

## Attention deficit hyperactivity disorder in children: moving forward with divergent perspectives

George Halasz and Alasdair L A Vance

AUSTRALIAN CHILDREN (mainly boys) are often referred to paediatricians and child psychiatrists with the question, "Does this child have attention deficit hyperactivity disorder (ADHD)?" We believe that the current controversy about diagnosis and management of these children's symptoms arises from the divergent perspectives of developmentally and non-developmentally minded professionals. Here, we discuss this divergence and illustrate the impact of the controversy on diagnosis with a case study.

### Scientific status of ADHD

In 2000, the US National Institutes of Health released a consensus statement on ADHD based on 31 expert "testimonies".<sup>1</sup> The statement concluded that the diversity of opinions about ADHD "raises questions concerning the literal existence of the disorder, whether it can be reliably diagnosed". In an accompanying commentary, Dr Peter Jensen, from the US National Institute of Mental Health, stated that according to the panellists ADHD remains of "unproven" status, which "should give pause to both researchers and clinicians who may have reified ADHD as a 'thing' or 'true entity' (rather than a *working hypothesis* that serves scientific, communication, and clinical decision-making purposes)".<sup>2</sup> He added the caveat not to confuse "unproven" with "disproved".

ADHD is currently an unsatisfactory umbrella term applied to children with widely differing temperaments and functional problems in home, school and social settings, but sharing certain core features: poor impulse control, motor overactivity and limited sustained attention span. Many have comorbidities, including oppositional-defiant disorder, conduct disorder, anxiety, obsessive-compulsive disorder, post-traumatic stress disorder, mood disorders, learning disorders, and substance misuse.

### Assessment of ADHD

#### The DSM-IV approach

No clinical or laboratory test can validly and reliably distinguish children with ADHD from those without

### ABSTRACT

- Current controversy about diagnosis and treatment of attention deficit hyperactivity disorder (ADHD) reflects the divergence between developmental and non-developmental approaches.
- While there is growing evidence for biological vulnerabilities associated with ADHD, we believe that environmental factors, including early problems in parental attachment, are also important in determining the type and timing of deficit that a child develops, the risk to academic and social performance and eventual outcome.
- We warn against labelling children with ADHD simply because they fulfil the cross-sectional diagnostic symptom criteria of the *Diagnostic and statistical manual of mental disorders — 4th edition (DSM-IV)*.
- We advocate an integrated biopsychosocial approach to diagnosis and management with a thorough developmental assessment to identify developmental factors, such as deficits in early attachment, contributing to the presentation.

MJA 2002; 177: 554–557

ADHD. Given the heterogeneity of the condition as currently defined, it seems unlikely that such a test will emerge.

In the absence of a specific test for ADHD, the *Diagnostic and statistical manual of mental disorders — 4th edition (DSM-IV)* bases diagnosis on the presence of specific clinical features — behavioural symptoms and signs<sup>3</sup> (Box 1). However, the DSM-IV taskforce chair observed that this approach perpetuates the lack of "developmentally sensitive, interactive or longitudinal perspective...[and thus] limits the useful[ness] of the categories for both research and clinical assessment and treatment of children and adolescents".<sup>4</sup> In other words, the DSM's neglect of developmentally sensitive interactive issues, such as attachment, creates the possibility of misdiagnosis.

#### The developmental approach

Developmentally informed psychiatric assessment of children is based on Professor Sir M Rutter's concept of development as "the crucial link between genetic determinants and environmental variables, between sociology and individual psychology, and between physiogenic and psychogenic causes".<sup>5</sup> For Rutter, developmental factors encompass both the "roots of behaviour in prior maturation" as well as the "modulations of that behaviour by the circumstances of the present". Recent advances in our

Department of Psychological Medicine, Monash Medical Centre, East Malvern, VIC.

George Halasz, MRCPsych, FRANZCP, Honorary Senior Lecturer;  
Alasdair L A Vance, MD, FRANZCP, Senior Lecturer.

Reprints will not be available from the authors. Correspondence: Dr George Halasz, Department of Psychological Medicine, Monash Medical Centre, c/- Burke Road Medical Suites, 30 Burke Road, East Malvern, VIC 3145. geohalasz@aol.com

### 1: Diagnostic criteria for attention deficit hyperactivity disorder from DSM-IV<sup>3</sup>

(A) Either (1) or (2):

(1) *Inattention*: Six (or more) symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level (eg, often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities; is often forgetful in daily activities).

(2) *Hyperactivity-impulsivity*: Six (or more) symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level (eg, hyperactivity: often fidgets with hands or feet or squirms in seat; impulsivity: often interrupts or intrudes on others).

(B) Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

(C) Some impairment from the symptoms is present in two or more settings (eg, at school [or work] or at home).

(D) There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

(E) The symptoms do not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder and are not better accounted for by another mental disorder (eg, mood disorder, anxiety disorder, dissociative disorder).

DSM-IV = *Diagnostic and statistical manual of mental disorders* — 4th edition.

understanding of the biopsychosocial correlates of the core symptoms of ADHD also highlight the interrelationship between the biological and the developmental perspectives. For example, neuropsychological deficits in verbal and visuospatial working memory have been proposed as core deficits in ADHD.<sup>6,7</sup> However, environmental factors are important determinants of the specific type of deficit that a child develops and its timing. Environmental factors are also often critical in promoting or diminishing the risk to a child's academic and social performance and eventual outcome.

As observed in the National Institutes of Health consensus statement, the medically based assessment appears "disconnected" from the developmentally based assessment.<sup>1</sup> We argue that this divergence can lead to misdiagnosis of ADHD and non-rational prescribing.

Accurate diagnosis and valid formulation of symptoms in children or adults demands that patients be considered in the context of the family, school or workplace, and community. Children's developmental "symptoms" (inattention or hyperactivity-impulsivity inconsistent with developmental level) may be necessary, but are certainly not sufficient, criteria to label them as having ADHD. Careful determination of functional impairment across settings, along with the time course of symptoms, is also required. We argue that such assessment cannot be completed in a single session and agree with the National Institutes of Health that the frequent prescribing of medication "may be due in part to the limited time spent making the diagnosis".<sup>1</sup> Yet, we often hear anecdotes of children being diagnosed with ADHD and even prescribed medication at the end of the first consultation.

Faced with such complexities in clinical assessment and diagnosis, Eisenberg observed more than 25 years ago that the ambiguity arises from two sources: "the insufficiency of information on which clinical decisions must be taken and... the frailty of the judgment we can, any of us, bring to bear on the human problems we face".<sup>8</sup> He further opined that a single cause is unlikely to be found for the hyperkinetic syndrome, and that the behaviours that constitute this condition are the expression of diverse pathologies.<sup>9</sup> The numerous clinical and research articles published since then have not provided cause to disagree.

### Applying the developmental approach

A case history illustrates these issues (Box 2). In this case, the non-developmental approach of applying the DSM-IV criteria alone to Nathan's presenting condition may lead to his unintentional misdiagnosis with ADHD.<sup>10</sup> Key environmental factors that also need to be considered during assessment include the early family "ecology": the impact of his mother's postnatal depression and antidepressant therapy on her attachment patterns when Nathan was three months old; stresses during parental separation and domestic violence when Nathan was a toddler; his father's binge drinking; and the family's level of emotional availability and adaptability during Nathan's different developmental phases. All these factors may affect a child's developing cognitive capacities, problem-solving skills, emotional involvement and communication.

Recent mother-infant research highlights the role of attachment in understanding developmental psychopathology. It also proposes mechanisms by which attachment problems may contribute to later disorders.<sup>11</sup> It is beyond the scope of this article to discuss early attachment disorders in detail. However, the absence of a developmental perspective in the DSM approach should not be equated with a lack of empirical support for the role of infant attachment problems in later maladaptation.

Applying the developmental perspective, Nathan's presenting problems can be seen as emerging from interactions between his biological vulnerabilities and a range of environmental influences from early childhood. His parents' reduced capacity to care for him during infancy in such a stressful, and possibly dysfunctional, family setting may have enduring developmental consequences.

Developmental factors possibly contributing to Nathan's presentation include:

- Innate developmental disability (possibly genetic);
- Disorganised-disoriented insecure attachment patterns;
- Post-traumatic stress disorder; and
- Environmental deprivation (maternal depression, absent father, witness or victim of domestic violence).

To fully assess Nathan's presenting problems and come to a rational diagnosis and treatment plan, a detailed developmental history is required. A developmentally sensitive assessment should detail the parenting style, qualities of early attachment, presence of parental and sibling physical or mental illness, and social and cultural influences (including academic and peer-group influences).<sup>12</sup> The diagnosis of

## 2: Case history — a difficult eight-year-old

"Nathan" is an eight-year-old boy referred to a paediatrician by his general practitioner. His teachers are concerned about poor academic progress and report that he is uncooperative, does not listen, distracts others, and has begun to act the "class clown". His mother is finding it increasingly difficult to cope with his behaviour as he is non-compliant, has aggressive outbursts and often seems unhappy. His father is not concerned, stating by telephone, "There's nothing wrong with him. I don't want him drugged!".

Nathan's parents separated when he was a toddler, and there may have been some domestic violence. He has a 13-year-old sister and a 10-year-old brother, who are both "doing well". All children live with their mother. Their parents do not speak to each other. Nathan did not see his father much between the ages of two and six years, but they have had semiregular contact over the past two years.

Nathan's mother suffered postnatal depression after the births of her children and was prescribed antidepressants when Nathan was three months old. She has taken them intermittently ever since. Nathan's father did not like school and left at age 15. He is literate but disorganised, and goes from one short-term job to another. He binge drinks.

Nathan was a challenging child to care for from the age of 10 months when he began walking. He was always different from his siblings. He had some delay in language development.

ADHD should be considered only if all DSM-IV criteria (Box 1) are met, including, importantly, criterion E — the symptoms are "not better accounted for by another mental disorder". In the case of very young children, it is essential to diagnose anxiety, mood or dissociative disorders associated with traumatic attachment disorders where these exist and not to misdiagnose them as ADHD (Box 3).

## Treatment

A range of psychological and psychopharmacological treatments are being investigated for children with ADHD. While the primary areas of current research are psychostimulant medication and behavioural interventions, there have also been major advances in family interventions.

### Psychostimulant medications

Psychostimulant medications are generally considered the primary treatment modality for children with disabling ADHD.<sup>13,14</sup> In the short term (up to four to six weeks), about 80% of treated children have improvements in the core behavioural features of ADHD-combined type (criteria for both inattention and hyperactivity-impulsivity are met for the past six months), as well as in executive functions (such as response inhibition and verbal and non-verbal working memory performance).<sup>15</sup>

In the longer term (over three months), the essential symptom domains of ADHD-combined type may be significantly ameliorated,<sup>16,17</sup> although there is some evidence that the short-term improvements may be attenuated over time.<sup>18</sup> In contrast, there is emerging evidence that executive functions, such as non-verbal working memory, remain improved, regardless of any attenuation of short-term improvements.<sup>19,20</sup>

Thinking in cognitive terms may help us understand the poor behavioural response of some children to psychostimulant medication in the longer term.<sup>21</sup> This poor response may arise from the complex interplay of the children's vulnerabilities caused by their executive functioning deficits with their comorbidities and psychosocial risk factors. This interplay is the final result of biological and psychosocial disturbances at particular developmental periods in early, middle and late childhood. The result is ADHD.

### Behavioural interventions

The chronic nature of ADHD and its associated comorbidities have also led to a range of behavioural therapeutic interventions, such as parent and teacher management training programs, which are combined with psychostimulant medication in clinical practice.<sup>22</sup> Yet, there are relatively few publications investigating the effectiveness of these interventions compared with those studying psychostimulant medication.<sup>23</sup>

A potential synergistic effect has been reported between the two types of intervention in the short term.<sup>24</sup> In the longer term, children treated with a combination of psychostimulant medication and behaviour therapy required lower doses of medication than those treated with psychostimulant medication alone in the 14-month NIMH Collaborative Multisite Multimodal Treatment Study of Children with ADHD.<sup>25</sup> The long-term effectiveness of behavioural therapeutic interventions remains unclear.<sup>26</sup> Research is needed to aid clinicians to tailor empirically validated behavioural interventions specifically to the nature and severity of the individual's impairments and disabilities.

### Family therapy

Recent developments in attachment research prompted Ladnier and Massanari to suggest that ADHD is an attachment deficit hyperactivity disorder, with its origins in early problems in parental attachment.<sup>27</sup> From the ethical perspective, they note that, in the United States, "Health insurance companies, for example, find it costs them less to pay a physician to treat the child pharmaceutically than for a psychotherapist to see the family for regular sessions. Physicians today generally assume that medication is the first and most effective treatment for a child diagnosed as having ADHD." This raises the question of whether financial considerations influence clinical decision-making and to what degree the principle of acting in the "best interest of the child" is upheld.

A recent South Australian Government inquiry into ADHD cautioned professionals about the "conflicting views about stimulant use and effectiveness".<sup>28</sup> The inquiry noted that "treatment" refers to symptoms and does not mean a "cure" for ADHD — so that it is possible for symptoms to return if medication is stopped. Although the effects of long-term intensive individual psychotherapeutic interventions are more difficult to measure, there is some research evidence to support this approach in selected cases.<sup>29,30</sup>

### 3: Differential diagnosis of attention deficit hyperactivity disorder (ADHD)

In differentiating ADHD, traumatic attachment and post-traumatic stress disorder of infancy, important considerations include:

- Detailed developmental history from early infancy
  - Experience of parenting
  - When did first concerns arise?
  - Nature of developmental and emotional or behavioural difficulties.
- Data from multiple sources (eg, parents and teachers), with use of standardised behaviour rating scales.
- DSM-IV diagnostic criteria are necessary but not sufficient criteria for the diagnosis of ADHD. These criteria require that symptoms:
  - Occur to a developmentally inappropriate level;
  - Have onset before age 7 (generally evident in pre-school years);
  - Be present in both home and school settings;
  - Cause impaired social or academic functioning; and
  - Not be better accounted for by mood or anxiety disorder.
- Identify comorbid conditions.

Applying Ladnier and Massanari's construct for treating the child with ADHD in the family context would suggest that these improved results could be attributable to the "repair" of deficits in impulse control, based on reversal of the underlying insecure attachment or attachment deficits.

### Conclusions

While genetic studies and response to psychostimulant medication provide strong evidence for biological vulnerabilities associated with ADHD, we advocate an integrated biopsychosocial approach to diagnosis and management to account for the heterogeneity of the condition's onset, clinical course and outcome.

In treating a child with ADHD, the aim is to maximise the child's cognitive, emotional, behavioural and interpersonal development. Thorough assessment is needed in the family and school contexts to understand the factors contributing to the child's behaviour and to formulate an individual management plan. We advocate careful and regular monitoring of the effectiveness of psychological and medical treatments and their "titration" according to response.

### Acknowledgement

We thank Jeannette Friedman (US) for her editing suggestions.

### Competing interests

None identified.

### References

1. National Institutes of Health Consensus Development Conference Statement: Diagnosis and Treatment of Attention-Deficit/Hyperactivity Disorder (ADHD). *J Am Acad Child Adolesc Psychiatry* 2000; 39: 182-193.
2. Jensen PS. Commentary: The NIH ADHD consensus statement: win, lose, or draw? *J Am Acad Child Adolesc Psychiatry* 2000; 39: 194-197.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders — 4th edition (DSM-IV). Washington, DC: American Psychiatric Association, 1994.
4. Frances AJ, Egger HL. Whither psychiatric diagnosis. *Aust N Z J Psychiatry* 1999; 33: 161-165.
5. Rutter M. Scientific foundations of developmental psychiatry. London: William Heinemann, 1980.
6. Tannock R. Attention deficit hyperactivity disorder: advances in cognitive, neurobiological and genetic research. *J Child Psychology Psychiatry* 1998; 39: 65-99.
7. Denney CB, Rapport MD. Cognitive pharmacology of stimulants in children with ADHD. In: Solanto MV, Arnsten AFT, Castellanos FX, editors. Stimulant drugs and ADHD: basic and clinical neuroscience. New York: Oxford University Press, 2001: 283-302.
8. Eisenberg L. The ethics of intervention: acting amidst ambiguity. *J Child Psychology Psychiatry* 1975; 16: 93-104.
9. Eisenberg L. The clinical use of stimulant drugs in children. *Pediatrics* 1972; 49: 709-715.
10. Halasz G, Anaf G, Ellingsen P, et al. Cries unheard. A new look at attention deficit hyperactivity disorder. Altona: Common Ground, 2002.
11. Greenberg MT. Attachment and psychopathology in childhood. In: Cassidy J, Shaver PR. Handbook of attachment theory, research, and clinical applications. New York: Guilford Press, 1999: 469-496.
12. Zeanah CH Jr, editor. Handbook of infant mental health. 2nd ed. New York: Guilford Press, 2000.
13. National Health and Medical Research Council. Attention deficit hyperactivity disorder (ADHD). Canberra: Commonwealth of Australia, 1997.
14. American Academy of Pediatrics Subcommittee on Attention-Deficit/Hyperactivity Disorder. Clinical practice guideline: treatment of the school-aged child with attention-deficit/hyperactivity disorder. *Pediatrics* 2001; 108: 1033-1044.
15. Wilens TE, Biederman J. The stimulants. *Psychiatric Clin N Am* 1992; 15: 191-222.
16. Gillberg C, Melander H, von Knorring A-L, et al. Long-term stimulant treatment of children with attention-deficit hyperactivity disorder symptoms. *Arch Gen Psychiatry* 1997; 54: 857-864.
17. Efron D, Jarman FC, Barker MJ. Medium-term are comparable with short-term outcomes in children with ADHD treated with stimulant medication. *J Paediatr Child Health* 2000; 36: 457-461.
18. Schachar R, Tannock R, Cunningham C. Treatment. In: Sandberg S, editor. Hyperactivity disorders of childhood. Cambridge: Cambridge University Press, 1996: 433-476.
19. Kempton S, Vance ALA, Maruff P, et al. Executive function and attention deficit hyperactivity disorder: stimulant medication and better executive function performance in children. *Psych Med* 1999; 29: 527-538.
20. Barnett R, Vance ALA, Maruff P, et al. Abnormal executive function in attention deficit hyperactivity disorder: The effect of stimulant medication and age on spatial working memory. *Psych Med* 2001; 31: 1107-1115.
21. Vance ALA, Luk ESL. Heart of the matter review: attention deficit hyperactivity disorder: progress and controversies. *Aust N Z J Psychiatry* 2000; 34: 719-730.
22. Swanson JM, Sergeant JA, Taylor E, et al. Attention deficit hyperactivity disorder and hyperkinetic disorder. *Lancet* 1998; 351: 429-433.
23. Zametkin AJ, Ernst M. Problems in the management of attention-deficit-hyperactivity disorder. *N Engl J Med* 1999; 340: 40-46.
24. Hinshaw SP, Whaien CK, Henker B. Cognitive behavioural and pharmacological interventions for hyperactive boys: comparative and combined effects. *J Consulting Clin Psychol* 1984; 52: 739-749.
25. The MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for ADHD. Multimodal Treatment Study of Children with ADHD. *Arch Gen Psychiatry* 1999; 56: 1073-1086.
26. Schachar RJ, Tannock R, Cunningham C, Corkum PV. Behavioural, situational, and temporal effects of treatment of ADHD with methylphenidate. *J Am Acad Child Adolesc Psychiatry* 1997; 36: 754-763.
27. Ladnier RD, Massanari AE. Treating ADHD as attachment deficit hyperactivity disorder. In: Levy TM, editor. Handbook of attachment interventions. San Diego: Academic Press, 2000: 27-65.
28. Social Development Committee. Inquiry into Attention Deficit Hyperactivity Disorder. Sixteenth Report of the Social Development Committee, Parliament of South Australia, January 2002.
29. Satterfield JH, Satterfield BT, Cantwell DP. Three year multimodality treatment study of 100 hyperactive boys. *J Paediatr* 1981; 98: 650-655.
30. Satterfield JH, Satterfield BT, Schell AM. Therapeutic intervention to prevent delinquency in hyperactive boys. *J Am Acad Child Adolesc Psychiatry* 1987; 26: 56-64.

(Received 6 Jun 2002, accepted 9 Aug 2002)

□