Mind the gap: the integrated management of cognitive and behavioural problems in childhood epilepsy

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Speakers
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Professor Chris Gillberg, Professor of Child and Adolescent Psychiatry, University of Gothenburg, Sweden
Professor Eric Taylor, Emeritus Professor of Child and Adolescent Psychiatry, King's College London Institute of Psychiatry
Dr Pamela Crawford, Consultant Neurologist and Director, Special Centre for Epilepsy, York
Professor Jonathan Green, Professor of Child and Adolescent Psychiatry, University of Manchester; and Honorary Consultant Psychiatrist, Royal Manchester Children’s Hospital
Professor David Taylor, Emeritus Foundation Professor of Child and Adolescent Psychiatry, University of Manchester
Dr Ingrid Olsson, Pediatric Neurologist and Associate Professor, Queen Silvia Children’s Hospital, Sahlgrenska University, Göteborg, Sweden
Professor Faraneh Vargha-Khadem, Professor of Developmental Cognitive Neuroscience, UCL Institute of Child Health, London
Dr Isobel Heyman, Consultant Child and Adolescent Psychiatrist, Maudsley Hospital and Great Ormond Street Hospital, London; and Honorary Senior Lecturer, Institute of Psychiatry and Institute of Child Health, London
Dr Rod Scott, Reader in Paediatric Neurosciences and Consultant Paediatric Neurologist, UCL Institute of Child Health, London; Research Associate Professor, Dartmouth Medical School, Hanover, New Hampshire, USA; and Young Epilepsy

Medical writer in attendance: Steve Chaplin
**Introduction**

This *Progress in Neurology and Psychiatry* report provides a summary of the second ‘Mind the gap’ meeting, which took place at the UCL Institute of Child Health, London, on 3 February 2011.

The first ‘Mind the gap’ meeting, held in March 2009, brought together neurologists, psychiatrists, representatives of the social services, carers and nurses to discuss how services for children with epilepsy and psychiatric comorbidities could work better. Introducing the second meeting, Professor Brian Neville said the effort to bring proper care to children with epilepsy and their families would continue. He hoped the day’s discussions would help to develop an appropriate training programme to deliver improved care.

Professor Neville said that the management of epilepsy in children is organised according to the model for adult epilepsy—that is, it focuses on seizure disorders. At least half of children with epilepsy also have cognitive and behavioural disorders, but child psychiatry is separated from paediatric services and there is a lack of psychology services for children. There is also a lack of home support for children and their families.

This model does not meet children’s needs. We must recognise that many children with epilepsy have cognitive impairments and also multiple behavioural and emotional difficulties, such as attention-deficit hyperactivity disorder (ADHD), autism spectrum disorders (ASD), depression, anxiety and tics.

These conditions are hard to diagnose and manage in children who have several comorbidities. Management guidelines from NICE do not include the identification or management of cognitive and behavioural impairments and, in delivering the medical model of care, doctors tend to select the problems they can do something about, Professor Neville continued. This medical model is unpopular with schools and social services, and does not meet the needs of families. It seems common sense that a composite approach is needed— it would be more efficient and less expensive— but what keeps us from implementing the obvious solutions?
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Biological basis of autism and ADHD

Autism is not a single entity but a group of disorders characterised by a lack of social instinct (a deficit in intuitive empathy, intuitive and active shared attention, and spontaneous intersubjectivity), said Professor Chris Gillberg. These traits persist from childhood into adulthood.

Professor Gillberg outlined the ESSENCE concept – Early Symptomatic Syndromes Eliciting Neuropsychiatric/Neurodevelopmental Clinical Examinations – as a predictor of disorders of empathy and conscience. These include ASD, ADHD, bipolar disorder, tics, language impairment, learning disability, developmental co-ordination disorders, behavioural phenotype syndromes and epilepsy syndromes. ESSENCE is not intended for the diagnostic manuals, he emphasised, but as a means of recognising that many disorders cannot be differentiated in early life because they have significant symptomatic and biological overlap. Children often present before age four years and the diagnosis they receive depends on which specialist they see (Table 1).

As an example, about 6% of all children screen positive for delayed language development at two and a half years. Of these, 70% have a ‘neuropsychiatric/neurodevelopmental’ diagnosis such as ADHD, ASD or learning disability (with clinical impairment) at age seven years, and virtually all have remaining speech/language problems. This means that all children with speech or language impairment at two and a half years need to be followed up carefully, and the vast majority will need additional services.

ASD is strongly associated with epilepsy: 45% of individuals with classic autism have or have had epilepsy by age 45 years, and 43–78% of individuals with autism have epileptogenic discharge in their EEG during sleep. Brain scanning research has demonstrated that children with autism have midtemporal (and midfrontal in most cases) single photon emission computed tomography (SPECT)-verified reduction in cerebral blood flow, whether or not they have epilepsy. Children with Landau-Kleffner syndrome have very high rates of autistic behaviour.

Professor Gillberg summarised the biological factors underlying ASD. The autisms are a group of multifactorially determined conditions. Synapse and clock genes probably play a major role (and often affect synapse formation and function, e.g. neurexin, neurexin, Shank 2 and 3, and melatonin genes), but environmental factors contribute to the clinical presentation and may themselves cause ASD. There is decreased and abnormal intra- and internetwork connectivity. The medial prefrontal, medial temporal, brainstem and cerebellar regions of the central nervous system are almost always affected, singly or in various combinations. These areas constitute a functional network – the ‘default network’ – which appears to be critically differentially functioning in ASD.

### Table 1. ESSENCE - where do we find the cases?

<table>
<thead>
<tr>
<th>Disorder of</th>
<th>Presenting at</th>
</tr>
</thead>
<tbody>
<tr>
<td>General development</td>
<td>Paediatrics, GP</td>
</tr>
<tr>
<td>Motor control/perception-sensory</td>
<td>Occupational therapy, paediatrics, GP</td>
</tr>
<tr>
<td>Communication/language</td>
<td>Speech and language therapy, CAMHS, paediatrics, neurology</td>
</tr>
<tr>
<td>Activity/impulsivity</td>
<td>CAMHS, paediatrics</td>
</tr>
<tr>
<td>Attention</td>
<td>CAMHS, neurology, psychology</td>
</tr>
<tr>
<td>Social interaction/reciprocity</td>
<td>CAMHS, autism centres</td>
</tr>
<tr>
<td>Behaviour</td>
<td>CAMHS, paediatrics</td>
</tr>
<tr>
<td>Mood swings</td>
<td>CAMHS</td>
</tr>
<tr>
<td>Sleep</td>
<td>Paediatrics, GP</td>
</tr>
<tr>
<td>Feeding</td>
<td>Paediatrics, GP, CAMHS</td>
</tr>
<tr>
<td>Any</td>
<td>Child health visiting, well-baby clinics, preschools, schools, and newborn CAMHS, children’s acute mental health service.</td>
</tr>
</tbody>
</table>
ADHD includes a largely genetic constellation of symptoms that can also be produced by environmental factors causing brain dysfunction. Most of the evidence implicates the frontostriatal (particularly the nucleus accumbens) and brainstem cerebellar neural circuitries.

In summary, Professor Gillberg said that children who present with major and impairing ESSENCE symptoms need to be followed up: most will need an intervention and many will have epilepsy or develop epilepsy with time. Clinicians should search for ESSENCE symptoms in every child they see in the longer term. Health visitors, preschool and school teachers, and social workers need to be very well educated about this concept. He hoped that the future would bring diagnoses based on genetics, networks and dimensional behaviour problems rather than categorically distinct syndromes.

ADHD and epilepsy: assessment and intervention

About one-third of children with epilepsy have ADHD, predominantly of the inattentive type, and it is often the most treatable component of a complex disorder, said Professor Eric Taylor. The assessment of ADHD involves detecting the core features (impulsiveness, inattention, restlessness) and making a differential diagnosis in the presence of associated problems. The developmental routes involved, and the causes, moderators and mediators, will influence management planning.

Rating scales can be useful for screening for ADHD, but the results from these need to be incorporated into a clinical assessment.

**Box 1. Treatment targets in children with epilepsy and ADHD**

- Reduce core symptoms
- Alleviate associated problems
- Promote realistic learning goals
- Reduce rejection by others
- Promote relationships with peers, family and at school
- Understand disability, value self

The cornerstone of assessment is therefore the clinical interview, and in particular the behavioural/observational interview. This should include assessment of what activities the child does, the time spent on them (excluding computer games and, in children under eight years, television), the amount of movement when the child would be expected to be calm, and their organisation of activities. Professor Taylor noted that parents’ perceptions of abnormality vary greatly, but they are reliable reporters of the child’s behaviour. The clinical interview for preschool children includes assessment of time on task and activity changes, waiting for reward and social disinhibition; for older children, it involves assessment of premature decisions in test situations, difficulty in slowing tempo and lack of reserve and discretion.

He warned that the novelty of the clinical interview and monitoring can markedly reduce hyperactivity, giving rise to false negatives.

Professor Taylor emphasised that diagnosing ADHD is about recognising a pattern of behaviour, not indicating a cause; the response to treatment does not depend on the aetiology. The diagnosis of ADHD is only the start of the assessment process, he continued. In addition to assessing epilepsy, every child should be monitored to assess their development, psychosocial stress, hearing, possible medication side-effects and comorbidities.

These assessments should be used to plan a programme of behaviour modification (parent training, social skills learning, classroom management), anticonvulsant medication (dose adjustment and minimising adverse effects), relationship education, school liaison and medication to reduce hyperactivity. Psychological therapies should include parent training and social skills; family education and school liaison are essential. The targets of treatment are listed in Box 1.

Professor Taylor advised that the drugs of choice for ADHD are those licensed for this indication (methylphenidate, dexamfetamine and atomoxetine). Others that are expected to be licensed in the near future include guanfacine, dexamfetamine complex, risperidone (for irritability) and modafinil. There is some evidence to support the use of imipramine, clonidine and bupropion, but the evidence for moclobemide and venlafaxine is weak. Other drugs may be indicated for comorbidities (e.g. a selective serotonin reuptake inhibitor for affective disorder/ anxiety or an atypical antipsychotic for schizophrenia disorder). The risk of adverse effects and drug interactions should be considered.

There are still avoidable gaps in management, Professor Taylor concluded. NICE guidelines are not fully implemented and there is large variation between services in the use of medication and psychological interventions. Interdisciplinary suspicion persists, the education system is not yet fully on board, and media accounts are sometimes untrustworthy. As a result, a treatable condition often goes untreated.
Generic substitution
A generic preparation is similar to, but not quite the same as, the original brand or other generic preparations, and the difference may be sufficient to threaten epilepsy control in some patients, said Dr Pamela Crawford.

The European Medicines Agency assesses the bioequivalence of a brand and a generic product on pharmacokinetic parameters: area under the curve, peak concentration and time of peak concentration must be within 80–125% (90% confidence limit) of the mean for the original brand, based on single-dose studies in 12–36 healthy volunteers. The differences may be clinically significant in some patients at the extremes of the permitted range, particularly for different generic products.

Although there are theoretical advantages to generic prescribing of antiepileptic drugs (AEDs), encouraging rational prescribing, Dr Crawford said the principal reason is to reduce costs. But, in addition to possible bioin-equivalence, the disadvantages to patients include confusion over different names or appearance; the generic name is often longer and harder to remember than the brand name; there are differences in excipients and colourants; and the source of a generic product cannot always be identified.

Dr Crawford emphasised that treatment with AEDs is carefully titrated to control seizures and to minimise the risk of adverse effects. About 30% of patients are on polytherapy and there is a high risk of clinically significant drug interactions. In a minority of patients, the difference in bioavailability between two formulations may increase the risk of adverse effects and breakthrough or more frequent seizures.

Conversely, of course, it could improve seizure control. Phenytoin’s non-linear pharmacokinetics, dose-dependent metabolism and narrow therapeutic range make it especially sensitive when generically substituted. A 20% increase in absorption can lead to a two- to three-fold increase in serum levels. A comparison of several generic tablet formulations of phenytoin found that bioavailability relative to the brand leader ranged from 76–107%, whereas that of an infant formulation was 121%.6

There is good evidence that differences in the bioequivalence of phenytoin formulations is clinically significant. In Australia, a change of excipient from calcium sulphate dihydrate to lactose in one brand led to increased bioavailability and an outbreak of toxicity.7 Reductions in serum levels have followed a switch from brand to generic (causing increased seizures) and from generic to branded phenytoin.8,9

Single-dose studies of five formulations of carbamazepine have found a 1.5-fold difference in maximum serum concentration and a seven-fold difference in time to maximum serum concentration. There are no differences in bioavailability, but brands with rapid absorption are associated with an increased risk of adverse effects. There are anecdotal reports of bioinequivalence between different formulations of valproate, lamotrigine and gabapentin.

The Yorkshire Community Study sought to determine the impact of changes in AED formulation in patients with epilepsy.10 Of 1333 patients who responded to a postal questionnaire, 18.7% reported a change in AED formulation. Of these, 29.5% reported problems including breakthrough seizures, more frequent seizures, or feeling worse or more adverse effects.

Following the introduction of generic substitution in Canada, 27.5% of patients treated with lamotrigine and switched to the generic product subsequently reverted to the brand (compared with 7.7–9.1% for drugs in other therapeutic areas).11 As a result, the anticipated saving from generic substitution was below expectations.12 It was estimated that the cost of increased consultations and hospital admissions during the period of generic substitution was one-third higher compared with use of the brand.13

Dr Crawford commended the recommendations of the Italian League Against Epilepsy (Box 2) as ‘very sensible’. She pointed out that the consequences of seizure breakthrough are profound and the savings from generic substitution are small. It is not possible to ensure continuity of supply with generic AEDs: patients should therefore be maintained on the same brand of AED.

Box 2. Recommendations of the Italian League Against Epilepsy on generic prescribing of antiepileptic drugs14

- Generic substitution not recommended for seizure-free patients
- Switches between generic brands should be avoided
- Sustained-release formulations should not be used interchangeably with immediate-release and generic formulations
- Patients should be informed

Transition to young/adulthood services
Adult epilepsy services are very different from those for children...
and generally less responsive, Dr Crawford said. Service provision varies between different areas: many are poorly served, resources are scarce, and some neurologists are not interested in epilepsy management. Patients are seen less frequently and most follow-up is carried out by specialist nurses; patients who are seizure-free are followed up in general practice.

The transition between children’s and adult services therefore requires a co-ordination between the lead paediatrician and the epilepsy specialist, and liaison with epilepsy specialist nurses. Each patient is different. Learning disability teams should be involved when necessary because adult services often know little about appropriate management. Girls with primary generalised epilepsy are at particular risk during the transition from children’s to adult services because of the need to ensure appropriate contraception and treatment during pregnancy.

Communication-focused interventions for autism

The results of the Pre-school Autism Communication Trial (PACT), the first large randomised trial of an early psychosocial intervention, were presented by Professor Jonathan Green. He said the rationale for this approach is that 80% of a preschool child’s communication is with his or her parents and, in families with a child with autism, communication is abnormal and the interactions between parent and child are unbalanced.

He noted that social brain development is in dynamic interaction with the environment and, by exploiting the plasticity of the developing brain, modification of an infant’s environment may reduce the interaction impairment associated with atypical development. PACT tested the hypothesis that a targeted, parent-mediated, video-aided intervention would enhance parental communicative responsiveness. In turn, this would elicit improved social responses/communication initiations from the child, reduce abnormality in communication and generalise to a reduction in autism social/communicative symptoms.

A total of 152 children, aged two years to four years and eleven months, were randomised to the PACT intervention plus treatment as usual, or solely treatment as usual, at three specialist centres in the UK. The primary outcome was severity of autism symptoms at 13 months, measured by social communication items from the Autism Diagnostic Observation Schedule – Generic (ADOS-G); secondary outcomes included parent–child interaction, language and social adaptation, and (non-blinded) parent-assessed language and early social communicative development.

After 13 months, there was no significant difference between the two groups in the primary outcome or parent–child interactions, although the non-blinded parent-reported endpoints improved significantly. Dr Green concluded that the results of PACT were consistent with other evidence that communication could be improved, but there is no generalisation to a reduction in symptoms.

Cognitive and behavioural aspects of absence epilepsy

Absence epilepsy accounts for 8–10% of epilepsies up to age 16 years. It is more common in girls than boys and has a strong genetic component.

Reviewing the evidence of the impact of absence epilepsy on children, Dr Ingrid Olsson said that long-term follow-up of children with childhood absence epilepsy has shown that 65% are in remission after 12–31 years; cognitive difficulty at diagnosis
suggests a poor prognosis. In young adults, absence epilepsy was associated with vocational underachievement and social isolation; it was considered to have a high impact on life, even among individuals who were seizure-free.

Absence epilepsy in children is associated with lower general cognitive functioning, lower scores on visuospatial skills, and memory disturbances. Deficits in cognitive function affect attention, verbal learning and memory, word fluency and controlled fine motor responses. Psychiatric comorbidity appears to be common, but definitive data are lacking. Dr Olsson was critical of a widely quoted study reporting a high prevalence of subtle cognitive defects, linguistic difficulties and psychiatric disorders, including anxiety, in children with absence epilepsy. She questioned the inclusion criteria with a bias towards not optimally treated, and pointed out that anxiety could be due to other factors such as persisting absence seizures. The authors concluded that few children had received any interventions. A study from Gothenburg of 20 six-year-old children with newly diagnosed epilepsy included five with absence epilepsy and, compared with controls, reported lower performance IQ and cognitive and linguistic dysfunction. Dysfunction of executive and related processes has also been reported compared with children with diabetes and healthy controls. Parental reports suggest higher rates of hyperactivity and inattention compared with controls.

Dr Olsson concluded that children with absence epilepsy have cognitive and behavioural problems, but whether this is due to frequent absences, AEDs or the underlying cerebral dysfunction is unknown. Whatever the cause, she said, these children need a multidisciplinary assessment and support at school. Only a minority receive this today.

Cognitive outcomes in paediatric epilepsy

Recent unpublished studies of the cognitive profiles of children with epilepsy undergoing neurosurgery were described by Professor Faraneh Vargha-Khadem. The first study involved assessment of preoperative cognitive function and educational status in 540 children with intractable focal epilepsy, and confirmation of a structural abnormality by MRI. The aim was to measure IQ, memory and language skills in relation to the seizure focus and the pathology of the lesion.

The mean age at onset of epilepsy was 3.7 years and the mean number of seizures per month was 171. The mean age at assessment was 7.5 years. There were many different pathologies and variation in the site of the lesion, although the majority were temporal (42%) or multilobal (28%). Most children had daily seizures.

Professor Vargha-Khadem presented data only for the children able to complete tests of intelligence, memory, literacy and numeracy. In 39% of children, IQ was within the normal range (80–103). Children with an IQ in the range 40–69 (38%) included the largest proportion with multilobal epilepsy, whereas IQ was highest in those with frontal, temporal or parietal-occipital lesions. Children with temporal lesions scored lower and those with frontal, parietal-occipital or multilobal lesions scored higher on verbal than non-verbal IQ. This, she said, suggested a selective problem with verbal intelligence in temporal lobe epilepsy; this was particularly associated with left-hemisphere lesions.

Moving to the relationship between language (as measured by verbal IQ) and memory, the memory quotient was lowest in the multilobal group. Memory quotient was slightly higher than full-scale IQ in children with frontal, parietal-occipital or multilobal lesions, but that relationship...
was reversed in children with temporal lesions. By contrast, there was no difference by lesion site or lateralisation in the relationship between IQ score, literacy and numeracy skills. IQ score was significantly lower than academic attainment, showing that children were able to achieve in spelling, comprehension and mathematical reasoning despite lower intellectual performance in this test.

Professor Vargha-Khadem concluded that the best way to determine the effects of temporal lobe epilepsy in children is to measure memory – academic attainment and IQ (which reflects language ability) are less useful.

The second study assessed seizure and cognitive outcomes after temporal lobectomy in 105 children between 1992 and 2006 (61 with left-sided lesions). In this group, the mean age at onset of seizures was 45 months and the mean duration of epilepsy until surgery was 90 months. Follow-up was available for 63 children: 72% of children were considered improved after one year and 71% after three years.

There was little difference in IQ outcome by lateralisation or pathology. Regression analysis demonstrated that, although preoperative IQ and preoperative duration of epilepsy were significantly associated with postoperative full-scale IQ, the strongest prognostic factor was age at onset of epilepsy.

Psychiatry within an epilepsy surgery programme

Discussing her experience as a psychiatrist in the epilepsy surgery programme at Great Ormond Street Hospital, Dr Isobel Heyman noted that up to three-quarters of children with severe epilepsy have a range of psychiatric disorders, notably autism spectrum disorders (Figure 1). Their epilepsy has far-reaching effects on behaviour, emotions, cognition and physical function (Figure 2). Psychological problems may cause greater distress or impairment than the epilepsy itself. Untreated, they are very persistent, but they often improve substantially with treatment.

Children coming into an epilepsy surgery programme

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therefore need a range of services and careful functional evaluation. Part of this process involves tackling unrealistic expectations that surgery will greatly improve cognitive or behavioural function.

It is difficult to predict psychiatric outcome after surgery. In one study of 60 children with temporal lobe epilepsy, 72% had a psychiatric diagnosis before surgery and 72% did so afterwards (although some had improved and others were newly diagnosed).\textsuperscript{28} Dr Heyman noted that, in her experience, very few children get worse after surgery. Quality of life may improve maximally in children who become seizure-free,\textsuperscript{29} and the benefits may be apparent in physical but not cognitive, social or behavioural functioning.\textsuperscript{30}

An uncontrolled case series of 13 children has demonstrated postoperative reductions in the severity and impact of emotional and behavioural symptoms during follow-up of up to eight and a half years (Figure 3).\textsuperscript{31} By contrast, an unpublished case note review of children with extra-temporal lobe epilepsy has shown no group effect of surgery on psychiatric disorders. In this study, several children with behaviour typically associated with a frontal lobe lesion (dissociation, aggression, lack of motivation) developed a new psychiatric disorder (particularly ADHD). Overall, these findings suggest that the decision to offer surgery should not be influenced by the preoperative psychiatric diagnosis.

Dr Heyman then discussed the management of children with psychogenic non-epileptic seizures. These paroxysmal episodes resemble epileptic seizures but do not have an organic aetiology. They are involuntary and are not wilful attempts to deceive. Studies of case series suggest that about one-third of affected children also have epilepsy, and a stressor provoking the ‘seizure’, such as school difficulties, abuse, family problems or anxiety, can be identified in almost all cases.\textsuperscript{32,33}

Dr Heyman said she had encountered several children with psychogenic non-epileptic seizures in the surgery programme, but the true prevalence is unknown. This condition may contribute to preoperative impairment in children who also have epileptic seizures. Further studies are needed to

**Figure 3.** Improvement in the severity and impact of behavioural and emotional symptoms after surgery (measured by the Strengths and Difficulties Questionnaire). Reproduced with permission from Hannan S, Cross JH, Scott RC, et al. The effects of epilepsy surgery on emotions, behavior, and psychosocial impairment in children and adolescents with drug-resistant epilepsy: a prospective study. Epilepsy Behav 2009;15:318–24. Copyright 2009 Elsevier\textsuperscript{31}
determine outcomes after surgery, and to discover whether non-epileptic seizures develop for the first time postoperatively.

Psychological adjustment varies after surgery. There is good evidence that seizure freedom is the best correlate of a good psychological outcome, but there is a subgroup of individuals who are seizure-free but having difficulty coming to terms with being well. There may be several reasons for this, but intense fear of seizure recurrence and the ‘burden of normality’ are important.34–37 Postoperative adjustment may therefore be slow, Dr Heyman concluded.

Towards a comprehensive childhood epilepsy service

We should not lose sight of the fact that cognitive and behavioural impairments significantly affect the quality of life of children with epilepsy, said Dr Rod Scott, and up to 60% of children with idiopathic epilepsy may be underachieving at school.

The Isle of Wight study found that one-third of children with epilepsy have a psychiatric diagnosis, with emotional problems, conduct disorders predominating, with over-representation of ASD and ADHD.38 It is tempting to think that these problems are due to the effects of epilepsy on a normal brain, but it is more likely that both epilepsy and adverse outcomes arise from an abnormal brain, and there is an interaction between epilepsy and the other events. This approach suggests that treating only the epilepsy will not improve other outcomes.

Dr Scott described an animal model of epilepsy in which environmental enrichment enhanced learning to an extent that is not seen with AEDs. This suggests, he said, that the environment in which children with epilepsy grow up should be modified to improve outcomes and that schools and social sciences have a lot to contribute to their care.

Professor Neville concluded the meeting by proposing a model for a comprehensive childhood epilepsy service. A multidisciplinary network should co-ordinate resources and adopt a uniform system of data collation, appraisal and annual review. Shared guidelines should be adopted covering diagnosis, treatment, assessment and audit. Psychologists should have a central role in secondary, tertiary and specialist services. Schools should be supported to play a greater role in management. Training in paediatrics and psychiatry should be improved and re-alignment of the specialties should be considered. A service should be developed for the assessment and treatment of young children, and all children should have access to a multidisciplinary team and social services. The financial and resource implications of these proposals should now be assessed.

References
Mind the Gap III

When: Thursday 8 March 2012
Time: 08:30–17:30
Topic: Very difficult behaviour in children with epilepsy
Venue: Kennedy Lecture Theatre, UCL Institute of Child Health, Guilford Street, London WC1H 1EH
Cost: £35.00

The speakers include Christopher Gillberg, Rod Scott, Eric Taylor, Philippa Russell, Heather McAlister, Mike Kerr and Brian Neville

To register an interest, please contact Young Epilepsy’s information service on 01342 832243 ext 296/424 or e-mail epilepsytraining@youngepilepsy.org.uk

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