Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

Hyptertrophic 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 fa-voured 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 1995 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 scopo-lamine nitrate intravenously as pharmacologi-cally this compound was reported to have a spasmyloic effect on gut two to three times greater than methyl atropine nitrate 3 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.

B Corner
Flat 4 Chartley, The Avenue, Sneyd Park, Bristol BS9 1PE

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 1995.2 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 butyrylmethide 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 butyrylmethide 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.

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

References

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; 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 ibufrofen, 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 3 days and antibiotics for 10 days. She recovered completely and continues to enjoy good health.

1-year-old 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. Nonetheless 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 overdose. Although patients may sometimes receive ibuprofen in toxic quantities, hypothermia is not a consistent feature. Hypothermia in overdose is attributed to central nervous system depression.8

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. The other known risk factors, such as preterm birth and young maternal age, are less susceptible to prevention campaigns.

Disordered autonomic function, including cardiorespiratory control, has been suggested to be involved in SIDS.1 Vagal overactivity (VO), characterised by breath holding spells and repeated syncope in specific circumstances, has been described as a manifestation of autonomic dysfunction.2 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. Patients 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 < 0.000011). Our result showed an overall significant excess of SIDS among siblings of children with VO (p = 0.001). Children 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 risk and high risk and the decrease 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.

1 T Shojaei-Brosseau, C Bonaiti-Pellie. Unité de Recherche en Epidemiologie des Cancers, INSERM U521, Villejuif, France
2 S Lyonn, J Feingold. Unité de recherche sur les Handicaps Génétiques de l’Enfant, INSERM U523, Paris, France
3 V Lucet. Centre de Cardiologie Infantile du Château des Côtes, Les Loges-en-Josas, France

Correspondence to: Dr T Shojaei-Brosseau, Service de Biostatistiques, Institut Curie, 70 rue Mouffetard, 75005 Paris, France; taraneh.shojaei@curie.net

References

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, intermittent 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 1-year-old girl presented with a 3-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 3.5–5.3), 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), albumin 29 mmol/l (40–50) and C reactive protein 4 mg/l (0–5). Liver function tests and clotting were normal. She was resuscitated with a total of 30 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 mol/l (10–100). She was discharged home on omeprazole. Upper endoscopy and 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 dicarboxylic aciduria (suberic accounted for 8% and adipic 6% of total organic acids); and a small but significant quantity of hexanoylglycine (2% total organic acids) in the absence of ketonuria.

Blood obtained a week after clinical presentation, when analysed by tandem mass spectrometry, showed oxoacids: 3-methyl-2-oxopentanoic acid 0.19 mmol/l (0.10–0.19), hexanoylglycine 0.67 mmol/l (0.29), and decanoylglycine 0.63 mmol/l (0.10–0.10), with a subnormal concentration of acetylcrementline 4.0 mmol/l (6.2–7.5). This profile was consistent with MCADD. Polymerase chain reaction/restriction digests based method revealed two mutations in the MCADD 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 MCADD patients during clinical attack, and also in a boy who died.1 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.1 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 decompen- sation 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.

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@hhct.nwts.nhs.uk

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1 Politte 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 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. 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, lactate 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.1 Thus, any child who has unexplained encephalopathy, regardless of its cause and clinical setting, should be screened for MCADD.

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.1 This 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; kohyama.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 antibiotics for urinary tract infections (UTIs) in children.1 Given the publication (in close succession) of two meta-analyses on the same question with (on the surface) strikingly different conclusions 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 disparity 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 up 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 frequently in recipients of short-course treatment, then Michael et al’s definition of treatment failure could have failed to capture the thera-}

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References


Authors' reply

In response to Keren and Chan's thoughtful letter regarding our recent systematic review, we need to emphasise that the study question we addressed was different from that addressed by Keren and Chan in their own systematic review 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. In addition, we chose to limit the review to trials in which the same antibiotic was used to treat each group, to avoid confounding.

We believe that short duration treatment may be used to treat children with lower tract UTI. E M Hodson, M Michael, J C Craig, V A Moyer Centre for Kidney Research, The Children’s Hospital at Westmead, Sydney, Australia

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 5–7 days</td>
<td>13</td>
<td>RR 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>

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. Only one study included in the meta-analysis, the 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. 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.

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

References


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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 do 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 (EKG) 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.

Touhy 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. 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.

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 workloads.

References


Newborn screening for Duchenne muscular dystrophy

Elliman, Dezaute, 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
SOMS and Institute of Medical Genetics, University of Wales College of Medicine, Cardiff, UK

D M Bradley
Department of Medical Biochemistry, University of Wales Hospital of Wales, Cardiff, UK

A J Clarke
Institute of Medical Genetics, University of Wales College of Medicine

Correspondence to Dr Parsons; parsonsep@cf.ac.uk

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 3 years of age. It has been estimated that more than 46 900 children died between January and August 1991.

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 infections 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.

Data for 1994–99 showed that mortality among 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 dehydration were well under control prior to the Gulf War; 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 from the family.

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 lice of spare parts and maintenance. All public and private hospitals and health centres were experiencing 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.

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.

Differential diagnosis of periodic fevers

We just read the short report of Galanakis et al. We have been involving in periodic fevers management for many years. At present, PFPA 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 PFPA. Among periodic fevers, cervical lymph adenopathies, polyarthritis, 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 Crohdi’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 PFPA; 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 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.

Mechanisms of pulmonary hypertension in Bordetella pertussis

Casano et al describe a case of refractory pulmonary hypertension with severe Bordetella pertussis infection. Their description of the literature is incomplete. In 1999, four cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B. pertussis. The cases that developed PHT all presented with severe hyperleukocytosis (WCC>100 × 10^9/L) which was unresponsive to all currently available modalities including extracorporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation (see table). There was no significant difference in the severity of disease or histological evidence such that extreme leucocytosis predisposes to the formation of lymphocyte aggregates in the pulmonary vasculature and increased pulmonary vascular resistance by obstruction rather than

References


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 leucocytosis; 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

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.

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.