香港兒童腦科及體智發展學會
HKCNDP

The Hong Kong Society of
Child Neurology & Developmental Paediatrics

ANNUAL SCIENTIFIC MEETING

10 - 13 November 2006

Hong Kong

Table of Contents

PROGRAMME-AT-A-GLANCE

Date	Time	Session	Topic	Speaker
10 Nov (FRI)	1800 - 2000	*Registration and Light Buffet Dinner*		
	2000 - 2200	Seminar I	Historical Perspectives and Evolution of the Concept of ADHD: The Spectrum of Attentional Disorders	Professor Drake Duane
11 Nov (SAT)	1230 - 1400	*Registration and Light Buffet Lunch*		
	1400 - 1500	Seminar II	From Anatomy & Physiology of CNS Attentional Systems to Aetiology (Primary and Secondary) of ADHD	Professor Drake Duane
	1500 - 1530	*Tea Break*		
	1530 - 1700	Local Presentation I	Multi-modal Interventions and Generalization of Classroom Behaviour of Children with ADHD in Hong Kong	Professor Vivian Heung
			Managing Disruptive Behaviour: From Research to Clinical Practice	Dr. Cheryl So
			Management of ADHD - The Role of Developmental Paediatricians in Hong Kong	Dr. Stephenie Liu
12 Nov (SUN)	0900 - 0930	*Registration*		
	0930 - 1030	Seminar III	Epidemiology, Clinical Manifestation and Co-morbidities of ADHD & Assessment of Executive and other Neuropsychological (Dys-) Functions	Professor Drake Duane
	1030 - 1130	Free Paper Session	Free Paper Presentations	
	1130 - 1200	*Tea Break*		
	1200 - 1300	Local Presentation II	Response Regulation: From Brain to Behaviour	Professor Tatia Lee
			The Concept of Situational Hyperactivity: A Re-examination	Dr. Ernest Luk
	1300 - 1430	*Light Buffet Lunch*		
	1430 - 1530	Seminar IV	A Psycho-educational Perspective on ADHD	Professor Che-kan Leong
	1530 - 1600	*Tea Break*		
	1600 - 1620	Local Presentation III	Towards a Model of Training Teachers of ADHD Students	Professor Ling-po Shiu
	1620 - 1700	Seminar V	Social and Emotion Implication of ADHD	Professor Drake Duane
13 Nov (MON)	1830 - 1900	*Registration*		
	1900 - 2000	Keynote Lecture	Treatment of ADHD: Medical, Behavioural and Educational & Prognosis	Professor Drake Duane
	2000 - 2200	*Chinese Banquet*		

Venues:

10 - 12 November 2006: Lecture Theatre, G/F., Block M, Queen Elizabeth Hospital, Jordon, Hong Kong

13 November 2006: Centenary Ballroom, G/F., The Marco Polo Hong Kong Hotel, Tsim Sha Tsui, Hong Kong

Course Director

Drake D. Duane, M.D., is a behavioral neurologist, with an additional interest in hyperkinetic movement disorders. A Regent Alumni Scholar of the University of Michigan in Psychology, Dr. Duane is a graduate with distinction and a member of the Alpha Omega Alpha medical academic honorary from Wayne State University College of Medicine in Detroit, Michigan. After a two year tour in the U.S. Army Medical Corps in Psychiatry, Dr. Duane entered a residency in Neurology at the Mayo Clinic in Rochester, Minnesota, and continued on the faculty of the Mayo Medical School, where he rose to the rank of Associate Professor of Neurology. At Mayo, he co-founded the Learning Disabilities Assessment Program and the Didactic Neurology Training Lecture series for residents, receiving the Teacher of the Year Award from the residents at the Mayo Graduate School of Medicine.

In 1987, he moved to Scottsdale, Arizona, to establish the Institute for Developmental Behavioral Neurology, and accepted an Adjunct Professorship in Speech and Hearing Sciences at Arizona State University. He is the Immediate Past President for the International Academy for Research in Learning Disabilities of which is a Founding Fellow, Past President of the Orton Dyslexia Society (now known as the International Dyslexia Association), Past Chairman of the National Joint Committee on Learning Disabilities, a Member of the President's Commission on Mental Health, and currently serves on the Board of Directors of National Center for Learning Disabilities in New York City, and The Board of Trustees of The Dyslexia Foundation. He is consultant to the National Institute of Child Health and Human Development. He is a Fellow of the American Academy of Neurology, the American Clinical Neurophysiological Society, and the American College of Physicians. He is a Member of the American Neuropsychiatric Association, Society for Behavioral and Cognitive Neurology, Child Neurology Society, American Association for the Advancement of Science, American Psychological Society, and American Sleep Disorders Association. He has edited four books; most recently "Reading and Attention Disorders: Neurobiological Correlates (York Press)". He has written 19 book chapters, the most recent on learning disabilities in Frank's "Pediatric Behavioral Neurology". He has published over 140 papers and abstracts as well as 13 published letters to the editor, including the *New England Journal of Medicine*, the *JAMA*, *Movement Disorders*, *Neurology*, and the *Journal of Child Neurology*. He is advisor to graduate students at Arizona State University and supervises psychiatry residents from the Maricopa Medical Center. His platform presentations include the American Academy of Neurology, American Psychiatric Association, the International Academy for Research in Learning Disabilities, the Explorers' Club in New York City, the Harvard Club in Boston (the first Cruickshank Lectureship), the Hong Kong Child Neurology and Developmental Pediatric Society, the Richter Memorial Lectureship at Indiana University School of Medicine, UCLA, University of Colorado, and the University of Uppsala, Sweden. He serves on the Education and Continuing Medical Education Committees at Scottsdale Healthcare Shea. He also serves on the Executive Council of the Physicians for the Phoenix Symphony, as well as the Board of Directors of the Phoenix Symphony.

Synopsis

Historical Perspectives and Evolution of the Concept of ADHD: The Spectrum of Attentional Disorders

Professor Drake Duane
Department of Neurology, Arizona State University, USA

There are at least two strands of history worthy of our attention in the issue of attentional disorders. The first is the historical progression of concepts in psychology of mechanisms and functional aspects of attention. The second is the historical evolution in medicine and clinical psychology of a specific disorder of attention of childhood onset and its differentiation from other conditions which may produce symptoms and signs of inattentiveness with and without clinical hyperactivity.

The psychological approach to attention issues begins in 1850 with von Helmholtz, who in 1850 felt that attention was concerned with *where* processing of information was to be directed. This is in direct contrast to a later concept of William James, who considered the function of attention was to answer the question *what* was to be processed. After von Helmholtz but before James, Lange in 1888 employed the concept of "attentional set", meaning an attention either to a specific cluster of incoming stimuli or to a subset of response alternatives. Attention therefore prepares the individual for a specific reaction. The Dutch physiologist, Donders, in 1868 found that the psychophysiologic property of information processing worthy of investigation was the *rate* of attentional processes employing *reaction time*. He proposed that measuring the reaction time to one task variable and then subtracting this from the time required to process the second task variable would infer the duration of the added task variable. Even today however the issue of reaction time is assessed by computerized measures of attention such as the Test of Variables of Attention (TOVA) and Continuous Performance Tasks (CPT).

James further defined attention as the "taking possession by the mind in clear and vivid form, of one out of what seems several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others and is a condition which is opposite to a confused, dazed, scatter-brained state, which the French call "distraction" and in German "Zerstreutheit". That is, one principal object comes then into the focus of consciousness, while others are temporarily suppressed". As we shall see, historical contributions to psychological concepts of attention include those of Titchener, 1924, Pavlov, 1927, Hebb, 1949, Skinner, 1953, Broadbent, 1953, Deutch and Deutch 1963, Schiffrin and Schneider 1977, Gibson and Rader 1979, Posner in 1988, Fuster 1989, Sergeant 1990, Mirsky 1991, whose neuropsychological model of attention will be addressed in our neuroanatomic discussions. The important contributions of Russell Barkley (1994) in which the notion of response inhibition to allow for prolongation of internal processing enhancing both perception and memory have had a significant impact on current concepts of Attention Deficit Disorder.

The group of disorders of attention commences back with the description in 1902 of Still, who described children with "defects in moral control". He recognized this conduct difficulty was more common in boys and that difficulties with hyperactivity, learning, conduct and attention were often coexistent. He conceptualized that the disorder he was encountering of childhood onset was likely of central nervous system origin, thus organic, but that there might be environmental factors that play a role.

Kahn and Cohen in 1934 described children who were the victims of the pandemic of encephalitis lethargica as organically driven. The postulation was that the damage was to the brain stem. These poor children were placed in special residential treatment centers and demonstrated behavioral problems improved by residential treatment but would relapse when returned to the general home or educational environment.

In 1937, Bradley observed the "paradoxical quieting" effect of Benzedrine, which is a racemic mixture of dextro and levoamphetamine. This perception led him to suggest the term "minimal brain dysfunction".

Based on work done by Strauss and Werner beginning in the 1930's, the speculation was the disorder was related to some form of damage in utero or perinatally, thus the term "MBD" reflected the sense of a minimal brain damage syndrome. Often retarded globally, these brain damaged children were described as hyperactive, distractible, impulsive, perseverative and as possessing cognitive deficits. In the late 1950's the work of Knobloch and Pasamanic suggested that perinatal stress was a causal factor in many cases of MBD.

The source of this disorder was debated as to how much was acquired and how much might be intrinsic. This notion was captured by Clements and Peters: "It is necessary to take into account the full spectrum of causality from the unique genetic combination that is in each individual to his gestation and birth experiences to his interaction with significant persons and finally to the stresses and emotional trauma of later life after his basic reaction patterns have been laid down".

It was Lauretta Bender (famous for her participation in the development of the Bender Visual Motor Gestalt Test) who suggested the term "developmental hyperactivity". In the late 1950's, Laufer and Denhoff suggested the term "hyperkinetic impulse disorder". The core symptoms of this disorder were restlessness and impulsivity. Etiology was not implied by this terminology. Dykman and Douglas suggested that faulty attention and disorders of inhibitory control were cardinal features of this hyperactive reaction of childhood, which is what was used in the DSM-II nomenclature. DSM refers in the United States to the Diagnostic and Statistical Manual of Mental Disorders. The terminology was changed by the DSM-III in 1980 to "attention deficit disorder, with or without hyperactivity". With its revision in 1987, DSM-III R employed the term "attention deficit hyperactivity disorder". In 1994, the fourth addition, DSM-IV, used the letters ADHD. This has most recently been updated by the DSM-IV TR diagnostic criteria for attention deficit disorder, there being three types, without hyperactivity or ADD, ADD with associated hyperactivity or impulsivity or the combination of these three components, combined type. In the United States these four AD(H)D diagnostic groups are digitally coded, two sharing the same five digit coding, the other two separate diagnostic codes.

Even of late there has been a debate as to the validity of the concept of attention deficit disorder. Hopefully, the data to be reviewed in the course of these seminars will clarify that although there is a broad spectrum of conditions affecting central nervous system function which may intrude on attention mechanisms, within that heterogenous spectrum there is a group of students who, even as preschoolers, but especially in the classroom environment, spreading eventually into the general social environment, encounter difficulty in directing their attention and as a consequence suffer with difficulties in learning, socialization and most importantly in controlling impulsive behavior.

To ensure as accurate a diagnosis as possible, both behavioral and cognitive measures are required. Rating scales are commonly used for the behavioral components and neuropsychological measures for the latter, these two superimposed upon a psychoeducational and intelligence assessment. The developmental history is essential as well, for among the appropriate criteria for diagnosis is that the symptoms should have been present at some level prior to the age of seven years. Furthermore, it is most helpful that the symptoms in question are global and not isolated to home or school alone. The behavioral criteria, through standardized rating scales, as well as the neuropsychological measures, will be among our topics of discussion in the course of these seminars.

From Anatomy & Physiology of CNS Attentional Systems to Aetiology (Primary and Secondary) of ADHD

Professor Drake Duane
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In contrast to anatomic studies in dyslexia, postmortem data is not yet available with regard to attentional disorders. However, in vivo studies by magnetic resonance imaging (MRI), functional MRI (fMRI), positron emission tomography (PET), have yielded trends in brain function and anatomic structures that separate individuals with attention deficit disorder from the general age matched population. A volumetric analysis of the cerebrum, caudate nucleus, putamen, globus pallidus, amygdala, hippocampus, temporal lobe, cerebellum and prefrontal cortex demonstrates in children with ADHD a 4.7% smaller cerebral volume, a significant loss of the normal right greater than left caudate asymmetry, a smaller right globus pallidus, a smaller right anterior frontal region, a smaller cerebellum, and reversal of the normal lateral ventricular asymmetry. Clinical studies of those with reversed caudate asymmetry, i.e., left greater than right, was associated with poorer performance on measures of inhibition. Similarly, in acquired disorders of attention such as head trauma, those who demonstrate attentional symptoms are more apt to have visible evidence by MRI of lesions in the right putamen.

Cerebellar processes of the cerebellum may be involved in executive function, including those attributed to the prefrontal cortex. The cerebellar vermis is smaller in its inferior posterior lobe in students with attention deficit disorder versus those without. Consequently, these suggest a cerebello-striatal-prefrontal circuit dysfunction which may underlie the loss of control of inhibition and executive dysfunction which occurs in AD(H)D.

Metabolic studies have documented basal state differences in regional blood flow in the prefrontal cortex and striatum (caudate, globus pallidus and putamen). Hypoperfusion of frontal lobes is common and when treated with stimulant drugs is reversed. Adults by PET studies have an overall 8% reduction in cerebral cortical glucose metabolism. These are most prominent in the premotor and superior prefrontal cortex. These adult based studies, when attempted in adolescence, showed that ADHD girls were more apt to demonstrate this anomaly than adolescent ADHD boys.

Studies of single photon emission computer tomography (SPECT) showed in ADHD a greater cortical asymmetry characterized by diminished left frontal and left parietal activity. An fMRI study showed elevations of T2 relaxation time in the putamen, suggesting reduced blood flow and as a consequence reduced neuronal activity. Hypoactivity in the putamen matched objective measures of hyperactivity and attention utilizing infrared motion analysis. Chronic treatment with methylphenidate alters these changes. A recent study suggests that the reduced volume of the right caudate nucleus is reversed with methylphenidate treatment, i.e., a normalization of volume associated with exposure to stimulant medication.

Functional imaging also reveals deficits in cortico-striatal activity during performance of cognitive tasks, particularly those which measure aspects of inhibitory control or working memory.

The above metabolic/neuroanatomic changes, in the absence of other disease states of the CNS, are apt to be related to genetic mechanisms, which are supported by a rate of 84% of adult ADHD sufferers having at least one affected offspring. Twin studies show an 80% concordance rate. Candidate genes have been suggested at DRD4, DAT1, D_2 receptor, DXS7 (X chromosome) with sites suggested at chromosome 3, 5, 11 and a possible link to autism on chromosome 16 and 17. Physiologic studies suggest, as is the case in dyslexia, higher rates of cortical over-excitability producing physiologic patterns reminiscent of those in epilepsy even without a clinical history of febrile seizure. In dyslexia, these are thought to be clinical physiologic manifestations of anatomic developmental lesions within the cortex. Whether such is the case in ADHD remains unresolved, but might in those with coexistent reading disorder and attention disorder.

Studies utilizing event related potentials, such as the N-100, P-300, suggest prolonged latencies are characteristic of up to 94% of affected students but do not distinguish between ADHD and other developmental disorders affecting educability.

Among the disorders in which attention mechanisms are impaired, disorders lateralized to the right hemisphere are potentially causal, e.g. in the nonverbal learning disabilities syndrome, as well as Tourette syndrome.

In addition to head trauma, other disorders which may be associated with attentional symptoms include the psychiatric states of anxiety, depression and obsessive-compulsive disorder, fetal alcohol syndrome, PKU, whole head irradiation, and maternal/infant malnutrition. Eastern European neglected children, adopted into US homes, demonstrate uniformly among the broad spectrum of deficits difficulties in attention. Anomalous sleep patterns, sleep deprivation or unexplained daytime hypersomnia is observed in a subset of attention disordered children and adolescents. Stimulants alert such daytime sleepy students. Whether these children/adolescents have primary or secondary attention deficit is unclear. Pupillometry measures pupil size and pupil size is inversely related to alertness, can discern sleepiness but not its cause.

These data suggest that whether the cause is intrinsic biological or extrinsic acquired that multiple mechanisms, both genetic and acquired may result in disorders of attention in which hyperactivity may or not be among its manifestations.

Epidemiology, Clinical Manifestation and Co-morbidities of ADHD & Assessment of Executive and other Neuropsychological (Dys-) Functions

Professor Drake Duane
Department of Neurology, Arizona State University, USA

It has been suggested that AD(H)D is a fabrication or a societal induced disorder of US American culture. This notion is not without merit. A society exhibiting progressively loosening family structure, two working parent households, high rates of divorce, a blitzkrieg of media stimulation, distracting sexual exploitation, news and computer information ever shorter in length with abbreviated and low level language, nutrition that is too often fattening, and an educational system only at the international median, could explain the higher than anticipated rate of occurrence of attention problems.

A refutation of the above hypothesis is: The disparate rate among males versus females, high frequency of occurrence in first degree relatives, morphometric brain asymmetries differential from control subjects, brain metabolic studies that distinguish those with AD(H)D from controls, physiological studies the results of which are similar to other organic developmental disorders, quantitative neurologic examination markers suggesting neuronal motor system anomaly, as well as the beneficial effects of stimulant and other medicinal therapies. These data suggest that biological factors are likely to play a major, perhaps primary role in the genesis of disorders of attention with early childhood onset and frequently associated with measurable increased motor activity (hyperkinesis).

Alternatively, it could be the case that biology may place one at risk and social factors may exacerbate that risk to the point of clinical dysfunction. As a result, the notion of "at risk" for AD(H)D has been proposed.

Worldwide, how prevalent is this American epidemic? In the UK, estimates of 3% agree with the conservative US frequency of 3%. That in the US the occurrence may be greater is suggested by the Mayo Clinic epidemiologic survey of Olmsted County, Minnesota, which found a frequency in school age children of 8%. Boys in that study outnumbered girls three to one. Olmsted County is a predominantly Caucasian community of Western and Northern European ancestry with middle class socioeconomic levels. Of further interest is the 1993 survey from mainland China, which observed an incidence of ICD-9 diagnosed ADHD of 3%.

Among the factors that could influence these estimates are the differential diagnostic criteria employed, as ICD-10 criteria are more stringent than ICD-9/DSM-IV TR. Increasingly, AD(H)D in developed countries is recognized as a major pediatric mental health problem.

The comorbidity of AD(H)D symptoms with other recognized central nervous system disorders further reinforces the notion that attentional disorders have a CNS origin. These include Tourette syndrome and the so-called nonverbal learning disabilities that overlap with Asperger syndrome, one of the pervasive childhood disorders.

High rates of anxiety, depression, obsessive worry and conduct disorder within the ADHD population cannot be ignored (see Seminar IV). These emotional concomitants offer however a confound to diagnosis, as disordered psychological states interfere with attention. As a result, assessment of attentional disorders should include measures of mood. For this purpose, our assessment includes the Achenbach Child Behavior Checklist (ACBCL), a parental survey that reveals a variety of personality traits including that common to ADHD children and adolescents. The Children's Depression Inventory (CDI) provides five dimensions of childhood depression, as completed by the student. For adolescents and adults, the Minnesota Multiphasic Personality Inventory (MMPI) provides an accurate reflection of one's emotional state on a multitude of dimensions. To quantify obsessive-compulsive behavior, the Yale-Brown Obsessive-Compulsive Rating Scale (YBOCRS) is a useful tool. The Spielberger Anxiety Rating Scale (Spiel-Anx) and Hamilton Depression Scale (Ham-D) are useful screening instruments for anxiety and depression.

The connection to psychiatric comorbidity extends beyond that observed in the patient himself. Our studies of family history, using DSM-IV criterion referenced techniques, suggests an unusually high rate, more common indeed than ADHD and other learning disabilities, of psychiatric disorders in first degree relatives that include depression, anxiety and obsessive-compulsive traits. This comorbidity may reflect neurochemical and/or neurophysiologic correlates binding these syndromes together.

Neuropsychological tests offer insight beyond that of the psychoeducational assessment. IQ measures such as the Wechsler Intelligence Scale for Children, Revised and III, more recently revised as the IV, provide measures of inattention (freedom from distractibility index) and social interpretive skills can be inferred from the performance on the picture arrangement subtest (not included in the WISC-IV kit).

Executive function, as defined by Martha Bridge Denckla, is a set of domain-generated control processes that involve inhibition and delay of responding for the goal of organization and integration of cognitive and output processes over time. Executive functions have distinctive future tense aspects and include such processes as anticipatory set, preparedness to act, freedom from interference of prepotent response tendencies and sequencing behavioral response. Russell Barkley distinguishes executive functions from other CNS functions as the primary means of self-regulation: the analysis, alteration and management of one's own behavior. Barkley further defines four aspects within executive function systems: separation of affect, prolongation, internalization and reconstitution. Anatomic localization of executive function is based on acquired brain disorders such as trauma and stroke, as well as metabolic studies of AD(H)D subjects, which suggest the prefrontal cortex and its connection to the striatum as a major locus of executive function.

A variety of neuropsychological tests within our battery inform one of the intactness of executive function. These include: The Wisconsin Card Sorting Test (WCST), Letter Cancellation Test (LCT), Digit Span (DS), the verbal recall of the Auditory Verbal Learning Test (AVLT), the Stroop Test, the Test of Variables of Attention (TOVA), and Conners' Continuous Performance Test (CPT). The WCST, TOVA and CPT are computerized investigations with national age and education-matched norms. The discussion within the seminar will include amplification of these instruments, their administration and interpretation of results.

Social and Emotion Implication of ADHD

Professor Drake Duane
Department of Neurology, Arizona State University, USA

It is hard to imagine that a disorder characterized by inordinate impulsivity, e.g., blurting out, pushing others aside in queue, and hyperactivity-motoric restlessness when others are still, would not unfavorably influence others perception of such behavior and consequently ignoring or rejecting the person committing such inappropriate behavior. Add to that a home in which one is raised in which at least one of the parents suffers with AD(H)D with consequent impairment of parenting skills and/or one or more members of that household with another diagnosable psychiatric disorder. The result is a person limited in their social emotional adjustment and themselves at risk for primary other than ADHD psychiatric disorder such as anxiety, depression and obsessive rigidity.

The social impact of the disorder is a limitation of empathy and capacity to initiate and maintain intimate relationships, perhaps except with another person with ADHD - so-called assortative mating.

The psychiatric comorbidity of depression in ADHD is matched by an equally high rate of depression in first degree relatives. As a result, it may not be only frustration over lack of success in school, work or home life that produces a state of despondency. Erratic employment with high rates of loss of employment is common in AD(H)D adults. Lack of school success is characterized by an average reduction in level of education of two years below even those with non-ADHD dyslexia.

Substance abuse, particularly alcohol, is more frequent in those with AD(H)D than in the general population. Abuse of cocaine and methamphetamine, both dopaminergic substances, may act as a form of therapy, self-administered.

Self-esteem is low in the majority of ADHD subjects, as measured by self report, rating scales and maternal impressions. The use of multimodal intervention for two or more years potentially improves self-concept, academic achievement and lowers the risk for substance abuse.

Social skills assessment using rating factors such as job interview, heterosocial interaction, and appropriate assertiveness have found significant differences in adult AD(H)D subjects versus controls.

As adolescent ADHD subjects mature, their cost for medical care markedly increases versus that of the general population, according to the Olmsted County study referenced in Seminar III. These costs were not primarily for medication or counseling but rather because of physical, usually accidental, injury, perhaps a measure of executive dysfunction, i.e. risk taking. The social emotional concomitants of ADHD suggest a need for early diagnosis and persistent treatment in order to maximize success and minimize the negative consequences on personality development and capacity for independent living and interpersonal relationships.

Treatment of ADHD: Medical, Behavioural and Educational & Prognosis

Professor Drake Duane
Department of Neurology, Arizona State University, USA

The management of disorders of attention is a hierarchical complex of options that begins with patient/family education. The student should feel part of the evaluation process and a team member in the decision regarding which treatment option to select. This empowerment of the student increases their compliance with treatment. Students should recognize that their condition is a natural human trait with positives as well as negatives but does not represent a "disease" or serious "malfunction" of the nervous system. Such fears are not uncommon in students who are underachieving.

In the United States, classroom "accommodation" can be implemented in a cost effective manner, which increases collaboration by schools. Accommodating measures include central classroom seating, increased times for test taking and reduction in homework, while enhancing, by phone and/or email contact, communication between the school and family as to assignments to be done as well as due dates for these assignments, providing assurance that they have been completed and that they will be returned to the school.

When emotional issues of anxiety, depression and obsessive worry arise, counseling as well as mood medications may further improve attention. Addressing psychiatric comorbidities, particularly if moderate or worse in severity, limits the extent to which psychostimulant medication can achieve optimal cognitive results. This is because psychiatric disorders themselves may impair cognitive function. However, in those situations where the emotional distress is primarily the result of AD(H)D induced school and/or social failure, remediating adverse effects of inattention on school performance may be sufficient in itself to rectify the inhibiting symptoms of reactive depression.

The above last observation may be why the Multimodal Treatment of ADHD study (MTA) which compared behavioral management alone, medication management alone, combined behavior and medication management, and community therapy alone, observed the best outcome in the combined behavioral/medication therapy group. Nonetheless, this investigation proved that for many students pharmacotherapy alone is sufficient to achieve excellent results in cognitive and school performance. Additionally, for those students whose family prefers that no medication be used, behavioral therapy alone is often sufficient to achieve modest success.

Pharmacotherapy, although relying primarily on the psychostimulants, methylphenidate and amphetamine, at times due to side effects or lack of efficacy may require alternative approaches. The dopaminergic antidepressant buprion (Wellbutrin®), particularly when depression is comorbid, can promote enhanced attention. The noradrenergic drug atomoxetine (Strattera®) holds popularity for those families preferring non-stimulant medication. However, this agent has side effects of nausea, headache, lethargy and potential mild liver function. In our experience, symptoms of hyperactivity are more responsive to atomoxetine therapy than inattention.

Pemoline (Cylert®) has been withdrawn in the U.S. because of the hazard of liver dysfunction. In my experience and the literature, the risk is low and for some students pemoline out performed the psychostimulants.

An interesting non-stimulant drug, proposed originally for adults with narcolepsy, namely modafinil (Provigil®), holds promise. A non-controlled, non-stimulant agent with few side effects, this drug may alert the subject, both in wakefulness and cognitive function. Unfortunately, a recent application by the manufacturer to the US Federal Drug Administration (FDA) was denied approval for ADHD. However, beyond puberty our data suggests it is an acceptable substitute for a stimulant. It may be helpful to younger students, but there is insufficient data to support its use without fear of potential adverse effects. Agents such as tricyclic antidepressants and the alpha 2 adrenergic agonists, clonidine and

guanfacine, may be useful, particularly for aggression. A limiting factor is the side effect of sedation. Atypical neuroleptics have been the supplemental agents of choice in our work, as they not only suppress stimulant-induced side effects of sleep disturbance, aggression, anxiety and tics, and at times seem to directly favorably affect attention. Their greatest limitation however is the symptoms of excess sedation and excess weight gain.

The informed use of the above pharmacotherapy, supplemented by school room accommodation and, where appropriate, treatment by counseling and supplemental medication of comorbid mood disorders, offers the best probability of enhancing student potential and limiting the adverse effects of AD(H)D on student outcomes.

Medication Options
In Developmental Disorders

Attention = Increase Dopamine

1. Psychostimulants
   - Methylphenidate: Ritalin - Concerta – Methylin Metadate – Daytrana
   - Amphetamine: Dexedrine - Adderall - Desoxyn

   → see chart

2. Psychotropics (antidepressants)
   - Wellbutrin - 100, 150, 200 mg SR; 150, 300 mg XL 1-2/d
   - Prozac, Zoloft → see SSRIs

   → 15-60 d

3. Provigil (modafinil) - 100, 200 mg ½ -2/d
4. Strattera* (atomoxetine) - 10, 18, 25, 40, 60 mg 1/d → 7-15 d

* May cause liver dysfunction

Psychostimulants
In Developmental Disorders

Name Trade (generic)	Dose/Form [1]	Effect Duration	Time for Effect	Side Effects [2]
Ritalin	5, 10, 20 mg tabs, 20 mg SR 10, 20, 30, 40 mg LA	3-4 hrs 6-8 hrs [3] 6-8 hrs	1 hour	- Aggression - Anxiety - Obsessive/ compulsive behavior - Tics - ↓ Appetite, weight - ↓ Sleep
Focalin (dextro)	2.5, 5, 10 mg tabs 5, 10, 20 mg XR	4-6 hrs 6-8 hrs	1 hour	
Concerta	18, 27, 36, 54 mg tabs	6-10 hrs	1 hour	
Methylin	5, 10, 20 mg; 10, 20 mg ER	3-4 or 6-8 hrs		
Metadate (methylphenidate)	10, 20, 30 mg CD	6-8 hrs		
Daytrana	10, 15, 20, 30 mg patch	9 hrs	1 hour	

1 - Any medication can be specially formulated into any size, liquid or spansule
2 - Except liver dysfunction, most can be "blocked"
3 - Often erratic blood levels during the day

*Possible liver dysfunction

Psychostimulants
In Developmental Disorders

Trade (generic) Name	Dose/Form [1]	Effect Duration	Time for Effect	Side Effects [2]
Adderall (mixed amphetamine salt)	5, 7.5, 10, 12.5, 15, 20, 30 mg tabs	4-6 hrs	1 hour	- Aggression - Anxiety - Obsessive/ compulsive behavior - Tics - ↓ Appetite, weight - ↓ Sleep
	5, 10, 15, 20, 25, 30, 40, 50 mg XR	6-10 hrs	"	
Dexedrine (d-amphetamine)	5 mg tablets 5, 10, 15 mg spansules	4-6 hrs 6-8 hrs	" "	
Desoxyn (meth-amphetamine)	5 mg tablets	6-8 hrs	"	

1 - Any medication can be specially formulated into any size, liquid or spansule
2 - Except liver dysfunction, most can be "blocked"

Non-stimulants
In Developmental Disorders of Attention

Trade Name (generic)	Dose/Form	Effect Duration	Time for Effect	Side Effects [2]
Tofranil (imipramine)	75, 100, 125, 150 mg caps 10, 25, 50 mg tabs	12 hour	2-4 wks	HA, ↑wt, drowsiness, dry mouth, ECG Δ
Norpramine (desipramine)	25, 50 mg caps 10, 25, 50, 75, 100, 150 mg tabs	24 hour	2-4 wks	" " "
Pamelor (nortriptyline)	10, 25, 50, 75 mg caps 2 mg/ml liquid	24 hour	2-4 wks	" " "
Elavil (amitriptyline)	10, 25, 50, 75, 100 150 mg tabs	12 hour	2-4 wks	" " "
Wellbutrin 100, 150 mg SR	75, 100 mg tabs 12-18 hour	8-12 hour	2-4 wks insomnia	Sz, agitation, HA, (bupropion)
Effexor (venlafaxine)	25, 37.5, 50, 75, 100 mg tabs 37.5, 75, 150 mg XR	12 hour 18 hour	2-4 wks	N, sedation, GI upset
Catapres (clonidine)	0.1, 0.2, 0.3 mg tabs 0.1, 0.2, 0.3 mg/d TTS patch	6-8 hour 24 hour/5-7 d	1-3 days 1-3 days	Sedation, orthostatic BP ↓ dry mouth, rebound ↑BP ↑ if quick withdrawal
Tenex (guanfacine)[1]	1 mg tab	6-8 hour	1-3 days	" " "

1 – Used as "add-on" Rx, may reduce aggression, tics – as may atypical neuroleptics Risperdal, Seroquel, Zyprexa & Geodon
2 - Except liver dysfunction, most can be "blocked"

Medication Options
In Developmental Disorders

***Anti-Anxiety** (-obsessional, -aggression, -tics)*

1. Atypical neuroleptics
   - Risperdal - .25, .5, 1, 2, 3, 4 mg tab - liquid; .5, 1, 2 mg M tab
   - Seroquel - 25, 100, 200, 300 mg
   - Zyprexa - 2.5, 5, 10, 15, 20 mg - 5, 10 mg Disintetab
   - Geodon - 20, 40, 60, 80 mg; liquid
   - Abilify - 5,10,15, 20, 30 mg - liquid 1 mg/ml

   → 1-3d ↑ appetite, drowsiness, –, nausea