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Adolescents may not be willing to give a true history in the presence of their parents. A 13 year old boy was brought by his mother to an accident and emergency department in London with facial lesions that he claimed were due to a fall (*Emergency Medicine Journal* 2004;**21**:631). It wasn't until his mother left the room (and after x rays had shown the diagnosis) that he admitted having been shot in the face with an altered blank bullet while playing with friends. As his symptoms settled and blood lead levels did not rise it was decided to leave the bullet where it was, near the coronoid process of the left mandible.

A large international study with data provided by almost 19000 people aged 20–44 years has given more evidence of the long-term effects of passive smoking in childhood (*Thorax* 2004;**59**:295–302). Men, but not women, exposed in childhood to parental smoking had more respiratory symptoms and poorer lung function as adults. Women, however, seemed to suffer more than men from the long term effects of maternal smoking in pregnancy.

Homozygous factor V Leiden mutation may lead to severe Perthes' disease. In Debrecen, Hungary (*Journal of Bone and Joint Surgery [British]* 2004;**86-B**:426–9) five of 47 patients with Perthes' disease had Factor V Leiden mutation, four homozygous and one heterozygous. The four patients with the homozygous mutation had the most severe form of the disease.

The long-term survivors of childhood cancer who have been treated with doxorubicin may show evidence of cardiac toxicity with heart failure and increased risk of sudden death. Researchers in the USA (*New England Journal of Medicine* 2004;**351**:145–53, see also perspective article, *ibid*: 120–1) have shown that the free radical scavenger, dexrazoxane, limits the elevations of serum cardiac troponin T concentration caused by doxorubicin without apparently detracting from the treatment of acute lymphoblastic leukaemia in children. The addition of dexrazoxane did not affect 2.5-year event free survival but longer follow up of both cardiac function and event free survival from leukaemia will be necessary.

Asking for allergen-specific IgE tests on the serum of children without allergen-specific symptoms and with a low serum total IgE concentration is likely to be a waste of time and money. In Portsmouth (*Journal of Clinical Pathology* 2004;**57**:956–9) 73 such children (no clue to allergen in the history, total serum IgE concentration <10 kU/litre) were tested and only three had positive allergen-specific IgE tests. The proportions of positive allergen-specific IgE tests in patients with total serum IgE concentrations of 11–20, 24–40, and 41–80 kU/litre were 13/73, 16/74, and 22/81. The authors of this paper suggest that allergen specific IgE testing should be done only if the total serum IgE concentration is >10 kU/litre or there is a history pointing to a specific allergen.

Haemopoietic stem cell transplantation after myeloablation may cure some children with severe non-malignant diseases but at a considerable cost in mortality and morbidity. Researchers in Chicago (*Lancet* 2004;**364**:156–62; see also Comment, *ibid*: 122–4) have used a less intensive preparatory regimen of fludarabine, busulfan, and anti-thymocyte globulin. They report on 13 patients treated for haemoglobinopathies (4), immunodeficiency diseases (6), and metabolic disorders (3). Full donor engraftment occurred in eight of 11 evaluable patients and three patients, two with hyper-IgM syndrome and one with X-linked lymphoproliferative disease, had complete resolution of their disease. There were two deaths in the first year, at 7 days and 3 months, and two later deaths, at 1 year 12 days and 2 years 1 month. Only one of the four patients with haemoglobinopathy had stable long-term engraftment. The authors of this paper commend the use of the reduced-intensity regimen except for children with haemoglobinopathy.

About 1% of children with diabetic ketoacidosis (DKA) develop clinically apparent cerebral oedema. More than two thirds of the children who develop symptomatic cerebral oedema either die or are left with severe brain damage. The causes of the cerebral oedema are not well understood but it may result from swelling of brain cells (cytotoxic oedema) as a result of cell damage or of osmotic imbalance, or from accumulation of intercellular fluid (vasogenic oedema) after breakdown of the endothelium that forms the blood-brain

barrier. Now researchers in California (*Journal of Pediatrics* 2004;**145**:164–71, see also editorial, *ibid*: 149–50) have shown that intercellular oedema occurs in DKA. Using diffusion magnetic resonance imaging during treatment and after recovery, they found that children without symptomatic cerebral oedema had, during treatment, a high apparent diffusion coefficient (indicating intercellular oedema) in all areas of the brain except the occipital grey matter. This suggests a vasogenic process rather than osmotic cellular swelling. They also showed a probable increase in cerebral blood flow using perfusion magnetic resonance imaging. They did not study children with symptomatic cerebral oedema so it is possible that such children may have a second and different insult. These results do not suggest alterations to present fluid and insulin management of DKA but they do suggest that research into early or preventive treatment aimed at the endothelium might prove fruitful.

The routine three-dose infant immunisation schedule against meningococcal serogroup C infection does not give long-lasting protection. After the introduction of the meningococcal serogroup C conjugate vaccine in 1999 the incidence of serogroup C disease in the UK fell sharply. Now long-term follow up data have been provided (*Lancet* 2004;**364**:365–7, see also Comment; *ibid*: 309–10). Up to the end of March 2004 there have been 214 cases of serogroup C disease in the target population, 53 of whom had been vaccinated (vaccine failures). There were four deaths among the vaccine failures and 21 of the 53 had received routine vaccination in infancy rather than catch-up vaccination. Vaccine effectiveness was 66% among infants who received the routine three doses at 2, 3, and 4 months, and 83–100% following catch-up vaccination at 5 months to 18 years. In the first year after vaccination vaccine effectiveness was high (87–98%) in all age groups. Beyond 1 year, however, vaccine effectiveness remained high in the catch-up vaccination groups (overall it fell from 95% to 90%) but was poor or nil after routine infant vaccination. Eighteen of 19 cases of serogroup C disease occurring in the routine vaccination age group after more than 1 year of follow up were in vaccinated children. Accelerated schedules may not be optimal for conjugate vaccines. Alternative schedules will need to be considered, possibly with a fourth dose in the second year.