Sanctions were imposed on the people of Iraq in 1990. Iraqi people are still suffering, especially children. Infant mortality (IM) has increased more than five times. Previously it had decreased from 139 in 1960 to 20 in 1989, which was comparable to developed countries. In 1992 it went up to 111. In 1999, a decade later, IM was still high at 104. The Gulf War and trade sanctions caused a three-fold increase in mortality among Iraqi children under 5 years of age. It has been estimated that more than 46,900 children died between January and August 1991.1

The study of the UN Food and Agricultural Organisation, published in a letter to the BMJ in 1995, concluded that deaths of more than 50% of those children could be attributed to UN sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.2 Data for 1994–99 showed that mortality rates for under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, IN child mortality for children under 5 years of age showed that the percentage of children below −2SD in urban Baghdad was 28% for stunting, 29% for underweight, and 12% for wasting, which was in the region of 9% in the period 1990–98.3

In 1995, the Baghdad nutrition survey of children under five years of age showed that the percentage of children below −2SD in urban Baghdad was 28% for stunting, 29% for underweight, and 12% for wasting. The survey by FAO in the year 2000 indicated the prevalence of wasting in children under 5 years of age was acceptably high level of 10% only a marginal difference from the 1995 survey.4

In school children aged 6–8 years the prevalence of wasting ranged from 1% in the upper class to 6% in rural areas. Similar differences were found for stunting and underweight.5 In a 1994 survey 1.6% of children under 5 years were reported to have night blindness, indicating vitamin A deficiency. A survey of school children in the north in 1994 showed a 30–50% prevalence of goitre, and evidence of iodine deficiency disease elsewhere throughout the country. Riches et al reported children were hospitalised from hospitals at a rate of 3–5 cases per week.6

Diarrhoeal diseases and mortality due to dehydration were well under control prior to the Gulf War; there was a threefold increase from May 1990 to May 1991. Other water born infections increased from 1990 to 1999, for example typhoid by 60% and cholera almost fivefold.7 A measles epidemic occurred in 1998. There were alarming rises in cases of malaria and leishmaniasis. Other infections like tetanus, poliomyelitis, diphtheria, and pertussis all showed an increase after the Gulf War.8

The National Immunisation Programme which had begun in 1985 came to a complete halt between January and April 1991.9 The percentage of fully immunised one year old children fell from 94 for tuberculosis, 83 for diphtheria, tetanus, and pertussis, 83 for polio, and 82 for measles to 79, 63, 64, and 68 respectively.10

A child psychology study (1991) revealed a level of psychological stress and pathological behaviour which was the highest the authors had seen in 10 years of conflict research. It revealed a highly disturbed population of children. Fear and anxiety were associated with memories of crisis. Seventy five per cent felt sad and unhappy, and four out of five expressed fear of losing their family by death or separation.11

There was a threefold increase in leukaemia in the southern provinces, sites of the Gulf War battlefield. A WHO investigation in 1995 suggested a possible link to products—now banned—in the local environment.12 A report in 1996 showed that one third of hospital beds were closed. More than half of all diagnostic and therapeutic equipment was not working due to lack of spare parts and maintenance. All public health facilities had experienced serious problems with lighting, cleaning, water supply, and sewage. The population had been burdened by a rapid rise in serious infections, nutritional deficiencies among children and pregnant women, and other treatable conditions for which neither drugs nor operations were available.13

Paediatricians have been isolated by the intellectual embargo from the international medical community. Physicians who wish to attend international conferences face travel restrictions, like denial of visas to European countries or the USA. In 1990, the delivery of European and American medical journals were abruptly stopped. This intellectual embargo served to undermine the care of patients, and denies Iraqi doctors the right to share scientific advancement and its benefits.14

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References
2 UNICEF. The State of the world’s children. 2001.
4 Court C. Iraq sanctions lead to half a million child mortality. BMJ 1995;311:1523.

Differential diagnosis of periodic fevers
We just read the short report of Galanakis et al.1 We have been involving in periodic fevers management for many years. At present, FPFA is an unclear entity classified among non-hereditary fevers. It is an unclear nosological entity. Pharyngitis, cervical lymphadenopathies and oral aphthae are exclusive findings in FPFA. Among periodic fevers, cervical lymphadenopathy, pyrexia and episodic fever can occur in patients with Hyperig D and periodic syndrome (HIDS), and less in Familial Mediterranean Fever (FMF). Oral aphthae (as minor sign), cervical adenopathies, and isolated fever can be in children affected by FMF. Pharyngitis, oral aphthae, cervical adenopathies, and recurrent fever also characterise Crohn’s disease (CD).

Lastly, oral aphthae and recurrent febrile attacks characterise the onset of Behcet’s disease (BD) in children. The efficacy of steroids does not confirm the diagnosis of FPFA; BD and CD are responsive to steroids, too. The lack of familiar involvement is not a criteria to exclude an inherited disorder, as FMF and HIDS are recessive and BD and CD are multifactorial diseases. Furthermore, the initial clinical picture of these disorders can be typifying and incomplete and can change during the clinical course.

So, considering the provenance of Galanakis’ series (Greece), we not be surprised if some cases had BD or FMF, that will be recognised in the future. Nowadays, with increased diagnostic sensitivity and multi-ethnic societies, periodic fevers are being recognised outside their traditional areas of incidence. Close follow up is essential in further years, in these patients. A possible genetic screening for gene causing FMF, HIDS, or immunological assay for HLA B27 could also be useful.

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Reference

Mechanisms of pulmonary hypertension in Bordeletta pertussis
Casano et al describe a case of refractory pulmonary hypertension with severe Bordeletta pertussis infection.1 Their description of the literature is incomplete. We have seen four cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B pertussis.2 The cases that developed PHT all presented with severe hyperleukocytosis (WCC>100 x 10^9/l) which was unresponsive to all currently available modalities including extra corporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation severity. Our experience suggests that increased pulmonary vascular resistance via obstruction rather than

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hypoxic vasoconstriction. Therefore Dr Casano's recommendation for the early use of pulmonary vasodilators is unlikely to be sufficient in this context. We are assessing the impact of strategies aimed at reducing lymphocyte numbers and adhesion in addition to standard treatments for pulmonary hypertension.

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References

Authors’ reply
As Peters comments in his letter, we know that hyperleukocytosis has been postulated as a factor for pulmonary hypertension in Pertussis infection, but necessary brevity did not make it possible to report. Nevertheless, our patient never reached these values of leucocytosis; it’s possible, as in many other diseases, that several pathogenic mechanisms contribute to pulmonary hypertension, making a concomitant treatment approach necessary.

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In the paper by Clarkson and Choonara in the December issue of ADC (Arch Dis Child 2002;87:462–7) the following corrections have been noted:
Results: first sentence: there were 331 deaths with 390 suspected drugs (not 390 and 389 respectively as stated in the paper).
Results: section “Corticosteroids”: the third sentence starting “No details were avail-
Results: section “Non-steroidal anti-
Discussion: fifth paragraph: the penulti-

The following figure should have appeared with the letter by Desai and Babu in the October issue of ADC (Arch Dis Child 2002;87:357).

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Figure 1 Scimitar syndrome. Chest x ray showing a curvilinear density which extends from the right hilum towards the right hemi-diaphragm which represents the anomalous pulmonary vein.