the stomach. She was referred to the surgeons who removed the toothbrush endoscopically the next day.

Two points struck me. First, how could she have swallowed a whole toothbrush accidentally? Second, if it was swallowed bristles first, one might expect the bristles to be at the pylorus rather than the fundus. The patient later admitted that she had been using the toothbrush to induce emesis.

Figure 1 **Foreign-body radiograph showing bristles in fundus**

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Shell shock

Dr Macleod (February 2004 *JRSM*[1]) whilst outlining the psychiatric effects of 'shell shock', overlooks some important clinical and experimental evidence of direct and indirect central nervous system (CNS) injury following exposure to explosive blast. The contribution of such injury to the psychiatric manifestations is unknown but worthy of consideration.

In his three Lettsomian lectures Mott referred to the concussion or *commotio cerebri* produced by 'aerial compression',[2] but probably wrongly attributed post-mortem findings of punctate haemorrhages and chromatolysis in soldiers to carbon monoxide poisoning. Further evidence of a primary blast effect on the brain was reported by Cohen and Biskind who showed scattered intracerebral and extracerebral haemorrhages in nine soldiers killed by air blast whose symptoms had included extreme restlessness, excitability and irrationality.[3]

Animal experimentation shows further evidence of cerebral injury following blast in air, Hooker having been the first to demonstrate focal damage to the CNS involving lethargy, paralysis and convulsions in frogs, rabbits and dogs.[4] More recently, an investigation of the causes of post-traumatic stress disorder showed evidence of neuronal atrophy in the cerebral and cerebellar cortices of rats surviving exposure to the detonation of a single TNT charge in an underground bunker, these changes being maximal in animals killed on day 14.[5] Axonal degeneration with chromatolysis and disruption of cellular function in the brainstem have also been observed as distant effects following penetrating ballistic injury to the thigh in pigs[6] and following pulmonary blast injury in rabbits.[7]

Cerebral air emboli have been demonstrated in animals sustaining pulmonary injury induced by blast in air and underwater, air gaining access to the circulation by traumatic alveolar-venous fistulae.[8–10] Reports of cerebral emboli in human beings are rare but emboli have been observed in retinal vessels at ophthalmoscopy in survivors of pulmonary blast injury[10] and in the brains of fatalities post mortem.[11,12]

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An unusual paracetamol overdose

A patient with complications from surgery was admitted to intensive care with sepsis, adult respiratory distress syndrome, and signs of impending renal failure. After 18 days of ventilation, further surgical intervention, and every trick in the microbiologist's book, he had recovered sufficiently for his sedation to be lightened. In addition, his persistent pyrexia gradually came under the control of paracetamol 1 g four times daily. Nutrition was supplied via a jejunostomy tube, and as his condition improved the paracetamol was administered in a soluble preparation by the same route.

By day ten he was much improved, with his sepsis resolving. Yet his sodium concentration was 159 mmol/L —13 mmol/L above our laboratory's upper limit. Changing jejunostomy feed to a low-sodium preparation, and swapping normal saline infusions to dextrose saline had no effect, and we assumed that a polyuric renal crisis was evolving.

Then one of the authors (MS), a senior staff nurse, remembered that soluble paracetamol contains sodium. Further investigation revealed that the preparation we were using contained 388 mg of sodium in each 500 mg tablet. In other words, the patient was receiving 3 g sodium a day, and had been doing so for 6 days. Back-of-envelope calculations then showed:

3 g Na=0.130 mmol Na
In a patient weighing 85 kg,
this gives a body water volume of 59.5 L.
Thus each day he received 0.130/59.5=218 mmol/L

Over 6 days the excess sodium equals 13 mmol/L, which happens to be the exact amount over the upper limit of normal.

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Putrid gums and 'Dead Men's Cloaths'

I enjoyed Dr Sutton's paper (December 2003 *JRSM*[1]): it is good that Lind and *Salisbury* are not forgotten, even though (or perhaps especially because) his work was neglected in his time. It is unlikely, for instance, that James Cook had ever heard of it.

Dr Sutton is quite right in emphasizing the disparity between ships' captains' views on sickness at sea and those of (especially present-day) medical opinion. Ships' captains set their threshold for sickness at the level where a man could no longer stand his watch, not at the level where his health was first impaired.

He also speculates on the reasons for the neglect of Lind. I think it can be put down to the fact that Lind had some science but little influence. David McBride (1726–1778), on the other hand, had influence but no science: he was brother to a naval captain who was friendly with Sandwich the First Lord and with Hugh Palliser (Cook's patron). He formed the idea that the fermentation of malt released 'fixed air' (CO_2) which was essential to the absorption of nutrients from the gut. He published his views with some detail lifted from Lind and with oblique homage to Sir John Pringle, and succeeded in having quantities of malt imposed on Cook with instructions to make a trial of it.

Indeed Cook was supposed to make trials of malt, of portable soup, of 'sour krout' and of a rob of oranges and lemons, which latter had been boiled down enough to destroy all its vitamin C. The further problem was that his surgeon in *Endeavour*, William Brougham Monkhouse, though briefed at the Admiralty on the use and assessment of these materials, neglected his medical duties to enjoy the excitement of ecotourism—and after Monkhouse's death

young William Perry inherited a muddle such that his report amounted to little more than deference to Pringle, McBride and malt.

Meanwhile Cook himself had achieved enough by his own empirical measures—cleanliness such as the British seaman was quite unaccustomed to; fumigation below decks; wherever possible setting three watches, which happened to reduce stress and the additional vitamin C demands it creates; and the gathering of fresh greens at every shore call—to reduce the impact of scurvy quite dramatically. His measures achieved two things. In the first place, they prompted the Royal Society to award him its Copley medal in 1776 for his work in combating scurvy at sea; and in the second, the very success he achieved, without an effective vitamin C preparation apart from his greens, arguably served to *delay* the routine use by the Royal Navy of citrus preparations such as the East India Company had employed for over a century.

In fact it was not until (Sir) Gilbert Blane, who combined science and influence, that the Navy 'caught up' in 1795.

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Role of thyroxine in chronic urticaria and angio-oedema

Dr Dunkley and Dr Jawad[1] propose that a patient's flare of chronic urticaria and angio-oedema (CUA) was related to a reduction in thyroxine dosage. We have seen a patient in whom CUA was apparently exacerbated by treatment of hyperthyroidism with carbimazole. The patient, a woman of 50, first consulted us after three weeks of generalized urticaria. She had no obvious precipitating factors and was taking no medications. Thirteen months previously an episode of acute urticaria had been precipitated by a viral illness. With a short course of prednisolone this had resolved completely within three weeks. Autoimmune serology and thyroid function tests had been normal at that time. The present episode of urticaria was controlled with cyproheptadine 4 mg four times a day and cimetidine 400 mg four times a day. A routine blood test revealed FT_4 27.1 pmol/L (normal range 9.0–26.0), TSH $<$ 0.02 m IU/L (0.3–5.5), T_3 28.8 nmol/L (0.8–2.5); thyroid autoantibodies were within normal range. For this biochemical hyperthyroidism she was started on carbimazole 20 mg twice daily. After three weeks she returned with generalized urticaria and angio-oedema. Sulfasalazine 1 g twice daily was added to the treatment and her rash resolved completely within days; four months later she is still in remission, taking low doses of cyproheptadine and cimetidine daily. She has stopped taking sulfasalazine. With thyroid function approaching normal, carbimazole has been reduced to 15 mg daily. We postulate that the flare of CUA was due to reduction of endogenous thyroxine by carbimazole. The possible role of endogenous thyroxine in suppressing urticaria deserves further investigation.

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Laennec's legacy

Where Laennec was a true pioneer was in correlating clinical signs with pathology. In the first edition of his treatise on mediate auscultation (1819) he analysed the physical signs of percussion and auscultation and substantiated them with their pathologo-anatomical relevance; and, in the second (1823) he provided a complete description of pathology, diagnosis and treatment. Writing about three early Welsh followers of Laennec (March 2004 *JRSM*[1]), Dr Morris acknowledges that Williams, Davies and Lucas were among many foreigners who came under the French doctor's influence. In England these included Heberden, Fothergill and the 'great men' of Guy's Hospital—Richard Bright, Thomas Addison and Thomas Hodgkin—who fostered the attitudes that were to make Britain a leader in clinical medicine. Reference must also be made to the Irish connection—John Cheyne, Abraham Colles, Robert Adams and John Corrigan from the great Dublin School, and the two leaders Robert Graves and William Stokes. Laennec's teaching had an immediate impact in Britain by the influential work of such physicians. Graves and Stokes, both graduates of Edinburgh and subsequently professors in Dublin, collaborated in a new system of clinical instruction. Stokes in 1825 published a small treatise on the stethoscope inspired by Laennec.[2]

The modern practice of a complete clinical examination derives from nineteenth century methods such as auscultation and percussion. In today's world of imaging techniques and other technologies, it is a challenge for teachers to decide which of the old techniques are worthy of preservation.

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Risk factors for neonaticide and infant homicide

Dr Michael Craig (February 2004 *JRSM*[1]) should be congratulated on his excellent review. My only regret is his failure to consider the vital role played by 'help seeking and warning behaviour' as a means of preventing such tragedies.

Under the rubric of the battered child syndrome there is a substantial but largely neglected literature on prodromal events culminating in infant deaths due to violence. In the context of evaluating dangerousness, Bennie and Sclare[2] reported that '. . . five of ten patients had seen a doctor immediately preceding the crime, in some cases to complain about the child's behaviour and to seek help in management.' Scott,[3], in his study of 29 fatal battered-baby cases, found that '. . . three quarters of the men gave unmistakable warnings of their subsequent actions. . . . '

The importance of 'open warnings' was a major concern of Ounsted and Lynch.[4] Lynch and Roberts[5] provide a typical example:

> 'Two weeks before a ten-week old girl arrived in hospital, moribund with bilateral subdural haematoma, her mother had caused a minor bruising to the baby's buttocks during a feeding battle. She had demonstrated the injury to the family doctor who, not realizing the significance of this ''open warning'', reassured the mother that she had done no serious damage and prescribed a tranquilizer to calm her down.'

It is also important to appreciate that some vulnerable children are able to signal their distress in the presence of danger. Ounsted's description of 'frozen watchfulness' is well known.[4] Unusual aggressive or antisocial behaviour, as a prodromal sign, is also reported.[5,6] Even with young infants these behaviours may have survival value. For example, in the first of several hospital admissions, a girl aged two months had a transverse fracture of the upper left humerus with displacement. The mother attributed this to a fall. Two months before her death, two social workers witnessed the child resting comfortably in the arms of her aunt. However, when her mother arrived to take her home, the child became exceedingly agitated and resisted her mother's attempts to feed her. The child's death, at the age of eighteen months, was caused by 'sub-arachnoid and subdural haemorrhaging due to blows to the head' and other findings included unexplained anal and genital injuries.[7]

Ounsted and Lynch's concept of a 'critical path' leading to the tragedy has been of inestimable value to my own studies of family violence.[7,8] This work has convinced me that the urge to kill or to severely injure a family member or members is, almost invariably, accompanied by an equal but opposite urge to be restrained from maiming or killing. However, as this insight is dependent on an unholy alliance of anecdotal reports and 20–20 hindsight, it is virtually impossible to prove. Nevertheless, effective clinical practice demands that physicians, nurses, social workers, police and others dealing with children at risk, should be extremely sensitive to help-seeking and warning behaviours.

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