Key messages

- Globally, grain yields are not falling, but their percentage rate of increase is seriously slowing
- If grain yields are to match future population growth they will need to double or triple—sustainably
- Like China, demographically trapped communities may need one child families
- If we are to encourage any community to have one child families we should have them ourselves
- The Cairo population conference in 1994 evaded local demographic entrapment and global food security and needs to be recalled

demographic entrapment—much more seriously, dealing as they do with the distribution of wealth and power and who eats what. It will also need to remind us that, if not physically tightening out belts, we need to eat a more photon (grain) efficient diet with fewer animal products, thereby easing both the upward trend in price of grain and our personal demands on the ecosystem.

There is much preparatory work to be done. This should include considering the legitimate incentives and disincentives that are now required to assure a one child world, in the difficult task of reaching the best compromise between the human rights abuses of China (resulting from too much population control) and those of Rwanda (where there has been too little). The UN now

needs to consider how much grain it can provide to trapped communities, while they bring their populations to within their carrying capacities, while it does all it can to improve their economies and increase their exports. Most difficult of all, CAIRO II needs to consider a sustainable Northern lifestyle in the context of the needs of the South and of the planet as a whole. The world's population is now growing by 10 000 people an hour—93% of it in the South. Each baby born needs to be assured of the 25 tonnes of grain that he or she will need during their lives. Both the forthcoming world food summit and CAIRO II must come to grips with how this is to happen—reliably.

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- 1 Dyson T. *Population and food, global trends and future prospects*. London: Routledge, 1996.
- 2 Alexandratos N, ed. *World agriculture towards 2010: an FAO study*. Chichester: Wiley, 1996.
- 3 Food and Agricultural Organisation, Rome. *Food Outlook 1996*;516:3.
- 4 King MH, Elliott C, Hellberg H, Lilford R, Martin J, Rock E. Does demographic entrapment question the 2-child paradigm? *Health Policy Plann* 1995;10:376-83.
- 5 United Nations in Malawi, Government of Malawi. *Situation analysis of poverty in Malawi*. Lilongwe, Malawi: Government of Malawi, 1993.
- 6 King MH, Elliott CM. UNICEF's call to greatness, an open letter to Carol Bellamy. *Natl Med J India* 1996;9:130-3.
- 7 McIntosh CA, Finkle JC. The Cairo conference on population and development: a new paradigm? *Popul Dev Rev* 1995;21:223-60.
- 8 Sen G, Germain AS, Chen LC. *Population policies reconsidered. Health empowerment and human rights*. Boston: Harvard University Press, 1994.

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Lesson of the Week

Thyrotoxicosis in a patient with multiple trauma: value of "AMPLE" history taking

Jo Fitz-Henry, Bernard Riley

A complete medical history should be taken from all patients with trauma using information from the patient, family and friends, and witnesses

In the highly charged atmosphere of resuscitating a critically injured patient the more mundane tasks such as taking a complete medical history may often be overlooked. We report a diagnostic conundrum which occurred because of this oversight.

Case report

A 26 year old tall, slim, white man was admitted to the accident and emergency department because of trauma to the left side of his body sustained in a motor-cycle accident. Initial assessment identified an intra-abdominal haemorrhage and fractured left femur. He had a Glasgow coma scale score of 15 and no evidence of any head or neck injury. A history from the patient showed that he had been fit and well and was not receiving any drug treatment. He was taken to the operating theatre for an urgent laparotomy, splenectomy, and orthopaedic fixation of the fractured femur. He had persistent tachycardia in the operating theatre (heart rate 120-150 beats/minute) but maintained a good blood pressure and urine output throughout. During the four hour procedure he was given 5500 ml of intravenous fluids and 9 units of blood.

He was subsequently admitted to the intensive care unit for assisted ventilation and cardiovascular stabilisation. His heart rate was 150 beats/minute, intra-arterial blood pressure 180/60 mm Hg, and central venous pressure 3 mm Hg. He was considered to be fluid depleted, and vigorous fluid resuscitation was undertaken.

Six hours after surgery he had a temperature of 39.4°C and was sweating profusely, with a heart rate of

140 beats/minute, blood pressure of 110/40 mm Hg, and an obviously hyperdynamic circulation with a visible apex beat and a systolic flow murmur. There was no continuing blood loss and he was normovolaemic with a central venous pressure of 8 mm Hg, urine output of >100 ml/hour, and serum osmolality of 297 mosmol/l. He was considered to be adequately sedated by morphine and midazolam infusions, having a Ramsay sedation score of 3.¹ As he had sustained an injury to the left side of the chest in the accident, echocardiography was performed and showed a mildly dilated left ventricle with normal function. An electrocardiogram showed only a sinus tachycardia; there was no arrhythmia throughout his stay in intensive care.

This condition persisted for the next three days. His temperature decreased with active cooling of his skin surface, but he had a rebound fever each time this was discontinued. Extubation was deferred because of his high metabolic rate, oxygen requirement, and persistent tachycardia. Repeat cultures of blood, urine, and tracheal aspirate showed no sign of infection, although he had a persistent leucocytosis.

We had considered his hyperdynamic circulation and fever to be caused by the systemic inflammatory response syndrome as the result of trauma.² A continuously monitoring Swan-Ganz pulmonary artery catheter gave his cardiac output as its upper limit default reading of 20 l/minute (cardiac index 9.8 l/minute/m², normal range 2.5-3.5 l/minute/m²) and a systemic vascular resistance index of 546 dyn.s.m².cm⁻⁵ (normal range 1900-2400 dyn.s.m².cm⁻⁵). A noradrenaline infusion was started, but this made little difference to his systemic vascular resistance index, even in doses of up

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to 3.2 µg/kg/minute. *Haemophilus influenzae* was isolated from bronchoalveolar lavage fluid; his gas exchange was normal with a fractional inspired oxygen concentration of 35%, and a chest x ray film looked normal.

By day 7 he was considered to be ready for extubation. At this time his temperature was 37.7°C and his cardiac parameters were: heart rate 110 beats/minute, blood pressure 120/50 mm Hg, cardiac index 8 l/minute/m², and systemic vascular resistance index 700 dyn.s.m².cm⁻⁵. He required reintubation after only 20 minutes, when his respiratory rate was 48 breaths/minute. The following day he underwent a percutaneous tracheostomy. Although he had a long slim neck with no obvious swelling, there was a definite sensation of transmitted pulsation from the thyroid gland and the procedure itself was unusually bloody.

We then considered the possibility of thyrotoxicosis as causing symptoms of the systemic inflammatory response syndrome. His parents gave a family history of thyroid disease, and his sister was hyperthyroid. On direct questioning his girlfriend revealed that he had recently consulted his general practitioner because of severe night sweats and a failure to gain weight despite having a voracious appetite. Thyroid function tests showed severe thyrotoxicosis: tri-iodothyronine concentration was 19.5 pmol/l (normal 3.3-7.5), thyroxine 42.4 pmol/l (normal 12-24), and thyroid stimulating hormone <0.1 mIU/l (normal 0.4-5.0).

After treatment with propranolol, carbimazole, and hydrocortisone his thyrotoxic symptoms slowly settled, and he was successfully weaned from the ventilator as his oxygen requirement diminished. During the weaning process he required haloperidol for acute agitation—this may have been another manifestation of his thyrotoxic state.

Discussion

In retrospect, the natural course of a thyrotoxic crisis had unfolded in a textbook manner in the pages of our notes. The commoner differential diagnoses for persistent tachycardia, fever, leucocytosis, and high cardiac output and low systemic vascular resistance in this patient after trauma were infection and the systemic inflammatory response syndrome. Intercurrent infection seemed unlikely in the presence of repeatedly negative results in cultures. The isolation of *H influenzae* from bronchoalveolar lavage fluid without clinical or radiological signs of chest infection suggested that this

was a commensal organism. The liberation of inflammatory cytokines by direct tissue trauma is a potent trigger of the systemic inflammatory response syndrome, which produces signs identical with that of classic sepsis² and thyroid storm.

The usual response to critical illness is a hypothyroid state.^{3,4} Studies have shown that diminished thyroid stimulating hormone activity precedes low concentrations of tri-iodothyronine and thyroxine. This hypothyroid state may be physiologically protective against excessive catabolism.

Direct trauma to the thyroid area (such as may be caused by the chin strap of a motorcycle helmet) has been documented as causing a traumatic thyroiditis, but is not known to cause an acute hyperthyroid state.⁵ From the history the family gave, the patient had obviously been thyrotoxic for some time before his accident.

This patient was fully alert and oriented on his admission to hospital and had histories taken by both the staff in the accident and emergency department and the emergency anaesthetic team, both of which failed to elicit his history of thyrotoxic symptoms. Though his family were interviewed by medical staff on many occasions in the first week, they were not questioned further about his medical history until the possibility of thyrotoxicosis was raised.

This case illustrates the need to obtain a full and accurate history. The *Advanced Trauma Life Support Manual* gives the mnemonic AMPLE for the essential information to be elicited from a trauma patient's history—that is Allergies, Medications, Past medical history, Last meal, Events or description of the injury.⁶ This information should be gained from the patient, their family and friends, and witnesses to the incident in all cases.

- 1 Ramsay MAE, Savage TM, Simpson BRJ. Controlled sedation with alphaxalone-alphadolone. *BMJ* 1974; ii: 656-9.
- 2 Bone RC. Sepsis, sepsis syndrome and the systemic inflammatory response syndrome (SIRS). *JAMA* 1995; 273:155-6.
- 3 Wehmann RE, Gregerman RI, Burns WH, Saral R, Santos GW. Suppression of thyrotropin in the low thyroxine state of severe non-thyroidal illness. *N Engl J Med* 1985; 312: 546-52.
- 4 Hamblin PS, Dyer SA, Mohr VS, Le Grand BA, Lim CF, Tuxen DV, et al. Relationship between thyrotropin and thyroxine changes during recovery from severe hypothyroxinemia of critical illness. *J Clin Endocrinol Metab* 1986; 62: 717-22.
- 5 Leckie RG, Buckner AB, Bornemann M. Seat belt-related thyroiditis documented with thyroid TC-99m pertechnetate scan. *Clin Nuclear Med* 1992; 17: 859-60.
- 6 American College of Surgeons Committee on Trauma. *Advanced trauma life support manual*. Fifth Edn. Chicago, USA: American College of Surgeons, 1993.

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ANY QUESTIONS

Are selenium zinc tablets protection against macular degeneration?

The logic of giving zinc for age related macular disease relates to the observation that there are higher levels of zinc in retinal pigment epithelium, although there is little evidence of zinc deficiency at a tissue level. In 1988 a study was undertaken and as a consequence it was concluded that oral zinc had an influence on the natural course of age related macular disease in that there was a significantly lower instance of visual loss in the treatment group compared with the placebo group.¹ There were, however, certain deficiencies in the trial and it was acknowledged by the authors that the observation had not been proved beyond question. Several further studies failed to support the original conclusion. In June 1966 a well conducted double masked randomised placebo controlled study was undertaken in Vienna and this failed to show any effect on the course of age related macular disease over a period of two years.²

There is little evidence that selenium deficiency has a role in the genesis of age related macular disease or that oral intake of selenium would be of benefit.

The major risk factors for age related macular disease are age and genetic predisposition. Nevertheless, there is strong evidence that there are environmental influences, but studies to date have failed to show any consistent strong risk factors other than these. Several studies have shown that smoking increases the risk of age related macular disease and two studies imply that the intake of carotenoids, particularly zeaxanthine, may be important, although the evidence is not overwhelming.—ALAN C BIRD is professor of clinical ophthalmology in London

- 1 Newsome DA, Swartz M, Leone NC, Elston RC, Miller E. Oral zinc in macular degeneration. *Arch Ophthalmol* 1988;106:192-8.
- 2 Stur M, Tittl M, Reitner A, Meisinger V. Oral zinc and the second eye in age-related macular degeneration. *Invest Ophthalmol* 1966;37:1225-35.