1 Historical development

Seija Sandberg[1] and Joanne Barton[2]

[1]Department of Psychiatry and Behavioural Sciences, Royal Free and University College Medical School, London, UK
[2]Department of Child and Adolescent Psychiatry, University of Glasgow, Glasgow, Scotland, UK

Introduction

The constellation of overactivity, poor impulse control and inattention has been given a variety of diagnostic labels over the years. Although at present attention deficit hyperactivity disorder and hyperkinetic disorder are popular diagnoses, they are relative newcomers in the diagnostic classification of child psychiatry. For example, even the latest (1957) edition of the child psychiatry textbook by Kanner contained no reference to hyperactivity as a diagnostic entity. The same applied to the 1969 edition of a widely used text on child psychology by Johnson and Medinnus (1969), with even the term 'attention span' only mentioned on two of the total of 657 pages. A well-regarded textbook on experimental child psychology (Reese and Lipsitt, 1970) did include a section on attention processes, but without any reference to attention deficit hyperactivity disorder. Yet evidence of attention psychology going back at least to the latter half of the nineteenth century has been well documented (James, 1890; Spearman, 1937). On the European continent, however, the condition was recognized and referred to as 'hyperkinetic disorder' in Hoff's (1956) major textbook of general psychiatry. So, whilst hyperactivity tends to be thought of as a particularly American phenomenon, the history of the use of the term tells us otherwise. Though new as a diagnostic entity, hyperactive behaviour in children has been detected and treated for much longer. Indeed, the diagnosis of 'dextro-amphetamine response disorder' was in common use in the former German Democratic Republic (DDR: Göllnitz, 1981).

The current conceptualization of the disorder represents a stage in a complex and varying developmental history, and therefore in order to appreciate our present perspective it is important to consider the chronological course in the unfolding of the concept itself. In this chapter, an overview of the history of hyperactivity and attention disorders will be presented, especially as they have appeared in the western texts. Relatively more attention will also be given to publications up to the 1960s, after which time studies on the condition began to

mushroom. This revised version contains some modifications and additions to the text of the first edition.

References to behavioural disturbance in childhood of a similar nature to that seen in hyperactivity disorders can be found in the writings of Hoffmann (1845), Maudsley (1867), Clouston (1899), Ireland (1877) and others from the middle of the 1800s. However, the earliest clear descriptions of the disorder are those by Still and Tredgold in the early 1900s. Their work will be discussed and examined in the context of the prevailing social and scientific climate. Both authors presented their analysis of the behavioural characteristics of a relatively small group of children, some of whom bear close resemblance to the hyperactive children of today. Still (1902) attributed such behaviour to a 'defect of moral control', believing it to be a biological defect, which was inherited or resulted from some pre- or postnatal injury. His ideas about causation are best understood in the context of the widespread support given at that time to social Darwinism.

The theory of damage occurring in the early stages of the individual's development, though often mild and undetected, was adopted by Tredgold (1908); and at a later stage by others, such as Pasamanick et al. (1956a), as an attempt to explain the behavioural problems seen in hyperkinetic children. The encephalitis epidemic in 1917–18 played a significant part in the history of hyperactivity. Following the epidemic, clinicians were confronted with children suffering from behavioural and cognitive sequelae, many presenting with the core features of what would today be termed hyperactivity.

For the first half of the twentieth century, the predominant view regarding the causation of hyperactivity was that of an association with brain damage. A plethora of terms such as 'organic drivenness' and 'minimal brain damage' were used to describe the disorder. During this period, the similarity between the behaviour of hyperactive children and that of primates subjected to frontal lobe lesions was noted. This was used by several investigators to support the idea that hyperkinetic disorders were due to defects in forebrain structures, despite the lack of evidence of such lesions in many children.

During the fourth and the fifth decades of the last century, a series of papers were published which marked the beginnings of child psychopharmacology in general, and the pharmacotherapy of behaviourally disturbed children in particular. By the end of the 1950s, the concept that brain damage was the single important factor in the development of hyperkinesis was challenged. 'Minimal brain dysfunction' was substituted for 'minimal brain damage'. At the time, a variety of hypotheses were put forward regarding the causation of hyperactivity, including the psychoanalytic theory of poor parenting. With the decline of

the concept of 'minimal brain dysfunction', the idea of the 'hyperactive child syndrome' was being developed, with Stella Chess (Chess, 1960) as one of its chief proponents. Chess differed from her predecessors in that she viewed the prognosis of hyperactive children as reasonably good, with the condition having resolved by puberty in most cases. By the end of the 1960s the prevailing view was that hyperkinesis represented a form of brain dysfunction, and presented with a variety of symptoms, of which motor overactivity was the most predominant.

It was during the 1960s that the split developed between Europe and North America over the conceptualization of hyperkinesis. Clinicians in Europe maintained a narrower view of the disorder, seeing it as a rare syndrome of excessive motor activity, usually occurring in conjunction with some signs of brain damage. In North America, on the other hand, hyperactivity was seen as being relatively common, and in most cases not necessarily associated with overt signs of brain damage. The differences became highlighted in the diagnostic classificatory systems used (*International Classification of Disease* (ICD); World Health Organization, 1992 and *Diagnostic and Statistical Manual of Mental Disorders* (DSM: American Psychiatric Association, 1980), respectively).

In the 1970s, the emphasis moved away from motor overactivity and began to focus on the attentional aspects of the disorder. A number of authors demonstrated that hyperactive children had great difficulty with tasks involving sustained attention. At the same time, there was also a growing belief that hyperkinetic behaviour was due to environmental causes. This coincided with a move towards a healthier lifestyle and disquiet about the 'drugging' of schoolchildren. The idea that hyperactivity was, at least in every other case, due to an allergic reaction to food substances, particularly food additives, became popular at this time. Technological advance and other cultural influences were also put forward as causative factors. An additional development of the decade was the increase in the number of studies investigating the psychophysiology of hyperactivity.

During the 1980s the explosion of research in the field continued. With this came the development of research criteria and standardized assessment procedures. Advancements were also made in the area of treatment, with new methods involving cognitive-behavioural therapies. Hyperactivity came to be seen as a condition with a strong hereditary component, chronic in nature and causing significant handicap in the areas of academic achievement and socialization; treatment required the complementary skills of a variety of professionals.

Throughout the 1990s hyperactivity and attention disorders continued to

generate more research literature than any other child psychiatric disorder. The breadth and range of the subject matter of this literature are enormous and encompass research into the genetics and neurobiological bases of hyperactivity, together with studies examining the relative efficacy of different treatment methods. The 1990s also saw the development of management guidelines, including the European Society for Child and Adolescent Psychiatry guidelines (Taylor et al., 1998), and the American Academy of Child and Adolescent Psychiatry practice parameters (Dulcan, 1997), in an attempt to facilitate standardization in practice. These guidelines emphasize the importance of individualized, multimodal, multidisciplinary assessment and treatment of hyperactivity and attention disorders by practitioners experienced in the field. Increasingly, it is also recognized that these disorders can and do persist through adolescence and into adulthood. Hence, the past decade has also seen a surge of interest in their presentation and treatment in adults.

Early medical explanations

The modern history of hyperactivity disorders is traditionally seen to begin with the writings of Sir George Frederick Still (1902) and Alfred F. Tredgold (1908), with many authors acknowledging that our present concept of hyperactivity has its foundation in their work (Ross and Ross, 1982). Preceding the studies by Still and Tredgold, extensive descriptions of hyperkinetic children, mostly in the form of individual case studies, had already appeared in the psychiatric literature of the nineteenth century (Hoffmann, 1845; Maudsley, 1867; Ireland, 1877; Bourneville, 1897; Clouston, 1899) and continued to be published in the early decades of the twentieth century (Pick, 1904; Boncour and Boncour, 1905; Scholz, 1911; Heuyer, 1914).

Overexcitability and mental explosiveness: forerunners of hyperactivity?

Clouston (1899) described a series of very difficult morbid conditions that 'occur in neurotic children, but lie on the borderland of psychiatry'. He hypothesized that the disorders were all due to some dysfunction in the 'brain cortex', and were pathogenically 'states of deranged reactiveness of the neurones of the higher regions of the brain'. The derangement had arisen because the higher centres of the brain, responsible for inhibiting activity, had in some way been weakened and therefore become unable to cope with the amount of 'energising they have to control'.

Clouston, however, emphasized that such disorders should not be classified as 'out and out mental disorders'. He believed that the conditions were

ultimately caused by 'hereditary and congenital peculiarities', with defects in the 'subtle and obscure process of central nerve development in childhood' acting as predisposing agents. He further postulated that certain areas of the brain 'running ahead' in their development, compared to others, were causing the derangement of its function. This in turn caused 'explosions which spread into other centres'.

A series of morbidities was outlined which, as Clouston proposed, all resulted from the same pathology; one of these resembles the attention deficit hyperactivity disorder of today. He called it 'simple hyperexcitability', arguing that the disorder resulted from 'undue brain reactiveness to mental and emotional stimuli'. It affected children from the age of 3 years until puberty, with overactivity and restlessness being the main symptoms. The disorder came in bursts and lasted from a few months to years. During the bursts the child would grow thin, not sleep and deteriorate in educational performance. Such clinical signs were ascribed to the children showing a 'delirium of pleasure in response to nice things', and interpreted as an exaggeration of the response one would expect from a child of nervous temperament.

The common feature of such disorders, according to Clouston, was the 'explosive condition of the nerve cells in the higher cortex'. In his view this was comparable to the overactivity of the motor cortex seen in persons suffering from epilepsy. Referring to the by then well-established findings that the process of brain development in childhood comprises rapid cell multiplication accompanied by the cells gradually becoming more stable, he argued that in the children suffering from 'hyperexcitability' this process of nerve stability does not occur, resulting in the children growing up with 'irregular explosive tendencies'.

The recommended treatment for such conditions was the use of bromides, 'fearlessly in large doses up to the point when symptoms of bromism are beginning to show themselves'. Clouston did, however, emphasize that the drugs should not be used in isolation. Instead, the children should at the same time be given a good diet, plenty of fresh air, 'suitable amusement, companionship and employment'. The aim of the treatment was to 'reduce the cell catabolism and the reactiveness of the cerebral cortex whilst not interfering with brain anabolism'. The treatment had to be carefully monitored in order to ensure that it did not 'go too far'.

Still and the defect of moral control

The first clear, systematic accounts of hyperactivity are attributed to Sir George Frederick Still (1868–1941), a paediatrician and the first professor of children's

diseases at King's College Hospital, London. Professor Still is, however, most notable for his description of chronic rheumatoid arthritis in children, commonly known as Still's disease. In 1902, Still presented the Cloustonian lectures to the Royal College of Physicians describing the case histories of 20 children, whose presentation was similar to what we at present would call hyperactivity. In his descriptions, features such as extreme restlessness and 'almost choreiform' movements were frequently mentioned. Another common characteristic was that of 'an abnormal incapacity for sustained attention', causing school failure even despite normal intellect. In their behaviour, many of the children were mischievous, destructive and violent, and appeared not to respond to punishment.

According to Still, this pattern occurred more often in boys than in girls, became frequently apparent by early school years, was sometimes accompanied by peculiarities of physical appearance (e.g. epicanthic folds and high-arched palate), seemed often to be a function of temperament, generally showed little relationship to the child's training and home environment, and commonly shared a poor prognosis. Thus, Still described many of the characteristics and associations we recognize today. Still suggested that the children he was describing suffered from a 'defect of moral control', whereby they demonstrated 'the reckless disregard for command and authority in spite of such training and discipline as experience shows will render a healthy child law-abiding'. Instead, such children displayed 'immediate gratification of self without regard either to the good of others or to the larger and more remote good of self'. Still also noted that, although many of the children with this condition came from chaotic families, a substantial proportion of them lived in homes where they appeared to receive an adequate upbringing. In fact, when refining his criteria for children with the disease, Still decided to exclude those who had been exposed to poor child-rearing. This led him to hypothesize that the 'defect of moral control' was due to some 'morbid physical condition', which was either inherited or resulted from a perinatal or postnatal injury.

The degree of uncertainty about the causation of the condition provided a logical basis for separating the children with such problems into subgroups. Still proposed a distinction between children 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, although none could be demonstrated; and those with hyperactive behaviours that could not be attributed to any known cause. Thereby, Still laid the groundwork for the historical equivalents of three major diagnostic categories of brain damage, minimal brain dysfunction and hyperactivity. In doing this, he also sowed the seeds for a terminologi-

cal confusion so prevalent in the literature of hyperactivity over decades to come, but confusion that also gave the impetus for much excellent research on the nature of hyperactivity and its treatment.

Still's theories about his patients are best understood in the context of the prevailing socioeconomic and scientific climate. During the nineteenth century the UK underwent significant economic, political and social change. The economy became increasingly centred on factories in small towns, moving away from farming and the land. Unemployment was common and those in employment often worked long hours and were poorly paid. A distinct class hierarchy dominated society, with the lower classes being perceived as immoral and inferior (Rowntree, 1901). Considerable adversity afflicted the lower classes as a result of the socioeconomic changes and this was reflected in rising infant mortality, poor physical health in general, and learning difficulties and delinquency in children. However, the intellectual and moral deficiencies of the lower classes tended to be identified as the cause, rather than the consequence, of their circumstances.

Concurrent with these developments was the rise of positivism in contemporary science, with beliefs that the progress of society could be achieved through the development of objective science. It was especially the theories of Darwin that provided a scientific rationale for various kinds of social deviance, with hypotheses suggesting that the environment conferred a selective advantage to some types of biological variation (Darwin, 1859). Deduced from his theories, the notion for the 'survival of the fittest' became elevated to the status of a 'law', in an attempt to explain social phenomena. Likewise, poor health could easily be viewed as a form of inherited weakness and inferiority. This social Darwinism soon found wide support among intellectuals and social reformers.

In keeping with the prevailing trend of his day, Still was keen to adopt the principles of social Darwinism and set out to explain the 'defects of moral control' of the children he was asked to treat. He claimed that moral consciousness and moral control were essentially innate characteristics. They were also 'the highest and latest product of mental evolution'. However, because they constituted a relatively recent evolutionary advance, they were also fragile and showed 'a special liability to loss and failure in development'.

Hyperactivity due to neuropathic diathesis

To support Still's reports of hyperactive behaviour pattern occurring when brain damage was suspected, but could not be substantiated, Tredgold (1908) presented further evidence in children. He proposed that some forms of brain

damage, such as birth injury or relatively mild anoxia, though undetected at the time, could express themselves as behaviour problems or learning difficulties when the child is faced with the demands of early school years. Alfred F. Tredgold was a member of the English Royal Commission on Mental Deficiency. His book, *Mental Deficiency (Amentia)*, was published in 1908, updated in 1914, and remained in publication until 1952. In the book, Tredgold described many children who exhibited features of hyperactivity, and he is attributed by a number of authors (Ross and Ross, 1982) as being the first to provide an account of 'minimal brain damage'.

Tredgold's descriptions of hyperactivity derived from his observations of a group of child patients whom he labelled as 'high-grade feeble minded'. Although incapable of making use of education as provided in school, such children would in his judgement nevertheless benefit from individual attention and instruction. He also noted that a number of the children exhibited a variety of physical anomalies, including abnormalities of the palate, soft neurological signs, abnormal head shape and size and poor coordination.

Apart from being educationally inferior, the children were also prone to criminal behaviour, despite having been raised in an adequate environment. Tredgold shared Still's belief that moral deficiency resulted from the effect of some 'organic abnormality on the higher levels of the brain', and argued that the areas of the brain where the 'sense of morality' was located were the product of the more recent development in the course of human evolution, and were therefore more susceptible to damage. Tredgold believed that such moral deficiency was caused by the inheritance of some brain defect that was being passed on from generation to generation: being able to take various forms, it resulted in hyperactivity, migraine, mild forms of epilepsy, hysteria and neurasthenia. He called the defect by various names, such as 'neuropathic diathesis', 'psychopathic diathesis', 'blastophoria' or 'germ corruption'. In his view, environmental circumstances played no significant role in the causation of such mental or moral deficiency.

> With regard to the influence of slum life and all its associated conditions in producing amentia, it is necessary to sound a note of warning. It does happen sometimes that the real mental defectives of our large towns hail from the slums, although I do not think such is disproportionately the case. Still, a sufficient number of defective children come from such areas to make the superficial enquirer content with that which is apparent, jump on the conclusion that the pernicious environment is therefore the cause of their defect. My own enquiries have convinced me that in the great majority of these slums cases, there is pronounced morbid inheritance, and that their environment is not the cause, but the result, of that heredity (Schachar, 1986, p. 23).

In the decades that followed, a wide range of deviance was attributed to the interaction of brain disorder and constitutional predisposition by leading medical authorities on both sides of the Atlantic. Such biological variation could in turn have several different outcomes, ranging from school failure (Cornell, 1912), to criminality (Healy, 1915). In contrast, psychological and social explanations for cognitive and behavioural deviations were explicitly rejected. The core debate revolved around the relative contributions of inheritance and birth injury as factors of prime importance in leading to disturbed adaptability towards one's surroundings (Henderson, 1913).

Sequelae of the encephalitis epidemic

The link between hyperactive behaviour and demonstrable brain damage was strengthened by the epidemic of encephalitis spreading across Europe and the USA in 1917–18. In its aftermath, many clinicians encountered children who, though having survived the infection, subsequently presented with behaviour problems and cognitive deficits. Hyperactivity, catastrophic changes in personality and learning difficulties were among the predictable sequelae of the disease (Hohman, 1922; Ebaugh, 1923). The term 'postencephalitic behaviour disorder' was adopted to encompass the various consequences. Observations of the child victims of subsequent outbreaks of encephalitis confirmed the same pattern of symptoms (Bender, 1943; Gibbs et al., 1964). Cantwell (1975) and many others date the development of North America's interest in hyperactivity to the encephalitis epidemic.

Hohman (1922), Ebaugh (1923) and Strecker and Ebaugh (1924) argued that the children who showed persisting problems with behaviour following the epidemic were also the ones who had been most severely affected by the disease, and in most cases were left with severe brain dysfunction. As such, however, only the problems of few of the children described would fit the present-day criteria for attention deficit hyperactivity disorder. It is also to be acknowledged that the available evidence was for an association between severe damage and severe behavioural disturbance. For some reason, this was subsequently extrapolated to claim that a similar connection existed between minimal brain damage and lesser degrees of disordered conduct.

As was the case with Still's disease, the influences of social Darwinism were also brought to bear on the sequelae of encephalitis, with the assumption that there was some inherited predisposition to developing the disease. People who contracted illnesses, such as encephalitis, were believed to be in some way constitutionally inferior (Bassoe, 1922; Bond and Appel, 1931).

Hyperkinetic disease

In the early 1930s, Kramer-Pollnow described a syndrome characterized by extreme restlessness, distractibility and speech disorder. He called it 'hyperkinetische Erkrankung' (hyperkinetic disease) and classified it as a form of childhood psychosis, usually of unknown origin (reported by Hoff, 1956, pp. 537–53). The extreme restlessness, commencing in the third or fourth year of life, often came on suddenly, 'after a period of quiet', and was frequently followed by an epileptic seizure. The restlessness reached its most severe form at the age of 6, and then gradually decreased, with most cases achieving almost complete recovery. Disturbances of speech and general mental development were often noted before the onset of restlessness.

The children were also described as being easily distracted. Their excessive motor activity, chaotic and aimless in nature, appeared to occur as a succession of uncorrelated impulses with no aim other than to respond to a stimulus. Their play also seemed to lack purpose, with toys tending to be broken rather than played with. The 'lack of discrimination', as noted, bears a resemblance to the 'impulsivity' described in children suffering from hyperkinetic disorders of today. The paucity of the children's interpersonal relationships was likewise remarked upon. They were also more often aggressive towards other children rather than playing with them. Any attempts to restrain the child would be met with opposition and struggling.

The speech disorders, as recorded, consisted of poor articulation and 'inarticulate labelling'; the children's vocabulary was thought not to increase until their recovery from the condition. However, their intelligence, when observed daily at home, appeared to be higher than that detected on formal cognitive testing. It was also argued that the disorder could be differentiated from others such as schizophrenia, dementia infantilism, encephalitis and schizophrenia-like psychoses.

In total, 15 children presenting with 'hyperkinetic disease' were described. Of these one died, three suffered from definite mental defect, three recovered from the restlessness but were left with permanently impaired intelligence, four recovered partially, whilst another two recovered completely. The remaining two children were less than 7 years old and were therefore felt still to be within the 'hyperactive stage'.

A small compilation of cases with similar presentation had also been published in Italy a few years previously (de Sanctis, 1925). Indeed, after having examined the evidence collected by both Kramer-Pollnow and de Sanctis, Hoff concluded the topic of 'Hyperkinetische Erkrankung', as part of his three-lecture series on child psychiatry to his Viennese medical students, by adopting