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 preferred procedure for the treatment of this condition. However, a variety of studies have shown that in 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, 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 regimens 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 pharmalogically 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.

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

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@peds.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; 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).
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 the rates 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 compared to siblings of 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 high risk and in the 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.

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. 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 the rates 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 compared to siblings of 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 high risk and in the 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.

T Shojaei-Brosseau, C Bonatti-Pellie
Unité de Recherche en Épidémiologie des Cancers, INSERM U521, Villejuif, France
S Lyonnnet, J Feingold
Unité de recherche sur les Handicaps Génétiques de l’Enfant, INSERM, U393, Paris, France
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

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 one year old girl presented with a three week history of corzyl 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 3.5–5.2), 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), magnesium 0.4 mmol/l (0.7–1.1), 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 (CL0) 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 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 accumulation of 5-hydroxyhexanoic acid (21% of total organic acids); a modest dicarboxylic aciduria (succinate 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 a clinical presentation, when analysed by tandem mass spectrometry, showed octanoylglycine 0.29 mmol/l (0.19), hexanoylglycine 0.67 mmol/l (0.29), and decenoylglycine 0.63 mmol/l (0.10), with a small but significant increase in 5-hydroxyhexanoic acid. 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,6 and also in a boy who died.7 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. The increased concentration of octanoyl 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.
Glucose metabolism in sleep disordered breathing

An association between sleep disordered breathing (SDB) and impaired glucose tolerance has been reported in adults. 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 guardians of each patient, and consent was obtained from the child if older than 5 years of age. Informed consent was obtained from the guardians of each patient, and consent was obtained from the child if older than 5 years of age. Glucose metabolism in sleep disordered breathing (SDB) and impaired glucose tolerance has been reported in adults. 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%) was significantly higher than that for the control 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. 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; kohyama.peidi md.ac.jp

References
(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 one compared a 7-day 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 &gt;5 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>

*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. 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. Therefore, we do not support Keren and Chan’s conclusion that clinicians should continue with short course 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

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 Hodson, Elissah@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 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 (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.

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. 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 having increasing demands of workload.

S J Murugan, J Thomson, J M Parsons
Yorkshire Heart Centre, Leeds General Infirmary, Leeds, UK

Correspondence to S J Murugan; jottdevi1@hotmail.com

References

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 preventative 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. The relationship 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 assessed by modalities such as dual energy x ray absorptiometry.

As illustrated by another article in the August 2002 edition of Archives, there is a lack of good evidence on which to base much preventative 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 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 assessed by modalities such as dual energy x ray absorptiometry.

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 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 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
2 Jarvinen O, Lehtojaaki AE, Lendal M, et al. Carrier testing of children for two X linked diseases: a retrospective study of the interpretation of bone density scans in children and the management of children with osteoporosis. Such individuals may not 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.

N J Shaw
Birmingham Children’s Hospital, Birmingham, UK
nick.shaw@bchumanchildrens.wmids.nhs.uk

References

www.archdischild.com

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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. The Gulf War and trade sanctions caused a three-fold increase in mortality among Iraqi children under 5 years of age. It has been estimated that more than 46,000 children died between January and August 1991.2 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 1991.3 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 due to 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 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 1991.5 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 due to sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.6 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 1991.7 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 due to sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.8 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 1991.9 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 due to sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.
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; mp@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 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).

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.
LETTER

Questions on questionnaire development

With interest I read the paper by Powell et al on the development of a questionnaire to describe respiratory symptoms in infants and preschool children. Because of the age of the children this is a difficult topic, and the authors are to be congratulated for their attempt. However, a number of questions arose when reading their paper.

First of all, the method for assessing test-retest reliability is questionable. The method, originally developed in the field of psychology, was used to see whether, when measuring some personality trait, assessing it repeatedly would give the same results. The kappa values for agreement in this area are usually in the range 0.70–0.90. Respiratory symptoms are not personality characteristics, and cannot be assumed to be stable. So when assessing symptoms over the previous three months, with two weeks interval, a change may be due to what statisticians call "measurement error", but also to a change in symptoms. A related issue is the interpretation of the results. In the paper, the authors mention one kappa score below 0.40; but they fail to mention that the majority of other items were below 0.60 (reliability results from tables 1 and 2, accessible from ADC Online). In the abstract the authors conclude that the short term reliability is good, but this certainly overstates the results. It is not clear why the authors have chosen to compare the 20 referred children in whom a diagnosis of asthma was made to the 42 children from the newborn cohort. Why not compare them to the referred children who were not labelled as having asthma? Finally, it is unclear why in table 1 (paper version) the reader cannot reproduce the figures in the last column from the previous columns.

J C van der Wouden
Dept of General Practice, Erasmus MC, Rotterdam, Netherlands; vandewouden@hag.fgg.eur.nl

Reference

Handbook of paediatric investigations


Stroobant and Field, perhaps the longest standing editorial partnership in UK paediatrics, have done it again. This time they shrewdly spied a gap in the market for a home grown concise book for everyday use aimed specifically at answering the question: "what tests?"

How best to review it? Why not try "road testing" it on a few problems this general paediatrician happens to have seen on the wards recently.

Firstly, a 12 year old with painless microscopic haematuria. We find the expected exhortation to take a full history and do a thorough examination, followed by a friendly table listing the more straightforward tests, and a discussion of the more fancy ones to be considered. The point about this sort of book, of course, is to supply reminders and hints about what to consider, rather than lists to follow slavishly.

Secondly, a pair of brothers whose bones keep breaking. Are there any tests worth doing to look for osteogenesis imperfecta? Nothing at all on this, but maybe that's a bit too specialised for this small book.

Next patient, one of those worrying "funny bruising" problems: is it NAI, or is there a rare clotting/platelet disorder? There's no schema for investigating easy bruising as such, but platelet function and coagulation disorders are discussed. There are useful tables of all the tests haematologists can do, and looking at these enables the paediatrician to sound less clueless when discussing them. There are also tips on how to take the specimens properly.

What about a child who has suddenly put on weight? What tests will rule out an organic cause? A brief paragraph helpfully distinguishes between tests to find the cause and tests to look for complications, and a table lists what investigations might be worth doing, including the rarities.

A 10 year old comes in with weak, painful limbs and unable to stand. Is it a viral myositis or something more sinister? Difficult to find all the answers in one place, but the tables on "acute generalised weakness" list some of the causes, including some one might not think of, and what tests might exclude them.

My conclusion? This handbook doesn't pretend to be a mini textbook, and within its limits achieves what it sets out to do very well. It's written in an accessible style with lots of quick reference boxes, and a few flow charts and illustrations. The index is somewhat limited and it may take a while to find what one is looking for. Some sort of index allowing cross reference by clinical presentation rather than by system would be a nice addition—for example, "gone off feet": what lists to look at? There are, inevitably, gaps, and bigger texts will be needed at times. That said, it is well suited for constant use by all in wards and clinics, is reasonably priced, and is already very popular.

R Scott-Jupp

In the letter by Murugan et al (Arch Dis Child 2003;88:91) the abbreviation ECG was used in error. Throughout the letter, "echocardiogram" should be used. The journal apologises for the error.

BOOK REVIEW

Handbook of paediatric investigations


Stroobant and Field, perhaps the longest standing editorial partnership in UK paediatrics, have done it again. This time they shrewdly spied a gap in the market for a home grown concise book for everyday use aimed specifically at answering the question: "what tests?"

How best to review it? Why not try "road testing" it on a few problems this general paediatrician happens to have seen on the wards recently.

Firstly, a 12 year old with painless microscopic haematuria. We find the expected exhortation to take a full history and do a thorough examination, followed by a friendly table listing the more straightforward tests, and a discussion of the more fancy ones to be considered. The point about this sort of book, of course, is to supply reminders and hints about what to consider, rather than lists to follow slavishly.

Secondly, a pair of brothers whose bones keep breaking. Are there any tests worth doing to look for osteogenesis imperfecta? Nothing at all on this, but maybe that’s a bit too specialised for this small book.

Next patient, one of those worrying “funny bruising” problems: is it NAI, or is there a rare clotting/platelet disorder? There’s no schema for investigating easy bruising as such, but platelet function and coagulation disorders are discussed. There are useful tables of all the tests haematologists can do, and looking at these enables the paediatrician to sound less clueless when discussing them. There are also tips on how to take the specimens properly.

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