Attention deficit hyperactivity disorder or hyperkinetic disorder in adults

B. K. TOONE and G. J. H. VAN DER LINDEN

Attention deficit hyperactivity disorder (ADHD) and hyperkinetic disorder are well recognised in children and adolescents, and are among the more frequently seen problems in child psychiatry. The criteria for ADHD are contained in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders*, DSM–IV (American Psychiatric Association, 1994), and for hyperkinetic disorder in the tenth revision of the *International Classification of Diseases*, ICD–10 (World Health Organization, 1992). The disorders are similar, but there is a greater emphasis on overactivity in hyperkinetic disorder. The core features of these disorders are hyperactivity, poor ability to maintain attention and poor impulse control. To make a diagnosis of either disorder, symptoms should be pervasive, give rise to significant impairment and be evident early in life, usually before school age; they should not be attributable to another psychiatric disorder.

There now appears to be little doubt about the validity of the concept despite past controversies. In the UK the condition is likely to be more frequently diagnosed in the future for three reasons: the ICD–10 definition of hyperkinetic disorder is more explicit than in previous editions; pressure from parent support groups has increased; and methylphenidate is now more generally available (Taylor & Hemsley, 1995).

What is the natural outcome of ADHD? The received view is that the disorder resolves spontaneously. Certainly ADHD is rarely diagnosed in adults. However, as children treated for ADHD grow up and continue to need treatment, we are becoming more aware that adults too can suffer from ADHD. There has been publicity in the media recently coinciding with the formation of a self-help and campaigning group in the UK (the National Learning Attention Deficit Disorder Association – LADDER) which has drawn attention to the problem. The result has been an increase in requests for expert psychiatric opinions from adult patients and from their general practitioners to evaluate ADHD. Some of these adults never received the diagnosis of ADHD as a child, but recognise their symptoms and request treatment. In the US, where some rights come with the diagnosis (Nadeau, 1994), this process is further advanced than in the US and should be a forewarning to psychiatrists to educate themselves about this disorder, its diagnosis and treatment.

Although in England and France severe overactivity has been recognised as a symptom in handicapped children since the 19th century, the introduction of hyperactivity as a disorder *sui generis* can probably be traced to Bradley's (1937) observation that amphetamine, when given to hyperactive children, had a calming effect. In America the concept was broadened and swiftly gained acceptance: near the end of the 1980s it was estimated that between 750 000 and 1.6 million (6%) of American public elementary schoolchildren were receiving stimulants (Safer & Krager, 1988). This was partly a result of federally mandated changes in the educational recognition of attention deficit. In the UK the diagnosis and treatment remained rarer events, more closely allied with overt neurological conditions and seen more as a symptom and less as the primary disorder.

EPIDEMIOLOGY AND LONG-TERM OUTCOME

In three American studies (a primary care paediatric sample, a pre-adolescent (aged 11) population sample, and a community survey of children aged 4 to 16) the prevalence of ADHD was estimated to be respectively 2%, 6.7% and 9.5% (Anderson *et al*, 1987; Bird *et al*, 1988; Costello *et al*, 1988). The disorder is more prevalent in boys in a ratio of 2.9:1 (Anderson *et al*, 1987; Bird *et al*, 1988; Safer & Krager, 1988). The point prevalence of the hyperkinetic disorder is about 1.5% in 7-year-old boys in British inner cities (Taylor *et al*, 1991).

The long-term outcome has been studied in New York (Gittelman *et al*, 1985) and in Montreal (Weiss *et al*, 1985). Gittelman *et al* followed up two cohorts of hyperactive 6–12-year-old boys until they reached between 16 and 23, and compared them with an age-matched control group. At follow-up approximately a quarter still met criteria for ADHD compared with 3% of the controls. A third met criteria for personality disorder and a sixth for substance abuse disorder. In the control group only 8% fell into each of those categories. There was considerable comorbidity, with half of the index group and a fifth of the controls attracting a DSM–III diagnosis. Those who did not were indistinguishable across a range of variables. Seven years later the proportion of ADHD had fallen to 8% and 1% respectively (Mannuzza *et al*, 1993). In a similar study, Weiss *et al* (1985) found that two-thirds of hyperactive children followed up into early adult life retained at least one disabling ADHD symptom, compared with 7% of a normal control group.

Extrapolations from these studies suggest that approximately 0.5% to 1% of the young adult population has symptoms associated with ADHD.

AETIOLOGY

The aetiology of ADHD remains uncertain. Genetic factors are likely to be important. There is an increased prevalence of ADHD in first-degree relatives of childhood ADHD probands. One study of monozygotic and dizygotic twin pairs has yielded estimates of heritability of trait measures of hyperactivity and inattentiveness between 30% and 50%; environmental factors accounted for only 0 to 30% (Goodman & Stevenson, 1989). Elsewhere the evidence for a major environmental contribution (such as pre- or perinatal complications, parental attitudes, environmental toxins, etc.) is unconvincing. The cerebral pathways and mechanisms through which ADHD symptoms are mediated are equally obscure. Computerised tomography and functional neuroimaging studies have so far proved negative, but structural magnetic resonance imaging has recently demonstrated abnormalities of the caudate nucleus (Castellanos *et al*, 1994) and corpus callosum (Giedd *et al*, 1994) that await replication.

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ASSESSMENT AND DIAGNOSIS

In common with most psychiatric disorders there is no specific cognitive, metabolic or neurological marker for ADHD. Diagnosis is beset by a number of difficulties. As it is a maturational disorder, a childhood history strongly suggestive of ADHD is mandatory for the adult diagnosis. Yet hyperactive children, on reaching adulthood, not infrequently fail to recall their childhood hyperactivity (Manuzza *et al*, 1993). Parents may provide a testimony that is more predictive of treatment response (Wender *et al*, 1981). Contemporaneous medical and educational records offer the firmest evidence, but the lack of such information is insufficient reason to discount a diagnosis of childhood ADHD; in schools and among general practitioners there has often been little awareness of these disorders. A retrospective diagnosis of childhood ADHD, essential to the adult diagnosis though it may be, must often and unavoidably seem insecure due to lack of reliable information. The assessment of ADHD symptoms in the adult is beset by difficulties of a different nature. DSM–IV which provides the most detailed account of ADHD as it presents in childhood, recognises the persistence of symptoms into adult life and has modified the wording of the criteria accordingly. An adherence to those criteria provides probably the best guide to diagnosis in the adult. A minority of patients continue to satisfy the full complement of ADHD criteria; others, though still symptomatic, fall short and are designated ADHD in partial remission or ADHD not otherwise specified, according to whether or not childhood ADHD can be diagnosed. The clinician will be more concerned to learn of the relationship between this diagnostic spectrum and the level of disability and probability of treatment response. Regrettably there is little information to hand. Standardised questionnaires are widely used in childhood ADHD to estimate the severity and pervasiveness of symptoms and disabilities. Similar instruments have been designed for use in adult ADHD, such as the Wender–Utah Rating Scale, a self-report of past ADHD symptoms (Ward *et al*, 1993), but experience of these is still limited. Their development will doubtless continue and should provide better means of quantifying clinical morbidity.

During adolescence, hyperactivity may become less prominent, and in young adults inattention, impulsivity and personal disorganisation may be the more salient characteristics. Adult ADHD may coexist with other psychiatric diagnoses, notably affective disorders, substance misuse, intermittent explosive behaviour and antisocial behaviour. Every attempt should be made to identify ADHD as a distinct nosological entity, while recognising that the presence of comorbidity may obscure the specific attribution of disability. Particular caution should be exercised in treating ADHD in the presence of substance misuse. A failure to attend is a feature of many psychiatric disorders, a number of which commonly present during early adult life. The distinction between primary and secondary attentional deficit may sometimes be difficult, but can usually be accomplished by attendance to accompanying symptomatology and to the evolution and course of the disorder. If doubt persists, further evaluation of ADHD symptoms should be deferred until treatment of the primary condition has been successfully accomplished.

NEUROPSYCHOLOGICAL TESTS

Measures of attention do not distinguish between primary and secondary deficits, and are therefore of limited value in diagnosis. But once diagnosis has been established they may be used to quantify the degree of impairment and to monitor change in response to treatment. In the assessment and management of childhood ADHD, the Continuous Performance Test (Rosvold *et al*, 1956) has the greatest predictive power and is widely used. The role of neuropsychological testing in adult ADHD has yet to be firmly established.

TREATMENT

Since the discovery in 1937 of the calming effect of amphetamines on hyperactivity, over 100 controlled studies of over 5000 patients have documented the efficacy of stimulant treatment in hyperactive children. Stimulant drugs that potentiate dopaminergic activity, such as methylphenidate, pemoline and amphetamine, are the mainstay of treatment of ADHD. These agents have been found to improve the core symptoms of ADHD in a dose-dependent fashion. They also improve self-esteem, academic and occupational function and social skills (see Wilens & Biederman, 1992, for review).

There have been few controlled trials in adults with ADHD. The first was reported by Wood *et al* (1976). Five further studies have since appeared (Wender *et al* 1981, 1985; Mattes *et al*, 1984; Gaultieri *et al*, 1985; Spencer *et al*, 1995). Four studies reported a significant and beneficial effect of methylphenidate. In the largest trial (Mattes *et al*, 1984), 37 patients meeting criteria for current symptoms of ADHD, but not the criterion for having symptoms as children, were also treated along with the 29 'true' ADHD patients. Unfortunately the difference between these two groups of patients in the rate of response to methylphenidate was not reported. It was concluded that although a quarter of patients benefited from methylphenidate, the majority did not.

The average dose of methylphenidate for the first four trials was 0.6 mg per kg per day in divided doses, which is more in line with the dose used in children (Wilens & Biederman, 1992). They achieved a higher rate of response, approximately 70%, compared with about 50% for the other trials. The average dose of pemoline used by Wender *et al* (1981) was 65 mg per day in a morning dose, and by Woods *et al* (1976), 56.25 mg per day.

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The drug trial literature therefore narrowly favours an effective role for methylphenidate and pemoline, both from the dopaminergic agonist group of drugs, in the treatment of adults with ADHD. This endorsement must be heavily qualified. Few studies have been reported; fewer still have used adequately sized patient cohorts; and of the six studies that reported positively, three were from the same centre.

CONCLUSIONS

It is more usual for the features of ADHD to be first recognised in childhood, but it has become increasingly clear that the symptoms may persist, at least in some, well beyond adolescence. ADHD may make an important contribution to personality disturbance in early adult life. The precise role and effectiveness of pharmacotherapy

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has yet to be fully established, but it seems likely that some individuals will benefit, and that change in behaviour and in lifestyle as well as symptom relief may ensue. Among clinicians, a recognition that ADHD symptoms may persist into adult life should be tempered by an awareness of the pitfalls that accompany any new diagnostic concept in the domain of personality disorder, and of the risks of self-diagnosis.

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