ORIGINAL ARTICLE

# Severe and unrecognised: pertussis in UK infants

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**Aims:** To diagnose pertussis using culture, polymerase chain reaction, and serology, in children admitted to intensive care units (PICUs) and some paediatric wards in London, and in their household contacts to determine the source of infection.

**Methods:** Infants <5 months old admitted to London PICUs between 1998 and 2000 with respiratory failure, apnoea and/or bradycardia, or acute life threatening episodes (ALTE), and children <15 years admitted to paediatric wards at St Mary's and St George's Hospitals between 1999 and 2000 with lower respiratory tract infection, apnoea, or ALTE were studied.

**Results:** Sixty seven per cent of eligible children (142/212) were recruited; 23% (33/142) had pertussis, 19.8% (25/126) on the PICU and 50% (8/16) on wards. Two died. Only 4% (6/142) were culture positive. Pertussis was clinically suspected on admission in 28% of infants (7/25) on the PICU and 75% (6/8) on the wards. Infants on PICU with pertussis coughed for longer, had apnoeas and whooped more often, and a higher lymphocyte count than infants without pertussis. Pertussis and respiratory syncytial virus (RSV) co-infection was frequent (11/33, 33%). Pertussis was confirmed in 22/33 (67%) of those who were first to become ill in the family. For 14/33 children the source of infection was a parent; for 9/33 the source of pertussis was an older fully vaccinated child in the household.

**Conclusions:** Severe pertussis is under diagnosed. An RSV diagnosis does not exclude pertussis. Future changes to the UK vaccination programme should aim to reduce pertussis transmission to young infants by their parents and older siblings.

Case reports, statutory notifications, and laboratory reports indicate that young infants are continuing to develop pertussis in the UK despite good vaccination coverage, with pertussis vaccine given at 2, 3, and 4 months of age.[1 2] Such traditional methods of ascertainment are known to underestimate the level of disease.[3] Doctors fail to notify even cases of clinically typical pertussis admitted to hospital. General practitioners are often reluctant to carry out appropriate investigations, such as pernasal swabs. Even if appropriate specimens are taken, culture has sensitivity as low as 20–40%,[4 5] because the organism is delicate and the likelihood of culturing it falls if there is any delay in processing specimens. Culture is also more likely to be unsuccessful the longer the time since the onset of illness. Diagnostic sensitivity can be maximised by supplementing culture with polymerase chain reaction (PCR) methods and serology. PCR is more sensitive than culture as it does not require organisms to be viable. Serology is particularly useful in diagnosing infection in patients who have been coughing for some weeks, when both culture and PCR would be anticipated to be unhelpful. Serology has undergone extensive evaluation and standardisation in recent years (ESEN). We applied PCR and serological diagnostic methods to find out the level of pertussis in hospitalised children and their household contacts. We aimed to determine whether parents or siblings infect infants too young to be directly protected by vaccination, in order to inform UK pertussis vaccination policy.

## METHODS

The main study was carried out in paediatric intensive care units (PICU) for two years from November 1998, with a recruitment break for the respiratory syncytial virus (RSV) season (November to February) of the 1999/2000 winter. A smaller study was carried out on paediatric wards starting in July 1999. Eligible infants for the PICU study were under 5 months of age and admitted between November 1998 and October 1999 or March and October 2000 with any of the following:

- Respiratory failure (defined as respiratory insufficiency requiring admission to PICU but excluding persistent pulmonary hypertension of the newborn, meconium aspiration, hyaline membrane disease, and respiratory failure due to known structural airway problem)
- Apnoea and/or bradycardia
- Acute life threatening episode (ALTE).

Eligible children for the ward study were under 15 years and admitted between July 1999 and October 1999 or March and October 2000, with lower respiratory tract infection (excluding asthma and croup), apnoea, or ALTE. All household contacts were included.

The study was carried out with research ethics committee approval from all participating centres and with written informed consent of participants. Research nurses collected information onto a standard questionnaire about the clinical features of illness in cases and contacts and the results of any hospital investigations from parents, clinical notes, and pathology systems. This included all hospital results of standard investigations including pertussis culture. Vaccination status of infants, children, and parents was based on parental reporting. The recommended number of doses of pertussis vaccine for the age of infants given in the national vaccination schedule was compared with the number of doses actually received.

**Abbreviations:** ALTE, acute life threatening episode; ESEN, European Sero-epidemiology Network; PCR, polymerase chain reaction; PICU, paaediatric intensive care unit; PT, pertussis toxin; ptxA, pertussis toxin gene; RSV, respiratory syncytial virus

**Table 1** Laboratory results for children with microbiologically confirmed pertussis (excludes nine epidemiologically linked cases)

| | Culture positive | | Culture negative | | | |
|---|---|---|---|---|---|---|
| PCR | Serology positive | Serology not received | Serology positive | Serology negative | Serology not received | Total |
| PICU | | | | | | |
| Positive | 2 | 0 | 3 | 4 | 4 | 13 |
| Negative | 0 | 0 | 2 | * | * | 2 |
| Not received | 1 | 0 | 1 | * | * | 2 |
| Ward | | | | | | |
| Positive | 1 | 1 | 2 | 0 | 1 | 5 |
| Negative | 0 | 0 | 1 | * | * | 1 |
| Not received | 1 | 0 | 0 | * | * | 1 |
| Total | 5 | 1 | 9 | 4 | 5 | 24 |

*Specimen results would not meet the diagnostic criteria for a case of pertussis and so would not appear in this table.

Research nurses obtained nasopharyngeal aspirate, and acute and convalescent sera from eligible infants on PICU. From the generally older eligible children on the wards, they took pernasal swabs. Pernasal swabs were taken from adult and child household contacts and a single blood specimen from adult contacts only. For mothers only, we obtained stored antenatal serum where available.

A case of pertussis infection was diagnosed if one or more of the following was found:

- *Bordetella pertussis* isolated by culture
- Polymerase chain reaction (PCR) positive for two targets—the pertussis toxin gene (ptxA) and insertion element IS481 sequences
- PCR positive with one target in duplicate samples
- Pertussis toxin (PT) IgG antibody levels greater than 100 U/ml.

If a child did not meet these criteria but one or more of their household contacts had been ill and met the diagnostic criteria for confirmed pertussis, the child was designated an epidemiologically linked case. Infants with pertussis were compared in the analysis with other recruited infants who did not meet the study diagnostic criteria for pertussis.

Household members were regarded as having a confirmed infection with *B pertussis* if they met the criteria above for a case or, in the absence of a clinical specimen, they had an illness compatible with pertussis and were epidemiologically linked to another confirmed case in the family.

The source of infection was defined by the individual in the household with the earliest date of onset of cough (or of admission for two infants with no cough). If household members became ill with dates of onset separated by five days or less, they were considered to be co-primary cases.

For the first year, samples were transported to the laboratory within four hours of collection and processed within one hour of delivery. Culture of these samples was carried out by standard PHLS methods.[6] In the subsequent year, samples were frozen rapidly to −70°C and transported frozen. Pertussis PCR was carried out using single round PCRs to minimise contamination risk, with two independent targets providing mutual confirmation and a range of controls. The ptxA PCR targets the pertussis toxin promoter region yielding a 191bp product and has a reported sensitivity of six bacteria per reaction.[7] The IS481 PCR targets the *B pertussis* insertion sequence IS481, yielding a 146bp product, and has a reported sensitivity of three bacteria per reaction.[8] Control measures included: for sensitivity, titration of a positive control within each run; for specificity, a dummy sample (phosphate buffered saline in place of the clinical sample) per run, and 2–3 water blanks per run. Serology for pertussis toxin (PT) IgG antibody using PT antibody as a marker of recent infection with pertussis was undertaken as previously described.[9] Use of paired and single high titre diagnostic criteria have been evaluated in the European Sero-epidemiology Network (ESEN) project and elsewhere.[10] RSV and influenza detection were carried out by multiplex nested PCR.[11] RSV positive results from nasopharyngeal aspirate which had been taken more than 48 hours after admission were excluded as potentially nosocomial infections. Clinicians were aware that the study was ongoing but laboratory results were not made available in real time.

For the data analysis, groups were compared for categorical variables using $\chi^2$ tests, and for continuous variables by *t* test or by Mann-Whitney test for non-parametric data.

## RESULTS

We recruited 126/183 eligible infants (69%) admitted to the PICUs and 16/29 children (55%) admitted to wards. Nurses obtained 79% specimens within two days of admission, with a median time of one day between admission and sampling. The mean duration of illness prior to taking specimens was 13 days for the ward cases and 18 days for PICU cases ($p = 0.4$). For the household contacts, questionnaire data were available for 282/300 adults (94%) and 186/192 other children in the household (97%). Specimens were obtained from 81% adult and 43% child contacts.

Pertussis PCR was positive in 18/138 (13%) specimens received from recruited children, 16/235 (7%) pernasal swabs from their adult contacts, and 4/85 (5%) pernasal swabs from child contacts. Pertussis was diagnosed according to the case definition in 25/126 (19.8%, 95% confidence interval (CI) 12.9% to 26.8%) infants on PICU and 8/16 (50%, 95% CI 24.7% to 75.3%) children on the wards. Of the 25 cases on the PICU, 17 were laboratory confirmed and eight were epidemiologically linked cases (table 1). Five infants with confirmed pertussis on PICU were diagnosed by pertussis PCR alone, and two were diagnosed on the basis of serology only (table 1). Specimens from 2/126 (2%) PICU infants and one ward infant were culture positive. Of the ward cases, 7/8 were confirmed and one was epidemiologically linked. Of the total of nine epidemiologically linked infants on the PICU and wards, three had equivocal PCR results and negative serology, and six were negative by PCR but no serum was obtained. Pertussis was suspected on admission in 7/25 (28%) infants who met the study criteria on the PICU and 6/8 (75%) infants on the wards. There was a tendency for the ward cases to have more "typical" features (table 2).

Antibiotics had been given prior to admission to seven children with pertussis and 15 with another diagnosis. This had included a macrolide antibiotic in one child with confirmed pertussis and three with other diagnoses. A further five children had specimens taken for the study after starting

**Table 2** Clinical features of children on PICU and wards with pertussis (including linked cases) compared with children on PICU with other diagnoses (categorical variables)

| | Ward: pertussis (n=8) | PICU: pertussis (n=25) | PICU: other diagnoses (n=101) |
|---|---|---|---|
| Cough | 8/8 | 23/25 | 82/101 |
| Paroxysmal | 6/8 | 14/24 | 34/89 |
| Whoop | 4/4 | 9/24 | 3/87 |
| Vomiting | 4/4 | 15/25 | 49/101 |
| Fever | 5/8 | 11/25 | 45/101 |
| Apnoea | 3/8 | 17/25 | 40/100 |
| Cyanosis | 8/8 | 16/25 | 51/100 |
| Pneumonia | 0 | 5/25 | 14/96 |
| Conjunctival haemorrhage | 0 | 1/25 | 3/101 |
| Death | 0 | 2/25 | 6/101 |

in-patient antibiotics. Pertussis was confirmed in none of these children. Four of these five infants received a macrolide antibiotic for one to seven days before the specimens were obtained. All infants with pertussis received antibiotics during the PICU admission, but for 7/25 this did not include a macrolide antibiotic.

Infants admitted to the PICU with pertussis were not more likely to cough than infants with other diagnoses (table 2). However, they were significantly more likely to have had apnoeas ($p = 0.03$), and to whoop ($p < 0.005$). Of infants with an admission diagnosis of apnoea alone, 3/10 (33%) had pertussis. Two infants died, both previously well infants born at full term, compared with six deaths of infants without pertussis. The duration of ventilation, stay on the PICU, and total hospital admission of the 25 infants with pertussis were not significantly different to those with other diagnoses, but they had longer durations of cough and higher lymphocyte counts (table 3). The ward children had a median duration of cough of 12 days (interquartile range (IQR) 7 to 19.3), median lymphocyte count of $15.6 \times 10^9$/l (IQR 2.3 to 7.8), and median length of stay of 7.0 days (IQR 10.5 to 23.5). Similarly to the PICU infants, the ward children with pertussis had a longer median duration of cough ($p = 0.03$), and higher lymphocyte count ($p = 0.004$) than children with other diagnoses, but their overall length of admission was not significantly different ($p = 0.9$).

Most PICU infants with pertussis were unvaccinated because they were too young; 16/25 were less than 2 months old. PICU infants with pertussis were as likely to have received fewer than the recommended number of doses of pertussis vaccine than those without pertussis (5/25 (20%) compared with 33/101 (32.7%); $p = 0.2$). Ward babies were "under vaccinated", with 5/8 (71.4%) with pertussis having received fewer doses than recommended for their age versus 1/8 (12.5%) without pertussis ($p = 0.1$).

In total, 26/289 contacts of recruited PICU infants and 6/39 children recruited on the wards had laboratory confirmed pertussis. The families of a baby with pertussis had a median number of laboratory confirmed cases (in addition to the hospitalised child) of one, with a range of 0–2 cases. Sixty of 111 (54%) contacts of children with confirmed pertussis had a cough versus 144/351 (41%) contacts of children admitted with other diagnoses ($p = 0.02$). Duration of cough was available for 168/204 coughing contacts. The median duration of cough in contacts of pertussis cases was 13.5 days compared with 7.5 days in other contacts ($p = 0.04$). A clinical case definition of 21 or more days coughing plus at least one of paroxysms, whooping, or vomiting was met by 10/111 (9%) contacts of pertussis cases compared with 9/351 (3%) other contacts ($p = 0.006$). Pertussis was confirmed in 6/17 (35%) contacts who met this case definition compared with 26/311 (8%) who did not ($p = 0.003$).

Primary cases (the source of infection) included parents and other children in the households (table 4); 67% of primary cases were laboratory confirmed. The greatest level of non-confirmation occurred when a child was the primary case, largely because we obtained fewer specimens from children. Of seven unconfirmed primary cases in child contacts, no specimens were obtained for four. Two of the three unconfirmed cases with a negative pertussis PCR result met a clinical case definition of coughing for 21 days or more, and coughed for 30 and 60 days respectively. Seven PCR positive contacts and a three contacts with serological evidence of recent infection were asymptomatic prior to and at the time of sampling and did not develop symptoms in the 6–8 weeks before follow up of the infant.

All siblings who were a possible source of infection were reported to be fully vaccinated. In total, 91% of adult contacts (30/33) and 97% of child contacts (29/30) of PICU infants with microbiologically confirmed pertussis reported having been vaccinated for pertussis in the past. This was not significantly lower than reported for contacts of PICU infants without pertussis (adults: 94%, 133/141; children: 95%, 120/127).

RSV co-infection occurred in nine PICU infants with pertussis and two ward children. Infants on PICU with

**Table 3** Clinical features of children on PICU with pertussis (including linked cases) compared with children on PICU with other diagnoses; continuous variables

| | PICU: pertussis (n=25) | | PICU: other diagnoses (n=101) | | Mann-Whitney test |
|---|---|---|---|---|---|
| | Mean | Median | Mean | Median | |
| Duration of cough (days) | 15.2 | 8.5 | 11.0 | 4.0 | 0.003 |
| Lymphocyte count (× $10^9$/l) | 8.8 | 7.8 | 4.5 | 3.5 | 0.003 |
| Duration of ventilation (days) | 4.6 | 3.5 | 4.8 | 3.0 | 0.4 |
| Length of stay on PICU (days) | 5.7 | 4.5 | 8.2 | 4.0 | 0.8 |
| Length of total hospital admission (days) | 15.6 | 13.0 | 15.2 | 10.0 | 0.2 |

**Table 4** Proportion of laboratory confirmed cases among primary (first) cases in families of pertussis cases in PICU and wards

| Relationship | PICU | Ward | Total |
|---|---|---|---|
| Parent | 10/11 | 2/3 | 12/14 |
| Sibling | 0/6 | 2/3 | 2/9 |
| Baby or co-primary | 6/8 | 2/2 | 8/10 |
| Total | 16/25 | 6/8 | 22/33 |

co-infection did not have more severe illness than those with other diagnoses, with no statistically significant difference in duration of ventilation, admission to PICU, or total hospital admission.

## DISCUSSION

Pertussis is a more frequent cause of admission to PICU than generally recognised. Although the numbers in this study are small, for most of the infants the presentation was not typical, the diagnosis was unsuspected, and the case would not have been investigated or notified as pertussis. The combination of pertussis PCR and serology greatly enhanced diagnostic sensitivity in young hospitalised infants, with implications for surveillance and infection control. Hospitalised infants with pertussis including fatal cases are under notified.[3 12] This study shows that, in addition to under notification, under ascertainment is occurring of severely affected infants requiring admission to a PICU. The true number of severe infections, particularly fatal cases, is extremely important in determining the likely benefits of booster vaccinations in modelling different policy options.[13] On the basis of this study, the Health Protection Agency now offers PCR and serology to improve diagnosis of pertussis for such infants, and the results are contributing to enhanced surveillance.[1]

Twenty eight per cent of infants with proven pertussis did not receive a macrolide antibiotic and risked transmitting the infection to staff and other patients. Pertussis is extremely infectious, and a missed diagnosis in PICU may lead to outbreaks among extremely vulnerable infants.

Infants with pertussis were not more ill than those with other diagnoses causing similar clinical syndromes. Co-infection with RSV occurred frequently but did not adversely affect outcome. Samples were collected too close to the point of admission for these co-infections to be nosocomial. Co-infection with pertussis and RSV has been described previously to cause severe infections.[14 15] The different findings in this study may be a chance result because the number of co-infections was small. Alternatively it may reflect greater sensitivity of diagnostic methods for both pertussis and RSV, which means that either or both may be detected outside the window of acute infection. In addition, either agent may influence the transmissibility of the other without influencing disease severity. It is important to recognise co-infections, both for infection control and clinical management. A diagnosis of RSV does not exclude pertussis, and vice versa.

Ten contacts had no symptoms, but *B pertussis* DNA was detected by PCR of nasopharyngeal swab, or PT IgG levels indicated recent infection. There are several possible explanations, including false positive results, "carriage" of *B pertussis*, modification of disease through vaccination, subclinical infection with immunological boosting, and incubating disease. While false positive results are always a risk of PCR, we applied stringent methods and we believe that the diagnostic criteria erred on the side of risking false negative results rather than false positive ones. Although *B pertussis* carriage has not been recognised previously,[16] we may need to change our perspective in the light of the results of highly sensitive diagnostic methods. If carriage does occur, this might explain persistence of the infection in the community despite sustained high vaccination coverage.

Although the primary (source) cases were defined only by date of onset of cough, most were also laboratory confirmed. The role of possible asymptomatic infections in spreading pertussis is unknown, but symptomatic ones are likely to be more important for transmitting the infection through droplets. Parents appear to be the most important source of infection, but siblings also appear to bring pertussis into families. Laboratory confirmation was less frequent in siblings than parents. This was partly because specimens were not obtained. In addition, as all siblings were vaccinated, they may have presented with milder disease that is less likely to be detected by PCR. For the cases where the source of the infection was not identified, the source may include visitors to or contacts outside the household. Nearly all household contacts reported having been vaccinated in the past, and yet infants were still infected, as has also been observed in France.[17]

The study was carried out during the inter-epidemic years of 1998–2000, in which notifications were at the lowest levels on record in the UK. Consequently, the findings represent a minimum estimate of the burden of disease. PCR and serology add considerably to sensitivity of pertussis diagnosis in PICU. These diagnostic methods should be used routinely, at least in this setting. There is considerable under recognised morbidity and mortality from pertussis in infants presenting to PICUs in London, despite high vaccination coverage in their household contacts. The finding that pertussis continues to affect young infants and the degree of its under ascertainment, as well as the source of infections in families, helped to inform the decision to introduce a preschool pertussis booster into the UK vaccination schedule from November 2001.[13] Any future changes to the immunisation programme may need to take into account the fact that in the UK, adults may be transmitting whooping cough to infants.

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Contribution of authors: NS Crowcroft took a lead in design, writing the protocol, securing ethics committee approval, coordinating the study, supervising the research nurses, assisting with recruitment and follow up, carrying out the epidemiological analysis, and writing the paper. R Booy had the idea to do a study, helped with study design implementation, and supervision of research nurses, and contributed to analysis and writing the paper. T Harrison helped with design of the study, was responsible for the bacteriological investigations, and contributed to analysis and writing the paper. L Spicer was the lead research nurse, secured ethics committee approval for the study extension, coordinated the second half of the study, and contributed to data entry, analysis, and writing the paper. J Britto helped with design and implementation of the study, and contributed to analysis. Q Mok helped with implementing the study, and contributed to analysis and writing up. P Heath helped with implementing the study, and contributed to writing up. I Murdoch helped with implementing the study, and contributed ideas and to analysis. M Zambon provided virology investigation, and helped with interpretation of RSV results and writing up. R George helped with design and implementation of the study, and contributed to analysis and writing up. E Miller contributed to protocol writing, analysis, and writing the paper.

Other contributors not listed as authors: Ting Li and Oceanis Tzivra carried out the pertussis laboratory work; Angela French carried out the RSV and influenza PCRs.

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eyesight in peril'' are two of the section headings. The symptoms of attention deficit disorder can be explained almost entirely by excessive or inappropriate use of television and computers, in Large's opinion.

The suggestion that we are being manipulated by advertisers and large television companies, whose main goal is, of course, that the TV is on for a longer rather than a shorter time, is thought provoking. Large proposes that television is, by its very nature, addictive. Advertising directed at children is not illegal in this country, although children younger than 8 are developmentally unable to understand the aims of advertising, simply accepting all claims as true. Children's programmes, such as *Teletubbies*, are marketed as educational when there is no evidence to support the suggestion that they have any beneficial effect on development.

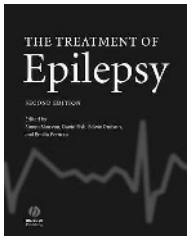
The final section of the book offers parents some practical advice on controlling and monitoring their children's TV and internet use. Large suggests that children younger than 7 should watch no TV, benefiting much more from creative play and adult interaction.

Awareness of the impact of the media on children is steadily increasing. *Set free childhood* presents an extreme view of the possible negative consequences of our current viewing habits. The issue is not as clear cut as Large suggests, but it is time that we take greater interest in the media habits of the children we see, and consider the ways this may be influencing their health or development. A media history may be as necessary a part of every clerking, as the social and family history.

**S Bowring**

## The treatment of epilepsy, 2nd edition

Edited by Simon Shorvon, David Fish, Edwin Dodson, Emilio Perucca. Oxford: Blackwell Publishing, 2004, £150.00 (hardback), pp 952. ISBN 0632060468




With recent studies showing that paediatricians make a diagnostic error in up to one in three children where epilepsy is considered (for none too complacent paediatric neurologists it is one in 15), it is a relief to know that there are texts available that might remedy the situation. As I eagerly turned its leaves, however, the realisation dawned that this book may not offer the whole cure. The first edition preface commended the text to ''specialists in'' … neurology, neurosurgery, psychiatry, paediatrics, alienist medicine (have they landed already and why were we not told?) and learning difficulty. The preface to the second edition outlines the book in its true colours, a résumé of the progress achieved to date by the International League Against Epilepsy (ILAE) captured in its 93 chapters and almost 1000 pages.

I hurried to chapter 5 on ''differential diagnosis of epilepsy'', the key to overturning diagnostic error. The actuality was definitely written for the adult physician. No mention here of blue or white breath holding syncope, masturbation, or simply being ''lost in his own thought'', these often presenting the greatest source of diagnostic difficulty. Refusing to be subdued I advanced quickly to chapter 14 (10 pages) on the management of epilepsy in infants, and chapter 15 (11 pages) on the management of epilepsy in children. Bearing in mind there are a number of thick tomes dedicated to childhood epilepsy, it was not surprising that these 21 pages, though broad in their scope were not comprehensive in their cover.

Despite the preface declaring the book ''patient orientated'' the text concentrates heavily on investigation and drug treatment. The equally important issue of how to give children and their families an understanding of their condition, how to aid adjustment, and to liaise with school are all passed by. Interesting too that a book which says it will be ''patient centred'' uses the word patient and not ''person with'', and where is the chapter by the ''person with''?

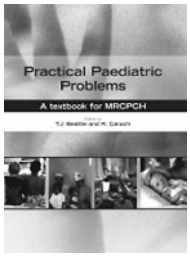
There are useful sections, of course. The chapter on ''definitions and classification'' summarises the latest ILAE classification, though none of the text carries detail on childhood epilepsy syndromes. There are sections on pharmacokinetics, with reference to childhood, and a useful section on the contraceptive pill. Two sections, 48 chapters and 578 pages, are devoted to résumés of 24 drugs and epilepsy surgery selection. Sadly, although the preface promises an ''evidence base'', there is no clear reference to levels of evidence and a dearth of Cochrane reviews (Cochrane does not appear in the index). Drug interactions and side effects (I prefer unwanted effects) are usefully defined.

This wonderful body of knowledge would be an important reference for paediatricians with special responsibility for epilepsy and paediatric neurologists, but will the general paediatrician want this book on the shelf? I suspect not, but it would be good for colleagues with the special responsibility for epilepsy to place the book on the floor just inside the office door. The tome's physical size will cause the general paediatricians to trip up frequently, serving to remind us all that there are frequent pitfalls in the diagnosis and management of epilepsy, and only through good liaison between colleagues and evolving clinical networks will this problem be overcome.

**R W Newton**

## Practical paediatric problems, a textbook for the MRCPCH

Edited by Jim Beattie, Robert Carachi. Hodder Arnold, 2005, £40 (US$70 (approx); €60 (approx)), pp 681. ISBN 0340809329




How large is your desk space? How many of us have placed a dozen new shiny books on our desk just hoping that the information will seep by osmosis into our brains while we snooze and drool over our revision? The MRCPCH examination is a beast that must be grabbed by both horns and beaten into submission by a multitude of weapons consisting of paediatric texts, anatomy and embryology, physiology and biology, not to mention the latest review articles and key paediatric papers. How else to win the battle but to buy a small (expensive) library of textbooks?

This new textbook, the publishers claim, will provide ''*all* the information that the senior house officer and specialist registrar in paediatrics will need during their training and when preparing for the MRCPCH examination''. Quite a claim to make, especially when the editors themselves acknowledge that there will be inevitable gaps in a book of this size. So is this claim justified?

This textbook approaches paediatrics in a structured and comprehensive manner, modelled on the ''core knowledge'' and ''particular problems'' style suggested by the RCPCH publication, *A syllabus and training record for general professional training in paediatrics and child health* (1999). The list of contributors is striking (each acknowledged specialists in their field): 34 in total, including 2 professors and 24 consultants (like reading the dedication page of a textbook, the numbers are important when one is revising). The book covers the expected major systems but also includes chapters on community child health, development and learning difficulties, clinical genetics, acute injuries and ingestion, ophthalmology, surgery, and tropical paediatric medicine.

Each chapter is divided into three elements: firstly covering the background science and relevant investigations critical to diagnosis, secondly the core system problem, and finally a bibliography incorporating suggestions for further reading and key primary papers and review articles. The background science section is excellent. It incorporates relevant embryology, anatomy, biology, and physiology, which really does negate the need to search out those old medical student textbooks to jog one's memory of basic sciences. Included in this section lies succinct summaries of appropriate investigations and their relevance. The core system problems are approached in a systematic and thorough way covering causes, classifications, differentials, clinical features, investigations, therapeutic options, and outcomes. Of particular attraction is the use of short case history boxes, key learning points, flow diagrams, tables, and photographs.

The editors have certainly been brave in trying not only to produce a textbook to cover the recommended RCPCH syllabus but also to help trainees achieve the required standards set out in *A framework of competences for basic specialist training in paediatrics* (2004). Their caveat of the ''inevitable gaps'' has been more than adequately addressed by the encompassing further reading section that includes pertinent and up-to-date book references, papers, reviews, and most importantly, useful websites.

Although this book is primarily aimed at trainees in the lead up to examinations, it is sure to be of value to those specialist registrars beyond this stage. The claim of relevance to all candidates preparing for the examination worldwide certainly does hold true, however some may be confused by the entirety of references to and from the Scottish Executive document of 2004 in the first chapter. This is in relation to *Health for all children* and child surveillance and is obviously due to the striking contributor list being almost exclusively Scottish in origin. Despite this I would urge readers not to be deterred and continue past the first 14 pages to where the Children Act is discussed in

terms of both the English and Welsh Act of 1989 and the Scottish Act of 1995. The rest of the book undoubtedly has worldwide relevance, especially with the chapter on parasites, nematodes, and malnutrition.

This text provides the trainee with a valuable reference source that certainly reinforces the suggestion that learning should be integrated. As to the claim of providing all the information a trainee could need, the authors and editors are to be congratulated on producing concrete foundations for paediatric education and learning. You may only need limited desk space after all, just enough room for this book.

**G Modgil**

## Towards MRCPCH Part II theory examination

Edited by Tapabrata Chatterjee. Hodder Arnold, 2005, £12.99 (US$23 (approx); €20 (approx)), pp 103. ISBN 0340905840

"How many?" I asked. "Oh, at least 3000 multiple-choice questions" said the experienced exam-positive senior registrar. That was the number of multiple-choice questions I should complete to achieve a successful result in my Part I MRCPCH. I never found out whether that meant actual questions or individual stems. Nevertheless, I completed well over this number during revision and did indeed pass. Whether my success had been related to question number or not, I sought to find just how many data interpretation and grey cases one must do in order to pass the next formidable hurdle.

The answer appeared to lie not in quantity but recognising patterns of questioning and developing the art of identifying pertinent information and clues within the questions. The topics chosen by Dr Chatterjee are representative of those that have been asked in the exam over the last five years. Although obviously dependent on candidate recall, the 75 data interpretation questions do appear to be typical of those in the examination. They include the obligatory electrocardiograms, family trees, and audiograms. There is the standard explanation section, which provides crisp answers with few pointers to further study.

The grey case section is superior with a good broad range of 50 cases. Incorporated are the deliberately misleading and irrelevant information typical of grey case questions. The explanations are more detailed, although unlike similar textbooks of its kind it does not include up to date references from textbooks or journal reviews. I particularly liked the tips on how to tackle grey cases and also the identification of the "clue" in many of the explanations.

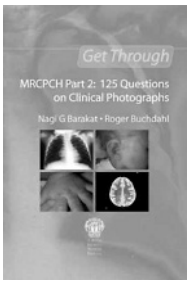
Overall, the grey cases cover the bulk of the diagnoses encountered in everyday paediatric practice. However, there are few esoteric cases (except case 23 where the poor girl with toxic shock syndrome turns mysteriously into a boy via pronoun misuse) and limited neonatal cases. I was pleased to see a case involving "Munchausen syndrome by proxy", although a little disappointed that the explanation did not support the abandoning of this term for the recommended description of "factitious illness by proxy".

Revising for the theory examination can be an arduous task. This book is not especially different to any of the other textbooks covering data interpretation and grey cases, however it remains a useful addition.

**G Modgil**

## Get through MRCPCH Part 2: 125 questions on clinical photographs

Edited by Nagi G Barakat, Roger Buchdahl. The Royal Society of Medicine Press, 2005, £19.95 (US$35 (approx); €30 (approx)) (paperback), pp 196. ISBN 185315685




"One picture is worth ten thousand words" (Frederick R. Barnard, 1921). Current studies of the human memory make a functional division of memory into short-term and long-term memory. Both types store and remember information as "chunks" but there is a distinct difference in the number of these "chunks" that can be retrieved. Short-term memory can retrieve a limited number at any one time (about seven plus or minus two) while long-term memory is not limited to number of recall.

The capacity for recognition of memory for pictures is limitless. Pictures have a direct route to the long-term memory. Pictures themselves make use of a massive range of cortical skills—colour, form, texture, visual rhythm, line dimensions, and especially imagination.

This book is an excellent compilation of clinical paediatric photographs consisting of 125 cases. The questions revolve around high quality paediatric and neonatal images of clinical cases including radiological and ultrasonographic scans. The question formats include the extended matching and "best of five" styles, which were introduced as part of the new examination in 2002. The explanations are concise and comprehensive, based on standard textbooks, which are referenced.

It was refreshing to see the breadth of cases covered from normal variants (answer: "do nothing") to the expected complex paediatric syndromes (answer: "refer to specialist"). I was encouraged to see cases covering child protection (both as the answers and as considered differentials) as well as, more unusually, cases touching on the issues of withholding and withdrawing life sustaining treatment in children. Acute APLS type situations are also encompassed. The most striking element of this book is the true to life way that each case is handled in terms of the presenting features, investigations, treatment, and further management. This surely reflects the fact that the images are derived from the authors' wealth of clinical experience and obvious strong desire to teach.

I remain in strong agreement with the comments in the foreword that this book will remain an invaluable reference for those that have already attained the MRCPCH examination as well as those still in training. These pictures are certainly worth far more than ten thousand words.

**G Modgil**

# CORRECTION

N S Crowcroft, R Booy, T Harrison, *et al*. Severe and unrecognised: pertussis in UK infants (*Arch Dis Child* 2003;**88**: 802–6).

In the process of carrying out further analysis of the data from this study and to examine the role of respiratory syncytial virus (RSV) the author uncovered a single data entry error in the date of onset of disease in one contact of a case when looking back at the original questionnaires. Unfortunately this changes the order of cases in one family, which affects table 4 (the corrected table 4 is shown below).

The penultimate and last sentences of the Results section of the Abstract should have read:

Pertussis was confirmed in 21/33 (64%) of those who were first to become ill in the family. For 13/33 children the source of infection was a parent; for 10/33 the source of pertussis was an older fully vaccinated child in the household.

In the third to last paragraph of the Results section the first sentence should read:

Primary cases (the source of infection) included parents and other children in the households (table 4); 64% of primary cases were laboratory confirmed.

The error has no implications for the methods, discussion or conclusions of the paper.

**Table 4** Proportion of laboratory confirmed cases amongst primary (first) cases in families of pertussis cases in PICU and wards

| Relationship | PICU | Ward | Total |
|---|---|---|---|
| Parent | 9/10 | 2/3 | 11/13 |
| Sibling | 0/7 | 2/3 | 2/10 |
| Baby or co-primary | 6/8 | 2/2 | 8/10 |
| Total | 15/25 | 6/8 | 21/33 |

## Pre-published book reviews

Book reviews that have been accepted for publication but have not yet been published in the print journal can be viewed online at http://adc.bmjjournals.com/misc/bookreviews.shtml