Attention deficit hyperactivity disorder

Attention deficit hyperactivity disorder is a highly heritable medical condition which mainly affects school-aged children although it is increasingly being recognized in adults. The exact cause remains unknown, but the condition responds well to evidence-based interventions.

Attention deficit hyperactivity disorder is a common neurobehavioural developmental disorder of childhood (Faraone et al, 2003). Core symptoms include hyperactivity, inattention and impulsivity. The terms 'attention deficit', 'attention deficit hyperactivity disorder', 'hyperkinetic disorder' and 'hyperactivity' are often used interchangeably by professionals to describe children who are overactive and have difficulty concentrating and controlling their impulses. Although hyperactivity, impulsivity and inattention are all attributes of childhood behaviour, they are considered to be features of a disorder when they are severe, developmentally inappropriate, pervasive in nature and cause functional impairment (Overmeyer and Taylor, 1999). This article defines the disorder and examines its origins from a historical perspective. The research literature relating to prevalence and causation (i.e. genetics, biological factors and psychosocial factors) is then summarized and current recommendations for the assessment and treatment of the disorder are briefly outlined.

Definitions

The *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM-IV) (American Psychiatric Association, 1994) recognizes attention deficit hyperactivity disorder as a mental disorder. In Europe, a similar constellation of symptoms is termed hyperkinetic disorder and is classified under 'Behavioural and Emotional Disorders' in the *International Classification of Diseases* (ICD-10) (World Health Organization, 1992). According to ICD-10, to make the diagnosis of hyperkinetic disorder, the onset of symptoms should be before the age of 7 years, the three core symptoms of inattention, hyperactivity and impulsivity need to have been present for at least 6 months, and the symptoms should be pervasive in nature. According to DSM-IV, however, attention deficit hyperactivity disorder can be diagnosed if either the inattention or hyperactivity-impulsivity symptoms or both have been present for at least 6 months but, again, the onset of symptoms should be before the age of 7 years and there must be evidence of impairment in two or more settings.

In DSM-IV, attention deficit hyperactivity disorder is classified into three subtypes: (1) predominantly inattentive type, (2) predominantly hyperactive-impulsive type and (3) combined type (American Psychiatric Association, 1994). Both diagnostic systems require the exclusion of other mental disorders which might better account for the symptoms, e.g. schizophrenia, other psychotic disorders, or anxiety and mood disorders.

According to the National Institute for Health and Clinical Excellence (2008), 'moderate ADHD [attention deficit hyperactivity disorder] in children and young people is taken to be present when the symptoms of hyperactivity/impulsivity and/or inattention, or all three, occur together, and are associated with at least moderate impairment, which should be present in multiple settings (e.g. home and school) and in multiple domains,' e.g. achievements at school and forming positive relationships with family and peers, and 'Severe ADHD corresponds approximately to the ICD-10 diagnosis of hyperkinetic disorder... when hyperactivity, impulsivity and inattention are all present in multiple settings and when impairment is severe (i.e. affects multiple domains in multiple settings).'

Historical view

Attention deficit hyperactivity disorder was probably first described in the medical literature in 1798 by Dr Alexander Crichton who named it 'mental restlessness' (Palmer and Finger, 2001). In 1845, Dr Heinrich Hoffman wrote a story for his son in which he described 'Fidgity Philip' who presented with symptoms of hyperactivity, inattention and impulsivity (Thome and Jacobs, 2004). In 1902, Sir George Fredrick Still published a series of Goulstonian lectures describing a group of 43 children who had significant problems with sustained attention and self-regulation, who were often aggressive, defiant and resistant to discipline, excessively emotional or passionate and could not learn from the consequences of their actions, although their intellect was normal. Although Still did not use the current terminology for this disorder, many suggest that the children he described would have fulfilled diagnostic criteria for attention deficit hyperactivity disorder (Barkley, 2006). In relation to the pharmacological treatment of the symptoms of attention deficit hyperactivity disorder, Bradley first described the beneficial effects of amphetamine on hyperactivity in 1937.

Prevalence

The prevalence of attention deficit hyperactivity disorder is estimated at 3–5% in school-aged children (American Psychiatric Association, 1994). In the UK, a survey of

Dr Muhammed Ather *is Specialist Registrar (ST4) in Child and Adolescent Psychiatry, Forth Valley Child and Adolescent Mental Health Service, The Manor, Falkirk FK1 4PX and* **Dr Gill Salmon** *is Consultant in Child and Adolescent Psychiatry, Trehafod Child and Family Clinic, Swansea*

Correspondence to: Dr M Ather

children age 5–15 years showed a prevalence of 3.6% in male children and less than 1% in female children (Ford et al, 2003). In England and Wales alone 366 000 pupils aged 6–16 years are estimated to have attention deficit hyperactivity disorder (National Institute for Clinical Excellence, 2000). Attention deficit hyperactivity disorder is more common in males with the ratio of males to females varying between 2.5:1 to 9:1 (Rappley, 2005). More than 70% of children diagnosed with attention deficit hyperactivity disorder still fulfil diagnostic criteria in adolescence, and up to 65% of adolescents still have the disorder as adults (Jadad et al, 1999).

Overlap with other conditions

Many children with attention deficit hyperactivity disorder have co-occurring psychiatric or other conditions. These can be of a mild to severe nature and include learning disability, oppositional defiant disorder, conduct disorder, anxiety, depression, Tourette syndrome, bipolar disorder and sleep disorders (Beiderman et al, 1991). Kaplan et al (2001) evaluated 179 school-aged children with rigorous research criteria for seven disorders: reading disability, attention deficit hyperactivity disorder, developmental coordination disorder, oppositional defiant disorder, conduct disorder, depression and anxiety; 50% met criteria for at least two diagnoses, with children with attention deficit hyperactivity disorder at highest risk (80% chance of having at least one other disorder). Ohlmeier et al (2008) reported that alcohol and substance misuse can also be highly co-morbid with attention deficit hyperactivity disorder. Co-occurring conditions need early diagnosis and prompt intervention and treatment.

Causation

Psychosocial, social and biological factors are all likely to contribute to the aetiology (Cantwell, 1996). Some important theories and explanations have been put forward.

Genetics

Attention deficit hyperactivity disorder runs in families – more than 76% of children diagnosed with attention deficit hyperactivity disorder have another family member affected by this disorder (Thapar et al, 1999). The interaction of several genes and the environment may contribute to attention deficit hyperactivity disorder symptoms.

Several gene candidates have been identified in families and children with attention deficit hyperactivity disorder. Molecular genetic studies support an association between attention deficit hyperactivity disorder and polymorphisms in DRD4, DRD5 and SLC6A3 which encode dopamine D4 and D5 receptors and the dopamine transporter respectively. Associations between SNAP-25 (synaptosomal-associated protein, 25kDa) and SLC6A4 (serotonin transporter) have also been reported (Thapar et al, 2005). Identification of susceptible genes is a significant development, but describing the molecular mechanisms, interaction between genes and environment and later development of attention deficit hyperactivity disorder is a far greater challenge for researchers.

Biological factors

Many biological factors have been proposed to have a causative role in the development of attention deficit hyperactivity disorder, including adverse events during pregnancy and birth (e.g. drug exposure in utero), brain infections (e.g. encephalitis), and neurotoxin exposure (e.g. lead poisoning) (American Psychiatric Association, 1994). One early theory was that attention deficit hyperactivity disorder was caused by brain injury although there is no conclusive evidence for this. Some children with brain injury show behavioural signs and symptoms similar to those seen in attention deficit hyperactivity disorder (Levin et al, 2007), but only a small percentage of children with attention deficit hyperactivity disorder have a history of traumatic brain injury (Keenan et al, 2008).

It has also been suggested that attention disorders are caused by consuming refined sugar or food additives. In 1982, the National Institutes of Health consensus conference concluded that dietary restrictions helped about 5% of children with attention deficit hyperactivity disorder, mostly young children who had food allergies (Consensus Conference, 1982). National Institute for Health and Clinical Excellence (2008) guidance is that elimination of artificial colouring and additives from the diet is not usually necessary for those with attention deficit hyperactivity disorder unless a clear link between a specific food or drink and behaviour has been established.

There is some evidence that symptoms and behaviours associated with attention deficit hyperactivity disorder may be the result of abnormal brain functioning or abnormal brain structure (Castellanos, 2001). The frontal lobes of the brain are involved in executive functions such as problem solving, understanding the behaviour of others and controlling impulses, and they communicate with each other through the corpus callosum. The cerebellum, which is responsible for motor coordination, is also connected to the frontal lobes. Functional magnetic resonance imaging, positron emission tomography and single photon emission computed tomography studies have shown noticeable deficits in the functioning of these brain parts in children with attention deficit hyperactivity disorder compared with other children (Valera et al, 2007).

Psychosocial factors

Psychosocial adversity, e.g. a history of child abuse or neglect, or multiple foster placements, has been thought to have a causative role in attention deficit hyperactivity disorder (Haddad and Garralda, 1992). There is a correlation between the use of cigarettes and alcohol during pregnancy and risk of development of attention deficit hyperactivity disorder in the offspring. Mick at al (2002) reported that the children of mothers who smoked during pregnancy were more likely to be diagnosed with attention deficit hyperactivity disorder. Button et al

(2005) found that smoking by the mother, the father or both during pregnancy has an independent influence on attention deficit hyperactivity disorder symptoms in offspring. Froehlich et al (2009) reported an association between prenatal tobacco exposure and attention deficit hyperactivity disorder in American children. However, the role of low socioeconomic status, bad housing and poverty remains uncertain (Taylor et al, 1991).

Assessment

The European clinical guidelines for hyperkinetic disorder (Taylor et al, 2004) recommend that children suspected of having attention deficit hyperactivity disorder should be first assessed in primary care before referral to specialist child and adolescent mental health services or paediatrics. There are no laboratory tests of diagnostic value and diagnosis is largely based on the clinical history and observations provided by parents, teachers and other professionals who know the child. Where co-existing learning difficulties are suspected, additional assessment by an educational or clinical psychologist may also be helpful.

A variety of rating scales are used to gather information from parents, teachers and young persons. Narrow band scales, e.g. the Conners Rating Scales-Revised (Conners, 1997), are most helpful when assessing children for attention deficit hyperactivity disorder (American Academy of Pediatrics, 2000). Free rating scales do exist, such as the SNAP-IV (DuPaul et al, 1998), but broad band scales, e.g. the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997), are useful to assess different areas such as prosocial skills. Obtaining information from different sources (e.g. parents and teachers) provides a more accurate overview of the child's behaviour in different settings.

Assessment is often carried out by a child psychiatrist or paediatrician but can also be done by a professional with appropriate expertise and training, e.g. a specialist nurse (National Institute for Health and Clinical Excellence, 2008). Assessment of attention deficit hyperactivity disorder consists of reviewing pre-assessment information received, e.g. Conners rating scales and SDQs completed by parents and teachers and written reports from teachers and educational psychologists. Parents and the child are interviewed to assess current symptoms and take a full developmental history. A physical examination is needed to exclude other medical explanations for the symptoms, e.g. impairments in vision and/or hearing. Observation of the child's behaviour in the clinic and/or school setting is also informative. Consideration of other clinically appropriate investigations is required to complete the assessment (Taylor et al, 2004), e.g. neuropsychological tests, electroencephalography, brain scans, audiograms, fragile X test, and speech and language assessment.

Treatment

Early recognition and treatment of attention deficit hyperactivity disorder can limit the psychological, social and academic impact of the disorder (Cantwell, 1996). Treatment aims to manage the core symptoms, address associated impairments and improve social and academic functioning. Options include behavioural or pharmacological interventions or both. A multi-focal psychological and behavioural intervention provided to affected children, their parents or carers and teachers can be more effective than intervention with children alone (Jensen et al, 1999).

Psychological treatments include parent training, behavioural therapy, psychoeducation, cognitive behavioural therapy, interpersonal therapy, classroom interventions and play therapy (Pelham et al, 1998; Taylor et al, 2004). Young and Amarasinghe (2010) have reviewed non-pharmacological treatments of attention deficit hyperactivity disorder for both pre-school and school-aged children as well as adults. The National Institute for Health and Clinical Excellence (2008) guidelines recommend that all children diagnosed with mild to moderate attention deficit hyperactivity disorder should first get appropriate behavioural intervention before considering pharmacological treatments, whereas children with severe attention deficit hyperactivity disorder can be given combined treatment, e.g. stimulant medication and psychological intervention. Parent training programmes using standard behavioural techniques should be offered in community and clinic settings: these aim to reduce the child's disruptive behaviour and family stress and may enhance parents' confidence in their parenting ability (Cantwell, 1996).

In the UK, atomoxetine, dexamphetamine and methylphenidate are licensed for the treatment of attention deficit hyperactivity disorder. Atomoxetine and methylphenidate can be used for children aged 6 years and older while dexamphetamine is licensed for use from the age of 3 years. Clonidine, risperidone, imipramine and bupropion are not licensed and are used less frequently, generally only in specialist tertiary centres.

Stimulant medications, e.g. methylphenidate, tend to be used as first line and are available in short- and long-acting formulations. Choice of medication depends on suitability, comorbidity and patient (or parent) choice (National Institute for Health and Clinical Excellence, 2006). Stimulant medications are very effective, with a response rate of up to 80% in treating hyperactive and inattentive symptoms (Swanson et al, 1995). However, these can cause increased pulse rate, high blood pressure, decreased appetite and sleep, drowsiness, emotional lability, headaches and stomach ache (Jensen et al, 1999). Rarely stimulants can cause psychotic symptoms or even sensitivity reactions requiring the medication to be stopped. Stimulant medication needs monitoring because of the side effects and length of time that people remain on the treatment (National Institute for Health and Clinical Excellence, 2008). Children receiving long-acting stimulant medication to treat attention deficit hyperactivity disorder are more likely to achieve remission, feel less stigmatized, remain adherent to medication and benefit from other interventions which can improve the long-term prognosis (Hosenbocus and Chahal, 2009).

Conclusions

Attention deficit hyperactivity disorder affects 3–5% of children (American Psychiatric Association, 1994), with symptoms including inattention, hyperactivity and impulsivity. The condition is often co-morbid with other neurobiological, psychiatric and behavioural disorders. The aetiology is not clear, but genetic, psychosocial and/or environmental factors may be responsible.

Assessment, diagnosis and management should be provided by a multidisciplinary team of professionals with appropriate training. Treatment is multimodal and includes medication and psychosocial interventions. **BJHM**

Conflict of interest: Dr M Ather has been supported by Shire to attend two international meetings on attention deficit hyperactivity disorder. Dr G Salmon has been paid to present at a meeting on attention deficit hyperactivity disorder by Janssen-Cilag.

American Academy of Pediatrics (2000) Clinical practice guideline: diagnosis and evaluation of the child with attention-deficit hyperactivity disorder. *Pediatrics* **105**: 1158–70

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders.* 4th Edition. (DSM-1V). American Psychiatric Association, Washington, DC

Barkley R (2006) The relevance of the Still lectures to attention deficit hyperactivity disorder: a commentary. *J Atten Disord* **10**(2): 137–40

Beiderman J, Newcorn J, Sprich S (1991) Co-morbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety and other disorders. *Am J Psychiatry* **148**: 564–77

Bradley C (1937) The behavior of children receiving benzedrine. *Am J Psychiatry* **94**: 577–85

Button T, Thapar A, McGuffin P (2005) Relationship between antisocial behaviour, attention-deficit hyperactivity disorder and maternal prenatal smoking. *Br J Psychiatry* **187**: 155–60

Cantwell D (1996) Attention deficit disorder: A review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* **35**: 978–87

Castellanos F (2001) Neuroimaging studies of ADHD. In: Solanto M, Arnsten A, Castellanos F, eds. *Stimulant Drugs and ADHD. Basic and Clinical Neuroscience.* Oxford University Press, New York: 243–58

Conners C (1997) *Conners Rating Scales - Revised.* NFER, Windsor

Consensus Conference (1982) Defined Diets and Childhood Hyperactivity. *JAMA* **248**(3): 290–2

DuPaul G, Anastopoulos A, Power T, Reid R, Ikeda M, McGoey K (1998) Parent ratings of attention-deficit/hyperactivity disorder symptoms: factor structure and normative data. *J Psychopathol Behav Assess* **20**: 83–101

Faraone SV, Sergeant J, Gillberg C, Biederman J (2003) The worldwide prevalence of ADHD: is it an American condition? *World Psychiatry* **2**(2): 104–13

Ford T, Goodman R, Meltzer H (2003) The British Child and Adolescent Mental Health Survey 1999: the prevalence of DSM-IV disorders. *J Am Acad Child Adolesc Psychiatry* **42**: 1203–11

Froehlich T, Lanphear B, Auinger P et al (2009) Association of tobacco and lead exposures with attention-deficit/hyperactivity disorder. *Pediatrics* **124**: e1054–e1063

Goodman R (1997) The strengths and difficulties questionnaire. A research note. *J Child Psychol Psychiatry* **38**: 581–6

Haddad P, Garralda M (1992) Hyperkinetic syndrome and disruptive early experiences. *Br J Psychiatry* **161**: 700–3

Hosenbocus S, Chahal R (2009) A review of long-acting medications for ADHD in Canada. *J Can Acad Child Adolesc Psychiatry* **18**(4): 331–9

Jadad A, Boyle M, Cunningham C, Kim M, Schahar R (1999) *Treatment of Attention-Deficit/Hyperactivity Disorder.* Evidence Report/Technology Assessment: Number 11. Agency for Health Care Policy and Research and Quality, Rockville, Maryland, USA

Jensen P, Arnold L, Richters J, Severe J, Vereen D, Vitiello B (1999) A 14-month randomized clinical trial of treatment strategies for children with attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry* **56**: 1073–86

Kaplan B, Dewey D, Crawford S, Wilson B (2001) The term 'co-morbidity' is of questionable value in reference to developmental disorders: Data and theory. *J Learn Disabil* **34**: 555–65

Keenan H, Hall G, Marshall S (2008) Early head injury and attention deficit hyperactivity disorder: retrospective cohort study. *BMJ* **337**: a1984

Levin H, Hanten G, Max J et al (2007) Symptoms of attention-deficit/hyperactivity disorder following traumatic brain injury in children. *J Dev Behav Pediatr* **28**: 108–18

Mick E, Biederman J, Faraone S, Sayer J, Kleinman S (2002) Case-control study of attention-deficit/hyperactivity disorder and maternal smoking, alcohol use, and drug use during pregnancy. *J Am Acad Child Adolesc Psychiatry* **41**: 378–85

National Institute for Clinical Excellence (2000) *Guidance on the use of methylphenidate (Ritalin, Equasym) for attention deficit/hyperactivity disorder (ADHD) in childhood.* Technology appraisal Guidance no 13. National Institute for Clinical Excellence, London

National Institute for Health and Clinical Excellence (2006) *Methylphenidate, atomoxetine and dexamphetamine for attention deficit hyperactivity disorder (ADHD) in children and adolescents.* Technology appraisal 98. National Institute for Health and Clinical Excellence, London

National Institute for Health and Clinical Excellence (2008) *Attention Deficit Hyperactivity Disorder: diagnosis and management of ADHD in children, young people and adults.* Clinical guideline 72. National Institute for Health and Clinical Excellence, London

Ohlmeier M, Peters K, Te Wildt B et al (2008) Comorbidity of alcohol and substance dependence with attention-deficit/hyperactivity disorder (ADHD). *Alcohol Alcohol* **43**(3): 300–4

Overmeyer S, Taylor E (1999) Annotation: Principles of Treatment for Hyperkinetic Disorder: Practice approaches for the UK. *J Child Psychol Psychiatry* **40**: 1147–57

Palmer E, Finger S (2001) An Early Description of ADHD (Inattentive Subtype): Dr Alexander Crichton and 'Mental Restlessness (1798). *Child Adolesc Ment Health* **6**(2): 66–73

Pelham W, Wheeler T, Chronis A (1998) Empirically supported psychosocial treatments for attention deficit hyperactivity disorder. *J Clin Child Psychol* **27**: 190–205

Rappley M (2005) Clinical practice. Attention deficit-hyperactivity disorder. *N Engl J Med* **352**(2): 165–73

Still G (1902) Some abnormal psychical conditions in children: the Goulstonian lectures. *Lancet* **i**: 1008–12

Swanson J, McBurnett K, Christian D, Wigal T (1995) Stimulant medication and treatment of children with ADHD. In: Ollendick TH, Prinz RJ, eds. *Advances in Clinical Child Psychology*, vol. 17. Plenum Press, New York: 265–322

Taylor E, Sandberg S, Thorley G, Giles S (1991) *The Epidemiology of Childhood Hyperactivity.* Oxford University Press, Oxford

Taylor E, Dopfner M, Sergeant J et al (2004) European clinical guidelines for hyperkinetic disorder- first upgrade. *Eur Child Adolesc Psychiatry* **13**(1): 7–30

Thapar A, Holmes J, Poulton K, Harrington R (1999) Genetic basis of attention deficit and hyperactivity. *Br J Psychiatry* **174**: 105–11

Thapar A, O'Donovan M, Owen M (2005) The genetics of attention deficit hyperactivity disorder. *Hum Mol Genet* **14**(spec no 2): R275–82

Thome J, Jacobs K (2004) Attention deficit hyperactivity disorder (ADHD) in a 19th century children's book. *Eur Psychiatry* **19**: 303–6

Valera E, Faraone S, Murray K, Seidman L (2007) Meta-analysis of structural imaging finding in attention-deficit/hyperactivity disorder. *Biol Psychiatry* **61**: 1361–9

World Health Organization (1992) *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines.* World Health Organization, Geneva

Young S, Amarasinghe JM (2010) Practitioner Review: Non-pharmacological treatments for ADHD: A lifespan approach. *J Child Psychol Psychiatry* **51**(2): 116–33

KEY POINTS

- Attention deficit hyperactivity disorder is a medical disorder which is common in school-aged children, with a prevalence of 3–5%.
- Attention deficit hyperactivity disorder is frequently co-morbid with other psychiatric or neurodevelopmental disorders.
- Psychosocial, social and biological factors all contribute to the aetiology, and attention deficit hyperactivity disorder runs in families.
- Effective evidence-based interventions are available.