Data from all children born in Denmark in the 11-year period 1990–2000 (New England Journal of Medicine 2004;350:1398–404; see also perspective article, ibid: 1380–2) have provided no support for the suggestion that childhood immunisations might cause type 1 diabetes. None of six different vaccines, live or inactivated, against eight diseases was associated with increased risk of later type 1 diabetes. Vaccination did not increase the risk for the siblings of children with type 1 diabetes and there was no clustering of cases 2–4 years after receiving any vaccine. Investigators have exaggerated the benefits of the newer antidepressants and played down their disadvantages in the treatment of children; these drugs cannot confidently be recommended as a treatment option for childhood depression. These are the conclusions of the authors of a review article (British Medical Journal 2004;328:879–83). In December 2003 the UK Committee on Safety of Medicines banned all selective serotonin reuptake inhibitors, except fluoxetine, for use in patients under the age of 18 years. A second systematic review (Lancet 2004;363:1341–5) has included published and unpublished data. The results suggest a favourable risk-benefit profile for fluoxetine but unfavourable profiles for paroxetine, sertraline, citalopram, and venlafaxine. Published studies may lead to wrong conclusions if the results of unfavourable studies are not published.

The entry of glucose into pancreatic beta cells leads to an increase in intracellular ATP and closure of ATP-sensitive potassium (KATP) channels. Via a series of steps involving calcium channels insulin secretion is then stimulated. Inactivating mutations in the gene encoding a subunit of the beta cell KATP channel (the KCNJ11 gene) cause congenital hyperinsulinism so activating mutations might cause congenital diabetes. An international team of researchers (New England Journal of Medicine 2004;350:1388–49) has reported finding heterozygous missense mutations of this gene in 10 of 29 patients with permanent neonatal diabetes. Sulphonylureas cause KATP channel closure and these ten patients secreted insulin in response to tolbutamide (but not to glucose) so sulphonylurea treatment may be feasible. The writers of a perspective article (ibid: 1817–8) warn, however, that sulphonylureas could have undesirable extrapancreatic effects.

With the increased provision of injector pens for children with food allergies there is a risk of accidental self-injection with adrenaline (epinephrine). Injection of adrenaline into the fingers may cause severe vasoonstriction. In Birmingham (Emergency Medicine Journal 2004;21:387–8) two 15-year-old boys each injected a thumb with discarded pens they had found and a 7-year-old boy injected his thumb with his own EpiPen. The youngest child may not have injected a significant amount of adrenaline. The other two boys had local vasoonstriction. One recovered slowly after warming the thumb and topical application of nitroglycerin paste. The other recovered after topical infiltration of 1.5 mg of phenolamine mesilate in 1 ml of 2% lignocaine (lidocaine). A literature review suggested that phenolamine, a short-acting α adrenergic antagonist, is the best treatment but there has been only one other report of its use in a child. The Birmingham team suggest using 1.5 mg of phenolamine mesilate (0.15 ml from a standard ampule containing 10 mg/ml) added to 1 ml of lignocaine 2%. After confirming local vasoonstriction from adrenaline injection the phenolamine solution should be slowly injected subcutaneously into the site stopping as soon as the skin becomes pink. The whole 1.15 ml will probably not be needed except in some adults. Unless the EpiPen is the child’s own the protocol for needle stick injuries should be followed.

Biphosphonates reduce osteoclast-mediated bone resorption and bone turnover. In the Netherlands (Lancet 2004;363:1427–31) 34 children with osteogenesis imperfecta (13 type I, nine type III, 12 type IV) were randomised to alendronate 10 mg/m2 daily or placebo for 2 years as well as taking calcium and vitamin D supplements. The risk of long bone fracture was reduced by 31% in the treated group and alendronate increased spinal bone mineral content and bone mineral density. There were no significant measurable improvements though, in function, body measurements, or radiologically measured heights of lumbar vertebrae, attributable to treatment.

Haemopoietic stem cells can change into cardiac myocytes. Now research in Florida (Lancet 2004;363:1432–7) suggests that they may also enter the brain and change into brain cells. Two women with leukaemia had each had peripheral blood stem cell transplantation from a brother, 22 days and 63 days before they died. A third woman with leukaemia had had two stem cell transplants from her brother, one of bone marrow cells and one, more than 6 years later, of peripheral blood cells. She died 13 days after the second transplant. At autopsy XY neural cells were found in the hippocampus in all three patients. In the first two patients the XY cells constituted 1% of microglia. In the third patient they constituted 2% of microglia, 1% of neurons, and 1% of astrocytes. There were no XX or XXXY fusion cells. None of the patients had had male children and a female control brain had no Y-containing neurons. Future research will show whether these cells can provide significant therapeutic neuroregeneration.

How many famous people have had Asperger’s syndrome? Quite a few if Professor Michael Fitzgerald is right. Together with Mohammed Arshad he has recently invoked the diagnosis to explain the unattractive features of Michaelangelo’s character (Journal of Medical Biography 2004;12:115–20), having previously written similarly about the architect John Nash, the mathematician Ramanujan, Irish President Eamon de Valera, Isaac Newton, and the philosopher Ludwig Wittgenstein. Michaelangelo was a bad tempered, uncommunicative, single minded genius. Are these features enough to make a diagnosis and is it helpful to group those who currently carry the diagnosis with some of the strange and eccentric characters of history? Aren’t those whose names ring down through the centuries necessarily different and psychologically “abnormal”? It would be interesting to know the “number needed to examine”. How many famous lives is it necessary to dissect in order to find one case of possible Asperger’s syndrome?

Children with Kawasaki’s disease and arterial aneurysms may develop persisting low grade inflammation and arteriopathy. Researchers in Hong Kong (Heart 2004; in press) studied 71 children who had had Kawasaki’s disease an average of 7 years previously and 35 control children. Forty-three of the Kawasaki group had had or still had aneurysms. Compared with the rest of the Kawasaki group and the controls these 43 currently had significantly higher serum concentrations of C reactive protein using a high sensitivity assay. They also had stiffer carotid arteries as assessed by ultrasound measurement of arterial distension during systole related to pulse pressure. Whether treatment at this stage would be beneficial remains to be shown.