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 appearances 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 intravenous methyl atropine nitrate intra venously as pharmacologically this compound was reported to have a spasmylic effect on gut two to three times greater than methyl atropine nitrate 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.

Author’s reply

We appreciate the interest shown by Dr Beryl Corner with regard to our article. 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. 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. 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.

H Kawahara
Consultant Paediatric Surgeon, Osaka Medical Centre and Research Institute for Maternal and Child Health; kawahara@pedsurg.med.osaka-u.ac.jp


Figure 1

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

Figure 1
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.

Hydrothorax 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. Nevertheless, we postulate that ibuprofen was responsible for hydrothorax in this case. We are not aware of any published evidence documenting hydrothorax after a single therapeutic dose of ibuprofen, but it has been recorded in a few cases of accidental and deliberate overdose. Although patients may sometimes receive ibuprofen in toxic quantities, hydrothorax is not a consistent feature.1 Hydrothorax in overdose is attributed to central nervous system depression.4

P R Desai
Southend Hospital, Room 2, Doctor’s Quarters, Chelmsford Accommodation, St John’s Hospital, Chelmsford CM2 9QB, UK, prpavin@yahoo.com

S Sriskandan
Department of Paediatrics, Southend Hospital, Prittlewell Chase, Southend on Sea SS0 8YK, UK

References

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. Nevertheless, we postulate that ibuprofen was responsible for hydrothorax in this case. We are not aware of any published evidence documenting hydrothorax after a single therapeutic dose of ibuprofen, but it has been recorded in a few cases of accidental and deliberate overdose. Although patients may sometimes receive ibuprofen in toxic quantities, hydrothorax is not a consistent feature.4 Hydrothorax in overdose is attributed to central nervous system depression.4

T Shojai-Brosseau, C Bonatti-Pellie
Unité de Recherche en Épidémiologie des Cancers, INSERM U521, Villejuif, France

S Lyonnet, J Feingold
Unité de recherche sur les Handicaps Génétiques de l’Enfant, INSERM, US933, Paris, France

V Lucet
Centre de Cardiologie Infantile du Château des Côtes, Les Loges-en-Josas, France

Correspondence to: Dr T Shojai-Brosseau, Service de Biostatistiques, Institut Curie, 70 rue Meuffelart, 75005 Paris, France; tareneh.shojai@curie.net

References

Perforated duodenal ulcer disclosing medium chain acyl-CoA dehydrogenase deficiency

Medium chain acyl-CoA dehydrogenase deficiency (MCADD) is an inherited metabolic disorder that affects the oxidation of medium-chain fatty acids. Patients with this condition may present with a wide range of symptoms, including hypoglycemia, shock, and death. The trigger may be fasting, intercurrent infection, or a drug that can induce it. The condition is caused by a deficiency in the enzyme medium chain acyl-CoA dehydrogenase, which is responsible for the oxidation of medium-chain fatty acids.

10 days. She recovered completely and continued to enjoy good health.

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The trigger may be fasting, intercurrent infection, or a drug that can induce it. The condition is caused by a deficiency in the enzyme medium chain acyl-CoA dehydrogenase, which is responsible for the oxidation of medium-chain fatty acids.
Thus, any child who has unexplained encephalopathy, regardless of its cause and clinical setting, should be screened for MCADD.

V Kairamkonda, M Dalzell
Department of Gastroenterology, Royal Liverpool Children’s NHS Trust, Alder Hey Children’s Hospital, Liverpool, UK

P D Losty
Department of Surgery, Royal Liverpool Children’s NHS Trust, Alder Hey Children’s Hospital

C Davidson
Department of Metabolic Medicine, Royal Liverpool Children’s NHS Trust, Alder Hey Children’s Hospital

Correspondence to: Dr M Dalzell, Department of Gastroenterology, Royal Liverpool Children’s NHS Trust, Alder Hey Children’s Hospital, Liverpool L12 2AP, UK, mark.dalzell@hcht.nwest.nhs.uk

References
1 Pollitt RJ, Leonard JV. Prospective surveillance study of medium chain acyl-CoA dehydrogenase deficiency in the UK. Arch Dis Child 1998;79:116–19

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, 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 adentosinilar 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.2 The desaturation time (percentage of total sleep time with oxygen saturation <90%) and minimum oxygen saturation level, and apnoea-hypopnoea index (AHI) were calculated. Complete blood count, blood gases, and blood chemistry (glucose, total protein, albumin, urca nitrogen, creatinine, uric acid, sodium, chloride, potassium, calcium, phosphate, lactate dehydrogenase, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, γ-glutamyl transferase, 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.3 Recent 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.

J Kohyama, T Hasegawa, J S Ohinata
Department of Pediatrics, Faculty of Medicine, Tokyo Medical and Dental University, Japan

Correspondence to: Dr J Kohyama, Department of Pediatrics, Faculty of Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Tokyo 113-8519, Japan; jkohyama.ped@md.ac.jp

References

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 antibiotic treatment 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 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 treatment 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-course (2–3 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 (RR ≤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 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 discrepancy was the use of different definitions of treatment failure. For our definition of treatment failure we pooled persistent infection and treatment failure to eradicate the organism after 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.

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.4–6

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 consequently increased cost) 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 treatment and that only moderate 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 their 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.

R Keren
Department of Pediatrics, The Children’s Hospital of Philadelphia, USA

E Chan
Department of Pediatrics, The Children’s Hospital of Boston, USA

Correspondence to R Keren; keren@email.chop.edu

References

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.

<table>
<thead>
<tr>
<th>Systematic review</th>
<th>Comparison of duration of therapy</th>
<th>Number of data sets</th>
<th>Risk for persistent bacteriuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tran et al, 2001</td>
<td>1–4 days v 5–9 days</td>
<td>13</td>
<td>RD 4.26 (95% CI 0.95, 9.48)</td>
</tr>
<tr>
<td>Keren &amp; Chan, 2002</td>
<td>3 days v 7–14 days</td>
<td>5</td>
<td>RR 1.36 (95% CI 0.68, 2.72)</td>
</tr>
<tr>
<td>Michael et al, 2002</td>
<td>2–4 days v 7–14 days</td>
<td>8</td>
<td>RR 1.06 (95% CI 0.64, 1.76)</td>
</tr>
</tbody>
</table>

*RD, risk difference; CI, confidence intervals; RR, relative risk.

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.1 Only one study 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 short 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.1 Therefore, we do not support Keren and Chan’s conclusion that clinicians should continue to treat UTI with standard duration treatment. Instead, we believe that short duration treatment may be used to treat children with lower tract UTI.

E M Hudson, M Michael, J C Craig, S Martin
Centre for Kidney Research, The Children’s Hospital at Westmead, Sydney, Australia

V A Moyer
Center for Clinical Research and Evidence Based Medicine, The University of Texas–Houston Health Science Center, Houston, TX, USA

Correspondence to: E Hudson, Elisash@chw.edu.au

References
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. The American Heart Association recommends echocardiographic (Ech) 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. Tuohy et al demonstrated, in their multi-institutional review of 356 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. 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 new coronary artery abnormalities and questioned the value of 6–12 month ECG in the same group.

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.

References

5 N J Shaw

Newborn screening for Duchenne muscular dystrophy

Elliman, Dezateux, and Bedford, 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. 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. More recently the full results of our prospective study have been published. 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.

E P Parsons, S J Murugan, J Thomson, J M Parsons

References

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. In 1999, a decade later, IM was still high at 104. 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. 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 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 among children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, and the mortality for children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, and the mortality for children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9.

A study in 1995 suggested that deaths of more than 46,000 children occurred in 1991 due to UN sanctions. In 1999, a decade later, IM was still high at 104. The Gulf War and trade sanctions caused a threefold increase in mortality among Iraqi children under 3 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 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 among children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, and the mortality for children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9.

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 health and community 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. 3

Paediatricians have been isolated by the intellectual embargo of 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 medical supplies was so difficult that it undermined the care of patients, and denied Iraqi doctors the right to share scientific advancement and its benefits. 4

L Al-Nouri
Q Al-Rahim
FRCPCH, Yarmouk, PO Box 15103, Baghdad, Iraq
Correspondence to: Dr Al-Nouri; al-nouri@uruklink.net

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; 311:1532.

Differential diagnosis of periodic fevers

We just read the short report of Galanakis et al. 5 We have been involving in periodic fevers management for many years. At present, PAPA is an unclear periodic fever, classified among non-hereditary fevers. It is an unclear nosological entity. Pharyngitis, cervical lymphadenopathies and oral aphthae are exclusive findings in PAPA. Among periodic fevers, cervical lymphadenopathies and oral aphthae can occur in patients with Periodic 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 PAPA; 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 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.

M La Regina, G Nucera, M Diaco, R Manna, G Gasbarrini
Centre of Periodic Fevers, Catholic University of Rome, Italy
Correspondence to: Dr La Regina; mman@uniroma1.it

Reference

Mechanisms of pulmonary hypertension in Bordetella pertussis

Casano et al describe a case of refractory pulmonary hypertension with severe Bordetella pertussis infection.4 Their description of the literature is incomplete, and their discussion of the four cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B. pertussis infection.5 The cases that developed PHT all presented with severe hyperleukocytosis (WCC>100 × 10^9/L) which was unresponsive to all currently available modalities including extra-corporal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation with hyperleukocytosis and extracorporeal life support.5 Their histological evidence 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.

M J Peters, C M Pierce
Paediatric Intensive Care Unit, Great Ormond Street Hospital, London, UK

N J Klein
Infectious Diseases and Microbiology Unit, Institute of Child Health, London, UK

Correspondence to: Dr Peters; m.peters@ich.ucl.ac.uk

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 leukocytosis; it's possible, as in many other diseases, that several pathogenic mechanisms contribute to pulmonary hypertension, making a concomitant treatment approach necessary.

M Pons, P Casano
Hospital Sant Joan de Déu, Unidad de Cuidados Intensivos Pediátricos, Passeig de Sant Joan de Déu, 2 080950, Esplugues de Llobregat, Barcelona, Spain
Correspondence to: Dr Pons; mpons@hsjdbcn.org

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-
able...” should be deleted.
Results; section “Non-steroidal anti-
flammatory drugs (NSAIDs)”: the second sentence “All reports for NSAIDs have oc-
curred since 1990” should be deleted.
Discussion; fifth paragraph: the penulti-
mate sentence should be “as recently as 1999 our study found a single fatality” (not 2 reported fatalities).

Discussion; fourth paragraph, second sen-
tence. 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).

CORRECTIONS

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.