

PostScript

LETTERS

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Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

Hypertrophic pyloric stenosis of infancy is a disorder of early infancy with typical clinical features and well-established radiological appearance of the pyloric canal. Many studies with surgical and medical treatment have been reported over the past fifty years. Pylorotomy has tended to become the favoured method of treatment as with expert paediatric, surgical, anaesthetic, and nursing services and specialised accommodation for infants, the outcome is good with low mortality, short stay in hospital and few complications. However, a variety of studies of medical treatment with anticholinergic drugs and successful outcomes in some large series of cases have also been reported from Sweden, United States of America and the United Kingdom.

Since 1996 this group of workers from Osaka, Japan, has revived an interest in medical treatment with reports of a new regime using methyl atropine nitrate intravenously. To achieve satisfactory short term outcomes considerable variation in drug dosage and modified feeding regimes were necessary which involved much medical supervision and careful monitoring for toxic effects of the drug, which were minimal. The treatment was successful in the relatively small number of infants in the trial (19) with two infants being referred for pylorotomy, no mortality and no serious complications. An interesting part of this paper is the long term clinical follow up of the successfully treated infants over two years and ultrasonography of the pyloric canal which demonstrated the changes in muscle thickness and length of the canal. The disadvantages of the treatment mentioned by the authors are length of stay in hospital and the necessity to continue atropine medication orally after discharge home.

Comparing the use of this anticholinergic drug intravenously with oral treatment using methyl scopolamine nitrate and similar restricted feeding regime, oral methyl scopolamine nitrate suppressed vomiting

more quickly and reliably, was also available for subcutaneous injection if vomiting recurred as size of feeds was increased, and no toxic effects were seen in any dosage used. It would be interesting if these workers would be prepared to try the use of methyl scopolamine nitrate intravenously as pharmacologically this compound was reported to have a spasmolytic effect on gut two to three times greater than methyl atropine nitrate¹ with lesser central nervous effects.

This paper serves to emphasise once more that these infants should always be treated in paediatric centres where there is a high level of experienced paediatric care and nurses trained for neonatal special care.

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Reference

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Author's reply

We appreciate the interest shown by Dr Beryl Corner with regard to our article.¹ Unfortunately, intravenous atropine therapy is not widely accepted in European countries or the United States; it is however now becoming popular in Japan.

We are truly honoured to receive the comments of Dr Corner, who is a pioneering neonatologist and reported medical treatment with methyl scopolamine nitrate for infantile hypertrophic pyloric stenosis (IHPS) in 1955.² She pointed out that methyl scopolamine might be better than atropine sulfate in terms of effectiveness and side effects. One of the reasons why atropine was used in our study is that methyl scopolamine is not available in our country. Scopolamine butylbromide is an available quaternary ammonium derivative of scopolamine and lacks toxic side effects. However, this agent tastes bitter and is difficult to give orally to infants. Therefore, this agent is only given intravenously in infants with IHPS.

We do not know if it is worthwhile to attempt combination therapy with intravenous scopolamine butylbromide and oral atropine rather than the intravenous and oral atropine therapy. Secondly, we already knew that an intravenous atropine injection of 0.01 mg/kg was effective enough to abolish transiently the phasic and tonic pyloric contractions characteristics of IHPS.³ We used an intravenous atropine injection of 0.01 mg/kg in our study to confirm that those pyloric contractions were the cause of disturbed transpyloric flow in this condition by seeing that their inhibition with the dose of atropine ameliorated symptoms.

We agree with Dr Corner's last comment, but believe that intravenous atropine therapy is possible not only in high level paediatric centres, but also in general hospitals where infusion therapy with intravenous atropine injections can be done safely in small infants. Clinical trials are now ongoing to establish

more efficient treatment strategy for IHPS with medical and surgical therapy in our country.

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Hypothermia in a child secondary to ibuprofen

A 7 year old girl was admitted with right lower lobe pneumonia. On admission her temperature was 39.7°C. After five hours she received ibuprofen (6 mg/kg). Subsequent to this single dose her temperature decreased to 33.5°C (core temperature 34.9°C) over four hours.

On examination her pulse was 90/min, blood pressure 90/50 mm Hg, SaO₂ 96% in air, and respiratory rate 20/min. Respiratory examination was consistent with signs of right lower lobe consolidation. The rest of the examination, including the central nervous system, was unremarkable.

Results of investigations included: Hb 125 g/l; white blood cell count 10.7 × 10⁹/l platelet count 81 × 10⁹/l; C reactive protein 180 mg/l; blood glucose 4.6 mmol/l. Electrolytes and all other biochemical investigations were normal. Thyroid and cortisol assays were normal. Results of all tests to determine possible bacterial or viral aetiology were all negative (blood and urine culture, viral serology, and tests for mycoplasma). Magnetic resonance imaging (MRI) of the brain was normal.

The hypothermia was so marked that we had to use a hot air spacer blanket to raise her temperature. Despite all the efforts she remained persistently hypothermic for four days (see fig 1).

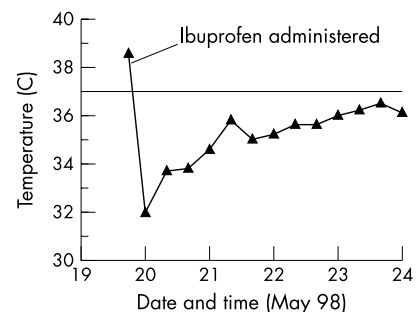


Figure 1 Temperature chart. After administration of ibuprofen, the temperature dropped considerably and remained low for five days.

A single dose of hydrocortisone and an albumin infusion were given initially. She was subsequently treated with warmed intravenous fluids for three days and antibiotics for 10 days. She recovered completely and continues to enjoy good health.

Profound hypothermia is extremely rare in children over 5 years of age. Results of investigations excluded infective and endocrine causes. A normal MRI brain scan showed there was no lesion of the hypothalamus or corpus callosum.

Ibuprofen is commonly prescribed for a raised temperature and is well tolerated in children. Side effects are not common, even in overdose.¹ Nevertheless we postulate that ibuprofen was responsible for hypothermia in this case. We are not aware of any published evidence documenting hypothermia after a single therapeutic dose of ibuprofen, but it has been recorded in a few cases of accidental and deliberate overdosage. Although patients may sometimes receive ibuprofen in toxic quantities, hypothermia is not a consistent feature.^{2,3} Hypothermia in overdosage is attributed to central nervous system depression.⁴

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Vagal overactivity: a risk factor of sudden infant death syndrome?

Since early 1990, the incidence of sudden infant death syndrome (SIDS) has dropped sharply because of public health campaigns decrying the dangers of the prone sleep position. The other known risk factors, such as preterm birth and young maternal age, are less susceptible to prevention campaigns.¹

Disordered autonomic function, including cardiorespiratory control, has been suggested to be involved in SIDS.^{2,3} Vagal overactivity (VO), characterised by breath holding spells and repeated syncope in specific circumstances, has been described as a manifestation of autonomic dysfunction.⁴ To investigate a possible relation between VO and SIDS, we investigated 65 children presenting documented VO; for example, clinical characteristics and a positive test for eyeball compression and/or electrocardiographic monitoring. Parents of these children were interviewed about their family history, especially with respect to the occurrence of SIDS among their other children.

Among their siblings, five of 126 had died of SIDS. All five children were full term infants. The average maternal age, birth weight, and age at death were respectively 27.4 (3.5) years, 3.3 (0.3) kg, and 3.5 (1.1) months. The rates of SIDS in siblings of children with VO were compared to those in the general population using the standardised incidence ratio (SIR), which is the ratio of the observed number to the expected number of cases of SIDS calculated by French incidence rates. The expected number of SIDS was 0.17 and hence the SIR was 29.4 (95% CI 9.5 to 68.6; $p < 0.00011$). Our result showed an overall significant excess of SIDS among siblings of children with VO. We verified that recruited children had not come to the centre because of a family history of SIDS. Since children with a positive family history of SIDS could be followed up more regularly than others, we estimated the SIR separately among siblings of children recruited during their follow up and those of children recruited during their first visit, and verified that there was no significant difference in SIR between these cases.

Despite the marked decline in SIDS, it is still the leading cause of postneonatal mortality. Better knowledge of other risk factors may allow identification of populations at high risk and a possible decline in infant mortality from SIDS through the implementation of appropriate prevention measures. Our findings suggest that VO may be involved in SIDS and that children with VO or a family history of VO may be a population at potential high risk of SIDS.

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Perforated duodenal ulcer disclosing medium chain acyl-CoA dehydrogenase deficiency

Medium chain acyl-CoA dehydrogenase deficiency (MCADD; McKusick 201450) typically presents in the first two years of life with recurrent episodes of hypoketotic hypoglycaemia, lethargy, coma, or sudden infant death. The trigger may be fasting, intercurrent infections, anaesthesia, or surgery. Incidence in the

UK is estimated at 0.45–1/10 000 live births.¹ We describe the case of a child who presented with marked encephalopathy unexplained by perforated duodenal ulcer, which led to the diagnosis of MCADD.

A 2 year old girl presented with a three week history of coryzal symptoms and three day history of frequent coffee ground vomiting. She was shocked, and had hepatomegaly and decreased conscious level. Blood glucose was 3.9 mmol/l (reference interval 3.3–5.5), plasma sodium 129 mmol/l (135–147), potassium 5.2 mmol/l (3.5–5.0), urea 17.8 mmol/l (3.3–6.6), creatinine 36 mmol/l (30–74), bicarbonate 15 mmol/l (21–28), base excess –5.4 mmol/l (–4 to +2) and C reactive protein 4 mg/l (0–5). Liver function tests and clotting were normal. She was resuscitated with a total of 50 ml/kg of colloid and crystalloid. The following day she relapsed with abdominal distension, shock, and deteriorating conscious level. Investigations showed glucose 14.2 mmol/l, amylase 20 IU/l (8–85), AST 186 IU/l (10–45), and ALT 129 IU/l (10–40). An x ray examination of the abdomen showed free air under the right hemidiaphragm. Emergency laparotomy revealed a single, 1 cm × 1 cm acute perforation in the second part of the duodenum. Histology and rapid urease test (CLO) of the duodenal biopsy for *Helicobacter pylori* were negative. Fasting blood gastrin was 20 mU/l (10–100). She was discharged home taking omeprazole. Upper gastrointestinal endoscopic biopsy (eight weeks later) for histopathology and CLO test from oesophagus, stomach, antrum, and duodenum were normal.

Analysis of urinary organic acids by gas chromatography and mass spectrometry, obtained a day after clinical presentation, revealed a marked increase in 5-hydroxyhexanoic acid (21% of total organic acids); a modest dicarboxylicaciduria (suberic accounted for 8% and adipic 6% of total organic acids); and a small but significant quantity of hexanoyl glycine (2% total organic acids) in the absence of ketonuria.

Blood obtained a week after clinical presentation, when analysed by tandem mass spectrometry, showed octanoylcarnitine 2.91 μmol/l (≤0.19), hexanoylcarnitine 0.67 μmol/l (≤0.29), and decenoylcarnitine 0.63 μmol/l (≤0.10), with a subnormal concentration of acetylcarnitine 4.0 μmol/l (6.2–27.5). This profile was consistent with MCADD. Polymerase chain reaction/restriction digests based method revealed two mutations in the MCAD gene.

The clinical details coupled with the absence of ketones and the increased 5-hydroxyhexanoic acid led us to look for an abnormality in the oxidation of fatty acids, and resulted in identification of the minor constituent, hexanoylglycine that is recognised as an indicative marker of MCADD. Increases in urinary hexanoylglycine and 5-hydroxyhexanoic acids in the absence of ketonuria have been reported previously in MCAD patients during clinical attack,² and also in a boy who died.³ Our case was unusual in that the amount of 5-hydroxyhexanoic acid was greater than even the sum of the individual dicarboxylic acids present, although high levels of 5-hydroxyhexanoic acids are reported in acute episodes.⁴ The increased concentration of octonoyl carnitine in blood was also consistent with a diagnosis of MCADD.

We believe that this is the first report of MCADD presenting with duodenal ulcer. It could be argued that the ulcer was the primary problem and that the decompensation was caused by the subsequent illness.

Thus, any child who has unexplained encephalopathy, regardless of its cause and clinical setting, should be screened for MCADD.

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Glucose metabolism in sleep disordered breathing

An association between sleep disordered breathing (SDB) and impaired glucose tolerance has been reported in adults.¹ Although SDB has been reported in diabetic children,² no data are available on glucose metabolism in children with SDB. We used glycated haemoglobin (HbA1c) for the preliminary assessment of glucose metabolism in paediatric SDB patients.

HbA1c was measured in 12 children aged 26–116 months (mean 63) with suspected SDB owing to adenotonsillar hypertrophy. Informed consent was obtained from the guardians of each patient, and consent was obtained from the child if older than 5 years of age. Overnight polysomnographic studies were performed once for each patient by the standard method described elsewhere.³ The desaturation time (percentage of total sleep time with oxygen saturation <90%), minimum oxygen saturation level, and apnoea-hypopnoea index (AHI) were calculated. Complete blood count, blood gases, and blood chemistry (glucose, total protein, albumin, urea nitrogen, creatinine, uric acid, sodium, chloride, potassium, calcium, phosphate, lactic dehydrogenase, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, γ -glutamyl transpeptidase, alkaline phosphatase, total bilirubin, total cholesterol, and triglyceride) were also determined.

The patients had no respiratory failure, heart failure, or coma. None of their weights exceeded 120% of their ideal weight for their

heights. Desaturation time clearly divided the patients into two groups: six patients whose desaturation time was 0 or 0.1 (mild SDB group); and six whose desaturation time exceeded 4.0 (severe SDB group). The average HbA1c value for the severe SDB group (5.0, SE 0.07) was significantly higher than that for the mild SDB group (4.6, SE 0.10) ($p = 0.01$), although the actual HbA1c values were all within normal range. No other items showed significant differences between the two groups.

The severity of respiratory disturbances during sleep in diabetic children has been known to correlate with the duration of diabetes and with the HbA1c value.² Recently, SDB parameters were found to be associated with worsening insulin resistance independent of obesity in adults.⁴ The current study shows that serum HbA1c is increased in association with the degree of desaturation in non-obese paediatric SDB patients; HbA1c levels should, however, be monitored after treatment. SDB and glucose metabolism are hypothesised to be closely associated in children as well as adults.

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Short versus standard duration antibiotic treatment for UTIs: a comparison of two meta-analyses

Having recently published a meta-analysis on the same clinical question,¹ it was with great interest that we read Michael *et al*'s systematic review of short versus standard duration antibiotics for urinary tract infections (UTIs) in children.² Given the publication (in close succession) of two meta-analyses on the same question with (on the surface) strikingly different results, we thought a comment was in order.

First, we applaud the authors on their methodologically sound review. The literature search was explicitly described and exhaustive. In fact, the authors identified a few studies that we had missed.^{3–6} The study outcomes for meta-analysis (frequency of positive urine cultures at 0–7 days after treatment and at 10 days to 15 months after treatment, and development of resistant organisms and recurrent UTI) were relevant and clearly defined.

The authors provided appropriate and important meta-analysis measures including summary relative risks (RRs) and a quasi-NNT calculation with varying risk of treat-

ment failure in the standard treatment group and confidence intervals corresponding to "best" and "worst" case scenarios.

For their primary outcome, frequency of positive urine cultures 0–7 days after treatment, the authors found no significant difference between short (2–4 days) and standard (7–14 days) treatment (RR 1.06; 95% CI 0.64 to 1.76). This is in contrast to our finding of a 94% increased pooled risk of treatment failure with short course treatment (≤ 3 days) compared to standard treatment (7–14 days) (RR 1.94, 95% CI 1.19 to 3.15; NNT=13, 95% CI 100 to 7). Why the discrepancy? We postulate a few possible explanations and conclude that the two meta-analyses, on closer inspection, actually have very similar results.

Our omission of certain studies identified by Michael and colleagues may have biased our results. However, of the three studies^{3–5} that we missed and that they included in their analysis of treatment failure at 0–7 days after completion of treatment, two favoured standard duration treatment, which would have supported our pooled RR result. Another possible explanation for the divergent results was the use of different definitions of treatment failure. For our definition of treatment failure we pooled persistent infection (failure to eradicate the organism within 1 to 2 days of initiation of treatment) and relapse (recurrence of symptoms and reinfection within 2 weeks of cessation of treatment after initial bacteriologic cure), whereas Michael *et al* used frequency of positive cultures 0–7 days after cessation of treatment as their primary outcome measure of treatment failure. If reinfections later than 7 days after cessation of treatment occurred more often in recipients of short course treatment, then Michael *et al*'s definition of treatment failure could have failed to capture the therapeutic advantage of standard duration treatment.

However, the most likely explanation for the divergent results was the different ways in which the study question was framed and the resulting differences in studies included in the meta-analyses. We compared ≤ 3 days of treatment to 7–14 days of treatment, whereas Michael *et al* compared 2–4 days of treatment to 7–14 days of treatment and excluded 11 studies comparing single-dose or single-day treatment to standard duration treatment.^{7–17}

The reasons for this exclusion are unclear, although we presume that they felt single-dose or single-day treatment was not a fair comparison with 7–14 day treatment. However, a number of randomised controlled trials (RCTs) made this comparison, suggesting that clinicians are, in fact, interested in the potential efficacy (and significantly increased ease and savings) of single-dose or single-day treatment. Inclusion of these studies in our analysis strongly influenced the pooled risk of treatment failure with short-course treatment. When we excluded these studies in a sub-group analysis of 3-day versus long course (7–14 day) treatment, the risk of treatment failure fell to 1.36 (95% CI 0.68 to 2.72) (NNT=50; 95% CI 33–13).

Thus, our meta-analysis demonstrates clearly that single dose or single day antibiotic treatment is not as effective as long-course treatment for UTIs in children. The two meta-analyses together suggest that:

- (1) "longer" short-course therapies may be as effective as 7–14 days of antibiotics and

(2) there is probably a duration of treatment threshold for "short-course" antibiotic treatment, above which longer duration of treatment confers no therapeutic advantage.

Michael and colleagues suggest that as little as 2 days of treatment may be sufficient. However, only one of the trials in their meta-analysis studied 2-day treatment⁴ and that one favoured long-course treatment with a RR of UTI 0–7 days after completing short course treatment of 2.17 (95% CI 0.48 to 9.76). The duration of treatment threshold may be 3 days, but the point estimate of relative risk of treatment failure with 3 day treatment in our meta-analysis suggests otherwise. If the duration of short-course treatment for which there is no difference in efficacy compared with standard treatment is actually greater than 3 days, then the added convenience and cost-savings of "short-course" treatment become marginal. In the absence of appropriately powered RCTs (or meta-analyses) examining outcomes (treatment failure, reinfection, emergence of resistant organisms and cost) with "longer" short course treatment regimens (3, 4, and 5 days), we think that clinicians should continue to treat UTIs in children with at least 7 days of antibiotics.

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Table 1 Results of three systematic reviews of randomised controlled trials comparing short duration with standard duration of antibiotic treatment for lower tract urinary tract infection.

Systematic review	Comparison of duration of therapy	Number of data sets	Risk for persistent bacteriuria
Tran <i>et al</i> , 2001 ³	1–4 days v >5 days	13	RD ^a 4.26 (95% CI ^b -0.95, 9.48)
Keren & Chan, 2002 ²	3 days v 7–14 days	5	RR ^c 1.36 (95% CI 0.68, 2.72)
Michael <i>et al</i> , 2002 ¹	2–4 days v 7–14 days	8	RR 1.06 (95% CI 0.64, 1.76)

^aRD, risk difference; ^bCI, confidence intervals; ^cRR; relative risk

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Authors' reply

In response to Keren and Chan's thoughtful letter regarding our recent systematic review,¹ we need to emphasise that the study question we addressed was different from that addressed by Keren and Chan in their own systematic review² of randomised controlled trials comparing short with standard duration treatment in the treatment of children with urinary tract infection (UTI). The aim of our study was to determine the relative efficacies of short (2–4 days) and standard duration (7–14 days) treatment with the hypothesis that short duration may be as effective as standard duration treatment and provide potential advantages such as improved compliance. Therefore, we did not include trials in which single dose treatment was compared with standard duration treatment. In addition we chose to limit the review to trials in which the same antibiotic was used to treat each group, to avoid confounding.

The response to single dose treatment appears different from short course, suggesting that it is inappropriate to pool studies comparing single dose and standard treatment with those comparing short course and standard treatment. Three systematic reviews^{1–3} have now demonstrated that there is no significant difference in the number of children with persistent bacteriuria after short duration or standard duration treatment (see table 1). In contrast, Keren and Chan² found that significantly more children had persistent bacteriuria following single dose compared with standard duration treatment (7 data sets: RR 2.73, 95% CI 1.38 to 5.40). Similarly, Tran *et al*³ in their meta-analysis of 22 studies comparing both single dose and short duration treatment with standard duration treatment found the latter to be more effective (risk difference 6.38; 95% CI 1.88 to 10.89).

Because there is no significant difference between short duration and standard duration treatment in the number of children with persistent UTI after treatment, it is not possible to calculate a number needed to treat to prevent one episode of persistent bacteriuria.

From our systematic review, we are not able to determine whether there is an "optimum duration of treatment threshold" as postulated by Keren and Chan.² Only one study⁴ included in the meta-analysis, examining the effects of short duration and standard duration treatment in clearing bacteriuria, compared 2 days of treatment with 10 days' treatment. In their letter above, Keren and Chan argue that this study favours standard duration treatment. However, there was no significant difference between treatments in the number of children with persistent bacteriuria at the end of treatment (RR 2.17; 95% CI 0.48 to 9.76) although the wide confidence intervals do not exclude the possibility that short duration treatment could be more or less effective than standard duration treatment.

No significant differences in the number of children with persistent UTI after treatment between short duration and standard duration antibiotic treatment have been found in three systematic reviews of randomised controlled trials despite different study inclusion criteria and definitions of persistent infection. As addressed in our review, the wide confidence intervals around the summary estimates indicate residual imprecision in the results. However, this statistical imprecision is of doubtful significance for most children, who are at a low risk (1–3%) of persistent UTI at the end of treatment following their first lower tract UTI.^{5,6} Therefore, we do not support Keren and Chan's conclusion that clinicians should continue to treat lower tract UTI with standard duration treatment. Instead, we believe that short duration treatment may be used to treat children with lower tract UTI.

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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 echocardiographic 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 similar 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 subspecialties 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 among 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 leishmaniasis.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 diphtheria, 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 involved 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 lymphadenopathies and oral aphthae are exclusive findings in PFAPA. Among periodic fevers, cervical lymphadenopathies 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 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 familial 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 multi-ethnic societies, 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 × 10⁹/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 evidence³ was such that extreme leukocytosis predisposes to the formation of lymphocyte aggregates in the pulmonary vasculature and increased pulmonary vascular resistance via obstruction rather than

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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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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CORRECTIONS

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 available..." should be deleted.

Results; section "Non-steroidal anti-inflammatory drugs (NSAIDs)": the second sentence "All reports for NSAIDs have occurred since 1990" should be deleted.

Discussion; fifth paragraph: the penultimate sentence should be "as recently as 1999 our study found a single fatality" (not 2 reported fatalities).

Discussion; fourth paragraph, second sentence. The word "seven" before "cases" should be deleted.

The journal apologises for the errors.

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).



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.