Recent studies have estimated the prevalence of specific phobias in children at between 3 and 4%. In Sweden (Journal of Consulting and Clinical Psychology 2001;69:814–24) 60 children aged 7–17 years were randomised to one session exposure treatment with or without a parent present or to a 4-week waiting list. Half of them had animal phobias (to dogs, spiders, snakes, ants, birds, snails, or insects) and half had other phobias (to injections, enclosed spaces, blood, thunderstorms, deep water, loud noise, mummies, and yoghurt). Treatment, which lasted for up to three hours, consisted of gradual exposure to the phobic stimulus, while allowing the child to correct false beliefs about the stimulus. One week after treatment most of the children had improved and the presence or absence of a parent during treatment made little difference. The improvements were maintained at 1 year.

In New South Wales a cohort of 8–10 year olds was followed for up to 15 years (Thorax 2002;57:104–9). The two main risk factors for the onset of wheeze were atopy (at least one positive skin prick test) at baseline and asthma in a parent. Late onset atopy (more than 2 years after baseline) developed in 13.7% and wheeze in 12.4% whereas atopy remitted in 3.2% and wheeze in 5.6%. Girls were more likely to develop airway hyper-responsiveness to histamine and boys were more likely to develop atopy.

Adults with Prader Willi syndrome are prone to severe psychiatric illness and the risk is greater for those with maternal uniparental disomy of chromosome 15. In the old Anglia and Oxford health region of the UK (Lancet 2002;359:135–6) severe affective disorder with psychotic features was found in one of 13 adults with deletions at chromosome 15 and in five of eight with maternal uniparental disomy. Over the age of 28 years the prevalence of psychosis was one of nine (deletion) and five of five (disomy).

Lucina is no greenhorn and she is not unacquainted with the nastier aspects of human activity. She has read some pretty disturbing stuff but never before has her limbic system reacted in a way that has made her physically unable to finish an article at a single reading. The subject that caused this reaction was infant rape (Lancet 2002;359:274–5 (editorial)). Three cases have been reported in the news media in South Africa in recent months and a review revealed 10 other cases over a period of 9 years. In order to commit this act it is, apparently, necessary first to destroy the division between vagina and anus by forced insertion of an instrument; life-threatening haemorrhage or sepsis may ensue. It seems likely that one factor behind the crime is a belief that sexual intercourse with a very young virgin is a cure for HIV infection. The South African leadership’s attitude to the cause of AIDS has been held to be partially responsible. In a national study (Lancet 2002;359:319–20) 1.6% of nearly 12000 15–49 year old women said they had been raped as children (under 15 years) and younger women were more likely to make this claim. (A recent US study is reported to have produced a figure of 1.9% before the age of 12 years.) In South Africa one third of the offenders were schoolteachers. Childhood rape was more than twice as likely to be reported by white women compared with black women.

About half of all cases of congenital hearing impairment are thought to be genetic in origin, mostly autosomal recessive. Twenty-eight loci for nonsyndromic autosomal recessive hearing impairment have been found and 10 genes have been sequenced. Up to a half of all cases have mutations in the gene for the gap-junction protein connexin 26 (GJB2) at chromosome 13q12 but many (10–42%) are heterozygous for this mutation. Now researchers in Madrid (New England Journal of Medicine 2002;346:243–9) have found a deletion in an adjacent gene encoding connexin 30 (GJB6) in 22 of 33 people with nonsyndromic congenital hearing impairment and one abnormal GJB2 gene. Two others were homozygous for the GJB6 mutation. It is suggested that the complex locus DFNB1 contains two genes, GJB2 and GJB6, and loss of any two of the four alleles results in congenital hearing impairment.

Each year in parts of the UK one woman in every nine is subjected to domestic violence. Now a report from Maputo, Mozambique (Br J Obstet Gynaecol 2002;109:5–8) raises the grim prospect of violence as a significant cause of maternal mortality. Over a 5-year period (1991–95) 27 maternal deaths in Maputo were classified as due to homicide (10), suicide (9), accident (6), or induced abortion (2). In a previous 5-year study (1989–93) at the same hospital there were also 27 maternal deaths from pregnancy-induced hypertension which ranked fourth in the causes of maternal death. Routine enquiry about domestic violence is advocated in the UK and women find it acceptable if it is properly done (Ibid: 9–16). Although the African data may not be relevant to the UK, the editor of BJOG describes it as worrying that the Reports on Confidential Enquiries into Maternal Deaths in the United Kingdom do not contain a separate section on maternal mortality due to violence (ibid: Editor’s Choice, page unnumbered). Men who abuse their partners are likely also to abuse their children.

For deprived, HIV-1-infected mothers in developing countries there is a difficult choice between the risk of transmitting the infection to their infants by breast feeding and the infection risks of formula feeding. A study in Kenya (Journal of the American Medical Association 2001;286:2413–20) has suggested that formula feeding is probably the better option provided mothers are adequately taught about it and have access to clean water. Three hundred and seventy-one HIV-1-positive mothers were randomised to breast or formula feeding and had complete data. The breastfed infants were better nourished in the first 6-months but more likely to become infected with HIV-1 in the first 2 years (37% v 21%). Two-year mortality (24.4% (Breast) versus 20% (formula)) was not significantly different between groups, though HIV-1 infection increased mortality ninefold. The incidences of diarrhoea and pneumonia were similar in the two groups.

Do toys in waiting areas pose an infection risk? In six general practices in Christchurch, New Zealand (British Journal of General Practice 2002;52:138–40) soft toys were found to be contaminated with bacteria. Two of 10 soft toys tested gave moderate to heavy growths of coliform organisms and nine moderate to heavy growths of any bacteria. Only one of 22 hard toys gave a moderate to heavy growth of coliforms and six of any bacteria. Hard toys are easy to clean and disinfect but soft toys are difficult to disinfect and rapidly become recolonised after cleaning.