HISTORY

<http://www.med.uottawa.ca/historyofmedicine/hetenyi/nefsky.htm>

A CONCEPTUAL HISTORY OF ATTENTION DEFICIT AND HYPERACTIVITY DISORDER

Colman Nefsky, B.Arts and Sc., Summer 2004

This study was funded by the Geza Hetenyi Memorial Studentship for the History of Medicine administered by the University of Ottawa.

Colman Nefsky is a first year student at the Faculty of Medicine, University of Ottawa

1.Acknowledgements

2.Introduction

3.What is ADHD

4.The changing concepts of ADHD

5.Interpretation of the history of ADHD

6.References

Acknowledgments:

This project was funded by the 2004 Geza Hetenyi Memorial Studentship for the History of Medicine in the Faculty of Medicine and Health Sciences at the University of Ottawa. I would like to thank my supervisor, Dr. Toby Gelfand, for his insightful feedback, encouragement and patience. It was a privilege to discuss the history of psychiatry with Dr. Gelfand, and his wealth of knowledge sent me towards interesting new sources and in unexpected directions. Though not involved in this particular project, Dr. William Mahoney guided me in my first academic exposure to ADHD. Dr. Mahoney fed my interest in the condition with many hours of discussion and feedback. His efforts in listening to and channeling my ideas not only helped me with my work at the time, but left me keen to learn more about ADHD.

Introduction

Attention Deficit and Hyperactivity Disorder (ADHD) and the treatment of children with Ritalin has become a highly publicized issue in recent years, however the disorder has a history that spans over a century. This is a particularly interesting history, as it has been characterized by constant, rapid change and a considerable amount of controversy. Although the disorder is widely regarded as a recent phenomenon, several authors have given historical accounts of ADHD. These authors can be categorized according to their academic disciplines and their approaches to the subject.

Most histories of ADHD are located in the beginning chapters of textbooks written by experts in the fields of child psychiatry and psychology, intended to be read by other clinicians as well as interested members of the general public. Such writers are often referred to by historiographers of medicine as “internal historians”, since they write from perspectives within the field that is the subject of historical study. Therefore, they are often considered to offer relatively uncritical histories that express reverence of the major figures of their field and faith in scientific progress. This stereotype is an exaggeration, as internal historians are often quite critical of their predecessors in their field, though they may not always approach the same degree of questioning and analysis that other historians bring. To find the major internal histories, one simply seeks the major textbooks, some of which contain quite detailed history chapters.

Histories written by people outside of the medical or mental health fields are less prevalent. Some accounts are given by writers who are skeptical about the very concept of ADHD. Although much writing from the popular press and academic fields such as sociology has questioned the legitimacy of ADHD diagnosis, most of these publications focus on contemporary rather than historical issues (Singh, 2002, 579). The few such publications that delve into the history can be called “anti-psychiatry” histories, since their accounts reflect the authors’ hostility to the psychiatry profession’s role of diagnosing and treating ADHD. Anti-psychiatry histories emphasize the importance of commercial interests in the establishment of a lucrative market for psychotropic drugs and political interests in the “social control” of deviant youth.

Finally, a handful of academic articles have been written by social scientists who avoid the extreme stance of anti-psychiatry historians, while taking more critical approaches than most internal historians. These social scientists attempt to better understand the processes involved in generating and popularizing psychiatric concepts. To do so, they examine the influences of professional interests, politics within the profession and social norms, as well as scientific discoveries and technological advances.

This paper examines the history of medical ideas about ADHD, and therefore is largely a synthesis of the major internal histories. I analyze these histories based on my own readings of primary sources: psychiatry journal articles and books published at different times in the history of the disorder. In my final discussion, I will integrate some of the insights offered by the social scientists. To limit the scope of the paper, I will not discuss the anti-psychiatry historians, whose interesting and controversial accounts are generally dismissed by the other schools of thought as biased by their strong anti-psychiatry positions. I will focus mainly on the development of concepts in North America, where ADHD has been found to be most prevalent1 and is most controversial. But one cannot isolate American thought about ADHD from European and especially British thought, as the two groups closely interacted and together built a common body of research. Before discussing its history, it is important to give an adequate definition of Attention Deficit and Hyperactivity Disorder.

1.Estimates for prevalence tend to range between 3 and 10% of American school-children, 5% being a widely cited figure.

What is ADHD?

Attention Deficit and Hyperactivity is a behavioural disorder characterized by problems with attention, impulsive behaviour and excessive motor activity. It is most prevalent in school-aged boys, though it is also diagnosed in females, adolescents and adults. Interest in these later groups has developed mainly in the past two decades (see Doyle, 2004; Pary et al., 2002; Mannuzza et al., 1998). Previously, it was frequently believed that children outgrew the disorder by adolescence. This belief was not completely misguided: many people diagnosed with ADHD as children indeed improve to the point where they experience no significant problems associated with the disorder. But currently, it is believed that most people are affected by the disorder throughout their lives. Nevertheless, ADHD is widely seen as a developmental disorder, as it typically slows the intellectual development and social adjustment of children from a young age. The term ADHD is used largely in North America, and has been adopted to a certain extent in some European countries as well in other countries such as Australia and New Zealand. The corresponding term used more widely in Europe is Hyperkinetic Disorder.

The Primary Symptoms

The main symptoms that define ADHD are inattention, impulsiveness and hyperactivity. These problems are considered in the context of “normal” or average behaviour for a given age and gender group. Attention, impulsiveness and hyperactivity are all multidimensional constructs, in that they each can be broken down into a collection of more specific features. Attention includes many dimensions such as alertness, selective attention and sustained attention (Barkley, 1998, chap. 2). In ADHD problems with attention primarily involve sustained attention. These differences can be observed in young children who rapidly shift from toy to toy when playing or in older children who show little persistence of effort in tasks that lack intrinsic appeal or immediate rewards. Such tendencies naturally contribute to the academic difficulties of many children with ADHD.

People with ADHD have difficulty inhibiting behaviour in response to situational demands. Like attention, inhibition can be broken down into more specific constructs. For example, momentary inhibition describes the ability to abruptly stop a planned action according to changes in a situation (Tannock, 1998). Ongoing inhibition involves suppressing an impulse over an extended period of time, which permits a delay in response in which a person can process information or make a decision so as to execute a more deliberate action. Problems with inhibition are manifested in a greater tendency to speak out of turn and to engage in unnecessary risk-taking behaviour. A lack of restraint with peers can lead to social problems. Impulsive behaviour can also result in discipline problems in school, and poor inhibition might relate to a tendency to work quickly and inaccurately and therefore to academic problems.

Hyperactivity can be the most obvious feature, especially in young children. It can include fidgeting, a need to move around excessively and a tendency to make unnecessary, unusual gross bodily movements. The term has also been expanded to include vocal “overactivity”; for example, the tendency to make constant “running commentaries”. This vocal form of hyperactivity is a useful concept, in part because it can be applied to adolescents and adults who are no longer physically hyperactive.

Not only are these symptoms all multidimensional constructs, they also overlap and interact with each other. For example, a short attention span can lead a child to move around excessively, and therefore can contribute to hyperactivity. Poor sustained attention on difficult or unrewarding tasks can be attributed to an inability to inhibit impulses to quit the dull task and find a new, more rewarding pursuit. Thus it is difficult to separate the elements of ADHD, and this can lead to confusion in studying the disorder and assessing children for diagnosis.

Diagnostic Criteria

The current diagnostic criteria for ADHD are outlined by the American Psychiatric Association’s fourth edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), published in 1994 and revised in 2000. The Manual splits ADHD into two dimensions: inattention and hyperactivity/impulsivity. It defines the dimensions by listing nine behavioural symptoms under each. For example, hyperactive symptoms include: “Often fidgets with hands or feet or squirms in seat,” and “Often runs about or climbs when and where it is not appropriate” (American Psychiatric Association, 1994). If the patient has demonstrated six of the nine symptoms of inattention persistently for the past six months then they are considered to have the Predominantly Inattentive Type of ADHD. If the patient has six of the nine symptoms of hyperactivity and impulsivity then they are considered to have the Predominantly Hyperactive-Impulsive Type of ADHD. If they demonstrate six symptoms in both dimensions then they are considered to have ADHD, the Combined Type (Barkley, 1998, ch. 2; American Psychiatric Association, 1994). This complex system of subgroups reflects the long-time dilemma of how to sub-classify the heterogeneous group of ADHD patients. The dimensions in the DSM-IV are based on statistical analysis of a large population study of the disorder. They were determined to be the most empirically supported subtypes, but do not represent any definitive, fundamental knowledge about the disorder. Therefore, child psychiatry experts expect the subtypes of ADHD to change in the future. Problems relating to the classification and sub-typing of ADHD constitute a prominent theme in the history of ADHD, especially in the recent decades.

Its symptom lists are not the only criteria required by the DSM-IV for the diagnosis of ADHD. The patient must also have presented some symptoms before the age of seven years2. This reflects an age of onset thought to be characteristic of ADHD in contrast to cases where behavioural symptoms are brought on by social influences. Symptoms must also be present in more than one setting, a further measure to differentiate ADHD from behavioural problems caused by problematic features of the environment at home or school. Finally, the symptoms must clearly cause “significant impairment in social, academic, or occupational functioning”(Barkley, 1998, p. 12). Thus a patient should not be diagnosed based on unusual behaviour patterns that seem to have little negative impact on them. The diagnosis of ADHD is meant to be an extensive process, utilizing at least one expert clinician, an office assessment of the patient and detailed information from parents and teachers. This is often accompanied by standardized, psychometric tests and observation of the patient by a clinician in different settings such as school and home.

2.According to Doyle, the text revised DSM-IV of 2000 creates a special subcategory for patients who meet ADHD diagnostic criteria except the age of onset. This can be seen as a recognition that a cut-off age of 7 was largely representative of the disorder, but arbitrary. Alternatively, it can be seen as a change that expands the ADHD category, thus enabling diagnosis of more patients.

The Changing Concepts of ADHD

Since early studies around the turn of the 20th century, hyperactive, impulsive children with poor attention spans have been described by a succession of names, such as Defect of Moral Control, Mild Mental Deficiency, Hyperactivity Disorder, Mild Brain Dysfunction, Hyperkinetic Syndrome and ADHD3. Similar but not identical groups of children were diagnosed under these categories, and so the prevalence of such conditions has naturally varied significantly according to time and place. The changing names mirror changing opinions about the nature and causes of the above behavioural symptoms.

A prototypical history of a disease might center around a major breakthrough in understanding that illuminated the cause of the problem. In the absence of such a breakthrough, a model history might at least involve a gradual process of gaining an increasingly specific understanding of the causal mechanism, facilitated by the development of new technologies and the accumulation of scientific knowledge. However, understanding of the causation of ADHD does not seem to follow either of these models. Rather, etiologic theories have constantly shifted between different causal mechanisms, implicating a wide range of brain structures, heredity, acquired brain injury and other environmental factors. Although there have certainly been major advances in the field due to a large body of sophisticated research, there remain numerous proposed mechanisms and a significant amount of contradictory research findings to this present day. This mysterious etiology is certainly not unique to ADHD, as many similar puzzles lie behind other psychiatric and somatic diseases. But such uncertainty would be particularly significant in the history of a controversial disorder such as ADHD, where some disbelievers consider the disorder to represent a social construct rather than scientific discovery.

Along with the shifts in etiologic ideas, focus has shifted between different clinical aspects of the disorder. Each of the major features of ADHD – hyperactivity, impulsiveness and inattention – have at different times been regarded as the central symptom that characterizes the disorder, with the other symptoms being regarded as peripheral. ADHD has proven so difficult to understand, in part because it is a heterogeneous disorder, with a wide range of severity and clinical presentations, and because it likely is brought about by a heterogeneous group of causes. Thus throughout the past century, researchers have struggled with two problems: firstly, defining and characterizing the disorder, and secondly, determining its etiology. At different points in the history, researchers were focused on one task or the other.

George Still and the “Defect of Moral Control”

Child psychiatrists writing about ADHD typically trace the study of the disorder to George Still, an English pediatrician at the turn of the 20th century and te first professor of children’s diseases at King’s College Hospital in London, most famous for his description of chronic rheumatoid arthritis in children (Still’s disease) (Schachar, 1986, 21). Still described and analyzed case studies of twenty children in a lecture series in 1902, which was published in the Lancet. Some of the children he described demonstrated very similar characteristics to children who currently fall under ADHD diagnosis. He observed them to be overly emotional or “passionate”, and many were noted for their lack of “inhibitory volition” and “lack [of] power of attention”. Some displayed exaggerated, “fidgety, almost choreiform” movements (Still, 1902, pp. 1077, 1080, 1082). They were unusually resistant to discipline, showing genuine fear at the time of punishment, but immediately repeating their misbehaviour afterward. Many of these children performed at a low level in school despite apparently normal intelligence. Some persistently stole, although not out of particular use or desire for the objects: for example one young child would present stolen objects to his parents every day, despite consistently being punished. He described their behaviour as mischievous, destructive, dishonest and spiteful.

Still considered this behaviour to represent a “defect of moral control”, which appears in nine behavioural qualities: “passionateness”, or excessive emotion; spitefulness towards other people and cruelty towards animals; lawlessness, or “frequent failure to conform with the law” and with rules at home and school; dishonesty, characterized by lies that did not seem to have a purpose, but resembled the fanciful stories of toddlers rather than careful deceit; destructiveness and wanton mischievousness; shamelessness and immodesty; sexual immorality, which was a label commonly applied to deviant girls at the time (Jones, 1999); jealousy; and viciousness (Still, 1902, p 1009).

He theorized that people are not born with “moral control” but develop this capacity in childhood (Still, 1902, 1078). It is affected by the environment, especially by parenting, but, at the core, determined by an inborn capacity for moral control that varies among individuals. While such variation naturally includes some people with slightly less ability to channel their behaviour according to moral and legal norms, Still suggested that there are some people whose level of moral control is much lower than that of most of the population, and therefore a reflection of pathology rather than normal variation. These children’s behaviours were distinctive enough to set them apart from hundreds of others in schools and institutions, leading Still to attribute such unusual behaviour to “a morbid defect”: “this excessive degree of the defect, the outrageous character of its manifestations, is one point which, although insufficient as evidence of morbidity when taken by itself, may be important evidence when taken in conjunction with other facts” (Still, 1079).

Still drew on his case studies to propose different causes of this defect in moral control. In some cases somatic disease, such as cerebral tumour, meningitis, head injury and epilepsy, caused brain damage and subsequent behavioural problems. The histories of other children revealed problems during infancy believed to have disturbed cerebral development. A large proportion of the children had no such histories, but had family members who were diagnosed with neuroses or with the then-popular label “feeblemindedness”4, or who had been drunkards or suicidal, thus suggesting familial transmission of mental or psychological weakness. He took care to show that in many cases the behavioural problems occurred in situations of apparent psychosocial advantage and stable, supportive families (Spencer, 2002). Thus Still deemphasized the possibility of psychosocial causes and proposed that defects in moral control could occur due to brain damage, abnormal cerebral development and heredity.

Russell A. Barkley, one of today’s most prominent ADHD researchers and the author of one of the most extensive internal histories seems to identify with Still’s work, as he draws parallels between Still’s focus on the internal control of behaviour and Barkley’s own current theory on ADHD that emphasizes problems with behavioural inhibition (Barkley, 1998, p. 5). Indeed, Still relates the behavioural characteristics that he observed to “the immediate gratification of self without regard either to the good of others or to the larger and more remote good of self” (Still, 1902, p. 1009). This corresponds with Barkley’s view of ADHD as problems with the executive functions that limit ability to plan and organize behaviour according to future consequences. Still’s observations also demonstrate a notable correspondence with our current view of ADHD in separating the disorder from intellectual deficiency. He maintained that a defect in moral control occurs independently of deficits of intellect, and focused on cases of children with normal intelligence. This is distinctive from the predominant medical approach at the turn of the 20th century, which grouped both the deviant and the mildly mentally handicapped under the previously mentioned label, “feeblemindedness”. Thus Still’s work perhaps represented an early stage in identifying a group of children with a distinctive pattern behavioural characteristics from the total population of “problem children” based on supposed evidence of biological causation.

On the other hand, Barkley notes "the degree of maliciousness and extreme misbehaviour” that was observed in these children, suggesting that many of them would currently be considered to have Conduct Disorder, today’s label for children and young adults with severe behavioural and attitude problems, for example those who commit criminal or violent acts (Barkley, 1998, p. 5). For the past several decades, categories such as Conduct Disorder, Hyperactivity and Attention Deficit Disorder have interacted with each other, since they describe children with many similar problems. While there currently appears to be a general consensus to separate ADHD and Conduct Disorder, researchers find correlations between the behavioural symptoms that define the two conditions. Some skeptical writers argue that ADHD and Conduct Disorder are not sufficiently distinguishable to be considered different conditions. Current diagnosis of ADHD leads to a significant amount of co-morbidity with Conduct Disorder and other psychological conditions. Many experts expect that ADHD’s boundaries with other developmental disorders and the subgroups within ADHD will change in the future. Thus beginning with Still, hyperactivity and impulsiveness was rarely, if ever, studied alone, but rather grouped with other similar childhood problems, such as aggression and learning disabilities.

Authors such as Russel Schachar demonstrate that Still’s theories were very influenced by the social prejudices in nineteenth century England. His emphasis on biological factors, most often familial inheritance, over environment reflected then-popular beliefs of Social Darwinism that were used to explain the urban social underclass created by the industrial revolution. According to these principles, many people of the working class lived under poor conditions and suffered from poor health because of an inferior inheritance of traits (Sandberg and Barton, 2002, p. 7). These attitudes made an impact on psychiatry, as Social Darwinism, combined with the lack of the therapeutic success and increasingly custodial role of asylums, led to increasingly pessimistic attitudes in the profession. In his influential “degeneration theory,” B.A. Morel portrayed mental illness as an inherited, incurable mental defect. Along with providing a medical theory about mental illness, this explanation rationalized the lack of therapeutic success and explained “social evils” seen in cities (Micale, 2000, 327).

These ideas seem to be integral parts of the theoretical framework behind Still’s writing. For example, as evidence of a biological disorder, he points to the presence of “stigmata of degeneration”, minor physical anomalies thought to be typical of the feebleminded (Still, 1902, 1079). To this day, scientists look for physical anomalies in children with ADHD and other psychiatric disorders. However the terminology used by Still and his contemporaries reveal the framework of degeneration theory, a scientific concept that would now be considered not only outdated, but also embarrassing for its relationship with the ideology of Social Darwinism. Moreover, Still drew more heavily on Social Darwinism when he explained the occurrence of children with normal intellect but defects in moral control. He proposed that moral control is the most recent feature of human evolution. But since it is such a recent development, it is also very fragile, and therefore vulnerable to loss or damage during abnormal childhood development. Thus deficiencies in self-control were implicitly associated with lower evolutionary status.

Still’s lectures did not seem to have a conscious social agenda, but, read a century later, display some of the idiosyncrasies in medical thought at the time. These idiosyncrasies were not necessarily later overcome by superior, objective science, but, were inevitably replaced by new science and new ideologies. Current ADHD experts like Barkley who face accusations that the ADHD diagnosis is largely a product of social and political forces may find comfort in the fact the same symptoms were identified by Still over a century ago, in a very different society than North America of the 1990’s and 2000’s. The age of a psychiatric disorder such as ADHD might lend it some legitimacy, by contributing to a perception that the disorder was “discovered” as opposed to “constructed”5. But, like Schachar, psychiatrists and psychologists may also be shocked at the social premises that lay behind Still’s research. Barkley recognizes that the influences of Social Darwinism are present in Still’s research, but counters that social influences are present in all research, and can be particularly evident in the behavioural sciences (1990, p. 5). Andrew Lakoff, one of the “social scientist” historians whose article I will discuss later, contrasts our current views of ADHD to those of Still, in order to demonstrate that the meaning of medical conditions at a particular time and place is reflective of the values, norms and expectations of the particular society.

Tredgold and “Feeblemindedness”

ADHD experts identify British physician Alfred Frank Tredgold’s major textbook “Mental Deficiency (Amentia)”, first published in 1910 and reprinted numerous times, as the next source that gives in depth descriptions of children that portray the traits of hyperactivity, impulsivity and lack of attention. He used the common term “feebleminded” to classify people considered to have a mental defect without severe intellectual impairment. Their deficiencies were considered to primarily be in commonsense, judgment, inhibitory control of primitive instincts and “active attention”, which left patients “lacking the ability to control, co-ordinate, and adapt their conduct to the requirements of their surroundings” (Tredgold, 1952, 162-3, 328). As a result they suffered from problems in managing themselves, to the extent that they usually lived dependent on family members, or else often encountered problems such as mismanagement of money, involvement in crime and being a victim of scams. Similar symptoms and social repercussions are now associated with ADHD. It appears that feeblemindedness had become a public concern, as the text cites government estimates of a prevalence, which ranged from 1.25 and 0.15 percent in urban areas and a slightly lower rate in rural areas. The incidence was found to be only slightly higher in boys.

Tredgold also described high-grade, but mentally unstable feebleminded patients who were susceptible to “restlessness, incessant chattering, numerous tricks and habit spasms, and general muscular overaction and inco-ordination” as well as “to fits of temper and sudden impulses, …general waywardness and unreliability” (p. 159-61). They generally showed higher intelligence than placid, mentally stable feebleminded children. This contrast resembles the distinction later made between Attention Deficit Disorder with and without Hyperactivity. Later, in his discussion of criminality, he differentiated between feebleminded criminals and “moral defectives”: those whose sole illness is a mild mental deficiency commit criminal and socially unacceptable acts simply because of their lack of control over their impulses, while moral defectives have strong antisocial instincts that are not controlled because of a mental deficiency (159-61). This suggests a distinction similar to our current separation of ADHD and Conduct Disorder that Still did not make.

Tredgold considered feeblemindedness to be the least severe degree of mental deficiency, or “amentia”, which also included the more intellectually limited conditions of “idiocy” and “imbecility”. Thus while the feebleminded were not necessarily less intelligent, but rather impaired in the highest functions of morality, judgment and self control, they were seen as a group continuous with the intellectually handicapped; all mentally defective but to different degrees. Like Still, he viewed morality as a recent evolutionary development and therefore more susceptible to damage, and he attributed this damage to a variety of causes, such as faulty genetic inheritance (“primary amentia”) and early brain damage (“secondary amentia”). He included descriptions and pictures of pathological and histological differences in the cortex of mentally deficient brains, though acknowledging that these changes do not occur consistently in amentia, are much more infrequent in mild feeblemindedness, and are not necessarily indicative of mental defect (Tredgold, 1952, pp. 110-122). He also found physical anomalies – still referred to as “so-called stigmata of degeneracy” – in his cases of amentia, including feeblemindedness: these included a smaller cranial circumference and abnormal head shape, along with anomalies in the palate, eyes and ears. While at the time interest in these anomalies may have been tied to theories of degeneracy, researchers would continue to look for such anomalies in their attempt to better identify what they believed was an organic condition.

Thus, Tredgold decidedly expressed an organic approach to problems with behaviour and social adjustment. While he acknowledged that family environment can influence the expression of mental deficiencies, he maintained that they must originally be caused by a weaker hereditary constitution or possibly by problems surrounding pregnancy. Thus in some families a brain defect or vulnerability to developing one was passed on, which could manifest itself in forms such as hyperactivity, subnormal intelligence, migraine, hysteria and epilepsy; he referred to this underlying defect by names such as “neuropathic diathesis”, “psychopathic diathesis”, “blastophoria” or “germ corruption”. Moreover, he argued that a faulty family environment was a result of an inferior heredity manifesting itself in family members and their ways of life (Sanberg and Barton, 2002).

Tredgold used these medical ideas to explain social problems, such as alcoholism, prostitution and pauperism. A strong supporter of the then-popular eugenics movement, he warned that degenerate people detract from the country, and advocated sterilization of the mentally deficient (Schachar, 1986, p. 23). Therefore he seems to be looked upon less fondly by the internal historians, in comparison to Still, whose ideological influences are less prominent. However, control of reproduction was not the only way he proposed to deal with mental deficiency. In his book, he discussed medical and psychosocial treatment, and emphasized modifications to education and training either at home or in institutions (Tredgold, 1952, p. 443). The book was dedicated to “all those persons of sound mind who are interested in the welfare of their less fortunate fellow-creatures”; a statement far from what we would now call empathetic, but certainly expressing some concern with the plight of people with mental deficiencies, rather than simply voicing intolerance towards them. Though Tredgold believed their conditions were incurable and rarely showed much improvement, he was interested in helping caregivers to improve patients’ lives. At the same time, he outlined only one approach to treatment for the wide range of “aments”, who varied considerably in their capabilities and needs. Tredgold regarded them as essentially variations of the same degenerative condition, and this seemed to have limited his discussion of treatment to one general approach. Tredgold’s eugenics attitudes did not completely undermine the scientific or therapeutic nature of his work, but they did exert significant influence on his scientific inferences and ideas for treatment.

The medical ideas in his book, based on case studies, data from clinical findings on a large sample of patients and contemporary psychological and medical understanding, gave confident medical explanations for social problems that were of great concern at the time. His focus on heredity and degeneracy has been influenced by the eugenics movement, and at the same time validated these ideas with scientific evidence. Thus clinical descriptions of patients that were in many ways very similar to later descriptions of hyperkinetic disorder and ADHD took on a much different meaning in this particular social and intellectual environment.

Approaches Following Still and Tredgold

Sandberg and Barton write that in the years following Tredgold’s publication, the mental health professions in North America and Europe explained a wide range of deviant behaviours in young people by “brain disorder and constitutional predisposition”. Debate “revolved around the relative contributions of inheritance and birth injury” rather than the significance of biological factors in comparison to psychosocial factors (Sandberg and Barton, 2002, p. 9). Social Darwinism and the related Eugenics movement continued to be popular, their influences possibly contributed to assumptions about the organic causality of deviance. On the other hand, in the early twentieth century United States, “child guidance” clinics run by psychiatrists, psychologists and social workers in co-operation with juvenile courts, had a largely social and, later, psychodynamic, approach to deviant behaviours in children (Jones, 1999, pp. 44-5). Sandberg and Barton cite the book The Individual Delinquent (1915) by William Healy, one of the founders of the child guidance movement, when they make their claim that there was already an emphasis on biological etiology in the 1910’s. However, this appears to conflict with Kathleen W. Jones’s portrayal of the Healy and the child guidance movement in her book about the history of this movement, Taming the Troublesome Child. Child guidance essentially marked the beginning of a cohesive child psychiatry profession in the United States. According to Jones, Healy’s work and the Child Guidance movement was based on assessing each individual child to understand the complex combination of causal factors that contribute to their deviance; sometimes these factors included psychological or mental disorders or somatic medical conditions, but they more consistently included social, economic and familial factors. Thus it is surprising that Sandberg and Barton cite Healy when discussing the organic explanations for behaviour problems. Perhaps there was a wider range of approaches to deviant behaviour in the early 20th century than they acknowledge, although this would not necessarily discount their claim that there was little debate between proponents of organic and social etiologies. It is very possible that different schools of thought did not interact and therefore did not engage in such debate. For example, the child guidance movement published in the specialized journals such as Mental Hygiene, and thus might have produced a literature separate from that written and read by neurological and biomedical schools of thought. At the least, such a lack of communication and mutual acknowledgement between different schools of thought would occur later in the history of the child-helping professions and ADHD.

Encephalitis Outbreak and Research Interest in the United States

An important event that gave momentum to the biomedical approach to hyperactivity and behavioural problems was the epidemic of encephalitis that spread through Europe and the United States in 1917 and 1918. Reports in the 1920’s described behavioural and neurological consequences in some children who survived encephalitis infections; these consequences included hyperactivity. Many authors believe that the encephalitis epidemic marked the beginning of North American interest in hyperactivity (e.g., Sanberg and Barton, 2002). While this may be the case, writers of internal histories, especially Schachar, point out that the behavioural consequences tended to be more severe than the symptoms of ADHD, “including carelessness with excreta, … hypersomnia and hysterical reactions” and were often accompanied by “unequivocal neurological signs such as epilepsy and paralysis” (Schachar, 1986, 24). Many of the children demonstrated oppositional, antisocial behaviour and some even tried to kill others. These behavioural and neurological problems, which were referred to as “post-encephalitic behaviour disorder”, followed some severe cases of encephalitis and were “clearly the result of CNS damage” (Barkley, 1990, 6). Schachar as well as Sandberg and Barton write that the linkage of severe brain damage with severe behavioural consequences was later extrapolated to associate less severe brain damage with moderate behavioural consequences; essentially a generalization or analogy that should not hold authority over scientific theory or medical practice (Sandberg and Barton, 2002, , p. 9; Schachar, 1986, 24). These authors also remind us that the understanding of post-encephalitic behaviour disorder was still influenced by the ideas of Social Darwinism, as scientists assumed that children with inherited predispositions were more likely to be infected by encephalitis and to develop the subsequent behaviour disorder. Thus these patients were not viewed simply as victims of a crippling disorder, but also as owners of inferior constitutions. Barkley mentions that despite this pessimism, some facilities that handled difficult post-encephalitic children “reported significant success in their treatment, using simple behaviour modification programs and increased supervision” (1990, 6).

Hyperkinetic Disease

Hyperactive behaviour unrelated to encephalitis was studied in Europe in the 1920s and 1930s. In the early 1930s, Kramer-Pollnow described several cases of young patients which he believed illustrated a syndrome of behavioural difficulties. The syndrome, which he named hyperkinetic disease, was “characterized by extreme restlessness, distractibility and speech disorder.” (Sanberg and Barton, 2002, 10) He classified the disease as a form of childhood psychosis, and argued that it was an independent condition, completely separate from encephalitis, and psychiatric illnesses like schizophrenia (Hassler, 1992, p. 147).

Kramer-Pollnow found that children tended to start to acquire these problems at the age of three or four, to become most severe at age six and then to improve, usually to near-normal. He depicted the children’s excessive motor activity as chaotic and aimless. He found the children to be easily distracted. He also noted a “lack of discrimination”, which Sandberg and Barton suggest resembles the current notion of impulsivity (2002, p. 10). This clinical picture and natural history largely corresponds to the current syndrome of ADHD. However, the speech disorder, which included a poor vocabulary and poor articulation, does not fit into our current concept of ADHD. Moreover, Kramer-Pollnow as well as Lederer and Ederer, who described a similar syndrome two years later, observed children who exhibited striking, extreme degrees of hyperactivity. Thus, their cases likely constituted only the most severely hyperactive children, perhaps corresponding more closely to the current European diagnosis, Hyperkinetic Syndrome, than to North America’s broader category of ADHD.

Similar hyperactivity disorders were studied in different places in Europe at the time: for example similar cases to those given by Kramer-Pollnow were discussed in an Italian paper in 1925. Hyperactivity appears to have been widely studied and treated in the Soviet Union beginning in the 1920s, where it was regarded as “a combination of a medical and a pedagogical problem” (Sanberg and Barton, 2002, 11). It was treated by placing children in special schools for “near-normal children with minor [nervous or neurological] aberrations”. They were eventually also treated medically with a combination of stimulants and tranquillizers, the proportions of which were determined by the ratio of excitatory and inhibitory processes believed to be occurring in the child’s nervous system.

Kahn and Cohen and “Organic Drivenness” and Other Biologically Oriented Research in the 1930’s

One influential study, which was guided largely by previous findings on encephalitis patients, was Eugen Kahn and Louis H. Cohen’s 1934 paper on ”organic drivenness”. Kahn and Cohen referred to a clinical and pathological study of post encephalitic nervous disorders, which identified changes in the brain-stem to be related to encephalitis-induced changes in personality and behaviour (Kahn and Cohen, 1934, p. 748), and then suggested that these ideas about the brain-stem can be applied to cases not involving encephalitis. They described cases of intelligent children and adults who were hyperactive, but for the most part had no history of brain injury. Kahn and Cohen found four elements that characterized the patients’ unusual behaviour: hyperactivity with “choreiform” or tic-like movements; an extreme difficulty in remaining quiet or still for even short periods of time; clumsiness in their movements; and an explosive quality to their voluntary motor activity (1934, p. 750).

They proposed that these four symptoms relate to functions of the brain-stem. They asserted that “the impression that one gets when observing these patients, that here is an ‘organic’ condition, is overwhelming”, where the “patient becomes a puppet at the mercy of its source, i.e. a defective brain stem” (p. 750). The reasoning was that the defective brain stem would send an abnormal quantity of impulses that overwhelm the efforts of the higher brain to control these impulses. This model, the authors believed, explained all the observed symptoms. The overwhelming of the patient’s control and inhibition by impulses manifests itself in the patient’s inability to suppress movements. The explosive nature of many of these actions, reflects the “force” of the brain-stem impulses released against the resistance of cortical inhibition. Different patients resisted and adapted to this excess of impulses in different manners, resulting in different types of behaviours. For example, one child exhibited spasms and twitches, constantly talked and whistled and frequently stole in order to give candy and toys to other children. An adult continually socialized and had many conversations with different people in a short time span, moving around a lot, smoking a cigarette one moment and rearranging furniture the next. Thus Kahn and Cohen did not believe that a subject’s behaviour was simply the product of a defective brain-stem, but rather of the patient’s personal response to this defect.

The authors proposed that “there are individuals who are possessed of organic drivenness from birth, either as the consequence of a prenatal encephalopathy or injury or of birth injury, or as a constitutional variant” (p. 752). They believed that the behavioural symptoms, soft neurological signs such as twitching, and the organic appearance suggested a brain-stem defect. While their study seems to have been influential and admired at the time, contemporary experts pointed out the lack of strong evidence supporting the proposed location of defect, which was based on “clinical impression” rather than more direct evidence such as a pathological study. A critic also pointed out that the current ideas of brain function did not strongly point to brain-stem defect as the source of “excess impulsion”; rather, in his opinion cortex lesions would better match the behavioural symptoms (Kahn and Cohen, 1934, p. 754).

Indeed, today scientists look for differences in the cortex to explain ADHD. Thus, Kahn and Cohen’s extension of findings in cases of encephalitis to more mysterious cases of hyperactivity produced a clever, intriguing paper, but one whose proposed neurological explanation was soon discarded. Kahn and Cohen’s contemporaries were skeptical about the study’s use of generalization and analogy to propose a brain-stem lesion, and therefore they debated about the location of defect. But they did not seem to question the larger assumption: that hyperactivity that gave an “organic impression” was indeed a result of a concrete brain defect. On the contrary, several contemporary studies linked hyperactivity in children to lead poisoning as well as to epilepsy and other brain disorders, thus further contributing to the association of hyperactivity with brain damage (Sandberg and Barton, 1934, p. 12). Researchers in the mid-1930’s drew parallels between the behaviour of primates who received frontal lobe ablations and that of hyperactive children, thus shifting scientific attention to frontal lobe structures in the brain cortex. Barkley writes that in the 1930s and 40’s “it became fashionable to consider most children hospitalized in psychiatric wards with this [hyperactive] symptom picture to have suffered from some type of brain damage”, with only a few writers raising questions about the assumption of brain damage without any such events noted in a child’s history (1990, pp. 7-8).

Schachar claims that Kahn and Cohen’s argument was “essentially ideological [in] nature”, citing their reliance on soft neurological signs and clinical impression in proposing a new etiology for hyperactivity and behaviour problems (1986, p. 25). Even if this is so, their theory at least reflected a strikingly different ideology than that of previous proponents of organic etiologies such as Still and Tredgold. In contrast to the Social Darwinism influences apparent in the work of Still, Tredgold and scientists studying encephalitis, Kahn and Cohen did not see their subjects’ behaviour problems as products of inferior constitutions. Rather, they depicted their patients as intelligent, well-intentioned, and often successful people who attempted to resist impulses that were out of their control and sometimes even managed to harness their “organic drivenness” for positive results. For example they described a banker who supposedly channeled his hyperactivity by taking on many hobbies and maintaining written correspondences with a very large number of friends (Kahn and Cohen, 1934, p. 752). They attributed the hyperactivity and behaviour problems to a pathological change that is peripheral to the patient’s constitution, and credited the patient’s personality and intelligence with resisting the impulses brought on by the disorder:

“in them also may be observed some fight, the fight of the organism against a kind of foreign body, the fight of the personality against something it cannot stop, something which is beyond the grasp of the individual because of its very nature. Simple and dull persons may readily succumb… but many, especially the more intelligent and sensitive resent their affliction and fight.” (pp. 750-1)

Thus while they did not necessarily withhold judgment of character from their assessments of patients, Kahn and Cohen certainly did not propose a biological model similar to that in the past, where patient’s behaviour disorder demonstrated an inferior, vulnerable constitution. On the contrary, their attitude more closely resembles the current approach in psychiatry of viewing a patient’s personality as separate from an unwanted, pathological behaviour disorder.

One could claim that Kahn and Cohen were influenced by ideological influences in a similar way to previous writers, in that they very readily looked for organic explanations for a behavioural disorder. Of course, this is a tendency that certainly occurs today, and is perhaps inherent to certain academic disciplines that study behaviour from a scientific point of view. And in their paper, Kahn and Cohen write in a tone that appears to be far from the extreme of biomedical attitudes. They approached hyperactivity in terms of the experience of the patient, and thus were concerned with broader psychological factors rather than a single organic mechanism: “however much certain factors (biological or psychological) may actually or apparently on occasion become more significant, it is always the personality as a whole which enters into the experiencing” (ibid.). While their proposal of an organic etiology without concrete, organic evidence was based on untested assumptions, Kahn and Cohen presented an ambitious attempt to combine neurology and psychology in their explanation of hyperactivity. Though their proposed mechanism was soon discarded, their more empathetic and optimistic approach to hyperactive behaviour and their careful integration of biological and psychological theory marked the beginning of a new approach to ADHD.

Bradley and Stimulant Treatment for Behaviour Disorders

According to Maurice Laufer, who himself would figure prominently in the later history of ADHD, Kahn and Cohen’s article made a considerable impression on the staff of the Emma Pendleton Bradley Home, the site where stimulant drugs were first used to treat behavioural disorders in children (Schachar, 1986, p. 25). Charles Bradley, a pediatrician and medical director of the institution, published the original articles documenting the effects of stimulants on children. Bradley expressed pride that the facilities, staff and programming met the young patients’ personal and psychological needs, describing the caring and patient nursing staff, the many outdoor activities offered, and the school classes with specially-trained and sympathetic but firm teachers and small student-to-teacher ratios (Bradley, 1937, p. 577; Bradley and Bowen, 1940, p. 783).

The efficacy of stimulants in modifying the behaviour of children was discovered by chance. Studies such as that of Kahn and Cohen, planted the idea among the staff “that structural abnormalities of the central nervous system might be responsible for children’s difficult behaviour” (Schachar, 1986, p. 25). Therefore Dr. Bradley routinely performed spinal-taps in his physical examinations of the children, which caused subsequent headaches. The headaches were often prolonged or severe and were thought to be due to loss of spinal fluid. Dr. Bradley speculated that he could use stimulant medication to induce quicker secretion of spinal fluid, and therefore administered Benzedrine, the most potent stimulator available. As physician Mortimer Gross recalls, “The effect on the headaches was negligible, but … the teachers reported major improvements in learning and behaviour in a number of children until the Benzedrine regimen was withdrawn” (Gross, 1995, p. 298).

This unexpected finding led Bradley to publish a series of studies on the effects of stimulants on children with a variety of behavioural disorders, beginning with a study on thirty children in 1937. He described the wide range of disorders that affected the children, “ranging from specific educational disabilities, with secondarily disturbed school behaviour, to the retiring schizoid on the one hand and the aggressive, egocentric epileptic child on the other” (Bradley, 1937, p. 578). After observing the children for a month and then for a week on regular doses of Benzedrine, he found striking improvements in school performance. Half of the children showed major improvements, displaying a greater interest in school, a stronger drive to complete work, and often increased speed in their comprehension and accuracy in their work. The other half either showed minor improvements or did not change in their performance, though often their behaviour changed in other respects. Bradley also documented their emotional response, and found that many of the children who had been noisy and aggressive became more placid, though at the same time seemed “more interested in their surroundings” (1937, p. 579). He explained the apparently paradoxical phenomenon of stimulants inducing subdued behaviour by proposing that they stimulate previously under-active parts of the cortex involved in inhibitory control of behaviour. Other findings included observations that several children seemed to gain “a sense of wellbeing, even to the point of mild euphoria” (p. 579), while three cried more and seemed more arousable emotionally.

In subsequent publications in the early 1940s, he gave a closer examination of the academic improvements with stimulant treatment and a more extensive, long-term study of overall behavioural effects on one hundred patients. He attributed enhanced academic performance to improvements in “emotional attitude” (Bradley and Bowen, 1940, p. 787). He commented that the more subdued behaviour of some normally boisterous children give a “general impression … that they [the children] were effectively exerting more conscious control over their activities and the expression of their emotions” (Bradley and Bowen, 1941, p. 95), thus, like Still in 1902 and like today’s ADHD scholars, relating hyperactivity to inhibitory control of behaviour. A smaller number of normally timid patients seemed to become less introverted and more stimulated: “more aggressive in competitive activities, and … an increased interest in what was going on about them” (ibid., p. 96). He illustrated such effects by describing the cases of individual patients.

When confronted with these improvements in withdrawn children as well as hyperactive types, Bradley largely abandoned his previous theory that stimulant drugs activated inhibitory centres6. He instead gave an explanation that integrated psychodynamic ideas with the biological theory behind stimulants. Children with behavioural disorders either suffered from a significant emotional conflict, an impaired ability to deal with such conflicts or both these factors (Bradley and Bowen, 1941, p. 101). Bradley proposed that the stimulants “imparted a sense of stimulation, well-being, and confidence” that lessened the stress of conflicts on the child and therefore the likelihood of a conflict provoking abnormal behaviour. Bradley maintained that one should attempt make adjustments to the child’s environment so as to remove sources of conflict before resorting to stimulants. Thus, while Schachar writes that “[t]he discovery of the therapeutic effect of amphetamines in 1937 was seen as a powerful validation of the hypothesized organic basis of hyperactivity”, it seems significant that Bradley’s papers also incorporated psychoanalytic theory. Ilina Singh, a social scientist whose work will be discussed later, suggests that his integration of biomedical and psychoanalytic perspectives represented a skillful way of “appeas[ing] potential critics from all sides” (2002, p. 589). In the early and mid-twentieth century psychoanalysis held major influence in the American psychiatric profession; Maurice Lauffer would later claim that stimulant drugs were not widely used following Bradley’s studies, because of the emphasis on psychoanalysis at the time (Schachar, 1986, p. 26; Sandberg and Barton, 2002, p. 13).

Bradley admitted that the relatively small numbers of patients in his studies limited the use of statistical analysis, although one of his later studies was on 100 patients and made use of charts and numbers. He believed that he was not able to find a correlation between type of disorder and response to stimulants, because the classification of behavioural disorders at the time was disorganized and inconsistent – a striking awareness of the limitations of his profession at the time (Bradley and Bowen, 1941, 101)7. While his use of charts, statistics and physiological measures such as the electroencephalogram gives his work a more modern appearance in some ways, he also readily used case studies and clinical impressions to make claims. His use of such methods to study drug treatment of childhood hyperactivity naturally associates Bradley with a biomedical school of thought, but his etiologic theorizing did not yield one consistent neurological mechanism to explain hyperactivity, and in the end made reference to the psychodynamic concepts of personal conflict.

Psychodynamic Approaches to ADHD

Sociologist Adam Rafalovich discusses the importance of psychodynamic perspectives to the conceptualization and clinical treatment of hyperactivity in the 1920s through 1950s. Psychoanalysts such as Anna Freud postulated that environmental conditions such as problematic family dynamics caused a fragmented ego, and that the resulting anxiety was expressed by children in the form of hyperactivity (Rafalovich, 2001, p. 399). As mentioned earlier, psychoanalysis was prominent in child psychiatry in the early twentieth century, in the form of the child guidance movement. Psychoanalysis suffered a sharp decline in prestige and authority in the 1960s and 1970s, and subsequently played little role in the treatment and conceptualization of hyperactivity. Social scientist Andrew Lakoff writes that the psychoanalytic approach to hyperactivity was overwhelmed by the popularity of new medical tools such as standardized questionnaires and the diagnostic criteria in the 1960s, which will be discussed later (Lakoff, 2000).

Many psychologists in the first half of the twentieth century sought a middle ground between the neurological and psychoanalytic approach. The resulting psychological model regarded hyperactivity to be a survival mechanism that children used to overshadow underlying anxiety, but this anxiety was thought to be a result from an organic cause, rather than family conflict. Treatment was not based on addressing the organic defect, but rather on making the child and family aware of the nature of their anxiety and compensating mechanisms. These psychologists found a professional niche practicing in cooperation with psychiatrists and pediatricians who took a more medical approach. These psychologists treated the child’s personal reaction to their brain abnormality, while the medical profession treated the underlying brain problem. Despite this partnership, there was a central contradiction between the medical and psychological perspective: the neurological approach postulated the central problem to be organic-based hyperactivity, and anxiety was regarded as a secondary reaction. Thus the neurological school of thought, believed their method to be more essential as it attacked the problem at its root. Raflovich portrays this tension as a competition between two conceptual models. Members of the neurological school of thought in the 1960s and 1970s, such as Paul Wender, advised that terminology and approaches toward childhood hyperactivity should be consolidated into a one unified approach. It was this medical model that became dominant. Consequently the medical and neuroscience rather than psychodynamic tradition is acknowledged in internal histories, as the internal historians, along with most other professionals who treat ADHD, belong to this medical tradition. Several ADHD experts anticipate that a developmental approach, which integrates different biomedical and environmental factors, will become more prominent in our understanding of ADHD (e.g., Tannock, 1998 and Olson, 2002). If this does indeed become the case, it will be interesting to see whether the much ignored role of psychodynamic psychiatry and psychology will be discovered in future internal histories of ADHD.

Strauss, Werner, Lehtinen and ‘Minimal Brain Damage’

Barkley suggests that the concept of ‘Minimal Brain Damage’ rose to prominence as a result of a collective lapse of logic and scientific rigor on the part of the child psychiatry, child psychology and pediatrics professions. He explains that in clinical practice cases of central nervous system damage that resulted in hyperactive behaviour patterns were “mixed in with those cases having a similar behavioural pattern but with an ambiguous history of damage” (1990, p. 7). This naturally led to assumptions about the etiology of the later types of cases, and thus to a type of logical circularity where the behaviour pattern hyperactivity and attention problems were attributed to undetected brain damage, simply by virtue of the presence of this behaviour pattern. The research of Alfred A. Strauss along with collaborators Heinz Werner and Laura E. Lehtinen further strengthened this link between behaviour problems and organic brain damage.

Strauss, Werner and Lehtinen worked at a special school in Michigan for children “of borderline intelligence, or ‘higher grade morons’,” (Sandberg and Barton, 2002, p. 14) and studied many such children who had additional behavioural and learning problems in the 1940s and 1950s. Strauss divided children of borderline intelligence into two groups. The endogenous group had no history of brain trauma during pregnancy, birth or infancy, but they tended to have family histories with other instances of intellectual deficiency (Schachar, 1986, p. 27). In the exogenous group, there was evidence of physical damage to the central nervous system, but no history of mental deficiency in the family. The exogenous group responded poorly to teaching, and appeared to be overactive and easily distracted” (Sandberg and Barton, 2002, p. 14). Strauss, in collaboration with Werner and Lehtinen, investigated the nature of these defects among the exogenous group and developed educational programs more suitable for these children.

Strauss’s interest in studying behaviour problems as products of organic damage rather than of psychosocial factors is apparent in how he chose his subjects: he excluded children affected by “traumatizing or limiting emotional relationships and those who required psychiatric treatment” (Schachar, 1986, p. 27). Under the assumption that children selected this way would have underlying brain differences causing their misbehaviour, Strauss and his colleagues attempted to gain a deeper understanding of the nature of these differences. To do this Strauss prepared experimental situations where children performed unusual, standardized tasks – for example, matching picture cards whose subjects relate to each other – in an isolated, laboratory environment, apart from the child’s usual settings of school and play (Strauss and Werner, 1942 and 1943).

The tasks appear to have been carefully chosen so as to correspond to certain psychological constructs, and thus enabled the researchers to simplify and abstract the children’s differences. Strauss and Werner observed the children’s responses to tasks in terms of constructs such as “forced responsiveness to stimuli” and “fixation and perseveration”. They used such concepts to explain differences in the responses of the “brain injured” children from those of non-brain injured children; for example, attributing unusual responses of brain injured children to increased “pliability” of concepts or an “inability to integrate two points of view, or to perceive a double relationship” (Strauss and Werner, 1943, 836-7). They compared such psychological findings to previous studies on adults who had sustained brain injuries, concluding that the supposedly brain-injured child shared “certain fundamental pathological characteristics” (1943, 837). To further explain the brain-injured child’s psychological differences, they postulated that all people share certain patterns of behaviour in response to stimuli, but that in the brain-injured child these responses were triggered by less intense stimuli, which then affect the child’s thought processes and behaviour.

Strauss and his collaborators attempted to apply their new understanding of the brain-injured child to devise educational programs and classroom environments adapted to their psychological differences. These adaptations largely included reducing stimulation in the classroom that might distract students. Barkley describes: “Strikingly austere classrooms were developed, in which teachers could not wear jewelry or brightly coloured clothing and few pictures could adorn the walls.” (1990, p. 8) In addition teaching methods were developed to help these children learn concepts or skills that posed particular problems for them.

By creating experimental tasks to be performed in an experimental environment, and measuring psychological constructs, Strauss treated behaviour and thought as subjects that can be observed scientifically outside of a psychosocial context. While previous researchers based their etiological hypotheses on their clinical observations of behaviour, medical history and physical examinations, Strauss designed experiments to dissect the children’s learning and behaviour into more specific, abstract elements. Thus clinical judgment was replaced by tests that seemed to evaluate fundamental properties of each child’s brain. By articulating behavioural and learning difficulties in terms of abstract psychological constructs, Strauss et al. transformed behavioural or clinical properties, into brain properties. This perhaps explains some of the circular reasoning behind the concept of minimal brain damage. Strauss’s intricate methodology, led him and his colleagues to conclude that the brain of the exogenously mentally defective child had an abnormal organization. But behind their careful method, they came to their conclusions largely by translating observations into scientific language; by rephrasing behavioural differences as cognitive and perceptual differences. While this method was biased towards accepting a biological etiology, it also represented something of a breakthrough, as an early attempt to gain a deeper understanding of the processes behind behavioural and learning problems through the application of the concepts of cognitive science.

One can admire Strauss’s detailed, conceptual investigations, but the internal historians point out several methodological problems with Strauss’s work. Perhaps because of the logistics of working at an institution designed for children with intellectual deficiencies, Strauss mainly studied such children as opposed to those of average or above average intelligence. However his conclusions were extended to children of normal intellect who had “behavioural or perceptual differences” (Barkley, 1990, p. 8; Schachar, 1986, p. 27). In this sense, Strauss’s work can be seen as continuing the confusing tradition, which can be traced to the writings of Tredgold, of studying children who have behavioural problems together with those who have intellectual deficiencies. Thus researchers such as Strauss did not isolate hyperactivity as a separate disorder from mental deficiency.

Schachar points out the most serious shortcoming of Strauss et al.’s approach: their use of “unclear, circular and impressionistic criteria … in distinguishing the brain-injured child” (1986, p. 28). While their psychological tests appeared to be very systematic, their methods for selecting brain-injured children to participate in their studies were inconsistent. Some subjects were selected based on neurological findings, while others were chosen based on psychological findings or simply referrals from other physicians. The diagnosis lacked rigidity to the extent that it “applied to children with virtually all types of behaviour problems, especially those who were overactive and inattentive” (Schachar, 1986, p. 28). The lack of validity of their results surfaced, as attempts by other scientists to replicate their findings failed. In addition, while the special educational programs they developed became very influential, they were frequently found to be ineffective. Strauss et al. perhaps neglected considerations of sample selection, because they were focused on explaining behavioural problems, rather than clearly defining them. Later researchers, such as Virginia Douglas, would follow Strauss’s example in applying psychology theory to the problems of hyperactivity, but would achieve more dramatic advances, because they were conscious of the need to improve the validity and reliability of diagnostic categories.

Despite these shortcomings, Strauss’s research had considerable appeal. In the “brain injured” child, he offered a scientific explanation for misbehaviour and school failure that avoided blaming heredity or social problems (Schachar, 1986, p. 29). This optimistic, non-judgmental attitude, comparable to that of Kahn and Cohen, was complimented by his detailed educational recommendations which attempted to use his scientific findings to help improve school performance. Schachar credits Strauss’ educational recommendations with providing the “guiding principles” for a powerful parent lobby, the Association for Children with Learning Disabilities” (p. 29). According to Barkley they “were the predecessors as well as instigators of the types of educational resources” mobilized in the 1970s when American laws mandated “special education of learning-disabled and behaviourally disordered children” (1990, p. 8)).Sandberg and Barton write that Strauss’s findings were contested, but were still very influential and essentially led to the adoption of the diagnostic term “minimal brain damage” to label children with hyperactive and impulsive behaviour patterns. German psychiatrist Frank Hassler writes that “it is due to Strauss and Lehtinen that the aetiological concept was developed into a diagnostic one” (1992, p. 147). Schachar, Werry and Sandberg and Barton explain the appeal of the concepts of “brain-injured child” and “minimal brain damage syndrome” in the context of the professions involved. According to Schachar, the psychiatry profession was largely focused on psychoanalytic psychotherapy, which was generally not found effective in treating hyperactivity8. As a result, paediatricians largely undertook the treatment of hyperactive and impulsive children. Werry claims that these cases generated tremendous interest in the paediatric profession, “ignoring the rarity of such disorders in children and secondly their refractoriness to any kind of medical intervention” (1974, p. 95). He further claims, though without citing specific evidence, that American professionals and parents were generally more eager to adopt the new idea of minimal brain damage than more conservative populations such as England. Sandberg and Barton suggest that disillusionment with the poor results of psychodynamic treatments made new biological explanations very attractive. These various professional factors likely made clinicians more ready to accept the idea of undetectable, minor brain damage causing childhood hyperactivity. Such glimpses at the influence of professional interests and biases on scientific theory appear very rarely in internal histories of ADHD, which normally focus on technological progress and changes in scientific methods. This exception reflects a degree of bewilderment about the widespread acceptance of Minimal Brain Damage, a concept which, in retrospect, was based on obvious logical fallacies.

Pasmanick, The Continuum of Reproductive Causality and Support for Minimal Brain Damage Syndrome

Benjamin Pasmanick and collaborators such as Hilda Knobloch provided further evidence for the link between brain damage and hyperactivity through their influential epidemiological studies in the 1950s and 1960s. They hypothesized that behavioural problems of hyperactivity and impulsiveness were part of a “continuum of reproductive causality”. This idea was based on the findings that premature births and pregnancy complications were associated with perinatal deaths and conditions such as cerebral palsy, and that these problems were usually caused by injury to the brain (Pasmanick, Knobloch and Lilienfeld, 1956, p. 597). Therefore, Pasmanick et al. reasoned that there was “a gradient of brain injury resulting in death of the fetus or newborn at one end and extending through cerebral palsy and going on to epilepsy, mental deficiency and possibly sufficient minor injury to so disorganize behaviour and lower thresholds to stress as to result in childhood behaviour disorder” (ibid., p. 597).

They tested this hypothesis by comparing a group of children referred to “special services” in the Baltimore Department of Education to controls, who did not receive such referrals. The children requiring special educational help were found to be much more likely to have histories of prenatal injury (Sandberg and Barton, 2002, p. 16). Moreover, a subgroup of children described as hyperactive, confused and disorganized had the strongest association with a history of birth complications (Schachar, 1986, p. 29).

Pasmanick also probed into the long-time association of neuropsychiatric entities, such as behavioural disorders, with socioeconomic status and race. This link had frequently been made by the medical profession – for example by Still and Tredgold – but was also expressed in popular beliefs and literature long before it was a subject of scientific investigation. Scholars such as Tredgold depicted behavioural problems as inherited qualities that limit certain families to the lower class. Pasmanick used his statistical findings and critical analysis of others’ methodology to reveal logical flaws in such studies. Instead, he argued that higher incidences of such neuropsychiatric problems in families of low socioeconomic class were a result of increased risks in pregnancy and birth, which were, in turn, attributable to social and economic disadvantages rather than faulty inheritance (Pasmanick, Knobloch and Lilienfeld, 1956, p. 600). Thus Pasmanick and colleagues studied behavioural disorders as manifestations of organic damage, but then argued that this perinatal damage was often a result of environmental factors dictated by socioeconomic conditions.

Pasmanick’s work can be seen as an effort to dispute the use of science to promote racial ideologies. However, this in itself is something of an ideological mission. He made logical arguments using his epidemiological findings to disprove previous ideas about the innate inferiority among lower classes, but these arguments – though possibly scientifically valid – were dictated by the agenda of dissociating science from racial theories. The ideological assumptions behind Pasmanick’s work are not nearly as noticeable as those of Tredgold, because they embody values of nondiscrimination and tolerance, which are widely accepted today and part of the background against which we judge any new idea. This is not to dismiss Pasmanick’s important work as science skewed by ideology. Rather, his work is an interesting illustration of the fact that science, no mater how sophisticated, is influenced by social values and assumptions, though these values may be admirable ones. Pasmanick, like Kahn, Bradley and Strauss, seems to have been part of a new tradition of doctors and scientists who looked upon behavioural disorders in an empathetic and largely nonjudgmental manner and were determined to study and treat these disorders without assigning blame or labels of inferiority. This attitude gave scholars new incentives to seek a biological explanation of hyperactivity. An underlying biomedical cause would solidify the status of the behaviourally disordered child as a patient and doctors as the guiders of therapy.

Beyond the influence of his conclusions about the cause of hyperactivity, Pasmanick’s work also appears to be significant, because it is among the first to use epidemiological methods and sophisticated statistical tools to study hyperactivity in children. Like Strauss’s use of psychological tests, Pasmanick’s statistical studies provided a new way of asserting conclusions about behavioural disorders, which contrasted with the traditional avenues of case studies and clinical impressions. Thus a new and varied group of literature was accumulating using distinctively scientific methods to link hyperactive behaviour in children to organic brain damage.

From Damage to Dysfunction, and the Decline of MBD

The concept of Minimal Brain Damage Syndrome was a diagnostic label for children with behavioural and learning problems, who were typically hyperactive and impulsive. This label assumed an organic etiology, on the basis of Strauss’s speculation about submicroscopic lesions and abnormal organization in the brain and Pasmanick’s contention that relatively small amounts of brain damage occurred in undetected or undocumented fetal and birth events. While this concept gained considerable popularity and influence in the 1950s and 1960s, its rise to prominence was immediately met by its widespread criticism in the scientific community.

Prominent critics such as Birch, Herbert and Rapin highlighted the logical fallacy in assuming that if some children had a history of brain trauma that caused a pattern of hyperactive behaviour, then all children with this pattern of behaviour must have brain damage (Barkley, 1990, p. 9). The Oxford International Study Group of Child Neurology released a publication in 1963 which asserted “that brain damage should not be inferred from behaviour alone” and suggested replacing the term “minimal brain damage” with “minimal brain dysfunction” or MBD so as to no longer imply the involvement of an organic lesion (Sandberg and Barton, 2002, p. 17). This change in terminology was widely adopted, but even the Oxford Study Group indicated that the concept of MBD would only be temporary. The Oxford Group along with other researchers at the time concluded that even the concept of MBD was “vague, overinclusive, [and] of little or no prescriptive value” (Barkley, 1990, p. 10). An American study by the National Institute of Neurological Diseases and Blindness noted the very large number of symptoms that had been attributed to the disorder. The Oxford Group recommended that the heterogeneous category of MBD eventually be replaced by more homogeneous subcategories. Indeed Mild Brain Dysfunction was eventually supplanted by more specific categories such as learning disabilities, language disorders and hyperactivity. This shift was gradual, as the term MBD faded from clinical and scientific usage by the 1970’s (Barkley, 1990, p. 12).

Laufer, Chess and Hyperactivity Syndromes

At the same time as the concept of MBD was coming under criticism, articles began to describe a “hyperactive child syndrome” (Sandberg and Barton, 2002, p. 10). The most prominent of these publications were studies by Maurice Laufer and Stella Chess. Laufer, who worked at the Emma Pendleton Bradley Home in Rhode Island, confirmed the findings of his mentor Charles Bradley that amphetamines can improve behaviour in hyperactive children. His publications in 1957 were the most famous of several studies at the time that investigated possible neurological mechanisms for hyperactivity (Barkley, 1990, p. 8).

Laufer and his collaborators conducted photo-Metrazol tests, in which they administered a medication called Metrazol while flashing lights at the subject (Laufer, Denhoff and Solomons, 1957, pp. 39-40; Barkley, 1990, p. 9). The amount of Metrazol required to induce a jerk of the forearms had previously been shown to be an indicator of function of the diencephalon, a subcortical structure. Levels that were below a normal range indicated diencephalon dysfunction. Laufer et al. selected samples of emotionally disturbed children from the Bradley home and divided them into two groups: an experimental group of children who presented with a hyperactive pattern of behaviour, which he categorized as “hyperkinetic impulse disorder”, and a control group of children who were not hyperactive. They outlined the behavioural characteristics of “hyperkinetic impulse disorder”: hyperactivity was “the most striking” feature but was also associated with impulsiveness and an inability “to tolerate any delay in gratification of their needs and demands” (Laufer, Denhoff and Solomons, 1957, p. 38). They specified that hyperactivity was typically detected around age five or six, though often would be prominent at an earlier age. These criteria for age of onset resemble the current approach to ADHD. Laufer et al. found that the children with hyperkinetic impulse disorder had a significantly lower threshold of response to Metrazol. Therefore they concluded that these children suffered from a dysfunction of the diencephalon. Moreover, they found that the hyperactive children’s Metrazol thresholds rose to the normal range after administration of amphetamines, which suggested that these medications enhanced the otherwise impaired functions of their diencephalons.

Laufer et al. proposed a possible mechanism, in which dysfunction of the diencephalon leads to decreased resistance to nerve impulses at synapses: “This would allow incoming impulses to spread out of usual pathways and irradiate large cortical areas.” (p. 45) Such poor filtering of stimulation overwhelms the cortex, leading the child to be “unusually sensitive to stimuli flooding in from both peripheral receptors and viscera”. Along with this proposed mechanism, Laufer et al. described an approach to treatment that incorporated amphetamine drugs and psychotherapy for the child and counseling for the parents. The psychotherapy would serve to treat psychological problems caused by the difficulties children encounter due to their frequently unacceptable and annoying behaviour or to help resolve other psychological problems exacerbating their hyperactivity. The counseling of parents was meant to relieve guilt by explaining the organic nature of their child’s disorder and to encourage them to adapt their parenting approach in a manner sensitive to their child’s hyperactivity.

Schachar and Barkley point to limitations in Laufer’s work. They mention that his results have not been replicated, Barkley adding that his work likely could not be repeated now, as it would probably not pass current standards of ethics (Barkley, 1990, p. 9; Schachar, 1986, p.27). Schachar points out that Laufer and collaborators never measured the effect of amphetamines on the photo-Metrozol thresholds of non-hyperactive children, and therefore did not determine whether stimulant medications function by correcting the underlying diencephalon dysfunction in hyperkinetic disorder or whether they simply increase inhibition of impulses in all children9. Schachar further argues that the photo-Metrazol tests did not constitute sufficient evidence to make conclusions about the etiology of hyperactivity. Laufer et al. acknowledged this in their publication, as they admitted that scientific knowledge about the diencephalon was still only in its earliest stages. Further they mentioned the possibility that “the apparent abnormal function of the central nervous system as depicted by the photo-Metrazol test might have been purely secondary to … emotional disturbance” (Laufer, Denhoff and Solomons, 1957, p. 45), though aside from this qualification they focused on studying hyperactivity as an organic disorder of the diencephalon rather than a condition secondary to emotional factors.

Laufer et al. appear to have followed Charles Bradley’s example of focusing on scientific approaches, but also integrating psychodynamic ideas. This can be seen as an attempt to appease all sides. As a result, their paper presents interesting contrasts. It displays an impressive array of electroencephalograms, graphs and charts, but also uses a case study to illustrate how a hyperactive boy could be treated effectively with stimulants and psychotherapy. The authors extensively cited recent neurology publications, but also cited Freud more than once. While Laufer et al. were clearly interested in finding a biological mechanism for hyperkinetic impulse disorder, they disapproved of “‘all or none’ type of thinking”, instead insisting on “continual consideration of both neurophysiological and psychological factors” (p. 47). They further warned that the success of stimulant treatment should not distract from the need for psychotherapy or environmental changes. They emphasized that “hyperkinetic syndrome is a very specific entity”, and that stimulants should not be used to treat other behaviour problems. The previously mentioned social scientist Ilina Singh suggests that like Bradley, Laufer’s references to psychodynamic theory were attempts to warm psychoanalysts to his new biomedical approach to behaviour problems (2002, p. 591). Still, Laufer’s studies represented not only an effort to use neurophysiological methods to understand the etiology of hyperactivity and validate the use of stimulant treatment, but also an attempt to take these steps cautiously.

Stella Chess, a psychiatrist at New York Medical College, paid little attention to theories of etiology in her influential 1960 publication, but rather focused on classification and clinical descriptions of hyperactive children. She offered a straightforward definition of hyperactivity: “The hyperactive child is one who carries out activities at a higher rate of speed than the average child, or who is constantly in motion, or both” (Chess, 1960, p. 2379). She studied the 82 children who were diagnosed as hyperactive out of a total of 881 children seen in her practice. She further divided these children into five diagnostic categories: physiologic hyperactivity, which presented without a history of brain injury or any other such pathology; hyperactivity with a history of organic brain damage; hyperactivity accompanied by an intellectual deficiency; hyperactivity determined to be a reaction to “environmental stress or neurotic patterns”; and hyperactivity as part of the more severe condition of childhood schizophrenia (Chess, 1980, p. 2379-81). Thus Chess was significant for her attempt to divide the heterogeneous group of hyperactive children into more homogeneous subgroups and for explicitly separating the concept of hyperactivity from that of brain injury (Barkley, 1990, p. 10).

Chess studied the 36 children with physiologic hyperactivity in detail. Of these cases, the majority (29) were males and were referred at the age of six or younger, findings similar to Laufer’s descriptions and to the current descriptions of ADHD. She maintained that the defining feature of the disorder was hyperactivity. Associated symptoms included impulsiveness, nonconformity to rules, short attention span and poor school achievement, though these depended on the age of the patient. Chess made suggestions for both diagnosis and treatment of the disorder. She insisted that doctors not depend on “subjective reports” of parents and teachers, but rather use “objective behavioural data … obtained from these sources and supplemented by the clinician’s own observations, by psychological tests, and by reports from other physicians” (1980, p. 2379). She recommended a treatment approach even more encompassing than Laufer’s suggestions, combining behavioural modification, medication, consultation with the school about special education measures, consultation with parents about their child’s disorder and psychotherapy in the many cases involving psychological problems secondary to the child’s hyperactive behaviour. Like Laufer, she emphasized the importance of relieving feelings of guilt from parents, by explaining the behavioural “symptoms to be as real as a paralyzed limb” (p. 2383). Chess did not make any scientific hypotheses about the etiology of these symptoms, and it is unclear whether her assumption that hyperactivity was a medical condition was in part due to an acceptance of previous neurological studies, such as that of Laufer. Interestingly, while Chess discussed using a variety of medications, including amphetamines on “preadolescent age groups”, she frequently recommended the use of tranquilizers.

While the concepts of hyperactivity disorders used in parts of Europe and the Soviet Union since the 1920’s applied to severe, “choreoform” conditions, Laufer and Chess depicted hyperactivity disorders as relatively benign conditions that resolve as the child matures. According to Sandberg and Barton, Chess’s concept of hyperactivity disorder became well established in the medical literature by the late 1960’s (p. 18). Barkley notes that the experts who drafted the second edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-II) of 1968 created the term “Hyperkinetic Reaction of Childhood Disorder”, though they provided little useful diagnostic information besides describing hyperactive behaviour (1990, p. 10). The work of Laufer and Chess is notable for classifying hyperactive, impulsive behaviour according to a central behavioural symptom, in this case hyperactivity. Chess in particular represents a shift from the use of diagnostic labels based on etiology to a behavioural definition of the disorder.

Divergence of European and North American Schools of Thought

Barkley writes that in the 1950s and 1960’s a different approach to hyperactivity began to develop in North America in contrast to the approach accepted in Britain and other European countries (1990, p. 11). In North America hyperactivity was conceptualized in a manner that corresponded with Chess’s work: “a relatively common disturbance of childhood” not typically associated with a history of symptoms of brain damage. Laufer et al. acknowledged that most normal children display some degree of hyperactive, impulsive behaviour (Laufer, Denhoff and Solomons, 1957, p. 39), and hyperactivity was generally regarded in North America as an extreme degree of such behaviour. By contrast there was a narrower view of hyperactivity in Britain, where hyperkinesis was seen as “an extreme state of excessive activity of almost driven quality. This was highly uncommon and usually occurred in conjunction with other signs of brain damage such as epilepsy or mental retardation or with a clearer history of brain insult” (Barkley, 1990, p. 11). These different definitions eventually contributed to large discrepancies in prevalence determined by North American and European epidemiologists. Attempts to discuss and reconcile these differences were not begun until the late 1980s.

Conners and the first Systematic Assessment Scales

Keith Conners, a researcher at Harvard, published influential studies in the late 1960’s and early 1970’s which used complex statistical methods and large samples to develop standardized behaviour rating scales. These forms, some designed to be filled out by parents and others by teachers, were meant to help doctors make more accurate assessments of childhood symptoms such as hyperactivity. They consisted of lists of behaviour descriptions, such as “hums and makes other odd noises” and “restless or overactive”. Parents were to check-off whether each behaviour was “not at all present, just a little, pretty much, or very much present” (Conners, 1969, p. 154; 1970, p. 670). These results were then converted to numbers added to provide a total score, which in turn could be used for quantitative comparisons and statistical analysis. Conners intended them largely for use in psychopharmacology trials, so as to better define and standardize experimental and control groups, as well as in daily clinical practice. The stimulant drug trials were aimed not only at hyperactive children, but also at children with other mild behavioural disorders and “poor attention spans associated with learning disorders” (Conners, 1970, p. 885).

Conners used methods such as “principal components factor analysis” to organize common behaviours typically associated with behavioural disorders into five statistically “stable” dimensions: an aggressive conduct dimension; a “‘day-dreaming-inattentive’ dimension”; “a shy, withdrawn and sensitive dimension”; a hyperactivity dimension, reflecting “restless, excitable and troublesome behaviour” lacking “aggressive components”; and a final dimension reflecting positive, healthy social interactions indicating relative psychological health and lack of symptoms (1969, pp. 154-5). Conners’ analysis found that it might not be appropriate to measure aggressiveness as a separate dimension from hyperactivity, as he found correlations between the two factors. Indeed, his scales were later criticized for “their confounding of hyperactivity with aggression” (Barkley, 1990, p. 19).

Conners did not just study hyperactivity and other disorders thought to be treatable with stimulant medications; much of his statistical research addressed the classification of all childhood disorders. He recognized the vagueness and confusion in the contemporary diagnostic system: “One of the fundamental unsolved issues in child psychiatry is the development of an adequate classification which would encompass in a unified system the wide variety of behavioural disorders, personality deprivations, and problems of social and educational development seen clinically”(1970, p. 667). Contemporary scientists, Peterson and Quay, proposed reclassifying all childhood behaviour disorders under the two broad groups of “conduct disorder” and “personality disorder”. Conners demonstrated the statistical validity of similar clusters – a neurotic group and hyperkinesis group – by analyzing a large sample of parent checklists. In the past, categories such as MBD and hyperactivity disorder certainly interacted with other behaviour disorders, in that they were part of a collection of labels used to classify children who were referred for psychiatric assessment. But often the disorder was discussed without consideration of this broader system of diagnostic labels. One possible exception is Tredgold who discussed mild mental deficiency in the context of all types of mental deficiency, but most writing on MBD and behavioural disorders paid little attention to the blurred boundaries between hyperactivity and other conditions. Researchers such as Conners became more conscious of this interaction among developmental disorders and of the need to limit overlap and inconsistencies by developing a coherent framework for all childhood disorders.

Conner’s work is considered most significant for his development of empirically-tested, standardized behaviour rating scales. This helped make diagnosis less dependent on the doctor’s clinical impression, which was supplemented by structured and seemingly more objective evaluations of abnormal behaviour (Barkley, 1990, p. 19). While this method had advantages, it also had limitations. Behaviour checklists were still based on subjective judgments, though disguised in the form of numerical scores that seemed to measure a child’s behaviour. But now these judgments would be made by multiple people, namely parents and teachers, who knew the child well and had observed the child extensively in different settings. His rating scales were widely adopted in North America and regarded for over twenty years as the “gold standard” for research and clinical assessment. They also were used as tools for epidemiological studies that attempted to determine the prevalence of hyperactivity in various populations.

Douglas and Attention Deficits

Research into childhood hyperactivity exploded in the 1970’s (Barkley, 1990, p. 12). As Barkley describes, a much higher volume of research papers were published in this decade relative to the amount of research produced earlier. This was accompanied by the release of many clinical and scientific textbooks, as well as an authoritative scholarly review of the literature by Dorothea and Sheila Ross. The high level of scholarly interest is apparent in the numerous special journal issues dedicated entirely to the topic. Summarizes Barkley, “hyperactivity had become a subject of serious professional and scientific, as well as popular attention”.

Not coincidentally, ideas about the disorder changed dramatically in this time. By the beginning of the decade, experts began to broaden the defining features of the disorder to include impulsiveness, short attention span, distractibility and low tolerance of frustration. Authorities such as Chess previously considered these to be “associated symptoms” peripheral to the central feature of hyperactivity. Sandberg and Barton write that this new emphasis on features such as impulsiveness and attention problems was particularly prominent in North America (p. 19). Virginia Douglas of McGill University was perhaps the most important figure in this shift, as she was the leader of a new group of researchers dedicated to reconstructing hyperactivity disorder into a more consistent and empirically-validated category.

Like Conners, Douglas recognized the confusion surrounding diagnostic terms used in child psychiatry:

“Like all reviewers in the area of childhood disorders, we have been continually hampered by the inconsistent and ill-defined methods used by investigators to select and label their subjects. Terms like minimal brain dysfunction, learning disability, and hyperactivity frequently are used vaguely and sometimes interchangeably.”(Douglas, 175)

For example, Douglas pointed out that the term learning disability was often used in a very general manner, despite evidence that some children exhibit “specific deficits in the manner in which they process information” (p. 176). These observations apply to most of the major studies in the first half of the twentieth century, as notable researchers such as Bradley, Strauss and Pasmanick tended not to differentiate hyperactive children from those with other learning problems. Therefore Douglas proposed that clinicians diagnose hyperactivity and learning disabilities independently of each other. Thus a child may be found to have both hyperkinetic disorder and a learning disability, but this would be determined by two separate decisions. This might be overlooked today as an obvious suggestion, but it was an important one, and was an essential step in clarifying the borders between hyperactivity and other disorders.

Like many other scientists at the time, Douglas pointed out the confusing nature of the term MBD:

“few would deny that attentional problems must ultimately be associated with some kind of dysfunction of the brain; however, no suitable criteria or norms are currently available for establishing neurological signs that could be used in applying the MBD. Thus, although promising work on this problem is under way, we believe that use of the MBD classification at this time is premature and likely to lead to further confusion” (p. 176)

The terms Mild Brain Damage and Dysfunction are often interpreted as embodying the medical community’s assumptions about the organic etiology of hyperactivity. However Douglas’s comment demonstrates that when the term MBD was abandoned, beliefs about the underlying organic nature of the disorder were retained. This is also evident in the very fact that a large volume scientific research was being conducted on hyperactivity disorder, compared with a smaller body of research that focused on psychosocial factors. But Douglas and other researchers became aware that defining the disorder by its supposed organic nature had no utility, as neurological theories provided no way in determining whether a child had the condition. Therefore Douglas set out to develop a clearer, more useful definition of hyperactivity through empirical research.

As well as being critical of the concept of MBD, Douglas was skeptical about the utility of the current definitions of hyperactivity disorder, such as that proposed by Chess. She noted that clinical measures of hyperactivity were fairly coarse and unreliable. But more importantly, hyperactivity no longer seemed to be the central symptom of the disorder: “the inability of these children to organize and sustain attention and to inhibit impulsive responding are the qualities that differentiate them most clearly from other groups” (Douglas, 1979, p. 177). Hyperactive children had long been found to have problems with attention, as evident in papers by Still, Bradley, Strauss and Chess. But researchers such as Douglas in the 1970s sought a more detailed understanding of these attention problems. They deconstructed the idea of “attention” into many different types of attentional processes: for example, vigilance or sustained attention and selective attention (ibid., p. 178). By separating and attempting to measure these different components – especially sustained attention and selective attention – Douglas and her colleagues sought to gain more insight into the nature of childhood hyperactivity.

Douglas and her team at McGill used a wide range of psychometric tests to measure cognitive and behavioural aspects of the disorder (Sandberg and Barton, 2002, p. 20). Like the experiments of Strauss et al. these tests consisted of unusual tasks to be performed in laboratory settings, and were designed to measure deficits at a more fundamental level than the regular observation of behaviour in an everyday or clinical setting. But unlike those administered by Strauss, these tests tended use audiovisual equipment, yielding quantitative results. Hence Barkley considers these results to be “objective measures of various behavioral and cognitive domains” (1990, p. 13). These tests included the Continuous Performance Test (CPT), Delayed Reaction Time Tasks (DRTT) and Matching Familiar Test of Reflection-Impulsivity10. In some studies, the McGill team took psychophysiological measurements such as skin conductance while children performed these tasks.

Like Strauss, Douglas and colleagues used psychometric tests to gain a deeper, more fundamental understanding of the disorder. But unlike Strauss and other predecessors such as Kahn and Cohen, Douglas’s theoretical explanations revolved around the goal of making the definition of the disorder more valid, coherent and empirical. This practical goal did not just compliment her theoretical work, but rather shaped it. By performing empirical research, guided by theory that could be tested and revised, Douglas could “rule in or out various characteristics felt to be typical for these children” (Sandberg and Barton, 2002, p. 20; Barkley, 1990, p. 13).

The results of this approach were publicized in her 1972 President’s Address to the Canadian Psychological Association, when she argued that hyperactive children were especially deficient in impulse control and sustained attention. The hypothesis that these deficiencies rather than hyperactivity were the core aspects of the disorder was strengthened, as Douglas’s colleague Gabrielle Weiss found that while hyperactivity diminished by adolescence, impulsiveness and poor vigilance often remained a problem. While they demonstrated difficulties in sustained attention, hyperactive children were found to be no more distractible than normal children. This finding was further supported by the fact that previous experiments with the stimulus-reduced classrooms suggested by Strauss failed to improve academic performance (Douglas, 1969, p. 187). On the contrary, children with hyperactivity disorder often became more overactive in environments that were stripped of all potentially distracting stimuli.

In this manner, Douglas and colleagues refuted the previous hypotheses of researchers such as Kahn and Cohen and Laufer that hyperactive children were “stimulus driven” because of defective “filter mechanisms” which allowed an excess of impulses to affect the cortex (Douglas, 1969, p. 174). On the contrary, such children were “stimulus seeking”, as evidenced by the fact that they became more hyperactive when given tasks to do in empty rooms stripped bare of interesting stimuli. While removing distractions did not necessarily improve the performance of hyperactive children on standardized tasks, Douglas et al. found that providing continuous rewards enhanced performance. This led Douglas to conceptualize hyperactivity as a disorder of the will, where symptoms could largely be attributed to defects in motivation and volition11. Her team also found that impulsive control and vigilance were improved with the use of stimulant medications. This helped validate the prescription stimulants. Symptoms appeared to be modifiable through both psychosocial and pharmaceutical methods.

As Douglas compiled a large body of research over the decade, she refined and expanded her theory. She explained symptoms by four fundamental deficits: “1. deficits in investment, organization, and maintenance of attention and effort; 2. inability to inhibit impulsive responding; 3. inability to modulate arousal levels to meet situational demands; and 4. an unusually strong inclination to seek immediate reinforcement” (Barkley, 1990, p. 14). Barkley as well as Sandberg and Barton write that the work of Douglas and colleagues was so influential that it was the main reason why the disorder was renamed Attention Deficit Disorder (ADD) with or without Hyperactivity in the DSM-III of 1980 (Barkley, 1990, p. 14; Sandberg and Barton, 2002, p. 20). The extent of the new focus on attention was apparent in the fact that children no longer needed to be hyperactive in order to be diagnosed with Attention Deficit Disorder, even though it was the successor of Hyperactivity Disorder. Later the construct of attention was criticized for many of the same reasons Douglas criticized that of hyperactivity: not specific to the disorder, no clear distinction between normal and abnormal levels and very dependent on the situation. But the name ADD might be a slightly misleading reflection of Douglas’s work, as she seemed to emphasize impulse control nearly, if not entirely as much as sustained attention. Besides her role in redefining the disorder, Douglas popularized a new ways of studying hyperactivity, based on psychological theory and empirical methods. Sandberg and Barton write that her systematic, detailed work “helped establish a tradition of high-quality research in the study of cognitive aspects of hyperactivity” (p. 20).

Psychophysiology

Along with the major developments made by Douglas and her team, the explosion of interest in the 1970s saw several other changes in the field. One such phenomena was the large number of attempts to study hyperactivity using psychophysiological measurements. These measures, such as galvanic skin response, heart rate acceleration and different electroencephalogram measurements, were used to test theories about differences in central nervous system arousal levels. Two such models had been proposed in the past: models of overarousal and underarousal. Authors such as Kahn and Cohen, Strauss and Lehtinen and Laufer et al. interpreted hyperactivity as a result of an over-aroused brain that is largely at the mercy of excess impulses, a view later refuted by Douglas (Sandberg and Barton, 2002, p. 19). Authors such as Bradley explained hyperactivity as a result of underarousal of areas of the brain responsible for impulse control.

Unfortunately, psychophysiological studies yielded little advancement in understanding. Their research was limited, in part, because they focused primarily on testing theories developed two to three decades earlier, rather than incorporating more recent approaches. They were highly criticized at the time for being methodologically flawed and difficult to interpret (Barkley, 1990, p. 20). The areas of psychophysiology and neuroscience eventually began to contribute prominently to ADHD by the 1990s. More fruitful areas of research in the 1970s focused on genetics, the question of how parenting and hyperactive behaviour interact, and the treatment of the disorder.

Debates on Causality, Nature and Nurture

Research in the 1970s did not just focus on the nature and definition of childhood hyperactivity, but many studies also attempted to gain a better grasp of how the disorder was caused. This would seem to have been pertinent goal, especially since the assumption that hyperactivity was usually caused by undetectable brain injury had recently been abandoned. Some researchers investigated the possibility of genetic contributions. Morrison and Stewart and Cantwell conducted family studies of the hyperactive child syndrome (Barkley, 1998, p. 36). They found that parents of hyperactive children had a higher incidence of psychiatric disorders such as alcoholism, sociopathy, hysteria and depression. These parents were also frequently assigned retrospective diagnoses of childhood hyperactivity by the researchers, based on their descriptions of their own childhood.

The finding that a variety of psychiatric illness in parents could lead to hyperactivity in a child was not new. Tredgold attributed such nonspecific “inheritance” to the existence of inferior, vulnerable germ-lines among some families, particularly of the working class. Pasmanick proposed that the living conditions associated with socioeconomic disadvantage can lead to birth complications, the end result being a greater prevalence of psychiatric disorders in poor families and communities. British child psychiatrist and ADHD researcher, Michael Rutter suggested in a 1966 paper that psychiatric problems in parents tend to exert nonspecific disturbing psychosocial influences on the child, leading to psychiatric symptoms. Morrison and Stewart explained this phenomenon at least in part by suggesting that hyperactive children are more susceptible to developing other disorders later in life (Morrison and Stewart, 1970, p. 194). Thus the high incidence of other psychiatric disorders in parents of hyperactive children was attributed in part to direct genetic transmission of hyperactivity from parents. They gave further evidence of genetic factors by finding higher rates of hyperactivity in biological parents than in adoptive parents of hyperactive children. Morrison and Stewart concluded that there is likely a “hereditary factor, possibly operating in combination with environmental factors” (p. 195). Genetic studies of ADHD became more popular in the 1990’s.

Claims about causation of ADHD were not restricted to genetic studies. Researchers in the 1960s such as H. L. Bee suggested that parental intrusiveness interfered with the development of abilities such as the child’s capacity to channel his or her attention and energy (Campbell, 1973, p. 341). Such claims continued to be made in the 1970’s from two very different angles. Psychoanalysts proposed that the negative reactions of parents intolerant towards moderately overactive children led to severe, clinical levels of hyperactivity (Sandberg and Barton, 2002, p. 21). Behaviourists blamed the ineffective use of parental commands and directions in conditioning children to control stimuli. Tizard and Hodges gave strong support for the importance of psychosocial factors when they found an association between institutional upbringing and hyperactive behaviour. This suggested that a lack of continuity in parenting may have a negative impact on control of attention and motor activity.

Other researchers in the 1970s such as Susan Campbell, one of Douglas’s colleagues, studied mother-child interactions in hyperactive children and concluded that difficulties in mother-child interactions were largely due to the child's impulsive behaviour (Campbell, 1973, pp. 347-8). Humphries et al. at the Hospital for Sick Children in Toronto supported Campbell’s conclusions that negative or directive parent-child interactions were effects rather than causes of hyperactivity by showing that mother-child cooperation greatly improved with the use of stimulant treatment to reduce the impulsivity and hyperactivity of the child (Humphries, Kinsbourne and Swanson, 1978, p. 14). Humphries et al. noted the circular reasoning used in the past in claims of cause and effect in the study of hyperactivity, stressing the need to restrict such claims to ones that can be tested empirically. Like Douglas, both Humphries et al. and Campbell used statistical analysis to derive much of their results and paid detailed attention to their methodology; for instance, Humphries using a “one-dose, double-blind paradigm”. In this way, many ADHD researchers were demonstrating a heightened self-consciousness with regard to their own experimental methods.

Around this time, ideas about causation of hyperactivity were being developed and adopted outside of the mainstream scientific community. Ben Feingold in his 1975 book Why Your Child is Hyperactive suggested that hyperactive behaviour was caused by an allergic or toxic reaction to substances such as food additives (Sanberg and Barton, 2002, p. 21)12. Many studies followed that searched for such a link, and the more highly regarded studies found no significant effect of such substances on children’s behaviour. Still, his ideas remained popular, as evidenced by the initiation of Feingold Associations of parents who lobbied for better control of artificial food substances. Other writers pointed to potential social causes of hyperactivity. For example, Block proposed that technological development and a rapidly changing culture contributed to an increased level of environmental stimulation, which would bring out symptoms in pre-disposed children. This view was refuted by hyperactivity experts Ross and Ross. But these scientists still suggested that culture might affect hyperactivity through inconsistencies between the demands on children made by families, schools and other “important institutions of enculturation” (Barkley, 1998, p. 16).

Developments in Treatment

Barkley also identifies several changes in how hyperactivity was treated in the 1970’s. Use of stimulant medication was becoming increasingly common (1998, p. 15). This was in part due to an increase in psychopharmacology research, typically using the rigorous scientific methodology exemplified in the studies of Conners, Douglas and John Werry of New Zealand. Despite the effectiveness of these drugs, the increase in prescriptions was met by reservations within the profession and especially among the public. In 1970, the Washington Post shocked Americans with a report that 5-10 per cent of school-children in Omaha were being prescribed Ritalin and other medications for their behaviour (Singh, 2002, p. 579). The resulting public uproar prompted a federal inquiry into the use of such drugs. Barkley associates the popularity of environmental explanations of hyperactivity such as those of Feingold and Block with the publics’ anxiety about the use of brain-altering medication on school-children. Prominent books were written by critics of the ADD diagnosis. Sociologist Peter Conrad argued that expansion of the diagnostic category and increase in prevalence were at least partly a result of the new availability of drug treatments for disruptive behaviours (Sandberg and Barton, 2002, pp. 21-2). Journalists Shrag and Divoky’s 1975 book gave a contentious account of the “myth” of hyperactivity disorder, implicating corporate and government interests in the creation of this myth. Such controversial publications, articles in the popular press and a passionate campaign by the Church of Scientology’s human right’s division stoked a polarized public debate about the issue of diagnosing and treating ADD. Anti-ADHD writers and activists accused psychiatrists of acting against patients’ interests by needlessly “drugging” children, while psychiatrists and psychologists dismissed their critics as misinformed, unscientific and reckless. Thus the use of stimulant medications likely led to heightened wariness of the medicalization of a type of childhood behaviour, which had in fact been medicalized for over half a century. This increase in public interest also corresponded with the increased research interest in the 1970s and at least a perceived increased prevalence of diagnoses.

A large body of research investigated possible methods of Behavioural Modification (Barkley, 1998, pp. 17-8). Successful results contributed to the consensus that stimulant drugs should never be depended on alone to treat hyperactivity. A 1975 public law in the United States mandated special educational services for children with behavioural disabilities and provided financial incentives to encourage the adoption of these policies by states. As a result, many states made such programs available to hyperactive children.

The 1980s and 1990s

Interest in ADHD exploded further in the 1980s and especially the 1990s. This time period will only be dealt with briefly here, as it is difficult to discuss such recent events as history. Moreover, the volume of research is so great, that it is difficult to identify a small number of major articles that can be interpreted as being representative of trends in the 1980s and 1990s. But it largely because of this recent high level of interest in ADHD that these years are important to deal with, at least briefly. To summarize more recent developments, I will largely draw on the major 1998 scholarly literature review article by Toronto psychologist Rosemary Tannock13.

Improving Diagnostic Criteria

In the 1980s many prominent researchers, such as Jan Loney, Michael Rutter and Barkley, focused on improving diagnostic criteria, by making them more empirically-based and more operational14 . This new emphasis seems to largely have been inspired by the work of Douglas and her colleagues, as experts became more concerned with the validity ADD’s definition than with its cause. Establishing clear and widely accepted diagnostic criteria was seen as necessary for further production of consistent research. One resulting change in clinical practice was that Conners Rating Scales were largely replaced by the Child Behaviour Checklist, which was viewed as a more comprehensive, rigorously developed scale. In addition, experts pointed out that validity of the DSM-III’s subtyping of ADD based on the presence or absence hyperactivity was based on little empirical evidence. In a revised edition of the DSM-III (DSM-III-R, 1987), the name of the disorder was changed to Attention Deficit and Hyperactivity Disorder (ADHD). ADD without hyperactivity was replaced by the vague, obscure category “undifferentiated ADD”. This was regarded as a temporary category, to be refined after further research into the relationship between attention problems and hyperactivity (Barkley, 1998, chap. 1). Indeed, further changes to classification and diagnosis arrived in the 1990’s, as the 1994 DSM-IV included non-hyperactive children back into the diagnosis, by subdividing ADHD into three subtypes: the predominantly inattentive, hyperactive-impulsive and combined subtypes. These subtypes were based on a large, multi-site statistical study, and therefore are regarded as more empirical, though they are not considered to be final, definitive subcategories. More probably they represent another temporary refinement to the definition and subclassification of hyperactivity.

In contrast to the 1970s and 1980s, when researchers largely focused on characterizing hyperactivity rather than determining its causes, the 1990s saw an increased focus on the etiology rather than definition and description of the disorder. In her review of the scientific literature of the 1990’s Tannock, divides this research into two different types: those developing cognitive models, based on psychological theory, and t those developing neurobiological models, which draw on different areas of biology to explain hyperactivity in terms of genetic influences as well as structural and biochemical abnormalities in the brain (Tannock, 1998, p. 65). These different approaches developed considerably in the 1980s, but exploded in the 1990s, as more and more energy was invested in attempts to understand the cause of ADHD.

Cognitive and Behavioural Theories

In the 1980’s, the behavioural inhibition system, became an increasingly popular subject of study for people trying to understand ADHD. Herbert Quay was a key figure in developing this theory, which described a balance between a behavioural activation system (BAS) and behavioural inhibition system (BIS), the later of which was regarded as under-active in ADHD. Alternatively, the disorder was conceptualized by some as a motivational deficit, which affected patient’s responsiveness to the anticipated consequences of their behaviour. Behavioural inhibition continued to be the focus of cognitive models of ADHD throughout the 1990s, and motivation has been discussed as an alternative way of understanding the disorder.

The majority of cognitive research in the 1990s has treated ADHD as a central defect of poor self-regulation and behavioural inhibition. Thus by the 1990’s, each of the three main symptoms that have characterized ADHD and its predecessors have also received attention as the “central defect”. Kahn and Cohen, Laufer and Chess focused on hyperactivity as the primary problem. Douglas and her contemporaries largely emphasized inattention in their research, although also were interested in impulsiveness. And finally, researchers in the 1990s decidedly shifted away from inattention, to concern themselves primarily with impulsiveness.

Several different cognitive models have been recently proposed, most of which relate ADHD to behavioural inhibition. Gordon Logan and Schachar conceptualize impulsive behaviour to be a result of an inefficient inhibitory control systems. In a model resembling Quay’s depiction of BIS and BAS systems, Logan and Schachar suggest that impulsive behaviour can either be caused by an excessively fast response to stimuli or excessively slow inhibitory processes. They implicate slow inhibitory processes to play a role in ADHD. Behavioural inhibition is considered to be one of the “executive functions” that are involved with the execution of complex, planned actions15. By contrast, Barkley, views response inhibition as a separate entity that functions in conjunction with the executive functions. In Barkley’s theory, the ability to inhibit responses to stimuli provides a delay between stimulus and action. This allows the executive functions to then control behaviour, so that actions are organized and guided towards future consequences. Thus according to Barkley, ADHD represents a primary defect in behavioural inhibition, which in turn, impairs the executive functions.

Another model, proposed by British researcher Edmund Sonuga-Barke, does not regard ADHD as an impairment in a particular psychological process. Rather, deficiencies in inhibition are viewed as deviance, or problems with “motivational attitude”. Sonuga-Barke arrived at this conclusion through analysis of the situational contexts in which impulsiveness appear most prominently. He concludes that the central problem of the disorder is “an aversion to delay or suppression of responses over time” (Tannock, 1998, p. 70). Thus impulsive behaviour represents an attempt to reduce the perception of delay.

A fourth group of theorists, including Sanders, Sergeant and Van der Meere, offer a model that is quite different from the others. Their model distinguishes between processes – discrete, short-term operations – and energetic mechanisms – conditions which modulate these processes. They have proposed three energetic systems: arousal, which alerts sensory systems; activation, which affects the readiness for motor action; and effort, which involves motivational factors. ADHD is then conceptualized as an underlying defect in these energetic mechanisms, quite a different conceptualization than the previous three approaches. Still, the most popular streams of thought on ADHD in the 1990’s focused on either behavioural inhibition or motivation. Both of these approaches were developed in the 1980s, but in the 1990s interest in etiology – and especially in behavioural inhibition and the executive functions – intensified.

Executive Functions and their Relationship to Cognitive Models of ADHD

Tannock claims that the cognitive theories of the 1990’s have been influenced tremendously by the recent popularity of the concept of “executive functions” in psychology. In current ADHD research, the executive functions are considered to be functions that enable people to execute complex actions that require planning. Barkley, a major figure in the current theorizing about ADHD, describes the executive functions as “self-directed actions of the individual that are being used to self-regulate” (Barkley, 1998, p. 233). According to Barkley, the four executive functions are non-verbal working memory, internalization of speech, or verbal working memory, the self-regulation of affect, motivation and arousal and reconstitution. These terms will not be explained here, but Barkley eloquently illustrates how they are relevant to normal function and the abnormal functioning seen in ADHD. He writes that the executive functions are self-regulating processes that “shift behaviour from control by the immediate environment to control by internally represented forms of information by their influence over … motor control” (ibid., 229). Behavioural inhibition is considered by some scientists to be an executive function and by others to be a separate entity that interacts with the executive functions. For example, according to Barkley behavioural inhibition allows us to utilize our executive functions: we first must inhibit our immediate, impulsive response to a stimulus before we use our executive functions to carry out a more deliberate, planned action.

Barkley’s depiction of the executive functions and their interaction with behavioural inhibition appears to be particularly relevant to ADHD, as it links impulsive behaviour to the problems with higher functioning that people with ADHD experience in day-to-day life, school and the workplace. But according to Tannock, this is far from the only use of the term executive functions. Rather the concept has been given diverse, inconsistent definitions, and therefore is point of frequent confusion within the scientific and medical communities. The term has been used inconsistently, largely because it is made up of constructs that are poorly defined and difficult to measure. Consequently, the popular but vague concept is interpreted in different ways in attempts to conceptualize different disorders. Thus Tannock contends that the concept of executive functions is being applied to almost every developmental psychiatric disorder. While the concept of executive functions offers great intellectual appeal to current behavioural scientists, it seems to suffer from many of the limitations that plagued previous theorizing about hyperactivity, in that it is vague and difficult to measure.

Neuroscience Theories

Researchers in the 1980s and 1990s also tried to explain ADHD at a more concrete, biological level: this biomedical research can be divided into neuroscience and genetic research. Neuroscience approaches have focused on neurotransmitters and imaging. Paul Wender was among the researchers who began to postulate connections between ADHD and neurotransmitter systems in the 1970s. In the 1980’s, deficiencies in neurotransmitters such as dopamine and norepinepherine were increasingly implicated in ADHD. Barkley was influential in linking possible deficiencies of a dopamine transporter with his cognitive model for the disorder. In the 1990’s scientists also began studying the molecular basis of ADHD from a genetic perspective. Many such researchers focused on genes relating to the dopamine system, such as dopamine transporter and receptor genes.

Neuroimaging gathered interest in the field of ADHD in the 1980’s, as scientists such as Hans Lou from Denmark detected different patterns of underactivity in the brain (Lou et al., 1984, 1989) Neuroimaging became a major field of science by the 1990’s, and a large number of specialized neuroimaging studies on ADHD have been published since. Structural imaging techniques such as Computerized Transaxial Tomography (CT) scans are used to search for abnormalities such as differences in volumes in specific brain structures. Functional imaging techniques such as Quantitative Electroencephalography (qEEG) are used to detect differences in brain activity in a variety of regions and during different cognitive activities. Abnormal volumes have been associated with structures such as the prefrontal cortex, basal ganglia and corpus callosum. Similarly, functional studies have found differences between ADHD patients and controls, but these differences have varied (Tannock, 1998, pp. 73, 77).

Tannock attributes this wide variety of results to several factors: diverse experimental methods, which are difficult to compare to each other; limitations of the various imaging techniques; and methodological limitations relating to sample size, subject selection, matching and recording of relevant information about the subjects. Thus it is difficult to make sense out of the neuroimaging findings to date. It is possible that abnormal findings in different brain areas may represent a dysfunction in a certain brain system, such as a group of frontal lobe-striatal networks, which involves the communication of multiple brain structures. Various imaging findings can be interpreted as supporting particular cognitive theories, based on the localization of different brain functions. However, it is difficult to draw such conclusions from neuroimaging findings, because normal variation exists in most brain measurements, and because of the difficulty of relating these measurements to cognitive functioning. It remains to be seen whether the different neuroimaging techniques begin to help piece together a convincing neurological model for ADHD, or whether they merely continue to present a diffuse collection of inconclusive correlations.

Genetics

Genetic studies of ADHD first appeared in the 1970s by scientists such as Morrison and Stewart16, but became a subject of much attention in the 1990s, as both the fields of genetics and ADHD research gained increased prominence. Genetic research typically uses statistical methods to analyze the occurrence of ADHD symptoms in families in order to determine to what extent the disorder is inherited. Twin and adoption studies are used to estimate the relative contributions of genetics and environment to this heredity. A strong genetic heritability has been found, but Tannock points out that these genetic studies have not been free from methodological limitations and biases (p. 85).

One such complicating factor involves the many cognitive and behavioural disorders that occur in high rates of co-morbidity with ADHD. All cognitive, imaging and genetic approaches must take diseases co-morbid with ADHD into account in their methods and theories. This issue certainly affected earlier research: for example, Still grouped impulsive behaviour with malicious and destructive tendencies while Strauss associated hyperactivity with learning disabilities and intellectual impairment. However, only recently have researchers become highly conscious of ADHD’s relationship with other developmental disorders. Douglas and Conners were among the first to identify the need to clearly differentiate ADHD from similar disorders. Subsequently, the issue of co-morbidity with other conditions only became a major research subject in the 1980s. High rates of co-morbidity with Conduct Disorder and Learning Disabilities complicate our interpretation of genetic studies.

Another issue important to genetic research is the question of whether to conceptualize ADHD as a categorical disease or behavioural dimension17. Statistical methods often treat ADHD as either a category or dimension, and methods which take the later approach often appear to be more powerful tools (though not necessarily representing the correct conceptualization). As mentioned above, some scientists search for the mode of inheritance on a molecular level, and have proposed a number of candidate genes, including some which relate to the dopamine system. The implications of this molecular approach are also drastically affected by our conceptualization of ADHD as a category or dimension, as differences in DNA can be conceptualized as representing variation or as genetic defects which identify a behavioural disorder.

While relatively new fields of study such as neuroimaging and genetics are being developed, still more approaches are emerging that promise to offer new perspectives regarding Attention Deficit and Hyperactivity Disorder. According to Tannock, insufficient attention has been given to understanding ADHD through a developmental approach. Such an approach considers abnormal behaviours in the context of normal developmental processes. In the case of ADHD, this involves looking at the development of self-regulation (Olson, 2002, pp. 248-50). This includes an interplay of neurological development, psychological maturing and caregiver-child interactions. Thus both inborn qualities, such as temperament and cognitive abilities, and environmental influences are considered together, in order to understand the process of abnormal development. Sheryl Olson from University of Michigan writes that such a developmental perspective has recently become valued as an important area of research (2002, p. 242). Tannock suggests that such an approach could complement the cognitive, neuroimaging and genetic approaches, by integrating their different findings into a broader model. This could help prevent researchers from adopting “single-cause, reductionist models that do not capture the complexity of the dynamic processes in developmental psychopathology” (1998, p. 66).

In the 1990s, several increasingly specialized and complex fields of science took part in the searching for the etiological basis of ADHD. This tremendous research yielded a wide variety of findings and possible mechanisms. A 1998 National Institutes of Health Consensus Development Conference statement concluded that “after years of clinical research and experience with ADHD, our knowledge about the cause or causes of ADHD remains largely speculative” (Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder, 1998). A more troubling conclusion of the symposium was that the diagnosis of ADHD lacked consistency, despite the various attempts to standardize the process in the DSMs III and IV. While research interest shifted from developing an empirically-based definition in the 1970s to establishing valid and operational diagnostic criteria in the 1970s and 1980s, and to understanding the underlying mechanisms in the 1990s, all three tasks appear to be far from complete18.

3.For readability, I tend to refer to these groups of children who would come to be referred to as having ADHD as “hyperactive children” throughout the paper.

4.Feeblemindedness referred to people with either mildly lower intelligence or delinquent behaviour believed to typically occur in the urban working class (Jones, 1999)

5.On the other hand, a behavioural disorder that emerged only recently might be viewed with more suspicion, due to an image of being “constructed” rather than “discovered”. This image of being “constructed” would be favoured, because people may assume that overt, behavioural manifestations of a pathology that has always existed would not have escaped notice until recent years. This points to one of the many stimuli that may inspire psychiatrists and psychologists to include history chapters in their textbooks: the chance to give an added sense of legitimacy to their area of study. Of course, regardless of its age, a scientific concept such as ADHD can be viewed as either “constructed or “discovered”, and both views contribute to our understanding of how scientific knowledge is created.

6. His opinion was also swayed by a lack of “physiological evidence” demonstrated in electroencephalogram studies (Bradley and Bowen, 1941, pp. 101-2)

7.The classification of developmental disorders became a focus of child psychiatry by the 1970s; this remains far from a resolved subject today.

8.Psychoanalysis lent itself to the treatment of so-called “internalizing disorders”, such as neuroses and anxiety, where patients repress feelings. On the other end of the spectrum of psychiatric problems, hyperactivity is regarded as an “externalizing disorder”, were patients seem to immediately act on their feelings and desires rather than repressing.

9. Barkley might remind Schachar that trying stimulants on non-hyperactive children might further dismay ethics committees.

10. It is worth illustrating an example of such psychometric tests. In the Continuous Performance Test (CPT) 12 letters would appear on a screen, one at a time, and the child was instructed to respond to the “significant stimulus”: the letter X, but only when immediately preceded by the letter A (Douglas, 1969, p. 203). On this test, hyperactive children were found to have a greater tendency to respond after the letter A before the next letter appeared, and generally displayed more errors of commission and more “random responses”. The CPT was later standardized and widely used in ADHD research and clinical assessment (Barkley, 1990).

11. Berrios and Gili discuss the history of the concept of volition and disorders of the will in their 1995 article. They argue that the concept was ignored during much of the 19th and 20th centuries largely because of professional politics and fashion, rather than inferior scientific validity.

12. It is worth noting that Feingold was a physician, though he did not seem to be one of the major hyperactivity researchers within the psychiatry or psychology communities.

13. Interestingly, internal histories largely resemble scholarly literature reviews, except that they review much older research.

14. Criteria that are more operational are more practical to apply in practice, often by being less vague or involving qualities that are reasonably straightforward to assess.

15. As suggested here, the executive functions are psychological constructs representing mental processes that enable people to plan and perform complex tasks. The concept of executive function will be discussed in more detail in the next section.

16. Their early genetic work on hyperactivity is mentioned earlier, in my discussion of the 1970’s, page 34.

17. A categorical disease is qualitatively different from the normal population, thus having some behavioural or pathological differences that make them a distinct group. Alternatively, disorders such as ADHD can be conceptualized as the extreme end of a behavioural dimension. According to this model, the disorder is essentially composed of qualities that exist in the normal population, such as overactivity or attention problems, but in excessive degrees. In this sense people with the disorder would not possess any qualitative distinctions from the rest of the population, but rather lie at the end of a continuum. Thus diagnosis is a somewhat less straightforward issue in a disease that exists on a continuum, since it would necessarily involve a somewhat arbitrary cut-off to separate the extreme from the normal population.

18.Treatment of ADHD has also been a major area of study in the 1990s and in the current decade. Large sample studies, most recently the major multiple center Multimodal Treatment Study (MTA) run by the National Institute of Mental Health, have tested stimulant medications such as Ritalin, other medications that used as alternatives to stimulants, behavioural modification therapy and consultation with parents and teachers (Jensen et al., 2001). The subject of treatment will not be discussed here; for my purposes, it is sufficient to mention that medication is regarded as the most effective treatment, although authorities such as the MTA group advise that medical therapy is most effective when complemented with psychosocial measures. Interestingly, the NIH consensus statement mentioned above points out the lack of any knowledge about prevention of ADHD, which is mainly due to our lack of understanding of the etiology, but also due to assumptions that it would be more effective to treat ADHD with medication than to try to prevent its occurrence.

Interpretation of the History of ADHD

This paper traced a history of medical conditions used to describe a childhood behaviour pattern of hyperactivity, impulsiveness and inattentiveness. These disorders were not identical, but were linked to each other by a continual process of redefinition by psychiatrists, psychologists and pediatricians. The shift in names reflects deeper conceptual changes. The central problem changed first from a defect in moral control to a deficiency in judgment, control and organization of behaviour on the part of the mildly mentally deficient. Following this, the central problem became hyperactivity, which at times was associated with cognitive differences, but this soon shifted to a motivational disorder primarily involving deficiencies in sustained attention and impulse control. Most recently, the disorder has been understood as a primary deficiency in inhibitory control linked with impaired “executive functions”. In the process, the diagnosis has expanded to include many children who are not hyperactive, as well as adolescents and adults. The cause of the disorder has shifted as well: from a largely inherited brain defect affecting some of the highest, but most fragile human functions to undetectable, microscopic brain lesions acquired in minor brain injury; then to a neurological, but admittedly unclear pathophysiology; and lately, to a largely heritable trait related to impaired cognitive processes and possible defects of several brain regions and neurotransmitter systems. The constant in this history has been the widespread though not unanimous belief that childhood hyperactivity is an organic condition that should be studied and treated medically.

Psychologists and psychiatrists writing internal histories describe an advancement towards superior technologies and research methodologies and the development of a progressively more detailed understanding of the disorder, though not without missteps in the course of scientific discovery. But a history of steady scientific progress does not seem to adequately account for the constant, rapid shifting between concepts seen in the history of ADHD. Therefore it is important to look at these conceptual shifts as a function of variables besides technological and intellectual advancement. The development of new technologies have certainly led to more sophisticated experimental methods, empirically-based diagnoses and new treatments. For example, the effects of technological changes can be observed by looking at the different ways the disorder has been studied over the past century. Hyperactivity was initially understood through observations in case studies. Later, standardized, psychological tests and neurophysiologic measures were adopted. By the 1960s and 1970s standardized questionnaires and statistical tools were used to make the diagnosis more empirical, and in the past two decades, neuroimaging has been used to understand brain mechanisms while statistical tools have been applied to study possible genetic causes. But these new technologies were not developed and adopted simply because they were more “advanced” or useful; rather, they thrived in appropriate social and political conditions. Thus the evolution of ADHD is not only a scientific process but a complex, historical process.

Professional Context

An integral part of Andrew Lakoff’s history of ADHD is the context of the child psychiatry profession. He writes that in the 1970s child psychiatry had reached a crisis. Up until this point the field had been predominantly based on the psychodynamic approach introduced by one of the major founders of the profession in America, Adolph Meyer. Psychodynamics was the focus of practitioners in Child Guidance Clinics of the 1920s to 1940s and Urban Community Mental Health Centers of the 1960s. This emphasis on analyzing a patient’s life history to understand maladjustment was seen as a superior, more patient-centered replacement of descriptive nosology. Descriptive nosology, introduced by Emil Kraepelin’s influential turn of the century work, limited psychiatry to describing and classifying diseases without hypothesizing about causes, and diagnosing patients based on this rigid, seemingly objective framework (Lakoff, 2000, p. 154). Kraepelin appears to have been a major influence on Tredgold, who rigorously classified different types of amentia based on clinical and pathological observation. On the other hand, this approach was absent in later work by Kahn and Cohen, Strauss and Laufer, who were primarily interested in understanding the causes – organic and, to a lesser extent, psychodynamic – of hyperactive, impulsive behaviour. Lakoff notes that the Kraepelinian approach would “return with a vengeance” by the 1970s, when Conners, Douglas and the DSM-III brought to the forefront concerns about the classification and definition of hyperactivity. This change took place at a time when the child psychiatry profession was at risk of losing much of its prestige and even its clientele.

By the 1970s, child psychiatry practices in Urban Community Mental Health Centers were no longer valued, as the empowerment movements of the 1960s drastically altered social welfare agencies and their staff (Lakoff, 2000, 157). Health budgets in the United States were tightened in the 1970s, and third-party payers increasingly demanded that treatments be proven effective and efficient by clinical trials. This attitude placed psychoanalysis under scrutiny, as lengthy, expensive therapy sessions were criticized as unproven and wasteful. This coincided with the declining prestige of the psychiatry profession, which could point to little evidence of progress in their understanding and treatment of mental disease. Psychopharmacology began to explode in the 1960s, and these medical treatments appeared to be more effective than the mainstay of psychiatry, psychoanalysis. Thus embracing biomedical approaches and the models of somatic medicine appeared to be an important adjustment for the psychiatry profession, one which could salvage the vitality of the field. In this sense, Lakoff suggests that changes in psychiatry in the 1970s can be viewed “as a kind of adaptive strategy” as opposed to the result of any technological breakthrough. Psychotropic medications had been prescribed and statistical studies conducted in the 1960’s and earlier, but these methods were embraced as central rather than peripheral parts of psychiatry in the 1970s.

This change of attitude is apparent in the professional politics of psychiatry and child psychiatry at the time. In 1974 Robert Spitzer, a disaffected psychoanalyst who had adopted the philosophy of a neo-Kraepelinian group of academics, was named head of the American Psychiatry Association DSM steering committee. Spitzer recruited like-minded colleagues to the committee to draft a DSM-III based on purely descriptive, objective diagnostic categories. Similarly, in 1976 Melvin Lewis became the editor of the Journal of the American Academy of Child Psychiatry, which had previously been dominated by psychodynamic articles (Lakoff, 2000, pp. 155-158). Lewis brought more biological research into the journal, and demanded that research meet scientific criteria such as being replicable.

In the early twentieth century, childhood hyperactivity was already studied in a biomedical fashion, by pediatricians along with psychiatrists. But researchers such as Kahn and Cohen, Bradley and Laufer incorporated psychoanalytic principles into their biomedical perspectives. While using some decidedly biomedical methods, including statistical analysis and neurophysiological measurement, their work was largely based on the case-study, which could impart the details of a life story that were relevant to psychoanalysts. Additionally, Lakoff claims, hyperactivity was a relatively obscure disorder in psychiatry at this time, possibly because it was less responsive than internalizing disorders to psychoanalytic treatment and often left for pediatricians to treat and study. However, by the time of Douglas’s research in the 1970’s, ADD was one of the most central, well known disorders in child psychiatry; a heavily discussed topic both within the profession and by the general public. Hyperactivity, which could be treated effectively with psychotropic medication, was a natural point of focus of a new, biomedical child psychiatry. By the late 1980s, child psychiatry had recovered well from its crisis, as Stella Chess declared at the 1988 meeting of the Child Psychiatry Association that the field had “come of age”. Thus the process of redefinition of ADHD is linked closely to the transformation of the child psychiatry profession.

The medical model brought many advantages. It brought new prestige to the discipline of psychiatry as a rigorous medical science. The medical model provided a convincing, nonjudgmental explanation for troubling behaviour to patients and parents. In contrast, psychodynamic approaches largely attributed hyperactivity to faulty parenting19. In addition, new statistical methods likely made hyperactivity more prominent, by simply yielding a clear definition of the disorder; professionals and interested lay people, could finally turn to specific, empirically derived diagnostic criteria to confidently differentiate between clinical presentations that were or were not ADD. Of course these criteria frequently changed and will undoubtedly continue to change. And they leave much room for interpretation and subjective judgment. But the existence of a diagnostic algorithm, albeit an imperfect and temporary one, inspires at least a certain degree of trust in diagnosis as a medical act.

Lawrence Diller, a physician and prominent author on ADHD, and Rosemary Tannock observe that medical, statistical approaches tend be reductionist, as they give focused biological explanations instead of more realistic, accounts of a disorder that is almost certainly heterogeneous in nature and cause20. Unlike the case study, statistical studies analyzing large numbers of patients, and so cannot narrate the subjects’ life histories and personal experiences; information gathered needs to be measurable and quantifiable. Thus rather than understanding hyperactivity in terms of a combination of organic pathology, environmental influences and the patient’s response to these conditions, statistical studies naturally focus on the first factor. But this reductionism is not simply a side effect of undertaking empirical, large-scale studies; rather, it is a function of the medical model. Scientific reductionism became the dominant perspective, because it offered advantages over the psychodynamic perspective. The psychodynamic model was unable to give a clear, specific definition for a disorder of which we, in truth, do not have a very clear understanding. Rafalovich writes:

“The psychodynamic perspective allows in the ambiguities which its discourse describes: an ADHD child may or may not have organic brain damage, may or may not need extensive therapy, may or may not need to be medicated. From the perspective of neurology these become non-issues once a positive diagnosis of ADHD is made.”

The unambiguous neurological model helped people “make cognitive and policy sense” of a complex problem (Rosenberg, 1989, p. 4). For the patient, “Previous incoherent signs took on biological meaning retrospectively when the correct diagnosis was made” (Lakoff, 2000, 162). Public health officials could assess the significance of the disorder and its long-term risks21. Treatments were proven effective in empirical ways that satisfied health care providers. And the condition was treated without blaming the child, the family or society. While the medical model was not necessarily more valid than psychodynamic models, it was more prestigious for psychiatrists and possibly more therapeutic for patients and their families.

Social Meaning

While professional interest contributed to the evolution of ADHD, on a different level, the disorder changed, because its social meaning changed. As articulated by Charles E. Rosenberg, any disease is defined not only by scientific knowledge and treatment, but also by social and economic conditions that affect the way people understand and experience the illness (Rosenberg, 1989). Lakoff further claims that psychiatric concepts reflect our values and social expectations – norms described by Norbert Elias as the “civilizing process” – since, after all, they involve the distinction between normal and “pathological” behaviour. Still’s concept of a “defect in moral control” reflected a concern with the effect of behaviour on the collective good. Channeling one’s behaviour according to the interests of others – being of “good character” – was viewed as an integral part of a person’s functioning in society: “In Still’s evolutionary framework, the badly behaving child was not yet properly civilized, was still incapable of the inhibitions necessary for social inclusion” (Lakoff, 2000, p. 165). IS THIS NOWADAYS WRONG?

By the early and mid-nineteenth century, hyperactivity and other developmental disorders took on a very different significance. At the time of the Child Guidance movement in the 1920s, increased antiauthoritarian “problem behaviour” in youth was linked to increased crime in American cities. Socioeconomic conditions, especially relating to inner city and immigrant life, was thought to largely be responsible for rising juvenile delinquency, which in turn threatened to lead to more serious crimes as young delinquents grew-up. Therefore treating children with behaviour problems – usually through counseling and psychosocial adjustments – was regarded a way to decrease crime and social problems such as drug use (Jones, 1999, p. 48). In the postwar period of the 1940s, psychiatric and behavioural problems were not only considered to be problems on the personal and community level, but also seen as threats to democracy. Consequently, a new National Committee on Mental Hygiene (NCMH) sent child psychologists and counselors into American schools, in order to detect the beginnings of psychiatric and personality abnormalities and help prevent their progression to greater problems. Ilina Singh, suggests that his goal of preventing psychiatric problems “likely helped encourage the scientific establishment of normative standards of children’s cognitive and emotional behaviours” (Singh, 2002, p. 584). Hyperactivity was soon understood in relation to these norms of cognitive and social functioning on behaviour rating scales.

In the 1970s and 1980s, community support groups of ADD patients and their parents, further altered how people with the disorder identified themselves. With the increasingly widespread use of Ritalin, ADD became a largely treatable condition. This medical treatment helped solidify ADHD’s status as medical condition22. The presence of an effective medical treatment drastically changed the social characteristics of the condition and the implications of diagnosis on the patient. As this treatment became common, one could observe ADD patients whose disorder was “corrected”. Thus one could seemingly separate the patient’s “true self” from the pathology that normally affected their behaviour. The effects of Ritalin provided a convincing demonstration of the belief espoused by the scientific community, first expressed by Kahn and Cohen, that the core personality of an ADHD patient was separate from their psychiatric disorder, which could be seen as an unwanted, foreign illness altering the patient’s brain (Lakoff, 2000, p. 166).

By the 1990s, hyperactivity was not seen primarily as threat to the collective good, urban safety or national security. Rather it was a problem for the individual child and family that threatened to prevent the child from reaching his or her professional and personal potential. Lakoff proposes that the disorder resonated so strongly in America of the 1990s, because recent social norms, the “civilizing process” of the 1990s, have been tied to principles of “rational self-management” and “the family investment in the future of the child” (p. 165). In this society of personal goals and rational, individualistic decision-making, people need to be effective in managing themselves and organizing their own behaviour in order to meet their own goals: in psychological terms, this means having good executive functions. In this social context, it is not surprising that many psychiatric disorders are understood in terms of executive functions, and that ADHD, which has had a particularly close association with the executive functions, is a subject of great scientific and lay interest. Thus Lakoff refutes the anti-psychiatry contention that ADHD became prominent, because it was used as a tool for social control of childhood deviance. Rather the disorder developed into an entity mainly important to the individual and family: “Police and school authorities did not so much impose the diagnosis as parents and children insisted on the validity of the designation” (p. 165)23. Professional and social climate, as well as the existence of an effective medical treatment that moderates troublesome behaviour with minimal side-effects, have combined to make ADHD a wildly successful psychiatric disorder at the beginning of the 21st century.

Conclusion

Ilina Singh points out another element that has helped define ADHD in the 1990’s. The center of an intensely publicized and polarized debate, ADHD has been “elevated to cultural icon status” in Western society, particularly in America (p. 598). Proponents of the scientific model of ADHD speak in a technical discourse of neurotransmitters, cognitive processes and genes, while skeptics speculate about corporate conspiracy, psychiatry as a form of social control and the psychological impacts of a modern, fast-paced society. From the perspective of neuropsychiatry, ADHD can be seen exclusively as a phenomenon of the brain. At the other extreme, one can turn to Foucault’s hostile view of psychiatry, where the establishment of a mental illness is a process of re-inscribing social norms on a society in the reified form of “objective” scientific fact. Both these sides “often lose sight of the grounded realities”: that ADHD and Ritalin are “phenomena grounded in time, space and people’s lives” (Singh, 2002, p. 598). Studying the conceptual history of ADHD does not reveal a story of scientific revelations or of conspiracy, but rather one of intellectual, technological, professional and social changes.

Purely scientific and anti-psychiatry views as well many other positions in between these extremes, coexist. But North American people have generally adopted the medical perspective espoused by the majority of their psychiatrists24. While people may worry that Ritalin is over-prescribed, this is seen largely as a problem of excessive referral and inconsistent diagnosis: parts of the medical processes that need to be refined. Deficiencies in the education system are implicated in contributing to high rates of referral for ADHD diagnosis, but ADHD remains primarily a medical entity. Thus the diagnosis and treatment of ADHD are regarded as medical issues. Technical, biomedical discourse inspires confidence through an image of meticulousness, objectivity and benevolence (Rafalovich, p. 412). But as this paper has attempted to illustrate, scientific disciplines, like all academic disciplines, exert influence not only by using technology to answer our questions, but also by selecting which technologies to use and which questions to ask. Medicine and science identify a limited range of moral concerns, which guide and limit clinical research and practice: for example, treating patients and subjects kindly and humanely, respecting patients’ right to make autonomous decisions and providing the best medical treatment possible to each patient. Besides such ethical considerations, the study and treatment of ADHD are considered to be biomedical topics. However, Charles Rosenberg reminds us that medicalizing behaviour changes the moral meaning this behaviour (1989, p. 10). Consequently, behavioural disorders are defined and redefined in a process of “social negotiation” between the medical community, social scientists, the government and the public (p. 11). Ideas and practices relating to ADHD reflect certain value judgments, whether these choices are made consciously or implicitly.

These value judgments can found in the assumption made by much of the psychiatry profession and general population that ADHD is primarily a biological disorder, which may be exacerbated, but rarely caused by social factors25. They are found in how we interpret the significance of cognitive and neuroimaging studies. And they are found in the belief that the patient’s core personality is separable from their disorder. This last assumption justifies psychopharmacologic treatment, by depicting medication as an agent which enables the patient to fulfill his or her true potential. By contrast, people skeptical about ADHD often view hyperactive and impulsive traits as part of the patient’s personality, and therefore believe that Ritalin alters the patient’s true personality. There does not seem to be much scientific evidence which would significantly favour one side of this central conflict; rather, the discrepancy seems to be one of belief and philosophy.

An important related issue is the question of whether ADHD is a category – representing a qualitative difference from the general population – or dimension – an extreme behaviour pattern existing on a continuum with “normal” behaviour 26. Some current philosophers argue that it would be more accurate and useful to view many psychiatric disorders through a dimensional rather than categorical perspective. This approach would better account for the wide variation in cases and avoid risks of stigmatization (Oepen, Harrington and Funfgeld, 1990). The conceptualization of ADHD as a category or dimension will depend on scientific evidence, philosophical stances as well as clinical and social utility. These possible conceptualizations have profound implications not only on how we understand ADHD, but also on how we understand the human capacities of behavioural inhibition and self-control. Thus moral and philosophical issues as well as scientific issues are central to the concept of ADHD27. Our understanding and treatment of ADHD is tied to our society’s morals and values; the concept of ADHD changes as these values change. In turn, developments in the diagnosis and treatment of ADHD are not just medical changes, but to some degree moral changes as well.

Like any psychiatric disorder, disorders of hyperactivity and impulsiveness reflect societal values and norms relating to certain behaviours. The evolution of these disorders is a historical process, a dynamic involving many variables, such as social conditions, professional culture, influential individuals, scientific and philosophical thought and technology. But, as Lakoff states, “to assert that the disorder is contingent is not to dismiss it as false. It is rather to ask why it took on a particular form and resonance at a given moment” (p. 165). By taking into account these different factors – factors described in both internal professional histories and in social histories – one can gain a fuller understanding of how the concept of ADHD developed and how this concept has impacted the children and adults diagnosed and their families.

19.The new interest in developmental models, would seem to have the same difficulties as previous psychodynamic approaches, as they give complex explanations for hyperactive behaviour, which consider parent-child interactions as important factors in normal and abnormal development.

20.The variety of behaviour profiles and presentations now diagnosed as ADHD lead many current experts to conclude that ADHD is heterogeneous group that will be more effectively subdivided in the future, based on nature of disorder and cause.

21.Such large scale, public health studies of childhood behavioural disorders include a major campaign started by the National Institute of Mental Health in 1990 (Lakoff, 2000, p. 161).

22.Of course strictly speaking, the existence of a biomedical treatment for the symptoms of ADHD is not necessarily imply that ADHD is a biological disorder. There is no evidence that stimulant medication corrects an underlying defect. It has been found to have the same effects of focusing and controlling effort on many people who do not have ADHD, and it does not improve symptoms in all patients with ADHD (Diller, 1998). Some raise the questions as to whether Ritalin and ADHD represent “a treatment seeking a disease” (Rafalovich, 2001, 414).

23.Singh would argue that families’ readiness to accept and even seek a diagnosis, and relinquish authority in determining their child’s needs has largely been due to insecurity and anxiety created by the psychiatric profession. Pressures and conflicting messages relating to parenting were disseminated through parenting magazines, whose articles were based on the latest scientific ideas by the psychiatry profession. Singh analyzes these articles to assess the history of hyperactivity at the level of the general population. She contends that psychiatry profession, especially psychoanalysis, discredited parenting, and in particular motherhood, as pre-scientific and often pathological (either overbearing or not disciplined enough), thus carving a niche for their profession as “parenting experts”. Anxiety about being viewed as bad mothers, made women more ready to accept treatments for their children.

24.Rafalovich claims that British psychiatrists are critical about the North American psychiatry profession’s treatment of ADHD, which is somewhat surprising, considering that the DSM and ICD definitions have recently converged somewhat.

25.Current use of the very term “pathology” for behavioural patterns seems to involve a type of circularity similar to that implicit in the old term Minimal Brain Damage. The medical community defines behaviour deemed to be problematic as pathological. But this designation does not simply imply a problem, but rather a medical problem. Thus behaviours identified by the medical community as problematic become medical conditions as well as being social troubles.

26.Some argue that behaviour now diagnosed as ADHD is no longer exclusively “extreme” patterns, but often includes people who behave and function quite normally.

27. For more depth on philosophical issues behind the treatment of ADHD, see Diller, 1998 and Gillet, 1999, chapter 7.

References

American Psychiatric Association. (1994) Diagnostic and statistical manual of mental disorders, 4th edition. Washington (DC): APA Press.

Barley, Russel A. (1990). Attention-Deficit Hyperactiviy Disorder: A Handbook for Diagnosis and Treatment. New York: Guilford Press.

Barley, Russel A. (1998). Attention-Deficit Hyperactiviy Disorder: A Handbook for Diagnosis and Treatment. New York: Guilford Press.

Berrios, G. E. and Gili, M. (1995). Will and its disorders: a conceptual history. History of Psychiatry, vi, 87-104.

Bradley, Charles. (1937). The behaviour of children receiving Benzedrine. American Journal of Psychiatry, 94, 577-85.

Bradley, Charles and Bowen, Margaret. (1940). School performance of children receiving amphetamine (Benzedrine) sulfate. American Journal of Orthopsychiatry, 10, 782-88.

Bradley, Charles and Bowen, Margaret. (1941). Amphetamine (Benzedrine) sulfate therapy of children’s behaviour disorders. American Journal of Orthopsychiatry, 11, 92-103.

Campbell, Susan B. (1973). Mother-child interaction in reflective, impulsive, and hyperactive children. Developmental Psychology, 8, 341-9.

Chess, Stella. (1960). Diagnosis and treatment of the hyperactive child. New York State Journal of Medicine, 60, 2379-85.

Conners, C. Keith. (1969). A teacher rating scale for use in doing studies with children. American Journal of Psychiatry, 126, 152-56.

Conners, C. Keith. (1970). Symptom patterns in hyperkinetic, neurotic and normal children. Child Development, 41, 667-82.

Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder. NIH Consens Statement Online 1998 Nov 16-18. 16(2): 1-37. Retrieved October 19, 2004, from http://consensus.nih.gov/cons/110/110\_statement.htm.

Diller, Lawrence H. (1998). Running on Ritalin: A Physician Reflects on Children, Society, and Performance in a Pill. New York: Bantam Books.

Douglas, V. I. and Peters, K. G. (1979). Toward a clearer definition of the attentional deficit of hyperactive children. In G. A. Hale and M. Lewis (Ed.s), Attention and the development of cognitive skills (pp. 235-253). New York: Plenum.

Doyle, Robert. (2004). The history of adult attention-deficit/hyperactivity disorder. Psychiatric Clinics of North America, 27(2), 203-14.

Gillet, Grant. (1999) The Mind and Its Discontents: An essay in discursive psychiatry. New York: Oxford University Press.

Gross, M. D. (1995). Origin of stimulant use for treatment of attention deficit disorder. American Journal of Psychiatry, 152, 298-9.

Hassler, F. (1992) The hyperkinetic child: A historical review. Acta Paedopsychiatr. 55(3),147-9.

Humphries, Thomas, Kinsbourne, Marcel and Swanson, James. (1978). Stimulant effects on cooperation and social interaction between hyperactive children and their mothers. Journal of Child Psychology and Psychiatry, 19, 12-22.

Jensen, Peter S. et al. (2001) Findings from the NIMH Multimodal Treatment Study of ADHD (MTA) : Implications and Applications for Primary Care Providers. Developmental and Behavioural Pediatrics, 22(1), 60-73.

Jones, Kathleen W. (1999). Taming the Troublesome Child: American Families, Child Guidance, and the Limits of Psychiatric Authority. Cambridge: Harvard University Press.

Kahn, E. and Cohen, L. H. (1934). Organic driveness: A brain stem syndrome and an experience. New England Journal of Medicine, 210, 748-56.

Lakoff, Andrew. (2000) Adaptive Will: The evolution of Attention Deficit Disorder. Journal of the History of the Behavioural Sciences, 36(2), 149-169.

Laufer, Maurice W., Denhoff, Eric and Solomons, Gerald. (1957). Hyperactive Impulse Disorder in Children’s Behaviour Problems. Psychosomatic Medicine, 19, 38-49.

Lou, Hans C., Henriksen, Leif and Bruhn, Peter. (1984). Focal Cerebral Hypoperfusion in Children with Dysphasia and/or Attention Deficit Disorder. Archives of Neurology, 41, 825-9.

Lou, Hans C. et al. (1989). Striatal Dysfunction in Attention Deficit and Hyperkinetic Disorder. Archives of Neurology, 46, 847-52.

Mannuzza, Salvatore et al. (1998). Adult psychiatric status of hyperactive boys grown up. The American Journal of Psychiatry, 155(4), 493-8.

Michale, Mark S. (2000). The Psychiatric Body. In Roy Cooter and John Pickstone (Ed.s), Medicine in the Twentieth Century. Singapore: Harwood academic publishers.

Morrison, James R. and Stewart, Mark A. (1971). A Family Study of the Hyperactive Child Syndrome. Biological Psychiatry, 3, 189-95.

Oepen, Godehard, Harrington, Anne and Funfgeld, Matthias. (1990). Normality and Mental Illness – Dimension Versus Categories: Theoretical Considerations and Experimental Findings. In Manfred Spitzer and Brendan A. Maher (Ed.s), Philosophy and Psychopathology (200-10). New York: Springer-Verlag.

Olson, Sheryl. (2002). Developmental perspectives. In S. Sanberg (Ed.), Hyperactivity and Attention Disorders of Childhood (242-89). Cambridge: Cambridge University Press.

Pary, Raymond et al. (2002) Attention Deficit Disorder in Adults. Annals of Clinical Psychiatry, 14(2), 105-11.

Pasmanick, Benjamin, Knobloch, Hilda and Lilienfeld, Abraham M. Socioeconomic Status and Some Precursors of Neuropsychiatric Disorder. (1956). American Journal of Orthopsychiatry, 26, 594-601.

Rafalovich, Adam. (2001). Psychodynamic and Neurological Perspectives on ADHD: Exploring Strategies for Defining a Phenomenon. Journal for the Theory of Social Behaviour, 31(4), 397-418.

Rosenberg, Charles E. (1989). Disease in History: Frames and Framers. The Milbank Quarterly, 67, suppl. 1, 1-15.

Sanberg, Seija and Barton, Joanne. (2002). Historical Development. In S. Sandberg (Ed.), Hyperactivity and Attention Disorders of Childhood (pp. 1-29). Cambridge: Cambridge University Press.

Schachar, Russell J. (1986). Hyperkinetic syndrome: Historical development of the concept. In E. Taylor (Ed.), The Overactive Child (pp. 19-40). Philadelphia: Lippincott.

Singh, Ilina. (2002). Bad Boys, Good Mothers, and the “Miracle” of Ritalin. Science in Context, 15(4), 577-603.

Spencer, T. J. (2002). Attention-deficit/hyperactivity disorder. Arch Neurol, 59(2), 314-6.

Still, George F. (1902). Some abnormal psychical conditions in children. Lancet, 1, 1008-1012, 1077-1082, 1163-1168.

Strauss, Alfred A. and Werner, Heinz. (1942). Disorders of conceptual thinking in the brain-injured child. Journal of Nervous and Mental Disease, 96, 153-72.

Strauss, Alfred A. and Werner, Heinz. (1943). Comparative psychopathology of the brain injured child and the traumatic brain injured adult. American Journal of Psychiatry, 99, 835-8.

Tannock, Rosemary. (1998). Attention Deficit Hyperactivity Disorder: Advances in Cognitive, Neurobiological, and Genetic Research. Journal of Child Psychology and Psychiatry. 39(1), 65-99.

Tredgold, A. F. (1952). A Text-Book of Mental Deficinecy (Amentia), eighth edition. London: Bailliere, Tindall and Cox.

Werry, J. S. (1974). Minimal Brain Dysfunction (Neurological Impairment) In Children. New Zealand Medical Journal, 80(521), 94-100.

©2004, Colman Nefsky, B.Arts and Sc.

HE DOESN’T CONSIDER THE POSSIBLE DEFENSIVE MEANING OF DIAGNOSIS

…………………………………….

References

American Psychiatric Association. (1994) Diagnostic and statistical manual of mental disorders, 4th edition. Washington (DC): APA Press.

Barley, Russel A. (1990). Attention-Deficit Hyperactiviy Disorder: A Handbook for Diagnosis and Treatment. New York: Guilford Press.

Barley, Russel A. (1998). Attention-Deficit Hyperactiviy Disorder: A Handbook for Diagnosis and Treatment. New York: Guilford Press.

Berrios, G. E. and Gili, M. (1995). Will and its disorders: a conceptual history. History of Psychiatry, vi, 87-104.

Bradley, Charles. (1937). The behaviour of children receiving Benzedrine. American Journal of Psychiatry, 94, 577-85.

Bradley, Charles and Bowen, Margaret. (1940). School performance of children receiving amphetamine (Benzedrine) sulfate. American Journal of Orthopsychiatry, 10, 782-88.

Bradley, Charles and Bowen, Margaret. (1941). Amphetamine (Benzedrine) sulfate therapy of children’s behaviour disorders. American Journal of Orthopsychiatry, 11, 92-103.

Campbell, Susan B. (1973). Mother-child interaction in reflective, impulsive, and hyperactive children. Developmental Psychology, 8, 341-9.

Chess, Stella. (1960). Diagnosis and treatment of the hyperactive child. New York State Journal of Medicine, 60, 2379-85.

Conners, C. Keith. (1969). A teacher rating scale for use in doing studies with children. American Journal of Psychiatry, 126, 152-56.

Conners, C. Keith. (1970). Symptom patterns in hyperkinetic, neurotic and normal children. Child Development, 41, 667-82.

Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder. NIH Consens Statement Online 1998 Nov 16-18. 16(2): 1-37. Retrieved October 19, 2004, from http://consensus.nih.gov/cons/110/110\_statement.htm.

Diller, Lawrence H. (1998). Running on Ritalin: A Physician Reflects on Children, Society, and Performance in a Pill. New York: Bantam Books.

Douglas, V. I. and Peters, K. G. (1979). Toward a clearer definition of the attentional deficit of hyperactive children. In G. A. Hale and M. Lewis (Ed.s), Attention and the development of cognitive skills (pp. 235-253). New York: Plenum.

Doyle, Robert. (2004). The history of adult attention-deficit/hyperactivity disorder. Psychiatric Clinics of North America, 27(2), 203-14.

Gillet, Grant. (1999) The Mind and Its Discontents: An essay in discursive psychiatry. New York: Oxford University Press.

Gross, M. D. (1995). Origin of stimulant use for treatment of attention deficit disorder. American Journal of Psychiatry, 152, 298-9.

Hassler, F. (1992) The hyperkinetic child: A historical review. Acta Paedopsychiatr. 55(3),147-9.

Humphries, Thomas, Kinsbourne, Marcel and Swanson, James. (1978). Stimulant effects on cooperation and social interaction between hyperactive children and their mothers. Journal of Child Psychology and Psychiatry, 19, 12-22.

Jensen, Peter S. et al. (2001) Findings from the NIMH Multimodal Treatment Study of ADHD (MTA) : Implications and Applications for Primary Care Providers. Developmental and Behavioural Pediatrics, 22(1), 60-73.

Jones, Kathleen W. (1999). Taming the Troublesome Child: American Families, Child Guidance, and the Limits of Psychiatric Authority. Cambridge: Harvard University Press.

Kahn, E. and Cohen, L. H. (1934). Organic driveness: A brain stem syndrome and an experience. New England Journal of Medicine, 210, 748-56.

Lakoff, Andrew. (2000) Adaptive Will: The evolution of Attention Deficit Disorder. Journal of the History of the Behavioural Sciences, 36(2), 149-169.

Laufer, Maurice W., Denhoff, Eric and Solomons, Gerald. (1957). Hyperactive Impulse Disorder in Children’s Behaviour Problems. Psychosomatic Medicine, 19, 38-49.

Lou, Hans C., Henriksen, Leif and Bruhn, Peter. (1984). Focal Cerebral Hypoperfusion in Children with Dysphasia and/or Attention Deficit Disorder. Archives of Neurology, 41, 825-9.

Lou, Hans C. et al. (1989). Striatal Dysfunction in Attention Deficit and Hyperkinetic Disorder. Archives of Neurology, 46, 847-52.

Mannuzza, Salvatore et al. (1998). Adult psychiatric status of hyperactive boys grown up. The American Journal of Psychiatry, 155(4), 493-8.

Michale, Mark S. (2000). The Psychiatric Body. In Roy Cooter and John Pickstone (Ed.s), Medicine in the Twentieth Century. Singapore: Harwood academic publishers.

Morrison, James R. and Stewart, Mark A. (1971). A Family Study of the Hyperactive Child Syndrome. Biological Psychiatry, 3, 189-95.

Oepen, Godehard, Harrington, Anne and Funfgeld, Matthias. (1990). Normality and Mental Illness – Dimension Versus Categories: Theoretical Considerations and Experimental Findings. In Manfred Spitzer and Brendan A. Maher (Ed.s), Philosophy and Psychopathology (200-10). New York: Springer-Verlag.

Olson, Sheryl. (2002). Developmental perspectives. In S. Sanberg (Ed.), Hyperactivity and Attention Disorders of Childhood (242-89). Cambridge: Cambridge University Press.

Pary, Raymond et al. (2002) Attention Deficit Disorder in Adults. Annals of Clinical Psychiatry, 14(2), 105-11.

Pasmanick, Benjamin, Knobloch, Hilda and Lilienfeld, Abraham M. Socioeconomic Status and Some Precursors of Neuropsychiatric Disorder. (1956). American Journal of Orthopsychiatry, 26, 594-601.

Rafalovich, Adam. (2001). Psychodynamic and Neurological Perspectives on ADHD: Exploring Strategies for Defining a Phenomenon. Journal for the Theory of Social Behaviour, 31(4), 397-418.

Rosenberg, Charles E. (1989). Disease in History: Frames and Framers. The Milbank Quarterly, 67, suppl. 1, 1-15.

Sanberg, Seija and Barton, Joanne. (2002). Historical Development. In S. Sandberg (Ed.), Hyperactivity and Attention Disorders of Childhood (pp. 1-29). Cambridge: Cambridge University Press.

Schachar, Russell J. (1986). Hyperkinetic syndrome: Historical development of the concept. In E. Taylor (Ed.), The Overactive Child (pp. 19-40). Philadelphia: Lippincott.

Singh, Ilina. (2002). Bad Boys, Good Mothers, and the “Miracle” of Ritalin. Science in Context, 15(4), 577-603.

Spencer, T. J. (2002). Attention-deficit/hyperactivity disorder. Arch Neurol, 59(2), 314-6.

Still, George F. (1902). Some abnormal psychical conditions in children. Lancet, 1, 1008-1012, 1077-1082, 1163-1168.

Strauss, Alfred A. and Werner, Heinz. (1942). Disorders of conceptual thinking in the brain-injured child. Journal of Nervous and Mental Disease, 96, 153-72.

Strauss, Alfred A. and Werner, Heinz. (1943). Comparative psychopathology of the brain injured child and the traumatic brain injured adult. American Journal of Psychiatry, 99, 835-8.

Tannock, Rosemary. (1998). Attention Deficit Hyperactivity Disorder: Advances in Cognitive, Neurobiological, and Genetic Research. Journal of Child Psychology and Psychiatry. 39(1), 65-99.

Tredgold, A. F. (1952). A Text-Book of Mental Deficinecy (Amentia), eighth edition. London: Bailliere, Tindall and Cox.

Werry, J. S. (1974). Minimal Brain Dysfunction (Neurological Impairment) In Children. New Zealand Medical Journal, 80(521), 94-100.

©2004, Colman Nefsky, B.Arts and Sc.