Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

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

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

Comparing the use of this anticholinergic drug intravenously with oral treatment using methyl scopolamine nitrate and similar restricted feeding regime, oral methyl scopolamine nitrate suppressed vomiting more quickly and reliably, was also available for subcutaneous injection if vomiting recurred as size of feeds was increased, and no toxic effects were seen in any dosage used. It would be interesting if these workers would be prepared to try the use of methyl scopolamine nitrate intravenously as pharmacologically this compound was reported to have a 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.

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

We are truly honoured to receive the comments of Dr Corner, who is a pioneering neonatologist and reported medical treatment with methyl scopolamine nitrate for infantile hypertrophic pyloric stenosis (IHPS) in 1955.2 She pointed out that methyl scopolamine might be better than atropine sulfate in terms of effectiveness and side effects. One of the reasons why atropine was used in our study is that methyl scopolamine is not available in our country. Scopolamine butylobromide is an available anticholinergic drug which lacks toxic side effects. However, this agent tastes bitter and is more quickly and reliably, was also available for subcutaneous injection if vomiting recurred as size of feeds was increased, and no toxic effects were seen in any dosage used. It would be interesting if these workers would be prepared to try the use of methyl scopolamine nitrate intravenously as pharmacologically this compound was reported to have a 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.

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

We agree with Dr Corner’s last comment, but believe that intravenous atropine therapy is possible not only in high level paediatric centres, but also in general hospitals where infusion therapy with intravenous atropine injections can be done safely in small infants. Clinical trials are now ongoing to establish more efficient treatment strategy for IHPS with medical and surgical therapy in our country.

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Hypothermia in a child secondary to ibuprofen

A 7 year old girl was admitted with right lower lobe pneumonia. On admission her temperature was 39.7°C. After five hours she received ibuprofen (6 mg/kg). Subsequent to this single dose her temperature decreased to 33.5°C (core temperature 34.9°C) over four hours. On examination her pulse was 90/min, blood pressure 90/50 mm Hg, SaO 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 platelet count 81 × 10⁹; E 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).
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.

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, however, are less susceptible to prevention or intervention. The other known risk factors, hypothermia and over-activity, apparent life-threatening event (ALTE), and a positive test for eyeball compression (MCADD; McKusick 201450) typically account for 8% and adipic 6% 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 hexanoylglutine (2% total organic acids) in the absence of ketonuria have been reported previously in MCADD.

Perforated duodenal ulcer disclosing medium chain acyl-CoA dehydrogenase deficiency

Medium chain acyl-CoA dehydrogenase deficiency (MCADD; McKusick 201450) typically presents in the first two years of life with recurrent episodes of hypoketotic hypoglycaemia, lethargy, coma, or sudden infant death. The trigger may be fasting, intercurrent infections, anaesthesia, or surgery. Incidence in the UK is estimated at 0.45–1/10 000 live births.1 We describe the case of a child who presented with marked encephalopathy unexplained by perforated duodenal ulcer, which led to the diagnosis of MCADD.

A 2 year old girl presented with a three week history of cortical symptoms and a 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 (22–28), calcium 2.4 mmol/l (2.0–2.6), 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 gasin was 20 mV/l (10–100). She was discharged home taking omeprazole.

Urea nitrogen in the serum was 9 mmol/l (2.5–7.5), bicarbonate 15 mmol/l (22–28), plasma sodium 129 mmol/l (135–147), potas-
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 3 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 calculated. Complete blood count, blood gases, and blood chemistry (glucose, total protein, albumin, urea nitrogen, creatinine, uric acid, sodium, chloride, potassium, calcium, phosphates, 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. Recent SDB parameters were found to be associated with worsening insulin resistance independent of obesity in adults. 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.

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 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 methodological 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 (3–5 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 (1.94; 95% CI 1.19 to 3.15; NNT = 13, 95% CI 10 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 (failure to eradicate the organism within 7 days) and relapse (reurrence of symptoms and reinfection within 2 weeks of cessation of treatment after initial bacteriologic cure), whereas Michael et al included frequency of positive urine cultures 0–7 days after cessation of treatment as their primary outcome measure of treatment failure. If reinfections later than 7 days after cessation of treatment occurred more often in recipients of short course treatment, our result would have been similar.

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 one showed 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 duration of treatment is actually greater than 3 days, then the added convenience and cost-savings of “short-course” treatment become marginal. In the absence of appropriately powered RCTs (or meta-analyses) examining outcomes (treatment failure, reinfection, emergence of resistant organisms and cost) with “longer” short course treatment regimens (3, 4, and 5 days), we think that clinicians should continue to treat UTIs in children with at least 7 days of antibiotics.

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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 treatment. In their letter above, Keren and Chan argue that the 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 UTI with standard duration treatment. Instead, we believe that short duration treatment may be used to treat children with lower tract UTI.

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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 vs &gt;7 days</td>
<td>13</td>
<td>RR: 4.26 (95% CI 0.95 to 9.48)</td>
</tr>
<tr>
<td>Keren &amp; Chan, 2002</td>
<td>3 days vs &gt;7–14 days</td>
<td>5</td>
<td>RR: 1.36 (95% CI 0.68 to 2.72)</td>
</tr>
<tr>
<td>Michael et al. 2002</td>
<td>2–4 days vs &gt;7–14 days</td>
<td>8</td>
<td>RR: 1.06 (95% CI 0.64 to 1.76)</td>
</tr>
</tbody>
</table>

*RR, risk difference; CI, confidence intervals; OR, relative risk*
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 (ECC) evaluation of the coronary arteries at presentation and follow up ECC 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.

Tyuoh et al demonstrated, in their multi-institutional review of 536 patients, that no patient with a normal follow up ECC, 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 ECC at 2 weeks to 2 months after the onset of symptoms had subsequent ECCs that revealed coronary artery abnormalities and questioned the value of 6–12 month ECC 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.

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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 preventive management. Although calcium and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density. The role of growth hormone in relation to growth hormone therapy are methodologically flawed because neither have accounted for the change in apparent bone density, which will occur in any child who grows better for any reason when assessed by modalities such as dual energy x ray absorptiometry 1.

As illustrated by another article in the August 2002 edition of Archives, there is a lack of good evidence on which to base much paediatric management and it is imperative that further research, especially randomised controlled trials, is undertaken in the area of prophylaxis against osteoporosis in children with chronic disease on steroids. Paediatric endocrinologists will be familiar with the flurry of small uncontrolled studies undertaken in numerous groups of children with short stature when recombinant growth hormone became available. Many reports of short term improvements in growth velocity have not been supported by long term outcomes in height.

There is a risk that a similar phenomenon will occur with the use of bisphosphonates in children with chronic disease and low bone density without properly designed studies and satisfactory outcome measures.

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

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References
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Sanctions 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 amongst 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 rate of low birth weight (<2500 grams) decreased from 139 in 1960 to 20 in 1989, increased more than five times. Previously it had been estimated that 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 1991.1 Data for 1994–99 showed that mortality among children under 5 years was 131 per 1000 live births, compared with 56 for 1984–89, before sanctions. The reasons for excess deaths are clear-economic collapse with plummeting wages, soaring food prices, poor sanitation, lack of safe water, and inadequate provision of health care.2

The rate of low birth weight (~<2500 grams) which was in the region of 9% in the period 1960–80, increased to 21% in 1994.3 The 1995 Baghdad nutrition survey of children under five years of age showed that the percentage of children below ~25SD in urban Baghdad was 28% for stunting, 29% for underweight, and 12% for wasting.4,5 Children ~2SD (ie ~5th centile), but clearly not underweight, were classified as mild stunting and underweight was defined as <3SD. The notification rate of severe anaemia among children under 5 years was 131 per 10000 population.6

A child psychology study (1991) revealed a level of psychological stress and pathological behaviour that was the highest the authors had seen in 10 years of conflict research. It revealed a highly disturbed population of children. Fear and anxiety were associated with memories of crisis. Seventy five per cent felt sad and unhappy, and four out of five expressed fear of losing their family by death or separation.7

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 banned—from the Gulf states.8

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 and private hospitals experienced serious problems with lighting, cleaning, water supply, and sewage. The population had been burdened by a rapid rise in serious infections, nutritional deficiencies among children and pregnant women, and other treatable conditions for which neither drugs nor operations were available.9 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 and American doctors. This has served to undermine the care of patients, and denies Iraqi doctors the right to share scientific advancement and its benefits.10

In school children aged 6–8 years the prevalence of wasting ranged from 1% in the upper class to 6.7% in rural areas. Similar differences were found for stunting and underweight.11 In a 1994 survey 1.6% of children under 5 years were reported to have night blindness, indicating vitamin A deficiency. A survey of school children in the north in 1994 showed a 30–50% prevalence of goitre, and evidence of iodine deficiency disease elsewhere throughout the country. Rickets has been reported from hospitals at a rate of 3–5 cases per week.12

Diarrhoeal diseases and mortality due to dehydration were well under control prior to the Gulf War; there was a threefold increase from May 1990 to May 1991.13 Other water born infections increased from 1990 to 1999, for example typhoid by 60% and cholera almost fivefold.14 A measles epidemic occurred in 1998.15 There have been alarming rises in cases of malaria and leishmanias. Other infections like tetanus, poliomyelitis, diphtheria, and pertussis all showed an increase after the Gulf War.16

The National Immunisation Programme which had begun in 1985 came to a complete halt between January and April 1991. The percentage of fully immunised one year old children fell from 94 for tuberculosis, 83 for diphtheria, tetanus, and pertussis, 83 for polo, and 82 for measles to 79, 63, 64, and 68 respectively.17

Differential diagnosis of periodic fevers

We just read the short report of Galanakis et al.1 We have been involving in periodic fevers management for many years. At present, PFAPA is an unclear periodic fever, classified among non-hereditary fevers. It is an unclear nosological entity. Pharyngitis, cervical lymphohaemopoiesis and oral aphthae are exclusive findings in PFAPA. Among periodic fevers, cervical lymphoadenopathy and isolated fever can be in children affected by PFAPA. Pharyngitis, oral aphtae, cervical adenopathies, and recurrent fever also characterise Crohn’s disease (CD). Lastly, oral aphtae and recurrent febrile attacks characterise the onset of Behcet’s disease (BD) in children. The efficacy of steroids does not confirm the diagnosis of PFAPA; BD and CD are responsive to steroids, too. The lack of familiar involvement is not a criteria to exclude an inherited disorder, as PFAPA and BD are recessive and BD and CD are multifactorial diseases. Furthermore, the initial clinical picture of these disorders can be atypical and incomplete and can change during the clinical course.

So, considering the provenance of Galanakis’ series (Greece), we not be surprised if some cases had BD or FMF, that will be recognised in the future. Nowadays, with increased diagnostic sensitivity and multi-ethnic societies, periodic fevers are being recognised outside their traditional area of incidence. Close follow up is essential in further years, in these patients. A possible genetic screening for cause gene FMF, HIDS, or immunological assay for HLA B5 could also be useful.

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

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Mechanisms of pulmonary hypertension in Bordetella pertussis

Casano et al describe a case of refractory pulmonary hypertension with severe Bordetella pertussis infection. Their description of the literature is incomplete. Four cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B pertussis.1 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 respiratory failure. The authors state ‘that histological evidence’ was such that extreme leukocytosis predisposes to the formation of lymphocyte aggregates in the pulmonary vasculature and increased pulmonary vascular resistance. This involves the restriction of blood flow in critical beds and the resultant hypoxic vasoconstriction (see figure).
hypoxic vasoconstriction. Therefore Dr Casa-
no’s recommendation for the early use of
pulmonary vasodilators is unlikely to be suf-
ficient in this context. We are assessing the
impact of strategies aimed at reducing
lymphocyte numbers and adhesion in addi-
tion to standard treatments for pulmonary
hypertension.

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in pertussis: does it have a role? Intensive

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 concomi-
tant treatment approach necessary.

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CORRECTIONS

In the paper by Clarkson and Choonara in the
December issue of ADC (Arch Dis Child 2002;87:462–7) the following corrections
have been noted:

Results; first sentence: there were 331
deaths with 390 suspected drugs (not 390 and
389 respectively as stated in the paper).

Results; section “Corticosteroids”: the third
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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).

Figure 1 Scimitar syndrome. Chest x ray
showing a curvilinear density which extends
from the right hilum towards the right
hemidiaphragm which represents the
anomalous pulmonary vein.
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The editors will decide, as before, whether to also publish it in a future paper issue.

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 kappas score below 0.40, but they fail to mention that the majority of other items were below 0.60 (reliability results from *AD* 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 the referred children who were not labelled as having asthma? Finally, it is unclear why in the newborn cohort. Why not compare them to the referred children who were not labelled as

**LETTER**

**Reference**


**Time for a randomised controlled trial of empyema treatment?**

We were interested to read Pierronpoint et al’s short report in October’s edition of *Arch Dis Child*, which concluded that first line treatment of empyema thoracis should be with a pigtail catheter drain and urokinase. However, there is still an ongoing debate as whether empyema thoracis is best treated like this or by open thoracotomy and decortication. It is interesting that the inpatient days for both therapeutic methods have been found to be similar. However, both reports are case series. Is it not time that a randomised control trial was performed comparing the two methods to aid paediatricians in the management of empyema thoracis?

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**References**


**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 NAIT, 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