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Is life long follow up for patients with Kawasaki disease indicated?

Brogan *et al* recommended life long follow up for patients with Kawasaki disease, including those who did not have coronary artery involvement. The reason they quoted was to document the blood pressure and provide general advice regarding other risk factors.[1] The American Heart Association recommends echocardiographic (ECG) evaluation of the coronary arteries at presentation and follow up ECG at 6–8 weeks and 6–12 months after the onset of symptoms for those who did not have or just have transient coronary artery involvement. They do not recommend follow up after first year unless cardiac disease is suspected.[2]

Tuohy *et al* demonstrated, in their multi-institutional review of 536 patients, that no patient with a normal follow up ECG, performed within 2 months following disease onset, subsequently developed echocardographic coronary artery abnormalities. Even those patients with initial echocardiographic abnormalities that became normal at 1–2 months remained normal thereafter.[3] Scott and colleagues showed that no patient with a normal ECG at 2 weeks to 2 months after the onset of symptoms had subsequent ECGs that revealed coronary artery abnormalities and questioned the value of 6–12 month ECG in the same group.[4]

Brogan *et al* did not make any comments about the adverse effects of life long follow up, such as anxiety and inappropriate restriction of activities. Finally, there were no comments about the cost and resources for providing life long follow up. The authors did not specify whether paediatric cardiologists, general paediatricians, or general practitioners would follow up; all of them already have increasing demands of workload.

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Management of childhood osteoporosis

I read with interest this recent review article that summarises current knowledge about this subject. I have a number of comments that are pertinent to the discussion. As the authors allude to, there is currently a lack of good evidence on which we can base preventive management. Although calcium and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density.[1] The two studies quoted in relation to growth hormone therapy are methodologically flawed because neither have accounted for the change in apparent bone density, which will occur in any child who grows better for any reason when assessed by modalities such as dual energy *x* ray absorptiometry[2 3]

As illustrated by another article in the August 2002 edition of *Archives*,[4] there is a lack of good evidence on which to base much paediatric management and it is imperative that further research, especially randomised controlled trials, is undertaken in the area of prophylaxis against osteoporosis in children with chronic disease on steroids. Paediatric endocrinologists will be familiar with the flurry of small uncontrolled studies undertaken in numerous groups of children with short stature when recombinant growth hormone became available. Many reports of short term improvements in growth velocity have not been supported by long term outcomes in height. There is a risk that a similiar phenomenon will occur with the use of bisphosphonates in children with chronic disease and low bone density without properly designed studies and satisfactory outcome measures.

The use of glucocorticoids in children with chronic disease occurs across many paediatric subspecialities and I would argue strongly that the management and prevention of osteoporosis requires specialist expertise just as the management of growth retardation currently does. It is important that in each tertiary centre such a specialist service is provided by one department that has expertise in the interpretation of bone density scans in children and the management of children with osteoporosis. Such individuals may not only be paediatric endocrinologists but may be a paediatric rheumatologist, a general paediatrician with a special interest in bone disease or a metabolic bone disease subspecialist. It is only in this way that we can learn more about the management of this condition and avoid children being treated inappropriately.

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Newborn screening for Duchenne muscular dystrophy

Elliman, Dezateux, and Bedford,[1] in their recent leading article on newborn and childhood screening, include reference to newborn screening for Duchenne muscular dystrophy (DMD). They argue that the main value of such a screening programme is to warn parents that future sons may be affected, and support this statement with reference to Jarvinen *et al*.[2] This paper does not report a newborn screening study but the results of a retrospective study of 23 females in Finland carrier tested for DMD during childhood. However, a newborn screening programme for DMD has been running in Wales since 1990 (1990–8 as a research evaluation and from 1998 health authority funded). During the research period interim evidence was published.[3–6] More recently the full results of our prospective study have been published.[7] Our evaluation has demonstrated that a newborn screening programme for DMD can be acceptable to both parents and health professionals, providing that a rigorous service delivery protocol is in place and the programme is supported by an effective infrastructure, in particular by paediatric and genetic services.

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The effect of sanctions on children of Iraq

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.[1] In 1999, a decade later, IM was still high at 104.[2] The Gulf War and trade sanctions caused a threefold increase in mortality amogn Iraqi children under 5 years of age. It has been estimated that more than 46 900 children died between January and August 1991.[3]

The study of the UN Food and Agricultural Organisation, published in a letter to the *BMJ* in 1995, concluded that deaths of more than 560 000 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.[4] Data for 1994–99 showed that mortality for children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, before sanctions. The reasons for excess deaths are clear—economic collapse with plummeting wages, soaring food prices, poor sanitation, lack of safe water, and inadequate provision of health care.[5]

The rate of low birth weight (<2500 grams) which was in the region of 9% in the period 1980–88, increased to 21% in 1994.[1] The 1995 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. Severe malnutrition (−3SD) was noted among children, 10% for stunting, 7% for underweight, and 3% for wasting.[6] The survey by FAO in the year 2000 indicated the prevalence of wasting in children under 5 years at the unacceptably high level of 10%, only a marginal difference from the 1995 survey.[7]

In school children aged 6–8 years the prevalence of wasting ranged from 1% in the upper class to 6.7% in rural areas. Similar differences were found for stunting and underweight.[7] 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. Rickets are still being reported from hospitals at a rate of 3–5 cases per week.[7]

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.[8] 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.[7] There have been alarming rises in cases of malaria and leishmiais.[1] Other infections like tetanus, poliomyelitis, diphtheria, and pertussis all showed an increase after the Gulf War.[1]

The National Immunization Programme which had begun in 1985 came to a complete halt between January and April 1991.[8] The percentage of fully immunised one year old children fell from 94 for tuberculosis, 83 for diptheria, tetanus, and pertussis, 83 for polio, and 82 for measles to 79, 63, 64, and 68 respectively.[1]

A child psychology study (1991) revealed a level of psychological stress and pathological behaviour that 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.[8]

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 incorporated in the food chain—which were derived from depleted uranium used in piercing artillery shells. There were staggering deficiencies in cancer treatment facilities because of UN sanctions which were intended to exclude food and medicines.[9]

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 hospitals 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.[10]

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 was 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.[11]

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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, PFAPA is an unclear cause of periodic fever, classified among non-hereditary fevers. It is an unclear nosological entity. Pharyngitis, cervical lymphoadenopathies and oral aphthae are exclusive findings in PFAPA. Among periodic fevers, cervical lymphoadenopathies 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 aphtae, cervical adenopathies, and recurrent fever also characterise Crohn's disease (CD). Lastly, oral aphthae and recurrent febrile attacks characterise the onset of Behçet's disease (BD) in children. The efficacy of steroids does not confirm the diagnosis of PFAPA; 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 atypical 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 multiethnic societes, periodic fevers are being recognised outside their traditional area 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 B51 could also be useful.

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Mechanisms of pulmonary hypertension in *Bordetella pertussis*

Casano *et al* describe a case of refractory pulmonary hypertension with severe *Bordetella pertussis* infection.[1] Their description of the literature is incomplete. We described 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 ($WCC > 100 \times 10^9/l$) 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 of illness. We suggested the existing histological evidence[3] was such that extreme leukocytosis prediposes to the formation of lymphocyte aggregates in the pulmonary vasculature and increased pulmonary vascular resistance via obstruction rather than