HANDBOOK OF
Attention Deficit
Hyperactivity Disorder
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Attention Deficit
Hyperactivity
Disorder

Edited by

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Preface

Neuroscience seeks to decipher the mystery of the most complex of all machines, the human brain. The brain has more than 10 billion neurons in a highly interconnected web governed by complex biochemical pathways. Disorders of the brain have particularly devastating consequences for patients, families, health and financial resources. Attention Deficit Hyperactivity Disorder (ADHD) is one of these conditions. ADHD is characterised by significant symptoms of inattention, hyperactivity and impulsivity. The impact of the condition on the individual, the family and society is enormous. It is associated with extensive use of health-related resources, it is a burden on the criminal justice system and confers significant social cost in terms of educational failure, family disruption, and marital breakdown.

The major events in the life of children and adolescents are educational and ADHD undermines this part of their life, leading to many secondary complications including bullying, school failure and poor self-confidence. ADHD has multiple negative impacts on education, sense of self, social relationships, and is often associated with depression, anxiety and suicidal behaviour. Increasingly, ADHD is being appreciated as a lifelong illness in perhaps as many 60% of childhood cases. This book includes much commentary on the clinical phenomenology, genetics and both pharmacological and non-pharmacological treatment of adult ADHD. Across the lifespan ADHD impacts on many professionals including general practitioners, psychiatrists, psychologists, social workers, lawyers, judges, paediatricians, neurologists, geneticists, pharmacologists, and neuroradiologists. We hope that professionals in each of these areas will benefit from this book.

ADHD represents one of the most controversial psychiatric disorders of our time. Controversy arises for at least two reasons. First there is the public perception that ADHD is a ‘new’ condition and that its diagnosis rates are ever on the increase. As reviewed in this book, reports of children presenting with inattentive or hyperactive/impulsive behaviour date back to 1798 when Alexander Crichton wrote of ‘mental restlessness’. Crichton wrote:

when born with the person it becomes evident at a very early period of life, and has a very bad affect, in as much as it renders him incapable of attending with constancy to any one object of attention. But it is seldom so great a degree as to totally impede all instruction; and what is very fortunate it generally diminishes with age. (Cadell & Davis, 1976, p. 271)

Nevertheless, any psychiatric disorder is a sign of our time, and current diagnosis rates undoubtedly reflect our modern world that calls for problem-solving and analytic abilities, focus of attention and restraint of impulsivity. As Klimkeit and Bradshaw point out in Chapter 21 of this book, in certain other historical settings,
the novelty seeking and impulsive behaviours of ADHD children, which in today’s society are seen as maladaptive, may well have been advantageous.

Controversy also arises from the treatment of children with ADHD with potentially addictive stimulants, such as methylphenidate and dextroamphetamine. Stimulant medications have now been the mainstay treatment for ADHD for more than three decades, and an overwhelming amount of data demonstrates a beneficial impact of these drugs on core symptoms of ADHD. However, as reviewed in Chapter 13 of this book by Solanto and colleagues, newer generation, non-stimulant medications have emerged that may help to allay some of the fears surrounding stimulants. Time will tell whether these newer treatments have comparable short- and longer-term efficacy in ADHD. Nevertheless, there is a growing appreciation that therapeutic response, even to stimulants, is somewhat variable in children with ADHD and so there is a push to identify individual difference factors which may predict drug response. In this endeavour, molecular genetics and pharmacology are interfacing in a new and important way. Pharmacogenetics is the study of how individual differences in drug response might depend upon underlying genetic factors. Barry and colleagues review current knowledge in this burgeoning area of research in Chapter 16.

Perhaps more than in any other neurodevelopmental disorder, our knowledge of ADHD is expanding rapidly. This book examines ADHD at many levels and represents an up-to-date description of our knowledge and understanding of the disorder. The book is divided into three sections, dealing with research findings from the clinical, neurobiological and treatment perspectives. The book begins at the bedside by reviewing the clinical description of child and adult ADHD and its key comorbid disorders (Chapters 1–6). It then moves to the bench to examine the key neurobiological findings from the fields of genetics, neuroimaging, neuropsychology and psychopharmacology (Chapters 7–16). Finally, the book makes a return from the bedside to the bench, describing the latest non-pharmacological treatment modalities that are being informed by our growing understanding of the neurobiology of the disorder (Chapters 17–20). Thus, the book tries to bridge the gap between basic neuroscience and clinical applications.

This *Handbook of Attention Deficit Hyperactivity Disorder* particularly focuses on recent developments in Attention Deficit Hyperactivity Disorder research. Wiley has produced previous handbooks of a similar nature on autism. The aim of this ADHD Handbook is to give the reader a rapid update on recent developments on ADHD research by an international panel of contributors. We hope that this book is as useful to the student as it is to the expert.

We have relatively effective interventions for ADHD but there is a great deal of extra work to be done in devising new pharmacological and non-pharmacological treatments. There is little doubt that the future lies in rigorous scientific research. Rigorous research has led to the abandonment of earlier views of ADHD as being due to minimal brain dysfunction or parental mismanagement, for example. The book emphasises solid scientific data where this is available. While there has been much progress in defining the ADHD phenotype across the lifespan, considerable challenges lie ahead for mapping the biological pathways that may lead from gene to disorder. While this may have been unthinkable even 15 years ago, we have little doubt that in time, such scientific advances will change the landscape for clinicians
and lead to improved treatment of the disorder. We are optimistic about the future of research and clinical practice in ADHD; we hope that the advances outlined in this book may inspire researchers or clinicians who are new to the area.

We would like to acknowledge the contributions of the many scientists and clinicians, from centres and universities around the world, who have taken time out of their busy schedules to contribute to this book. We would also particularly like to thank the many children with ADHD and their families, who have participated in research studies that informed this book. This book is dedicated to you all. Finally, we would like to acknowledge the editorial staff of John Wiley & Sons for their assistance and patience during the preparation of this book.

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REFERENCE

I Clinical Perspectives
1 The History of Attention Deficit Hyperactivity Disorder

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1.1 OVERVIEW

The condition now referred to as Attention-Deficit/Hyperactivity Disorder (DSM-IV) (American Psychiatric Association, 1994) or Hyperkinetic Disorder (ICD-10) (World Health Organization, 1992) was first described by George Still in 1901 (Still, 1902). In his lectures to the Royal Academy of Physicians he described a case series of 20 children presenting with problems of overactivity, inattention and deficits in ‘volitional inhibition’. He also described symptoms of aggressiveness, defiance, resistance to discipline and dishonesty, which in today’s nomenclature would be diagnosed as Oppositional Defiant Disorder or Conduct Disorder which are often comorbid with ADHD. Subsequent to Dr Still’s lecture a number of different diagnostic labels were assigned to the same symptoms, including Minimal Brain Damage and Minimal Brain Dysfunction to refer to children presenting with overactivity and inattention, subsequent to a pandemic of encephalitis lethargica in 1917. The condition which we now refer to as ADHD was first included in the second edition of the \textit{Diagnostic and Statistical Manual of Mental Disorders} (DSM) in 1968 and labelled ‘Hyperkinetic Disorder of Childhood’. The definition of the condition changed in subsequent editions of DSM, in keeping with changes in diagnostic nomenclature and delineation of subtypes. The most recent edition, DSM-IV, requires pervasive symptoms of inattention or inattention, hyperactivity and or impulsivity, which are clinically impairing with an age of onset prior to age seven. The diagnostic criteria used by DSM-IV are similar to the criteria for Hyperkinetic Disorder used in the current edition of the International Classification of Diseases (ICD-10) in that specific behaviour symptoms of inattention and hyperactivity-impulsivity are recognised and both are required for a diagnosis to be made. ICD-10 does not recognise predominantly inattentive or predominantly hyperactive-impulsive subtypes, and requires symptom onset prior to age six. In addition, ICD-10 requires a direct observation of symptoms by the clinician together with parental and school reports.

The concept of the diagnosis of ADHD has evolved through a complex developmental trajectory dating back to Greek times. The focus of this chapter is to present an overview of the developmental course and unfolding of our current
understanding of hyperactivity and attention disorders. We will present a chronological account of the literature referring to symptoms of inattention, hyperactivity and impulsivity and comorbid behaviour disorders, that have contributed to our current understanding of the condition ADHD.

1.2 PREHISTORY AND HISTORY OF ATTENTION DEFICIT HYPERACTIVITY DISORDER

1.2.1 EIGHTEENTH CENTURY

The earliest literature referring to the inattentive subtype of ADHD dates back to the writings of the physician, Alexander Crichton in 1798. In his paper ‘Mental Restlessness’, Dr Crichton described all the essential features of the inattentive subtype of attention deficit hyperactivity disorder which were almost entirely consistent with the criteria for the inattentive subtype as portrayed in DSM-IV (APA, 2000) (Palmer & Finger, 2001). He saw it as a nervous problem which may be born with the person or be the effect of accidental disease . . . when born with the person it becomes evident at a very early period of life, and has a very bad affect, in as much as it renders him incapable of attending with constancy to any one object of attention. But it is seldom so great a degree as to totally impede all instruction; and what is very fortunate it generally diminishes with age. (Cadell & Davis, 1976, p. 271)

Crichton further wrote:

every impression seems to agitate the person, and gives him or her an unnatural degree of mental restlessness. People walking up and down the room, a slight noise, too much light or too little light all destroy constant attention in such patients, in so much as it is easily excited by every impression.

He went on to say that when people are affected in such a way ‘they have a particular name for the state of their nerves, which is expressive enough of their feelings. They say they have the fidgets’ (p. 272). Crichton suggested that these children needed special educational intervention.

1.2.2 NINETEENTH CENTURY

John Haslam in his book Observations on Madness and Melancholy (1809, p. 120), described the case of a child who from the age of two was

mischievous and uncontrollable . . . a creature of volition and a terror of the family . . . he had limited attention span, being only attracted by ‘fits and starts’. He had been several times to school and was the hopeless pupil of many masters, distinguished for their patience and rigid discipline.

This poor child also had a tendency to break things, was very oppositional and cruel to animals. While Haslam paints a picture of a young boy with conduct disorder,
a diagnosis of ADHD, ODD, dyspraxia and specific learning difficulties would have to be included in the differential diagnosis.

A number of descriptions of hyperactive children mostly in the form of case reports appeared in the psychiatric literature towards the second half of the nineteenth century. The German physician Henrich Hoffman described the ‘hyperkinetic syndrome’ in a case report of a young boy presenting with symptoms of hyperactivity, impulsivity and inattention (Clements & Peters, 1962).

Maudsley (1867) described children as ‘little more than an organic machine automatically impelled by disordered nerve centres’. He discussed their ‘absence of mind’ and ‘an actual abnormality underlying children’s problems’. Albutt (1892) reported these children as ‘having an unstable nervous system’.

Clousten (1966, pp. 481–90) described a disorder which he referred to as ‘simple hyperexcitability’, caused by ‘undue brain reactivity to mental and emotional stimuli’. The condition he reported was characterised by symptoms of overactivity and restlessness and it primarily affected children from the age of three years until puberty. It occurred in bursts, lasting from a few months to years, adversely affecting academic performance and emotional well-being. Anorexia, weight loss and insomnia were associated features. The symptoms of ‘simple hyperexcitability’ that Clousten described shared a marked resemblance to DSM-IV ADHD, but also shared many of the features of early onset bipolar affective disorder. Clousten recommended a multimodal treatment approach for these children, including high dose bromides, good nutrition, fresh air, ‘companionship and employment’. The aim of treatment was to ‘reduce cell catabolism and the reactivity of the cerebral cortex whilst not interfering with brain anabolism’.

In 1870 an Education Act was passed by Parliament in Britain that made school attendance compulsory. This had a significant impact on the recognition of symptoms of inattention and hyperactivity as more than just extremes of normal childhood behaviour, and brought the condition increasingly to the attention of the medical profession. This may be one of the reasons why most of the literature pertaining to ADHD dates from 1900.

1.2.3 TWENTIETH CENTURY

1900–10

The birth of the new century witnessed the birth of the recognition of a disorder which was to become the most diagnosed child psychiatric disorder. Although some attribute the first clear accounts of hyperactivity to Dr Alexander Crichton (1798), most of the psychiatric literature credits Sir George Still, a paediatrician and first professor of childhood diseases at King’s College Hospital, London. In 1902 Still presented the Goulstonian lectures entitled ‘Some abnormal psychical conditions in children’ to the Royal College of Physicians. He described a case series of 20 children manifesting a deficit of ‘moral control’. The children he described experienced extreme restlessness and an ‘abnormal capacity for sustained attention’, impacting on academic performance and social relationships, despite normal intellectual functioning. Their behaviour was described as violent, destructive, oppositional and non-responsive to punishment. It occurred more frequently in boys and
first manifested in the early school years. The defect of moral control was not thought to be a result of adverse social circumstances which were common in society at the time, but rather was thought to be a neurobiological affliction due to ‘some morbid physical condition’. He defined three subgroups of hyperactive behaviour:

those with demonstrable gross lesions of the brain; those with a variety of acute diseases, conditions and injuries that would be expected to result in brain damage; and those with hyperactive behaviours that could not be attributed to any known cause. (Sandberg & Barton, 1996, pp. 5–7)

Alfred Tredgold (1908), a member of the English Royal Commission on Mental Deficiency, extended Still’s biological theory. He suggested that some forms of brain damage, resulting from birth injury or mild anoxia, though undetected at the time, could present as behaviour problems or learning difficulties in the early school years. He was the first to propose the concept of ‘minimal brain damage’. In addition to symptoms of hyperactivity and educational difficulties, the children he observed exhibited soft neurological signs and motor clumsiness.

1910–20

Neve and Turner (1913, p. 385) described Still’s ideas as a ‘contemporary and perhaps logical, extension of that put forward by James Crichton-Brown, as a newer neurological account of phenomena once seen as immoral, while still using the older language of morality (e.g. vicious, depraved) to describe abnormal psychological function’. In this same year the Dublin-born paediatrician, Robert Stein (1913, pp. 478–86) discussed ‘children saturated with insanity while still in the womb’, with ‘badly built minds’ and ‘a kind of partial moral dementia’. He observed that children with these afflictions presented with pervasive disruptive behaviour problems, evident in the early school years resulting in educational underachievement and relationship difficulties. It is possible that the children he described would today fulfil criteria for ADHD, and his phrase ‘badly built minds’ could equate with current neurobiological findings underlying the disorder.

In 1917 a pandemic of encephalitis lethargica swept Europe and North America. In its aftermath clinicians encountered children who having made a full recovery from the infection, presented with overactivity, distractibility, poor impulse control and cognitive deficits. This period gave rise to theories of Minimal Brain Dysfunction (MBD) (Kessler, 1980), and is regarded by many clinicians as the beginning of North America’s interest in hyperactivity (Cantwell, 1975).

1930–40

The paediatrician D.W. Winnicott (1931, p. 654) gave a very good description of the ‘hyperkinetic child’. In his words

such a fidgety child is a worry, is restless, is up to mischief if left for a moment unoccupied, and is impossible at table, either eating food as if someone would snatch it from him, or else liable to upset tumblers or spill tea . . . sleep is usually restless . . . . These children are over-excitable, or ‘nervy’ rather than nervous.
In 1934 Kramer-Pollnow described a condition which he referred to as ‘hyperkinetische Erkrankung’ (hyperkinetic disease). The syndrome he described was characterised by symptoms of extreme restlessness, distractibility and speech disorder, ‘a condition of persistent motor unrest which makes its appearance between the ages of 2 and 4 years’ (reported by Hoff, 1956, pp. 537–53). Kramer-Pollnow described a case series of 15 children who were symptomatic by the age of six, and in addition to the syndrome described, presented with aggressive behaviour, impulsivity and learning difficulties. In many cases the extreme restlessness was followed by an epileptic seizure. Kramer-Pollnow clearly described a cohort of children with complex neurodevelopmental difficulties of which ADHD appears to have been a comorbid condition.

Kahn and Cohen (1934) described a case series of three children with symptoms of overactivity, impulsivity, clumsiness and soft neurological signs. They argued that the symptoms were caused by ‘organic driveness, or a surplus of inner impulsion’ stemming from a defect in the organisation of the brain stem, caused by trauma, birth injury or a congenital abnormality.

Although Kanner’s third edition of the child psychiatry textbook (1957) made no references to hyperactivity as a diagnostic entity, he discussed a syndrome which bears a strong resemblance to the hyperactive subtype of ADHD as early as 1935. He described the ‘extreme of restless, fidgety, Hyperkinetic child who is always on the go, can never sit still, always must be doing something’ (Kanner, Tindal & Cox, 1935, p. 253). He subsequently described a syndrome characterised by daydreaming, lack of attention, and lack of concentration, which is similar to the DSM-IV definition of Attention Deficit Disorder.

In 1937 Charles Bradley, working at the Emma Pendleton Bradley Home in Providence, Rhode Island, USA, demonstrated the efficacy of Benzedrine, a central nervous system stimulant, in the treatment of ADHD. He administered benzedrine to children suffering with headache and noted a marked improvement in their behaviour and school performance (Bradley, 1937). This discovery marked a major milestone in the history of ADHD, and led to the use of dexamphetamine and methylphenidate in the treatment of hyperactivity.

1940–60

Despite the significant discovery of the use of psychostimulants in the treatment of ADHD, drugs were not widely used until the late 1950s. This, it was believed, was due to the psychoanalytic climate which prevailed in society during the 1940s and 1950s (Laufer et al., 1957; Laufer, 1975), which resisted the idea that hyperactive behaviour had a biological basis.

1960–70

*From minimal brain damage to minimal brain dysfunction*

During the early 1960s several clinicians began to question the concept of brain damage as the only cause of childhood hyperactivity. Kanner recommended that ‘lay persons should be discouraged from the much too frequent practice of using
the term brain damage or brain injury as an everyday cliché’. Birch (1964), Herbert (1964) and Rapin (1964) questioned the assumption that brain damage caused behaviour problems on the basis that most children with behaviour problems demonstrated no physical evidence of brain damage. In 1963 the Oxford International Study Group of Child Neurology (MacKeith and Bax, 1963) stated that brain damage could not be inferred from behaviour alone, and recommended that the term ‘minimal brain damage’ be replaced by ‘minimal brain dysfunction’ (MBD). In the USA, a national task force devised an official definition (Clements, 1966):

The term minimal brain dysfunction refers to children of near average, average or above average general intelligence with certain learning or behavioural disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system. These deviations may manifest themselves by various combinations of impairment in perception, conceptualisation, language, memory and control of attention, impulse or motor function.

The term MBD emphasised the role of organic factors in the aetiology of ADHD and challenged the prevailing psychoanalytic theories of the time that proposed that the disorder was due to poor parenting.

During the late 1950s and early 1960s, clinicians such as Laufer (1957) and Chess (1960) started introducing terms such as ‘hyperkinetic behaviour syndrome’. They began to recognise the key symptoms of hyperactivity and impulsivity, and moved away from the prevailing theories of brain damage or dysfunction. The disorder hyperkinetic reaction of childhood first appeared in DSM-II *Diagnostic and Statistical Manual of Mental Disorders* in 1968 (APA, 1968). The term emphasised overactivity as the cardinal feature of the syndrome rather than minimal brain damage or dysfunction.

The 1960s also saw the development of parent and teacher rating scales for diagnostic assessment of symptoms of hyperactivity and monitoring response to treatment. These questionnaires allowed for a standardised assessment of children’s behaviour in home and school settings.

1970–80

Interest in the concept of hyperactivity mushroomed in the 1970s, particularly in the USA. Symptoms such as inattention, overactivity and impulsivity began to be recognised as the core symptoms of the disorder. The shift to an emphasis on inattention began when Virginia Douglas and her team at McGill University suggested that deficits in the ability to sustain attention underlay the observed symptoms of hyperactivity and poor impulse control. She contended that these were the areas in which stimulant medication was most effective (Douglas, 1972).

The work of Douglas and her team was influential in the re-categorisation of the disorder in DSM-III (APA, 1980) as Attention Deficit Disorder with and without hyperactivity, thus emphasising the attentional aspects of the disorder, rather than hyperactivity. DSM-III defined ADD with hyperactivity as a tri-dimensional disorder characterised by developmentally inappropriate inattention, impulsivity and hyperactivity with symptoms and cut-offs to operationalise the diagnosis.
Coinciding with the work of Douglas, researchers in Northern Europe became more interested in the concept of hyperactivity as a diagnostic entity. 1977 marked the inclusion of ‘Hyperkinetic syndrome of childhood’ in ICD-9 (WHO, 1977), as a disorder in which the essential features are ‘short attention span and distractibility’.

1980–90

DSM was revised in 1987 (DSM-III-R, APA, 1987). The revised edition listed 14 symptoms, some referring to attention and some to hyperactivity and impulsivity, requiring eight symptoms for a diagnosis. The criteria also necessitated onset of symptoms prior to age seven. DSM-III-R also included a category of Undifferentiated Attention Deficit Disorder which excluded hyperactivity and impulsivity. There was no subtyping in DSM-III-R.

1990–2005

In preparation of the ICD-10 and DSM-IV the working parties of the WHO and the APA liaised closely in drawing up diagnostic criteria for childhood hyperactivity. Although the newest editions of both systems are almost compatible, significant differences remain between the definition of Hyperkinetic Disorder (HD) and the criteria for ADHD, in their diagnostic criteria, definition of pervasiveness, the role of inattention and the inclusion of comorbidity.

The ICD definition of hyperkinetic disorder emphasises the presence of at least six inattentive, three hyperactive and one impulsive symptom in home and school settings, together with the direct observation of this behaviour (WHO, 1992). DSM in contrast requires that symptoms of hyperactivity, impulsivity or inattention must be present in two or more settings, but does not require direct observation of the symptoms by the clinician.

In addition, ICD requires that anxiety disorders, mood disorders, pervasive developmental disorders or schizophrenia pre-empt a diagnosis of hyperkinetic disorder, while DSM allows for comorbid mood, anxiety and psychotic disorders, as long as the symptoms are not better accounted for by, or occur exclusively during the course of these other diagnoses.

ICD also describes a Combined Hyperkinetic Conduct Disorder category, which is classified as ADHD plus comorbid Oppositional Defiant Disorder or Conduct Disorder in DSM. The current classification system will be described in the next chapter. While similarities and differences between the two classification systems will be discussed, the focus of the chapter will be on DSM-IV.

1.3 CONCLUSION

This chapter outlines the history of the evolution of ADHD as a valid diagnostic entity. Clinical interest in the disorder has mushroomed over the past century, and this is reflected in the systematic increase in scientific literature. The future for ADHD looks bright. The nineteenth and twentieth centuries have a lot to show for
themselves. Standardised rating scales have been developed to validate the diagnosis, and multimodal treatment approaches are available. Scientific literature continues to blossom and children are being maintained in mainstream education. The twenty-first century has a lot to offer and we look forward with optimism to further developments.

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2 Diagnosis and Classification of ADHD in Childhood

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2.1 OVERVIEW

Attention Deficit Hyperactivity Disorder (ADHD) is a persistent and impairing disorder resulting from abnormal levels of inattentive, hyperactive and impulsive behaviour. By definition, its onset is prior to age seven, mostly before age five. It often persists into adolescence and adult life and puts sufferers at risk of a range of adverse outcomes, including educational and occupational underachievement, antisocial behaviour and delinquency. As a condition, ADHD affects 8–12% of children worldwide (Faraone \textit{et al.}, 2003) and represents up to 40% of referrals to child psychiatric clinics (Safer & Allen, 1976). Despite the high prevalence of this disorder ADHD remains under-diagnosed and under-treated and its validity as a diagnostic entity is frequently challenged. The focus of this chapter is to review the current understanding of the diagnosis and classification of ADHD in childhood. In addition, we will describe the rating scales used in aiding diagnosis. We begin by tracing the evolution of our understanding of the syndrome and examining the different subtypes.

2.2 WHY CLASSIFY?

Classification attempts to group cases according to distinguishing patterns of symptomatology. Classification of illness (nosology) is essential in order to categorise the observed symptoms, to communicate about the illness, to form a treatment plan, to determine prognosis and to inform scientific research. The merits of a good classification system are comprehensiveness, acceptability to users, clarity and the ability to change with emerging scientific evidence. Critics of classification argue that applying a diagnostic category stigmatises a patient and implies that all persons with this label are the same. This serves to distract from understanding the person’s unique personal difficulties, which can impact on prognosis and dictate treatment regimens.

Most medical conditions can be classified on the basis of aetiology; for example, tuberculosis and coronary artery disease. While some psychiatric
diagnoses have recognised physical aetiology (such as Down’s Syndrome, Fragile X syndrome), most can be classified only on the basis of observed symptoms. This is most problematic in child psychiatry, particularly in the diagnosis of ADHD, which is viewed by some as being an extreme of normal childhood behaviour (Baughman, 2001), caused by normal childhood energy, overstressed parents or restrictive classroom curriculum (McCubbin & Cohen, 1997; Breggin, 2001).

Clinicians need a classification framework to clarify misconceptions about ADHD. Such a framework proves that psychiatrists have rules of evidence for establishing the validity of disorders and that these rules have established ADHD as a valid psychiatric diagnosis.

DSM-IV (American Psychiatric Association, 1994) and ICD-10 (American Psychiatric Association, 1994) constitute the two major psychiatric classification systems used throughout the world. The DSM system is used mainly in the USA, whereas ICD is used predominantly in Europe. ICD refers to ADHD as Hyperkinetic Disorder (HKD). While similarities and differences between the two classification systems will be outlined, for the most part we adopt the DSM convention of using the term ADHD to refer to both systems.

2.3 THE EVOLUTION OF ADHD AS A DIAGNOSTIC ENTITY

Attention Deficit Hyperactivity Disorder (ADHD) was initially described by George Still in 1901 (Still, 1902). Dr Still recounted problems of overactivity, inattention, and poor inhibitory volition in a case series of 20 children. He also observed aggressiveness, defiance, resistance to discipline, lawlessness, spitefulness and dishonesty. In today’s nomenclature the latter would be diagnosed as Oppositional Defiant Disorder or Conduct Disorder, which are often comorbid with ADHD.

In 1917 a syndrome of overactivity and distractibility was described following a pandemic of Encephalitis Lethargica. Attention focused on the causal role of brain damage arising from infection and named Minimal Brain Dysfunction (Kahn & Cohen, 1934; Clements, 1966) with inattention, hyperactivity and impulsiveness seen as evidence of brain damage.

Since then successive editions of the DSM have revised the diagnostic criteria and subtyping associated with ADHD. DSM-II (APA, 1968) recognised a disorder known as Hyperkinetic Disorder of Childhood with hyperactivity as the principal symptom. DSM-III described operational criteria for diagnostic categories of ADD with and without hyperactivity, with a requirement for three inattentive, three impulsive and two hyperactive symptoms to be present to attain a diagnosis. This distinction was abolished in the revised edition that described a single list of 14 items incorporating symptoms of inattention, hyperactivity and impulsivity, with an eight-item cut off for diagnosis. This change implied that symptoms of ADHD were on a continuum from low to high numbers of symptoms. DSM-IV, based on factor analysis of field trials, returned to a categorical classification describing three subtypes of ADHD: