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REVIEW ARTICLE

Antecedents of ADHD: a historical account of diagnostic concepts

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Abstract The concept of ADHD has evolved gradually and still carries some traces of its origins. The idea of uncontrolled behaviour as a medical problem arose in eighteenth and nineteenth century accounts. It raised cultural issues about how far control was expected of children. This article traces the development of ideas with particular references to Hoffman's "Struwwelpeter", Frederick Still's "Disorders of Moral Control", minimal brain damage, and the hyperkinetic syndrome.

Keywords ADHD · History · Concept · Minimal brain damage · Struwwelpeter

Introduction

Attention Deficit Disorder (ADHD) continues to be a divisive and controversial topic. Different views about the nature of the disorders can divide psychiatrist from psychiatrist, psychiatrists from social workers, and mental health from education. Current disputes have been seen in negative reactions to the draft for DSM-V; adverse commentaries on the NICE guidelines (Indeed, NICE found it necessary to hold a conference on whether ADHD is a valid disorder); and a BBC apology that a documentary in its flagship programme, ("Panorama"), had misled the public in an attack on medication for ADHD treatment.

The debate on the conceptual nature of ADHD has oscillated for years between two extreme poles. "Brainless" and "mindless" opinions are both being held and guiding practice.

At one extreme, ADHD is seen as a biological condition of the brain, resulting from genetics and the physical environment, based in cognitive changes and requiring physical (diet or drug) treatments and directive behaviour modification. There is evidence for this from neuroimaging, molecular genetics, and experimental psychology, and clinical trials.

At another extreme, ADHD is seen as a psychological variant rather than a disorder, with any problems deriving from societal intolerance, based in emotional changes and requiring supportive and educational measures. There is evidence for this from the great increases in prevalence over time, the very great differences between countries, the presence of emotional upsets and the inconstancy of changes in performance.

This selective account aims to describe the prehistory of ADHD, in order to clarify the roots of the concept. Views about childhood disorders have developed with changing beliefs about childhood itself, and the latter need to be understood as the background to current concepts of mental disorder. Tracing lines of thought is inevitably a rather subjective process, but I have tried to consider how writers influenced each other from their citations and how the concepts developed from the words that the writers used.

Developing ideas of childhood

A prolonged childhood is a rather modern idea. In medieval and early modern times (up to about 1800 AD in England), most children older than about 7 were labourers (and of course still are in many parts of the world) (Hopkins 1994). Concerns about them were often indignation at

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unruly behaviour without any notions that mental disorder might be to blame.

Rousseau provided the voice for the enlightened understanding of childhood. For him, children are good by nature, curious and self-directing, rational from the age of 12 and need protection from a corrupting society[1] (Rousseau 1762). Through the nineteenth century, attitudes fluctuated from idealising childhood to exploiting it, but there was a growth through the century of an interest in the mind of the child (Shuttleworth 2005). Wordsworth famously saw the child as "father of the man" and capable of greater insights than the adult. By the end of the century, Sigmund Freud was taking a very different view of childhood virtue, and equally famously destroying some of the idealisation of childhood, but he took even further the idea of the child's mind as active and creating the mental life, and troubles, of the adult (Freud 1917).

This increasing appreciation of children as active and capable agents, and not simply as immature adults, came at the same time as the economic status of children was changing. Industrialisation created a massive move of peoples into cities and away from agricultural labour and created a need for literate and educated employees. Social justice and economic advantage conspired to change the roles of young people. By 1900, most children in England were students (Hopkins 1994).

Early descriptions of disorder: uncontrollability

In this social context, views of childhood disorder altered markedly. Early professional views (e.g. of Esquirol and his followers) regarded mental disorders as failures of reason, which could not apply to children as they were thought to lack reason anyway (Parry-Jones 1989; Walk 1964). Where disadvantage did not account for disturbed behaviour, psychiatrists thought that there must be an extreme constitutional cause (Ireland 1898). Contemporary accounts of cases can make uncomfortable reading, partly because of the extent to which the problems are seen as inherent in the child's personality and uncorrectable (Brandon 1986; Von Gontard 1988). They do nevertheless reflect enduring concerns—about the desperation of parents and educators and indeed the suffering children themselves. The key problems picked up in case histories are those of lack of control and regulation; they strike a chord with later ideas of hyperactivity.

Crichton (1798) in Scotland described a child born uncontrollable and 'raving mad', who had been affected continuously from 4 days old, and required four adults to hold him down. (He also showed uncontrollable fits of laughter, which to modern readers suggests a gelastic epilepsy) (Crichton 1798). Haslam (1809) provided a case history of a ten-year-old boy, who was indulged, mischievous, and uncontrollable. There was evidence of limited attention, and resistance to punishment, which made him the despair of skilled teachers. He had been cruel and destructive and threatened suicide. Haslam described him as "the creature of volition and the terror of the family"—a formulation clearly implicating a lack of impulse control. Treatment failed. Poignantly, Haslam adds that "When I showed him a mischievous maniac who was…strictly… confined, he [the boy] said 'this would be the right place for me'". For the doctor, this is taken as evidence of incorrigibility; to a modern eye, it reads more as evidence of desperation in the boy.

By 1812, that excellent clinical observer Benjamin Rush in the USA was adding the idea of a constitutional deficit of control: "One of them was addicted to every kind of mischief. Her wickedness had no intervals while awake, except when she was kept busy in some study or difficult employment. There is probably an original defective organisation in those parts of the body which are occupied by the moral faculties of the mind," (Rush 1812). As the nineteenth century unfolded, there was both a greater willingness to protect children from brutality (Hopkins 1994) and an increased inclination to attribute disorderly behaviour to insanity, so that admissions of young people to psychiatric institutions, such as the Bethlem Royal Hospital, rose (Wilkins 1993).

Struwwelpeter

It is conventional for historians of ADHD to begin with Heinrich Hoffman's great book "Struwwelpeter"—which translates as slovenly Peter, or Peter the slob (Hoffmann 1845). Indeed, a few unwary historians have obviously not actually read it because they refer to it as a case history by a psychiatrist. In fact, and even though Hoffman did indeed become a psychiatrist later, it is much more interesting than a case history. In 1845, when the first version of the book appeared, Hoffman was a physician and anatomy demonstrator; in 1852, he took over an asylum and did well in spite of no previous experience. He was regarded as a gregarious and popular man and also wrote as "Heinrich Kinderlieb" (Henry, lover of children). He sought to write for children in more child-centred ways. As "Peter Struwwel, Demagog" he published, a "Handbűchlein Fűr Wűhler" (little handbook for disturbers). This was not to glorify mischievous anarchists, but to satirise left-wing agitators. Hoffman's own politics were probably on the liberal side of conservative. He also wrote another picture

[1] His consignment of his own children to institutional care was widely cited against him, but did not discredit his views.

Fig. 1 An illustration from "Der Struwwelpeter" by Heinrich Hoffman (1809–1894). The picture is in "Die Geschichte vom Zappel-Philipp" and shows Fidgety Phil who cannot keep still; indeed, he creates a mess and upsets his parents

book ("King Nutcracker and poor Reinhold") which featured a boy so lazy that he learned nothing and became homeless.

This biography needs stressing, because there is an academic school that gives a psychoanalytically informed account of the book as a sadistic attack on childhood (Smith Chalou 2007). This seems to me to be a perverse judgement. His own account was that he had found existing children's books as "altogether too enlightened and rational, falsely naive, unchildlike, untruthful, artificial". He was, therefore, writing not for an idealised and wholly innocent child but for real children with the capacity to survive and enjoy scary stories. The book, directed originally to his own son and "good children", includes all sorts of catastrophes befalling children as a result of their own actions. One of them is "fidgety Phil"—who moves around so much that he brings down the dinner table and annoys his parents very much (Fig. 1).

Plainly, this is not a description of illness. In modern terminology, Phil might well be restless but unimpaired[2]—and the need to distinguish between the symptoms and their impact remains a living question for clinical science. Hoffman is emphatically not trying to pathologise. Rather, he presents an interesting and influential picture of children as active agents, making their own lives, and as complex beings, in contrast to the age's contradictory and polarised views of children as angels or as savages. He is allowing children to have their own taste for the macabre. And one must add how wonderful the pictures are by contrast with typical graphic representations of families in the first half of the nineteenth century.

[2] Or, indeed, showing multiple tics—is there a suggestion of jaw protrusion?—or a Weinberg syndrome with a sudden sleep attack? One could multiply explanations; it is simply not a case history.

The early twentieth century: Still

The next section of the usual historical chapter goes on to describe George Frederick Still (the founder of paediatrics in England) as having characterised ADHD and started modern understanding. His descriptions of problem behaviour certainly overlap with ADHD, but do not give primacy to impulsiveness, overactivity, or inattention. Rather, he aims to describe "defects of moral control"—in effect, extending the concepts of Rush—and the importance of his papers is that he is attributing behaviour problems to constitutional medical conditions, not to the "abstruse speculations" of psychologists.

Still was England's first professor of childhood medicine. He was a bachelor, extremely reserved with adults, and famous for not laughing. He loved children, particularly little girls with long hair, but generally could not stand their mothers. His patients included the present Queen of England, and he wrote poetry in English and Latin. The poetry can seem sentimental nowadays, but clearly stems from deep feeling (Still 1941):

> "For my garden is the garden of children
> Cometh naught there but golden hours,
> For the children are its joy and its sunshine,
> And they are its heaven sent flowers".

In his Goulstonian lectures in 1901, he described 43 children who presented with poor "moral control", in that they were often aggressive, defiant, resistant to discipline, and excessively emotional or passionate (Still 1902). Furthermore, they had problems with sustained attention and could not learn from the consequences of their actions. He regarded them as showing little inhibitory volition, and the emphasis was on constitutional deficit. With hindsight, this is taking medicine closer to both the symptomatology of hyperactivity and a biological formulation of the origins of disruptive behaviour.

I cannot find, however, that his work did in fact have any influence at the time. He is not cited by anyone until the 1970s and 1980s—when hyperactivity research is taking off and writers [such as Ross and Ross (1976) and Schachar (1986)] look back and find early writings to help legitimise their late twentieth century approach. His work does, however, reflect a view of the time. Strongly genetic ideas,[3] and the recognition of early brain damage, were leading to concepts of Minimal Brain Damage ("MBD")—considered below. There were stronger intellectual influences than Still's on the psychology of the early twentieth

[3] Deriving ultimately from Galton and Social Darwinism; and also giving rise to the eugenics movement. Tredgold's writings were influential in Britain and the USA; they did not seem to catch on in France.

century (Nissen 1991). Developmentalists such as Gesell (1925) and Piaget (1926) were describing the stages of childhood in ways that allowed individual differences to be studied systematically. Syndromes of disorder were being described (Kanner 1943) and the emotional roots of disturbance emphasised (Bowlby 1953; Winnicott 1964). These took the mainstream of child psychiatry away from ideas related to ADHD and towards the movement of 'mental hygiene' (Burke and Miller 1929).

Minimal brain damage (MBD)

The idea of MBD flourished in the early twentieth century and had considerable strengths. It emphasised cognitive processes and physical causes, at a time when thinking was very polarised and there were influential professional groups prepared to regard all behavioural problems as reflecting emotional processes and psychological causes. Sociological critiques have insinuated that the rise of MBD was motivated by neurologists' desire for power and status, but sociologists too have their vested interests.

Tredgold (1908, 1914) usually called the conditions "amentia", rather than "dementia", because they represented a developmentally static condition. He divided them into hereditary ("primary") conditions and those following an early environmental insult ("secondary"). In the 1940s, Strauss and his colleagues were propounding a similar set of neurodevelopmental conditions (as we would call them today) and a similar distinction into exogenous and endogenous (Strauss and Lehtinen 1947). They were, however, quite at odds over where to fit in motility disturbances such as hyperactivity:

Alfred D Tredgold

Primary: *hereditary, 'feeble-minded': clumsy, inattentive, distractible, defiant, hyper- or hypo-active*
Secondary: *non-hereditary, following birth injury etc., severe deficits*

Strauss & Lehtinen

Endogenous: *hereditary, borderline retarded*
Exogenous: *birth injury, cerebral palsy, epilepsy, hyperactive, confused, disorganised*

For other reasons as well, the idea of minimal brain damage fell into desuetude. There had been empirical objections to it all along as a good account of hyperactive and inattentive symptomatology. Childers (1935) had reported a sizeable series and concluded that hyperactive children were not more 'organic' than other disturbed children, but that the hyperactive were more likely to come from broken homes. More broadly, neurologists and psychiatrists came to find it unhelpful to lump all the neurodevelopmental disorders (specific learning difficulties, motor abnormalities, ADHD) together (Bax and MacKeith 1963) and to object that there was usually no other evidence of "brain damage" and that many of the conditions were devastating and not "minimal" at all (Bax and MacKeith 1963). The first thorough epidemiological surveys (Rutter et al. 1970) concluded that there was no characteristic syndrome of brain damage and therefore that it was usually invalid to conclude the presence of brain damage from nothing but the psychological presentation.

A pandemic of encephalitis appeared, like that of influenza, around 1917. It was first described in Vienna, in soldiers who had survived the war. It affected all ages and produced a bewildering variety of neurological syndromes—classically, lethargy and catatonic symptoms, but sometimes excited overactivity, involuntary movements, sleep disorders, mood changes, and manic behaviour. The pandemic faded after about 10 years, but left important sequelae. One part of the heritage was perplexity about the causes (which remain unknown). A second part of the legacy was the later emergence of neuropsychiatric syndromes—famously, parkinsonism, but children also developed psychiatric conditions that included severe conduct problems, emotional instability, compulsive misbehaviour, and "mental defects" (Anderson 1922). Accordingly, physicians would have been vigilant for the possibility of a brain cause of disruptive behaviour problems. This may have contributed to a third legacy—a readiness to attribute the symptoms, which one would now call ADHD, to acquired neurological disorder.

From MBD to attention deficit

The heir to MBD had the same acronym but a different emphasis: Minimal Brain Dysfunction. It was taken to comprise: Normal IQ, problems in perception and/or conceptualisation, language, memory, and impulse control (Clements 1966). This was prone to some of the same counter arguments as the former MBD, and since all psychological disturbances must have some basis in brain function, it was also rather tendentious in assuming physical aetiology.

The next step was therefore to move to a definition that would be based on psychological changes rather than unknowable neurological ones. North American research by this time was developing the tools for experimental investigation. A powerful step came from Dykman et al. (1971) in formulating MBD as a disorder of attention—still supposed to comprise 'organically based' deficits, but emphasising poor performance, increased reaction time, and decreased physiological reactivity and relating it to Luria's "cerebrasthenia"—which could be either hyperactive or hypoactive. In the same vein, Virginia Douglas

provided concepts of attention deficit (Douglas and Peters 1979), and Paul Wender (1971) regarded attention deficits as the key to understanding MBD. These North American authors were citing each other, and clear influences can be traced, but they were not citing the more neurological writings of the past.

The stage was set for the development of the key idea of Attention Deficit Disorder. It came with DSM-III (the third edition of the Diagnostic and Statistical Manual of the American Psychiatric Association) (American Psychiatric Association 1980). This was of great importance in the development of child psychiatry, because it replaced the aetiological formulations of the past (which were unreliable because of the difficulty of assigning cases to causes) with a simple description of observable behaviours. At this point, we have reached the end of the prehistory of ADHD and therefore of this article.

The story cannot, however, be allowed to stop there because the new ADDH category continued to carry many of the overtones of the MBD that it replaced. It is to this history that we can attribute many of the successes and limitations of subsequent practice and research. It helped to energise the application of neuroscience techniques—which have been notably successful in establishing genetic influences, identifying molecular genetic associations and finding structural, functional, and chemical brain abnormalities giving highly plausible accounts of the routes into disorder (Taylor and Sonuga-Barke 2008). But the nature of the concept also led to limitations, among which I would include the assumption of "deficit", the focus on "attention" at the expense of other cognitive changes, the lack of operational definitions of the behavioural criteria, the "universal" approaches to psychological treatment (e.g. training cognitive attention in all subjects, regardless of whether they actually had a difficulty in that domain), the confounding of behavioural attention with cognitive function, and the scarcity of work on the determinants of course, cultural effects on prevalence, or metacognitive and self-perception changes. MBD, and ADDH after it, had corrected "brainlessness"—in Leon Eisenberg's words—but perhaps helped to create "mindlessness" and a marked reliance on physical treatments (Eisenberg 1969).

It is necessary therefore to consider two other strands involved in the development of the concepts of disorder. First, the extent to which drug companies and pharmacological treatment conditioned the concept of disorder needs considering, in case a bias can be recognised. Second, a very different sort of concept developed in Europe and led to the idea of "hyperkinetic disorder"—was this a mere synonym or a different condition? The existence of hundredfold differences between different parts of the world, in the application of diagnoses involving hyperactivity, impulsiveness and inattention, suggests at the least that there is still something less than total consensus about the concept of the disorder(s).

The rise and rise of medication

The successive diagnoses of the two kinds of MBD, ADDH, ADHD, and AD/HD have been intimately linked with the use of medication. It was not, however, always so. Bradley (1937) discovered the effects of Benzedrine (racemic amphetamine) by accident. The story has often been told, at least at second hand, of how he was trying to use the newly synthesised compound to treat the headaches that followed lumbar puncture and noticed a beneficial effect on the mental state of the treated children. He was then able to report systematically about the effects on children, and it is clear that his observations were about learning and emotional state. This characterised the earliest reports of effects; it was not until the 1960s that hyperkinesis and overactivity emerged as targets for amphetamine medication.

The outcomes described in successive studies of medication were:

1937	Bradley	school performance better; subdued; relaxed
1940	Cutler et al.	reading better; little intellectual gain
1942	Bender and Cottington	relaxed; sociable; less aggressive
1962	Knobel	hyperkinesis improved
1963	Eisenberg et al.	learning, disobedience, overactivity improved

The suggestion is therefore that investigators' perceptions of the nature of drug effects gradually converged on ADDH, and it may well be that the growing knowledge about drug effects contributed to an intimate relationship between those effects and the conceptualisation of hyperactivity. The pharmaceutical companies, however, should not be held responsible: they were not the funders of the early medication trials, and it was not until about 1970 that advertisements to doctors started to promote Ritalin (methylphenidate) for "MBD".

The subsequent spectacular rise in prescription for children has divided the professions and the nations. Very few people argue that stimulants are ineffective or should never be used, but there remains a strong polarisation of professional opinion and practice about how widely they should be prescribed. This polarisation results in cultural attitudes in which ADHD is seen as that which stimulant drugs treat. The rejection of the concept of ADHD in

countries such as France was therefore very closely bound up with the rejection of medication solutions. In strict logic, the diagnosis and the treatment could have been divorced. The cross-national situation emphasises how a historical understanding is necessary to understand why we are where we are today.

European attitudes and 'hyperkinetic syndrome'

Kramer and Pollnow in 1932 described a series of cases that appear very organic in type (Neumärker 2005). Their position in the literature is the opposite of that of Still: they were very influential at the time and frequently cited by medical writers in the 1930s, but ignored by modern writers until rediscovered by neuropsychiatrists. They described children affected by severe and chaotic overactivity (sometimes associated with seizures), who often showed learning problems and/or anxiety and in whom there was often a definable onset around 3 years. Some recovered completely; 7/15 were seriously impaired afterwards.

Their terminology, and emphasis on underlying brain problems, seems likely to have energised a school that influenced the WHO's International Classification of Disease. In the United Kingdom, for example, their work was taken up by Rey at Guy's Hospital in London—a centre that influenced child neurologists such as Ingram (1956) (describing hyperkinesis in cerebral palsy) and Ounsted (1955) (describing hyperkinesis in epilepsy). "Hyperkinetic" was preferred linguistically as well as in its associations and took over from MBD in much European discourse in a way that ADDH was doing in North America. ICD-8 included "hyperkinetic disorder", and successive revisions (ICD-9 and ICD-10) have maintained it as the name of the category into which hyperactive, impulsive, and inattentive children should be fitted. They have maintained it, too, as a more restricted and severe version than ADHD has become.

Modern consequences

This selective historical account has described several different traditions leading to modern ideas of ADHD and hyperkinetic disorder. The concepts were conditioned by the struggles to identify and understand minimal brain damage, by the search for a neurobiological understanding of common disorders that would supplement or replace a psychodynamic account, and by the need to understand and use the effects of stimulant medication.

The modern heir is a neurobiological school of psychiatry with a strong commitment to medical treatment. It has some impressive successes in research and impressive support from clinical trials for the value of medicines. There are known changes in the structure and function of brain systems and in neurotransmitter changes in diagnosed people. Genetic influences are known to be strong. Nevertheless, naïve reductionism is unlikely to be enough for understanding. What is inherited appears to be a set of traits rather than an illness and a set of dispositions to react to the environmental associations of ADHD (Taylor and Sonuga-Barke 2008). Environmental contributions have been rather under-researched and under-emphasised until recently, and I would argue that social and cultural influences have yet to be given their full importance. There are probably strong cultural influences, not on the neurobiological variation but on the extent to which it leads to impairment or to disability and especially to diagnosis.

The differences between cultures in recognition and acceptance are not just trivial nuisances in ascertaining a "true" prevalence rate. They emphasise that the extent of impairment is a function not only of the children but of the context in which they are growing up. I suggest that public and professional attitudes can be helped by learning from history the interplay between scientific understanding and social change.

Conflict of interest None.

References

American Psychiatric Association (1980) Diagnostic and statistical manual of mental disorders, 3rd edn. American Psychiatric Association, Washington

Anderson GH (1922) The Sequelae of epidemic encephalitis in childhood, with notes on the prognosis as regards complete recovery. Q J Med 16:173–192

Bax M, MacKeith R (1963) Minimal cerebral dysfunction. Little club clinics in developmental medicine no. 10. Spastics Society with Heinemann, London

Bender L, Cottington F (1942) The use of amphetamine sulphate (Benzedrine) in child psychiatry. Am J Psychiatry 99:116–121

Bowlby J (1953) Child care and the growth of love. Penguin Books, Harmondsworth

Bradley C (1937) The behaviour of children receiving Benzedrine. Am J Psychiatry 94:577–585

Brandon S (1986) The early history of psychiatric care of children. In: Turner T, Cule J (eds) Child care through the centuries. STS Publishing, Cardiff, pp 61–78

Burke NHM, Miller E (1929) Child mental hygiene: its history, methods and problems. I. The history of the movements towards child mental hygiene. Br J Med Psychol 9:218–242

Childers AT (1935) Hyper-activity in children having behavior disorders. Am J Orthopsychiatry 5:227–243

Clements SD (1966) Minimal brain dysfunction in children; terminology and identification, phase one of a three-phase project. NINDB Monograph No. 3. US Department of Health, Education

and Welfare, Public Health Service publication no. 1415, Washington
Crichton A (1798) An inquiry into the nature and origin of mental derangement. T. Cadell & W. Davies, London
Cutler M, Little JW, Strauss AA (1940) The effect of Benzedrine on mentally deficient children. Am J Ment Defic 45:59–68
Douglas VI, Peters KG (1979) Toward a clearer definition of the attentional deficit of hyperactive children. In: Hale GA, Lewis M (eds) Attention and cognitive development. Plenum Press, New York, pp 173–247
Dykman RA, Ackerman PT, Clements SD, Peters JE (1971) Specific learning disabilities: an attentional deficit syndrome. In: Myklebust HR (ed) Progress in learning—disabilities, vol 2. Grune & Stratton, New York
Eisenberg L (1969) Child psychiatry: the past quarter century. Am J Orthopsychiatry 39:389–401
Eisenberg L, Lachman R, Molling P, Lockner A, Mizelle J, Conners K (1963) A psychopharmacologic study in a training school for delinquent boys. Am J Orthopsychiatry 33:431–447
Freud S (1917) Introductory lectures on psychoanalysis. Penguin Books Ltd, London
Gesell A (1925) Mental growth in the preschool child. Macmillan, New York
Haslam J (1809) Observations on madness and melancholy including practical remarks on these diseases together with cases. J. Callow, London
Hoffmann H (1845) Lustige Geschichten und drollige Bilder mit 15 schön kolorierten Tafeln für Kinder von 3–6 Jahren. Privately published. Renamed Struwwelpeter for the third German edition (1958)
Hopkins E (1994) Childhood transformed: working-class children in nineteenth-century England. Manchester University Press, Manchester
Ingram TTS (1956) A characteristic form of overactive behaviour in brain damaged children. J Ment Sci 102:550–558
Ireland WW (1898) The mental affections of children, idiocy, imbecility and insanity. J. & A. Churchill, London
Kanner L (1943) Autistic disturbances of affective contact. Nervous Child 2:217–250
Knobel M (1962) Psychopharmacology for the hyperkinetic child. Arch Gen Psychiatry 6:198–202
Neumärker K-J (2005) The Kramer-Pollnow syndrome: a contribution on the life and work of Franz Kramer and Hans Pollnow. Hist Psychiatry 16:435–451
Nissen G (1991) The history of child and adolescent psychiatry. In: Seva A (ed) The European handbook of psychiatry and mental health, vol 2. Editorial Anthropos, Barcelona, pp 1459–1467
Ounsted C (1955) The hyperkinetic syndrome in epileptic children. Lancet 2:303–311
Parry-Jones WLl (1989) The history of child and adolescent psychiatry: its present day relevance. J Child Psychol Psychiatry 30:3–11
Piaget J (1926) The language and thought of the child (trans: Gabain M). Routledge & Kegan Paul, London
Ross DM, Ross SA (1976) Hyperactivity: research, theory and action. Wiley, New York
Rousseau JJ (1762) Emile: or, on education. La Haye, Jean Néaulme (trans: Bloom A) (1979). Basic Books, New York
Rush B (1812) Medical inquiries and observations upon the diseases of the mind. Macmillan-Hafner Press, New York, 1962
Rutter M, Graham P, Yule W (1970) A neuropsychiatric study in childhood. Clinics in developmental medicine, nos. 35/36. S.I.M.P with Heinemann, London
Schachar RJ (1986) Hyperkinetic syndrome: historical development of the concept. In: Taylor E (ed) The overactive child. Spastics International Medical Publications, Oxford, pp 19–40
Shuttleworth S (2005) The mind of the child. Oxford University Press, Oxford
Smith Chalou B (2007) Struwwelpeter: humor or horror? 160 years later. Lexington Books, Lonham
Still GF (1902) The Goulstonian lectures on some abnormal psychical conditions in children. Lancet 1:1008–1012, 1077–1082, 1163–1168
Still GF (1941) Childhood and other poems. John Murray, London
Strauss AA, Lehtinen LE (1947) Psychopathology and education of the brain-injured child. Grune & Stratton, New York
Taylor E, Sonuga-Barke E (2008) Disorders of attention and activity. In: Rutter M, Bishop D, Pine D, Scott S, Stevenson J, Taylor E, Thapar A (eds) Rutter's child and adolescent psychiatry, 5th edn edn. Blackwell Publishing, Oxford
Tredgold AF (1908) Mental deficiency (Amentia). W. Wood, New York
Tredgold AF (1914) Mental deficiency (Amentia), 2nd edn edn. W. Wood, New York
Von Gontard A (1988) The development of child psychiatry in 19th century Britain. J Child Psychol Psychiatry 29:569–588
Walk A (1964) The pre-history of child psychiatry. Br J Psychiatry 110:754–767
Wender PH (1971) Minimal brain dysfunction in children. Wiley, New York
Wilkins R (1993) Delusions in children and teenagers admitted to Bethlem Royal Hospital in the 19th century. Br J Psychiatry 162:487–492
Winnicott DW (1964) The child, the family and the outside world. Penguin Books, Harmondsworth