CHAPTER 1
INTRODUCTION

In recent years attention-deficit hyperactivity disorder (ADHD) has received a great deal of clinical, scientific, and public attention. In fact, as Margaret Weiss and Gabrielle Weiss point out, between 1957 and 1960, thirty-one articles were published on hyperactivity in children, whereas since 1996 there have been about 400 articles a year on ADHD are published. This increase in the rate of publication is continuing (Hechtman, 2005).

One should be aware that it is not a new condition as in 1902, George Still described children who were restless, impulsive, and inattentive, with intense affect and conduct problems. Still it is believed that a combination of organic and environmental factors resulted in lack of inhibitory control and inattention, which are thought to be the primary deficits of the syndrome (Hechtman, 2005).

After the influenza pandemic and the epidemic of encephalitis lethargica in 1919 to 1920, children who survived frequently developed severe behavior problems similar to those described by Still. The condition was now thought to be caused by organic brain damage, and “minimal brain damage syndrome” was frequently used to diagnose such children, even though such damage could not be demonstrated. This diagnosis was quite stigmatizing without clear proof of brain abnormality (Hechtman, 2005).

Bradley in 1937 showed that Benzedrine (D and L-amphetamine) reduced restlessness and improved concentration and motivation in children with behavior problems in a residential treatment center. It was the first evidence of the effectiveness of stimulants for these symptoms. However, this finding was largely ignored for almost three decades, until Keith Conners and his colleagues in 1967 used dextroamphetamine (Dexedrine) in a double blind placebo trial for children with learning disabilities and school behavior problems. In the early 1960s, in an attempt to veer away from the “brain damage” concept, the condition was renamed “minimal brain dysfunction.” However, this too was stigmatizing, and the specific “brain dysfunction” could not be documented.
Hechtman (2005). In the late 1960s, there were attempts to create more scientifically valid and reliable classification, and both the ninth revision of the International Statistical, Classification of Diseases and Related Health Problems (ICD-9) and the second edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-II) adopted more descriptive terms for the condition — e.g., hyperkinetic syndrome of childhood or hyperactive child syndrome. These terms reflected the prevailing belief that hyperactivity was the condition’s key disabling symptom.

In the 1970s, further research suggested that the main difficulties of children with this condition involved problems with sustained attention and impulse control, with hyperactivity being secondary. Thus, the DSM-III renamed the condition attention-deficit disorder. The diagnosis of attention-deficit disorder in the DSM-III included three symptom categories and required a number of positive items in each category — e.g., inattention (three of five items), impulsivity (three of six items), and hyperactivity (two of five items). There were also three distinct subtypes: attention-deficit disorder with and without hyperactivity and the residual subtype that include adults or others who had disabling symptoms but who no longer met full criteria for the condition (Hechtman, 2005).

In the 1987 revised version of the DSM (DSM-III-R) the name of the disorder changed slightly to attention-deficit / hyperactivity disorder, and the criteria changed significantly. Patients receiving the diagnosis had to have at least eight of 14 possible symptoms of hyperactivity, impulsivity, and / or inattention in any combination. These behaviors need to be present before 7 years of age and occur considerably more frequently in patients than in most people of the same mental age, and they needed to persist for at least 6 months. There were no subgroups, and attention-deficit disorder without hyperactivity was no longer a diagnostic category. A committee of experts in the field constructed the DSM-III and DSM-III-R diagnostic criteria. The outcome was a criteria which reflected the consensus and compromises of these experts but were not based on objective controlled field trials. In preparation for the fourth edition of the DSM (DSM-IV), multisite field trials involving 600 clinic-referred children were initiated to
help define the validity of proposed ADHD symptoms through structured diagnostic instruments and unstructured clinical interviews.

Definition in the fourth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) is based upon extensive field testing that led to grouping symptoms in to two primary categories: inattentive and hyperactive impulsive symptoms. Six symptoms from a number of symptom categories is required for the diagnosis. Three different types of categories of Attention Deficit Hyperactive Disorder (ADHD) on the basis of number and type of symptoms from each category with subtypes referred to as either ADHD, inattentive type; ADHD, hyperactive-impulsive type; or ADHD, combined type. According to DSM-IV, symptoms of ADHD must be evident by age 7 years and have a minimum duration of 6 months. In the 10th revision of International Statistical Classification of Diseases and Related Health Problems (ICD-10), the category hyper kinetic conduct disorder overlaps considerably with ADHD (McCracken, 2000).

Attention deficit hyperactivity disorder is believed to be the most common neurobehavioral disorder of children characterized by developmentally inappropriate levels of inattention, impulsivity and motor activity that appears in at least two context (e.g., home and school), and is present for at least six months before the age of seven, (American Psychiatric Association, 1994).

Ellis (1985) suggested that ADHD children differ from each other on the degree of manifestation and not in the presence or absence of it. Douglas (1985) proposed that children with attention deficit experience a constitutional predisposition to experience problems with attention, effort, inhibitory control, and poorly modulated arousal and have a need to seek stimulation. Goldstein and Goldstein (1990) put forth the common sense definition of ADHD. They hypothesized that attention deficit may be a disorder of faulty selective attention. ADHD comprises of a group of children who experience difficulty deciding what is most important to pay attention to and investing in that particular stimulus. The current conceptualization of this definition contains five components: impulsivity, inattention, hyperactivity and over arousal, difficulty with stratification, and emotions and locus of control (Manchanda, 2010).
History of ADHD

Historically, there has been controversy regarding the labeling and clinical description of ADHD and the diagnostic criteria that have undergone several revisions over time. The history of ADHD has the nascent concepts that serve as the foundation for the current conceptualization of the disorder as largely involving poor inhibition and self regulation. One of the first references to a child with hyperactivity or ADHD was in the poetry of the German physician Heinrich Hoffman in 1865. But George Still (1902) and Alfred Tredgold (1908) were the first authors to focus serious clinical attention on the behavioral condition in children that most closely approximates what is today known as Attention Deficit Hyperactivity Disorder. Still (1902) argued that a defect in motor control could arise as a function of three distinct impairments: defect of cognitive relation to the environment; defect of moral consciousness; and defect in inhibitory volition.

Interest in ADHD in North America can be traced back to the outbreak of an encephalitis epidemic in 1917 to 1918 when clinicians were presented with a number of children who survived this brain infection yet were left with significant behavioral and cognitive sequelae. At that time, such children were described as being impaired in their attention, regulation of activity, and impulsivity, as well as in other cognitive abilities, including memory; these were often noted to be socially disruptive as well. It was then known as “Past encephalitic behavior disorder” and was thought to be a result of brain damage (Bender, 1942; Bond & Appel, 1931).

Over the years, behavioral characteristics like “organic drivenness” (Kahn & Cohen, 1934) and “restlessness syndrome” (Childers, 1935; Levin, 1938), were introduced and the concept of the “brain injured child” was born in this era (Strauss & Lehtinen, 1947). Later on it was named as “minimal brain damage” and eventually as “minimal brain dysfunction” by 1950s and 1960s. In the 1950s Laufer, Denhoff, and Solomons (1957) referred to ADHD children as having Hyperkinetic Impulse Disorder. By the end of this era it seemed well accepted that hyperactivity was a brain damage syndrome, even when evidence of damage was lacking. During the years 1960-1969, the definition of hyperactivity was given in the second edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-II).
In early 1970s the defining features of the hyperactive or hyperkinetic child syndrome were broadened to include what investigators previously felt to be only associated characteristics, including impulsivity, short attention span, low frustration tolerance, distractibility, and aggressiveness. At the end of decade the prevalent view was that hyperactivity was not the only or most important behavioral deficit seen in hyperactive children but poor attention span and impulse control were equally important in explaining their problems. Nevertheless, the view that attention deficits were equally as important in the disorder as hyperactivity was beginning to make its way into the European taxonomies like the International Classification of Diseases-9 (ICD-9).

In 1980's emphasis was laid on attempts to develop more specific diagnostic criteria, the differential conceptualization and diagnosis of hyperactivity from other psychiatric disorders and later in the decade, critical attacks on the notion that inability to sustain attention was the core behavioral deficit in ADHD. The hyperkinetic reaction of childhood was named as attention deficit disorder (ADD) with or without hyperactivity in the DSM III (Manchanda, 2010).

ADHD was viewed as far more influenced by neurological and genetic factors than by social or environmental ones (Manchanda, 2010). There was also a major shift over this decade towards the recognition that a deficit in behavioral inhibition may be the characteristic of ADHD that distinguishes it most clearly from other mental and developmental disorders and that this deficit is associated with significant disruption in the developmental disorders and that this deficit is associated with significant disruption in the development of typical self-regulation.

ADHD is now enjoy the status as a universal disorder; with an ever-growing international acceptance of both its existence and its status as a chronic disabling condition for which combinations of medication and psychosocial treatments and accommodations may offer the most effective approach to management (Manchanda, 2010).
Symptoms of ADHD

Inattention

Attention is a generic term used to designate a group of hypothetical mechanisms that collectively serve an important function for the organism (Mesulam, 1985). Skinner (1953) defined attention as a functional relationship between stimulus and response. Attention is not a thing, entity, or mental function but a description of a set of relations between stimulus or events and responses to them. Attention continues to be a multidimensional concept, likely to be impacted by physiological, cognitive, emotional and behavioral variables. Goldstein and Goldstein (1990) theorized that attention deficit may be a disorder of faulty selective attention. Whereas, Vander Meere and Sergeant (1988) suggested that sustained attention may represent the core deficit for children with attention deficit (Manchanda, 2010).

Children who ADHD display difficulties with attention relative to normal children of the same age and gender. Parents and teachers often described these attention problems in terms such as “does not seem to listen”, “fails to finish assigned tasks”, “often loses things”, “and cannot concentrate”. The construct of attention as studied in neuropsychology is multidimensional and can refer to alertness, arousal, selectivity or focus execution, encoding, sustained attention, distractibility, or span of apprehension, among others. Individuals with ADHD have greatest difficulties with aspects of attention related to persistence of efforts, or sustaining their attention to tasks (vigilance) (Newcorn et al. 2001).

Other problem is distractibility, or the likelihood that a child will respond to the occurrence of extraneous events unrelated to the task. It is also seen that the children with ADHD have the worst performance when the stimulation is emended in the task materials itself (Brodeur & Pond, 2001). Children with ADHD spend much more time engaged in off task behavior instead of attending to their assigned tasks (Sawyer, Taylor & Chadwick, 2001). Most studies document greater difficulties with attention or continuous-performance tasks or vigilance tasks (Sideman, Biederman, Foraone, Weber & Oullette, 1997).
IMPULSIVITY

In common parlance and popular perception an impulsive individual is someone who acts with some degree of frequency or impulsive or unplanned thoughts that others either or do not have but do not act on (Goslin, 1969). Whereas impulse refers to thoughts, impulsivity refers to a constellation of repeated behaviors that are somehow related to these thought (Stanford & Barrett, 1992). Impulsivity has been described as a number of overlapping definitions including human behavior without adequate thought (Smith, 1952), behavior with no thought what so ever (English, 1928), action of instinct without recourse to ego restraint (Demant, 1933), and the absence of reflection between an environment stimulus and an individual’s response (Doob, 1990). Clinically, individuals with ADHD are often noted to respond quickly to situations without waiting for instructions to be required in the setting. These children may also fail to consider the potentially negative, destructive, or even dangerous consequences that may be associated with particular situations or behaviors. Accident poisonings, injuries, causing careless damage to property are very common in children with ADHD (Hartsough & Lambert, 1985; Mitchell, Aman, Turbott & Manku, 1987).

Impulsivity is multidimensional in nature. These dimensions include executive control, delay of gratification, effort, and even compliance. These forms impulsivity often associated with ADHD involved the under-control of behavior, poor sustained inhibition, the inability to delay a response or defer gratification, or the ability to inhibit dominant or prepotent responses (Nigg, 1999, 2000, 2001). It is commonly noted that children with ADHD have problem in dealing with delay, they find waiting as painful and aversive. These children are often noted to say things indiscreetly and without regard for the feelings of others or for the social consequences to themselves. Blurting and interrupting others conversations are commonly noticed symptoms of ADHD impulsivity. It is difficult to distinguish impulsivity dimension from hyperactivity, as overactive children are also impulsive and vice versa.

HYPERACTIVITY

The third primary and characteristic feature of those with ADHD is their excessive or developmentally inappropriate levels of activity, whether motor or verbal.
Restlessness, fidgeting and generally unnecessary gross bodily movements irrelevant to the situation, are often seen. Parents often describe these children as "always on the go", "acts as if driven by a motor", "climbs excessively", "can't sit still", "talks excessively", and is "squirmy". At school, these children are often seen out of their seats, moving about the class without permission, moving their arms and legs, while working, talking out of their turn to others (Barkley, Du Paul, & M C Murray, 1990). Social interactions of these children often indicate generally excessive speech and commentary (Barkley, Cunningham, & Karlson, 1983; Berk & Potts, 1991; Copeland, 1979; Zentall, 1988). It is seen that ADHD children are more active, restless and fidgety than normal children throughout the day and even during sleep (Barkley, & Cunningham, 1979; Porrino et al., 1983; Teicher, Ito, Glod & Barber, 1996). Several studies have revealed that these symptoms increase in frequency in boring or under-stimulating environments and decrease when stimulation is added to the setting (Antrop, Roeyers, Vanoast, & Buysse, 2000; Zentall, 1985).

**DSM-IV TR Diagnosistic criteria for attention. Deficit/Hyperactivity Disorder**

A. Either (1) or (2):

(1) Six (or More) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

**Inattention**

(a) Often fails to give close attention to details or makes careless mistakes in school work, work, or other activities.

(b) Often has difficulty sustaining in tasks or play activities.

(c) Often does not seem to listen when spoken to directly.

(d) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the work place (not due to appositional behavior or failure to understand instructions).

(e) Often has difficulty organizing tasks and activities.
(f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as school work or home work).

(g) Often loses things necessary for tasks or activities (e.g., toys, school assignments pencils, books, or tools).

(h) Is often easily distracted by extraneous stimuli.

(i) Is often forgetful in daily activities.

(2) six (or more) of the following symptoms of Hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

**Hyperactivity**

(a) Often fidgets with hands or feet or squirms is seat.

(b) Often leaves seat in class room or in other situations in which remaining seated is expected.

(c) Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feeling of restlessness).

(d) Often has difficulty playing or engaging in leisure activities quietly.

(e) Is often “on the go” or often acts as if “driven by a meter”.

(f) Often talks excessively.

**G3. Impulsivity**

(g) often blurts out answers before questions have been completed.

(h) Often has difficulty awaiting turn.

(i) Often interrupts or intrudes on others (e.g. butts into conversations or games).

B. Some hyperactive – impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g. at school [or work] and at home).

D. there must be clear evidence of clinically significant impairment is social, academic, or occupational functioning.

E. the symptoms do not occur exclusively during the course of a pervasive development disorder, schizophrenia, or other psychotic disorder and are not better accounted for by
another mental disorder (e.g. mood disorder, anxiety disorder, dissociative disorder, or a personality disorder).

**Code based on type:**

Attention-deficit/ hyperactivity disorder, combined type: if both criteria A1 and A2 are met for the past 6 months.

Attention-deficit/ hyperactivity disorder, predominantly inattentive type: if criterion A1 is met but criterion A2 is not met for the past 6 months.

Attention-deficit/ hyperactivity disorder, predominantly hyperactive-impulsive type: if criterion A2 is met but criterion A1 is not met for the past 6 months.

Coding note: for individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, “in partial remission” should be specified, (APA, 2000).

**ICD-10 Diagnostic Criteria for Hyperkinetic disorder**

The research diagnosis of hyperkinetic disorder requires the definite presence of abnormal levels of intention, hyperactivity, and restlessness that are pervasive across situations and persistent over time and that are not caused by other disorder such as autism or affective disorders (WHO, 1993).

**G1. Inattention.**

At least six of the following symptoms of inattention have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the development level of the child.

1) Often fails to give close attention to details, or makes careless errors in school work, work, or other activities;

2) Often fails to sustain attention in tasks or play activities;

3) Often appears not to listen to what is being said to him or her;

4) Often fails to follow through on instructions or to finish school work, chores or duties in the workplace (not because of oppositional behavior or failure to understand instructions);

5) Is often impaired in organizing tasks and activities;
6) Often avoids or strongly dislikes task, such as homework, that require sustained mental effort;

7) Often loses things necessary for certain tasks or activities, such as school assignments, pencils, books, toys, or tools;

8) Is often easily distracted by external stimuli;

9) Is often forgetful in the course of daily activities.

G2. Hyperactivity.

At least three of the following symptoms of hyperactivity have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the development level of the child:

1) Often fidgets with hands or feet or squirms on seat;

2) Leaves seat in classroom or in other situations in which remaining seated is expected;

3) Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, only feelings of restlessness may be present);

4) Is often unduly noisy in playing or has difficulty engaging to quietly in leisure activities;

5) Exhibits a persistent pattern of excessive motor activity that is not substantially modified by social context or demands.

Impulsivity:

At least one of the following symptoms of Impulsivity have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the development level of the child;

1) Often blurts out answers before questions have been completed;

2) Often fails to wait in lines or await turns in games or group situations;

3) Often interrupts or intrudes on others (e.g. butts into others conversations or games);

4) Often talks excessively without appropriate response to social constraints.

G4. Onset of the disorder is no later than the age of 7 years.
G5. Pervasiveness.

The criteria should be met for more than a single situation, e.g. the combination of inattention and hyperactivity should be present both at home and at school, or at both school and another setting where children are observed, such as a clinic. (Evidence for cross-situational will ordinarily require information from more than one source; parental reports about classroom behavior, for instance are unlikely to be sufficient.)

G6. The symptoms in G1 – G3 cause clinically significant distress or impairment in social, academic, or occupational functioning.

G7. The disorder does not meet the criteria for pervasive developmental disorders, manic episode, depressive episode, or anxiety disorders.

Many authorities also recognize conditions that are sub threshold for hyperkinetic disorder. Children who meet criteria in other ways but do not show abnormalities of hyperactivity-impulsiveness may be recognized as showing attention deficit; conversely, children who fall short of criteria for attention problems but meet criteria in other respects may be recognized as showing activity disorder. In the same way, children who meet criteria for only one situation (e.g. only the home or only the classroom) may be regarded as showing a home-specific or classroom-specific disorder.

These conditions are not yet included in the main classification. Because of insufficient empirical predictive validation, and because many children with sub threshold disorders show other syndromes (such as oppositional defiant disorder) and should be classified in the appropriate category (WHO, 1993).

Models of ADHD

Early, research on ADHD was theoretical, at least in regard to its basic nurture. That research is mainly exploratory and descriptive, with two exceptions. One is Quay’s, (1988 a, 1988b, 1996) use of Grey’s (1982) neuro psychological model of anxiety to explain the origin of the poor inhibition seen in ADHD. This Quay-Grey Model states that the impulsiveness arises from an under functioning of the brain’s is behavioral inhibition system. That system is said to be sensitive to signal conditioned punishment, and the mallels predicts that those with ADHD should prove less sensitive to such signals, particularly in passive avoidance paradigm (Milich, Hartung, Martin, & Haiger,
1994; Quay, 1988b). The second exception is the work of Sergeant and Vander Meere, (1988; Sergeant, 1995a, 1995b, 1996; Vander Meere, Van Baal & Sergeant, 1989), who successfully used information processing theory and its associated energetic model (arousal, activation, and effort) for isolating the central deficits in ADHD with in that paradigm (Sergeant, 1995b) (Barkley, 1997).

Douglas’s, (1980) earlier model of ADHD is not actually a theory; it is mainly descriptive and was arrived at inductively from a review of the research findings ADHD in which Douglas, (1980, 1983; Douglas & Peters, 1979) discerned a pattern among due findings consistently noted in this field. That pattern comprised the four deficiencies noted earlier. Although it was tremendously helpful at the time, such pattern discernment remains at a descriptive level’s albeit more synthetic than prior efforts at conceptualizing ADHD (Barkley, 1997).

A second reason why a theory of ADHD is sorely needed is that the current clinical view of ADHD (i.e. that of the DSM –IV), being purely descriptive of two behavioral deficits (inattention and hyperactivity- impulsivity) also cannot readily account for many cognitive and behavioral deficits associated with ADHD. To account for such findings any model must fulfill at least five key requirements as noted by: (a) It must explain why an actual deficit in attention in children with ADHD has not been found (Schachar et al. 1993, 1995; Sergeant, 1995b; Van der Meere, Press; Van der Meere and Sergeant, 1988a, 1988b, 1988c) even though research on parent and teacher ratings of ADHD repeatedly identifies a forcenent (positive and negative) is available or has been previously associated with that response. The inhibitory process involved in interference control may be separable from that involved in the delay or cessation of a response. Nevertheless, the previous neuropsychological models on which the present one is based clustered these processes together. Barkley, (1997) suggested that all three inhibitory activities are impaired in ADHD and it has led to the construct being taken as single.

Distinctiveness of the model offered by Barkley, (1997) is its linkage of the deficiency in inhibition to the distruption of five other neuropsychological abilities that depend on inhibition for their efficient execution. Four of these abilities are critical for
self-regulation and goal-directed persistence, so they are called executive functions here. ADHD is believed to disrupt these executive functions because the first executive, self-regulatory act must be inhibition to respond that is used for further self-directed, executive actions. It is not to say that behavioral inhibition directly causes these executive or self-directed actions to occur. However, it does set the occasion for their performance by providing the delay necessary for them to occur these four executive functions, therefore, it should be viewed as neuropsychological systems, separate from the behavioral inhibition yet hierarchically (or pyramidally) perched on it to assist in self-regulation (Barkley, 1997).

Kanfer and Karoly, (1972) have reported that self-regulation is any response, or chain of response, by the individual that serves to alter the probability of the individual’s subsequent response to an event and, in so doing, functions to alter the probability of a later consequence related to that event (Skinner, 1953). These self-directed behaviors need not be publicly observable, although it is likely that in early development many of them are over development, they may become progressively more private, or internal-cognitive, in form. The development of internalized, self directed speech to be discussed later, may serve to exemplify this process. Although eventually private, these actions remain essentially self-directed forms of behavior. The term executive function refers to these mainly private (cognitive) self directed actions that contribute to self-regulation. So defined, the term incorporates most of the attributes often ascribed to it by others (Denckla, 1994; Stuss & Benson, 1986; Torgesen, 1994; Welsh & Pennington, 1988), including (a) self-directed actions; (b) the organization of behavioral contingencies across time; (c) the use of self-directed speech, rules, or plans; (d) deferred gratification; and (e) goal-directed, future-oriented, purposive or intentional actions.

Barkley, (1997) reported that the third reason for a new model of ADHD is that the current view treats the subtypes of ADHD as shaving qualitatively identical deficits in attention while differing only in the presence of hyperactive–impulsive symptoms. As noted above, it is doubtful that the problems with inattention associated with hyperactive-impulsive behavior lie in the realm of attention, whereas these are seen in the predominantly inattentive type of ADHD appear to do so. The predominantly
hyperactive-impulsive type actually seems to be a developmental precursor to the combined type. In the field trial for ADHD in the DSM-IV, this hyperactive-impulsive type was chiefly found among preschool children (Applegate et al., 1995). In contrast, the combined type was far more represented in school-aged children, as was nearly the entire sample of the inattentive type. This relationship of ADHD type to the ages associated with it likely arises from a simple observation made in prior studies. The hyperactive-impulsive behavior pattern seems to emerge first in development, during the preschool years, whereas the symptoms of inattention associated with it appear to have their onset several years later, (Applegate & Frick, 1995; Loeber, Green, Lahey, Christ & Frick, 1992). Moreover, the type of problems with inattention seen even later than those that would eventually be associated with hyperactive-impulsive behavior (Applegate et al., 1995).

In fact much of the present model linking inhibition to four executive functions was set forth by Bronowski (1967) 30 years ago. However the present explication differs substantially from the initial application of Bronowski’s ideas to ADHD (Barkley, 1994, 1995) in the following respects: (a) the incorporation of portions of Fuster’s (1989, 1995) theory and the views of others (Knights, Grabowecky, and Scabini, 1995; Milner 1995) on the neuropsychological functions subserved by prefrontal cortex into a new hybrid model; (b) the inclusion of more precise definitions of behavioral inhibition and self-regulation; (c) the addition of a motor control-fluency-syntax component to the model; (d) the inclusion of the self-regulation of drive and motivation as well as that of emotion in the model; (e) the reconfiguration of the model components more logically than before (Barkley,1994); (f) the addition of numerous recent findings bearing on the linkages among these components and their applicability to ADHD, and (g) additional predictions about ADHD (Barkley, 1997).

Bronowski’s Theory on the Uniqueness of Human Language

Bronowski, (1977) identified four unique properties of human language that distinguish it from the languages of animals. He argued that human language is distinctive because it is not simply a means of communication but of reflection, during which plans of action was proposed, play out and tested. Reflection can only happen if
there is a delay between the arrival of a stimulus or event and the response to that event. Bronowski treated this capacity to inhibit and delay responses as the central and formative feature in the evolution of the unique features of human languages. It is not just the response that is being delayed but decision to respond (Bronowski, 1976). Four consequences flow from the evolution of this ability to inhibit and delay responses: prolongation separation of affect, internalization and reconstitution.

Barkley, (1997) attributed the capacity to delay responses as well as the four consequent mental functions flowing from it are attributed to the brain's pre-frontal cortex. Prolongation, which is the ability to refer backward and forward in time and to exchange messages with others that propose action in the future. This prolongation of reference, or the relation of post event to future actions, requires a special form of memory. During the delay in responding, the feature of the signal, situation, or event must be briefly prolonged, fixed, and held in some symbolic form, so they can be retained for later recall when they will serve to revive the responses associated with them in the future. The recall of the past and the manipulation of the imagery of recall permit the construction of hypothetical situations and their associated consequences. Plans can be formulated from such conjecting and anticipatory behaviors initiated. It is similar to the contemporary concept of working memory in neuropsychology (For reviews, see Becker, 1994). For instance, Goldman-Rakic, (1995) defined working memory as the ability to keep on item of information in mind in the absence of an external cue and utilize that information to direct an impending response.

Separation of affect refers to the separation of the emotional charge from the content of a message or event and, as a result, the separation of the emotional charge from the content of the response to the event. It involves the self-regulation of emotion apart from motor behavior, and it affords the generation of neutral responses despite emotionally provocative events that may elicit highly charged feelings within the individual (Barkley, 1997).

The delay between event and response also permits time for the event to be referred to more than one center in the brain and gives rise to an inner discussion of alternatives before a response is formed. This internalization of language gives a unique
form to human thought and speech. During the delay in responding, language comes to be turned on the self. It thereby moves from being primarily a means for communication with others to one of communication with the self, a means of reflection and exploration that permits the construction of hypothetical messages or responses before one is chosen to utter or perform. It also permits the creation of self-directed instructions and thereby becomes a fundamental tool for self-control. In supporting his assertions, Bronowski, (1967, 1977) referenced the views of Vygotsky, (1978, 1987).

Berk, (1994) reported that Vygotsky’s, (1978, 1987) theory on the development of private speech remains the most accepted view on the topic at this time. Such speech starts out as Speech uttered aloud by children that is addressed either to the self or to no one in particular (Berk & Potts, 1991). In its earlier stages, it is thought spoken out loud that accompanies ongoing action. It functions as a form of self-guidance and direction by assisting with the formulation of a plan that will eventually assist the child in controlling his or her own actions as it matures (Berk & Potts, 1991). Slowly and gradually, speech becomes progressively more private or internalized, and behavior comes increasingly under its control; private speech thus become internal verbal thought that can exert a substantial controlling influence over behavior. This internalization of speech proceeds in an orderly fashion. It seems to evolve from more conversational, task-irrelevant, and possibly self-stimulating forms of speech to more descriptive, task-relevant forms and then on to more prescriptive and self-guiding speech. It then progresses to more private, inaudible speech and finally to fully private, subvocal speech (Berk, 1994; Berk & Garvin, 1984; Berk & Potts, 1991; Bivens & Berk, 1990; Frauenglas & Diaz, 1985; Kohlberg, Yaeger & Hjertholm, 1968).

The internalization of language brings with it the fourth consequence of inhibition, which is referred to by Bronowski, (1977) as reconstitution, it comprises two processes. The first is analysis, which is the decomposition of sequences of events or messages into their parts. It allows the progressive redistribution of the event or message to other parallel information-processing systems within the brain (Barkley, 1997). The second process is synthesis, where in these parts can be manipulated and used to construct or reconstitute entirely new messages can represent and initiate units of
behavior, behavioral units also can be reconstituted into entirely novel behavioral structures. It gives a synthetic and increasingly hierarchical structure to both human language and behavior. Increasingly complex, novel units comes to be formed out of more elemental ones, and thus a layered structure to behavior is created. Reconstitution, it is argued, that reinstitution creates the potential for original productivity in human language and hence in the human actions controlled by that language (Bronowski, 1977). Barkley, (1997) reported that the rules or syntax for the sequencing of these verbal and behavioral productions are an inherent part of the process of reconstitution.

Reconstitution is quite evident in verbal fluency and discourse because they represent the capacity to rapidly access and reconstitute parts of speech into complete messages for others. The speed, accuracy, fluency, syntax, and general efficiency with which cognitive content is translated into units of speech and then into whole messages to others reflect the synthetic function of reconstitution. Verbal reconstitution should be most evident in confrontational language tasks or in goal-directed speech or writing, where ideas must be rapidly conveyed to achieve the goal of the task. However, it should also be evident in goal-directed behavioral creativity in general because this reflects the capacity to generate a variety of novel, complex sequences of behavior directed toward goals (Barkley, 1997).

**Fuster’s Theory**

Fuster’s (1989, 1995) proposed a theory of prefrontal functions which was apparently independent of Bronowski’s (1997) model, yet the two have much in common. Fuster reported that the overarching function of the prefrontal cortex is the formation of crosstemporal structures of behavior that have a unifying purpose or goal. It is the novelty of these behavioral structures, and especially the temporal discontiguities among their elements that makes the prefrontal cortex essential in their formation. To a lesser extent, their complexity may necessitate the involvement of the prefrontal cortex. However, complexity alone is not sufficient to place such acts within the purview of the prefrontal cortex, on the other hand, time being inserted between the elements of the contingency would be sufficient to do so. Similarly, novelty of the response would also lead to involvement of prefrontal lobes (Barkley, 1997). However several functions must
occur for behavioural structures to be linked across time for example the functions which or temporally symmetrical and are called retrospective and prospective functions are examples. The retrospective function entails the retention of information about post events that are held in their temporal sequence as they pertain to a goal. Such memory is provisional, having timeliness and term and permits the referring of current events to previous events in a sequence as well as the retention of action-related information derived from that analysis. The retrospective function gives rise to formulation and retention of a goal-directed behavioral structure. This forms the prospective function leads to a preparation to act in anticipation of events or an anticipatory set. The behavioral scheme and its relevant events and temporarily represented, deployed in the preparation to act and the execution of these actions, and retained until the goal has been accomplished. These functions are identical to Bronowski's, (1977) concepts of prolongation, hindsight, and forethought, as well as to neuropsychological concept of working memory (Fuster, 1989, 1995).

Fuster, (1989, 1995) reported that the proficiency of working memory is dependent on response inhibition and interference control, just as Bronowski, (1977) had done. It is in working memory that goals and intentions to act are retained and that action plans are formulated and used to guide the performance of the goal-directed responses. The delay in responding, during which the cross-temporal behavioral structures are being formed and retained, is a critical time that requires protection from a variety of sources of interference that can prevent, distort, or completely disrupt the planning taking place. Internal sources may also interfere, such as traces of information still held in working memory from the formation of immediately previous behavioral structures. Old habits more familiar to the individual or having similarity to ongoing behavior may likewise disrupt this synthetic, goal-directed function, as might impulses to immediate gratification (Barkley, 1997).

The dissociation of an inhibitory function from a working memory function is not only conceptual but neuroanatomical as well. Iversion and Dunnett, (1990) argued that the inhibitory functions are ascribed to the arbital-frontal regions of the prefrontal cortex and its reciprocal inter-connections with the ventromedial regions of the striatum. The
functions of working memory are subserved by the dorsolateral region of the prefrontal cortex and its reciprocal connections to the more central region of the striatum (Iversen & Dunnett, 1990). Substantial evidence from neuropsychological and neuroimaging studies provide support to this dissociation (D'Esposito et al., 1995; Fuster, 1989, 1995; Goldman Rakic, 1995; Iversen & Dunnett, 1990; Knights et al., 1995; Milner, 1995; Vendrell et al., 1995; Williams & Goldman-Rakic, 1995).

Michon, (1985) argued that this capacity for holding events in mind in a correct temporal sequence may give rise to the psychological sense of time. If so, time perception would be directly dependent on the integrity of working memory, (Bronowski, 1977). A subjective sense of time would seem to be critical in Fuster's (1989, 1995) model as well, given his emphasis on the cross-temporal structure of the complex behavioral chains generated in the service of goal attainment (Barkley, 1997). The initiation and maintenance of cross-temporal, goal-directed actions require that the prefrontal cortex assist in regulating basic drive or motivational states in the service of such goal-directed acts. Otherwise, new behaviors would rarely be initiated or sustained on the way to their intended goal. Hence the self-regulation of drive and motivational states in the service of goal-directed actions appears to be another function of the prefrontal cortex. Fuster, (1989, 1995) also recognized that disorders of the prefrontal cortex often give rise to disturbances in the regulation of affective and emotional states, yet he found these difficult to interpret within his model. Bronowski, (1977), in contrast, made the separation and self-regulation of affect one of the major consequences of delayed responding in his model.

The functions of the prefrontal cortex clearly influence motor control. The prefrontal cortex is unnecessary for the performance of any motor act or even complex, over learned responses. It is essential for the orderly execution of novel, complex behaviors having a cross-temporal structure. Thus, working memory, hindsight, forethought, sense of time, anticipatory set, and the goal-directed behavioral structures they create influence motor control, fluency, flexibility, syntax, and persistence as they pertain to goal-directed actions. This influence of executive functions over motor control could be seen in three ways as argued by Fuster (1989). (a) in the retention of information
about past events and acts already executed that than feeds forward to influence subsequent responding (i.e., a sensitivity to errors), (b) in the anticipatory setting of the premotor and motor functions (i.e. a preparation to act), and (c) in the inhibition of motor impulses inappropriate to the goal or task. Lack of the inhibitory control that provides for the delay of responses and protection of the delay from interference would have many manifestations, Fuster reasoned, including distractibility, hyperactivity, and impulsivity – the very symptoms attributed to ADHD (Barkley, 1997).

**Behavioral Inhibition**

Behavioral inhibition refers to three inhibitory functions. These exert a direct controlling influence over the motor system, hence the direct downward arrow in figure between behavioral inhibition and motor control-fluency-syntax. Behavioral inhibition, however, does not directly cause the four intermediate executive functions to occur but merely sets the occasion for their performance. Visibly representing this crucial point, the line connecting inhibition to those four executive functions are blunted. But because those executive functions produce direct and casual effects on motor control, arrows connect each executive functions with motor control (Barkley, 1997).

**Working Memory**

The hybrid model predicts that poor behavioral inhibition, as in ADHD, should lead to secondary deficiencies in working memory and its subfunctions. (1) Children with ADHD should be more influenced by context and less controlled by internally represented information that same-age peers without ADHD. (2) Children with ADHD should be more influenced by immediate events and their consequences than by those more distant in time. (3) Those with ADHD should be less likely to recall and hold in mind information about the past (hindsight) for the formulation of a plan in the future (forethought and planning). (4) Anticipatory or preparatory behaviors founded on such planning should be less evident in those with ADHD, so motor presetting in anticipation of the arrival of future events should likewise be less proficient. (5) A form of temporal myopia should exist in children with ADHD, in that behavior is more controlled by the temporal "now" than by internally represented information pertaining to the post, the future, and the sense of time. (6) Children with ADHD should exhibit less control of
behavior by time and more deficient organization of behavior relative to time. (7) Performance under cross-temporal (if-then) contingencies should be less effective in those with ADHD because they cannot bridge the delays in the contingencies, using internally represented information. And (8) the larger the delays in time that separate the components of a behavioral contingency (events, responses, and their consequences), the less successful those with ADHD should be in effectively managing those tasks. Barkley, (1997) argued that there should also be less ability to successfully persist in goal-directed behaviors in those with ADHD. And even when those with ADHD undertake goal-directed behavior, it should be subject to greater interference by sources of disruption in both the external and internal environments and result in less success at goal attainment.

The behavioural inhibition model predicts six additional deficits in association with ADHD: (1) There should be an inability to imitate lengthy sequences of goal-directed behavior demonstrated by others, given that such sequences cannot be held in mind as well for the orchestration of their execution. (2) The sense of time is impaired. (3) Information recalled from memory (retrospective function) temporally disorganized – that is, the very syntax of recall is deficient. (4) Consequently, the syntax of motor planning and execution should likewise be disorganized. (5) Discourse with others reflect fewer references to time, the past, and especially the future. And (6) significant deficiencies exist in the performance of those social skills (i.e. sharing, cooperation, etc.) as well as other adaptive behaviors (i.e., concern for safety, health consciousness, etc.) that are predicated on the valuation of future personal and social consequences over immediate ones. The knowledge of those social and adaptive skills or behaviors is not at issue here; that knowledge may not be deficient in those with ADHD. It is the application of that knowledge in day-to-day functioning that impaired. The problem, then, for those with ADHD is not one of knowing what to do but one of doing what you know when it would be most adaptive to do so. This same problem is typical of patients with injuries to the prefrontal cortex (Delhi, Squire, Bihrl, & Massman, 1992; Stuss & Benson, 1986).

Self-regulation of affect-motivation-arousal

(Dimasio, 1994) believed that affect may not be completely separable from the decision to respond or even from the response itself 4Instead, a more self-regulatory role
of the executive system is stressed here in that emotions, once elicited, come to be moderated or regulated by self-directed, executive actions. Included in this component is also the self-generation of drive or motivational and arousal states that support the execution of goal-directed actions and persistence toward the goal. This combines into a single component and makes some sense. Lang, (1995) cogently argued that the array of human emotions can be reduced to a two-dimensional model, of which one dimension is motivation (reinforcement and punishment) and the other, level of arousal. So the ability to self-regulate and induce emotional states as needed in regulation and induce motivation, drive, and arousal states in support of such behavior. Thus, children may learn to create more positive emotional and motivational states in themselves when angered, frustrated, disappointed, saddened, anxious, or bored by learning to manipulate the variables of which such negative states and their positive alternatives are a function (Cole, Zahn-Waxler, & Smith, 1994; Eisenberg et al., 1993; Kopp, 1989). Such self-directed actions may involve efforts at self-comforting, self-directed speech, visual imagery, and self-reinforcement, among other means (Kopp, 1989). As per Stifter and Braungart, (1995) this process of self-regulating affect begins as early as 5-10 months of age. It is also conceivable that children may learn to self-regulate arousal levels for the purposes of goal accomplishment. Barkley, (1997) argued that this component of the model, therefore, includes the following subfunctions, all of which are performed in the service of goal-directed actions: (a) the self-regulation of emotion, (b) a capacity for objectivity and social perspective, (c) the self-regulation of drive and motivational states, and (d) the self-regulation of arousal.

**Internalization of Speech**

In fact, Fuster’s, (1989) model had little to say about the internalization of speech as a function of the prefrontal cortex Bronowski (1977), however, stressed the uniqueness and importance of the self-direction and internalization of speech and profound control it may exert on the individual’s behavior. Developmental psychologist (Berk and Potts, 1991; Kopp, 1982) and developmental neuropsychologist (Vygotsky, 1978, 1987) have likewise emphasized the importance of this process for the development of self-control.
directed behavior occur, the individual is able to disengage, respond to the interruption and re-engage the original goal-directed sequence because that plan has been held in mind despite the interruption.

Extension of the Model to ADHD

Tremendous progress has been made in the past 2 decades in understanding the neuropsychological functions served by the prefrontal cortex. It has led to the development of theories for organizing and explaining these functions (Fuster, 1989, 1995). Evidence is suggestive of the proposition that ADHD appears to arise from abnormalities in the structure and function of the prefrontal cortex and its networks with other brain regions, especially the striatum (Castellanos et al., 1994; Heilman et al., 1991, Lou et al., 1984, 1989; Rapoport, 1996; Seig et al., 1995; Zamelkin et al., 1990). A model of prefrontal executive functions, therefore, offers some promise as a model for understanding ADHD as well.

The hybrid model predicts that the deficiency in behavioral inhibition that characterizes ADHD diminishes the effective deployment of the four executive abilities that subserve self-control and goal-directed behavior. This inhibitory deficit thereby indirectly disrupts the control of goal-directed motor behavior by its influence on these executive functions. As a consequence, the behavior of those with ADHD is controlled more by the immediate context and its consequences than is the behavior of others. In contrast the behavior of others is more controlled by internally represented information, such as hindsight, forethought, time, plans, rules, and self-motivating stimuli that ultimately provide for the maximization of future net outcomes (Barkley, 1997).

Etiology

Considerable progress has been made toward revealing the pathophysiological basis of ADHD. Studies of ADHD neurochemistry, neuroimaging epidemiological risk factors and genetics have supported the notion that ADHD is a familial disorder involving differences in monoamine regulation and frontal striatal neural circuitry. Additional studies should soon add detail to early findings and improve early detection of risk and intervention (McCracken, 2000).
It was hypothesized in 1971 that the disorder was a genetically transmitted disorder of catecholaminergic functioning. (c/f McCracken, 2000) A number of studies have documented the genetic basis of ADHD. The inferences on genetic transmission as drawn were based on two crucial findings: the higher incidence of ADHD in siblings of children and the increased incidence of other psychiatric conditions, particularly alcohol abuse and antisocial personality disorder in their parents. The latter relationship may have been mediated by the well known comorbidity of ADHD and conduct disorder in child probands. Since one half of conduct-disordered children go on to develop antisocial personality disorder in adulthood, an increased frequency of alcoholism and antisocial personality disorder would be predicted in adults who had ADHD in childhood. Moreover, the clustering of alcohol abuse, antisocial personality disorder, somatization disorder agrees with results of the family studies of Samuel Guze at Washington University. Familial clustering can obviously result from genetic or psychosocial transmission. Studies of the concordance of ADHD in monozygotic and dizygotic twins found greater concordance in the former and no evidence for psychosocial transmission. Finally, adoption studies support the genetic hypothesis. Daniel ADHD children were twice as likely to have ADHD as their half-siblings, while Helene Deutsch found a higher frequency of ADHD symptoms in the biological relatives of ADHD children than in the adopting parents of these children.

The catecholaminergic hypothesis also drew early support from the development of ADHD like symptoms in children who recovered from von Economo’s disease. Pathological studies by Constance von Economo of children who died during the course of the encephalitis showed that they had damage to a variety of structure including the substantia nigra, the major site of origin of dopaminergic cells. The biochemical and genetic basis for ADHD is also supported by the fact that such children and adults often show dramatic improvement when treated with two indirect dopamine agonists, the amphetamines and methylphenidate (Ritalin). The availability of adults with ADHD allowed studies ethically precluded in children. The only study of cerebrospinal fluid (CSF) in adults with ADHD demonstrated a lower concentration of homovanillic acid (the principal metabolite of dopamine) in patients than in controls. Further,
administration of amino acid precursors of dopamine revealed that tyrosine, the immediate precursor of dopamine, decreased ADHD behavior in adult patients. Dopamine is metabolized by monoamine oxidase (MAO) type B (MAO_B). Administration of two inhibitors of this enzyme, pargyline and selegiline (Eldepryl), improved the behavior of ADHD subjects, however these studies were not placebo controlled and must be replicated. Finally, there are an increasing number of imaging studies of children with ADHD and presumably such studies will soon be conducted in adults.

**Neurochemistry**

The incontrovertible benefit of psychostimulants on hyperactive and impulsive behaviors has resulted in research into neurochemical differences in children with ADHD, principally among the monoamines dopamine and norepinephrine. In general, earlier studies of peripheral and cerebrospinal fluid monoamine measures in ADHD were considered inconclusive; similarly, studies on the effects of medications on monoamine metabolism in children with ADHD initially yielded variable results. However, studies McCracken, (2000) have also yielded significant evidence supporting the involvement of both the dopaminergic and catecholaminergic systems in ADHD. For example, one study showed a significant correlation between the pre-treatment cerebrospinal fluid concentrations of the major dopamine metabolite homovanillic acid (HVA) and response to psychostimulant administration. Alternatively, recent studies of changes in peripheral monoamine regulation reveal differences in catecholamines in children with ADHD and normal controls (McCracken, 2000).

Though difficult to extrapolate to the human disorder, animal models incorporating neonatal lesions of dopamine tracts, rodent strains with allelic variants at the dopamine transporter (DATI) and SNAP-25 proteins, and inbred strains such as the spontaneously hypertensive rat (SHR) also support the prominent role of monoamines in the regulation of activity. Taken together, these clinical and basic studies support the importance in ADHD of differences in the dopaminergic innervation of mesolimbic and cortical areas as modulators of persistence, distractibility, motivation, and motor control. Overactivity or poorly modulated responses of the noradrenergic locus coeruleus to
novelty or challenge may elicit the poorly regulated behavioral responses seen in children with ADHD. Increasing dopamine availability and inhibition of locus coerulus activity via adrenergic agonism are hypothesized to be required for maximal therapeutic efficacy on ADHD symptoms, as is found with medications such as the rapidly acting stimulants, (McCracken, 2000).

**Neuroimaging**

Both structural and functional neuroimaging studies have contributed to elucidating the etiology of ADHD. Early studies (McCracken, 2000) using Xenon-133 regional cerebral blood flow pointed to brain areas that form the crux of current neuroanatomical models of pathology in ADHD. Although complicated by the co-occurrence of specific developmental problems in some samples, reduced perfusion in bilateral frontal areas, the caudate nuclei, and additional basal ganglia areas, partially increased by methylphenidate administration, have been reported. The apparent reductions in blood flow in frontal and basal ganglia regions were consistent with the emerging view that many of the self-regulation difficulties seen in children with ADHD resembled the behavior seen in classic frontal lobe damage syndromes. The first positron emission tomography (PET) study of adults with familial ADHD also found both global and specific patterns of reduced metabolism in the brains of patients compared with controls. Compared with normal control adults, those with ADHD showed global reduced metabolism bilaterally, as assessed by a fluorodeoxyglucose (FDG) tracer. In addition, a survey of specific regions found significant metabolic reductions in superior prefrontal and premotor cortices. Attempts to replicate these findings in samples of adolescents with ADHD have found fewer differences; global metabolism did not differ, although metabolism in six specific regions (an equal number from right and left brain areas) was significantly lower than that in controls in one study. Gender apparently strongly influences regional cerebral blood flow, as only females with ADHD differed in group effects from controls in some studies. In general, the initial functional brain imaging studies indicate differences in brain activity associated with ADHD, which may vary with age. Reduced activity in brain areas associated with executive functions is consistent with the types of behavioral problems seen in the disorder.
As McCracken, (2000) concluded the available neuroimaging data strongly support the existence of differences in brain structure and, to a lesser extent, brain function in individuals with ADHD and normal controls. Rather than finding global brain involvement in ADHD, most studies identify relatively specific areas difference associated with the disorder, mainly the richly interconnected areas of the frontal lobes and the striatum. However, additional research is needed before these findings can be fully integrated with other knowledge about the disorder (McCracken, 2000).

**Neurological Factors**

Earlier brain damage, due to brain infections trauma, injury or birth complication, was proposed to be the chief cause of ADHD (Cruickshank, Eliason, and Merrifield, 1988). The structural or functional differences in the frontal lobes, basal ganglia and cerebellum are thought to be associated with ADHD. Research suggests that distinhibition of behavioral responses, difficulties with working memory, planning, verbal fluency, behavioral timing, motor coordination and sequencing are all evident in ADHD individuals (Hervey et al., 2004; Seidman, 1997). It has also been seen that their siblings with or without ADHD also have milder impairments in these executive functions (Seidman et al., 1997).

Studies using psycho-physiological measures of nervous system point to impaired right prefrontal mechanism underlying response inhibition (Pliszka, Liotti and Woldroff, 2000). Whereas, studies examining of cerebral blood flow indicate reduced flow to the frontal lobes striatum, and cerebellum, consistent with underactivity in these regions (Lou, Henriksen, and Brukhn, 1990). The degree of blood flow in the right frontal region and cerebellum are related to motor impairment (Gustafsson, Therlund, Ryding, Rosen and Cederblad, 2000).

(Hynd, Semrud-Clikeman, Lorys, Novey and Eliopulos, 1990) that children with ADHD have smaller right hemisphere plana temporal. Studies have indicated smaller anterior right frontal regions, smaller size of the caudate nucleus, reserved symmetry in the size of head of caudate and smaller globus pallidus regions in children with ADHD (Castellanos et al., 2002; Hynd et al., 1993; Singer et al., 1993). Reduced neurometabolite activity in the right frontal lobes is also found to be associated with the degree of
impairment in the inhibition and attention in children with ADHD (Semrud Clikeman et al., 2000). The ADHD children are also found to have a smaller cerebellar volume which causes deficits in major executive functions (Castellanos et al., 2002).

Pregnancy complications have also been reported to be associated with risk for ADHD. Claycomb, Ryan, Miller and Schanakenberg-Ott, (2004) found that maternal age at delivery, time between onset of labor and birth, and other birth complications account for 42% of ADHD. Other factors contributing to ADHD are fetal distress, forceps delivery, foxtemia or eclampsia, and low birth weight (Sykes et al., 1997; Szatmari, Saigal, Rosenbaum and Campbell, 1993).

**Environmental Factors**

Researchers have also linked ADHD to environmental factors to which children are exposed e.g. Feingold (1975) suggested that a food allergy created hyperactivity in children. Exposure to food colorings, additives, and salicylates in foods would result in overactivity, impulsivity, and learning problems. Research has not confirmed this contention. It has been demonstrated, however, that children with behavioral symptoms of ADHD who also have allergies show improvement when they are placed on a food-elimination diet. Studies examining the effect of ingestion of food colorings have provided only weak evidence that food colorings, especially in preschool and early elementary school age ADHD children, will cause behavioral deterioration. Studies that have examined for the effects of sugar on ADHD children have not shown any deleterious effects on children’s behavior when they consume various amounts of sugar. In general, a dietary explanation of hyperactivity and inattention is not sufficient for all cases of ADHD. Several other studies examining for the environmental effects of leads toxicity, maternal smoking and fluorescent lighting have demonstrated minimal effects of various environmental factors that can, in susceptible children, create a worsening of already existing behavioral symptoms of ADHD (Garfinkel, Wonder, 1989).

However, findings in longitudinal studies of children who have experienced repeated loss, family breakdown, and disruption in bonding in the first 3 years of childhood demonstrate many of the same symptoms of ADHD. It seems difficult to interpret these findings, as these environmental factors may be social sequelae of this
residual state in the parents. Families that are at risk for this disorder may undergo a
greater number of chaotic family situations. Further research into the independent
psychosocial causation of ADHD should control for these situational conditions and for
existing psychiatric disorders. At the present time, studies have not controlled for the
effects of the pre existence in both children and parents of ADHD, conduct disorder,
antisocial personality disorder or ADHD (residual state) and their association with
diverse and family disruption (Garfinkel, Wonder, 1989).

**Genetic Factors**

Efforts have been made to link ADHD to genetic factors e.g. in one study
compared ADHD children with normal children and demonstrated a much higher rate of
antisocial personality were compared and alcoholism in the fathers of ADHD children
and Briquet’s syndrome in the mothers (Garfinkel and Wonder, 1989). In their own
childhood, the fathers were also noted to have a much higher rate of ADHD. Other
studies have also demonstrated the aggregation of alcoholism, antisocial personality
disorder and Briquet’s syndrome in the families of ADHD children compared with
normal children. Yet another study demonstrated that there was a threefold greater
likelihood of identifying ADHD in the brothers of ADHD probands as compared with
normal children. Affective symptomatology was also noted more commonly in siblings
and ADHD probands as compared with the control group. It has also been noted that in
the families of children who have the combination disorder of ADHD and conduct
disorder that the adult relative are most at risk for antisocial personality, alcoholism, and
Briquet’s syndrome. The determining factor may be the presence of the conduct disorder
and its association with adult forms of antisocial personality disorder and substance
abuse.

(Garfinkel, Wonder, 1989) reported two adoption studies which indicated were
done indicating that there was an increased rate of alcoholism, antisocial personality
disorder and Briquet’s syndrome in the biological parents of ADHD children and with the
parents of matched control group of children. Those parents who had adult psychiatric
diagnoses were often hyperactive and conduct disorder as children. These studies also
demonstrated the high rate of psychiatric diagnoses in the biological parents of ADHD
children. It also has been observed that full siblings of ADHD children had a much higher rate of ADHD approximately five to six time greater when compare with half siblings. These studies plus a number of twin studies, reinforce the importance of genetic factors rather than other environmental events (Garfinkel and Wonder, 1989).

**Developmental and Seasonal Factors**

Reports in the literature state that September is the peak month for births of children with ADHD with and without comorbid learning disorders. The implication is that prenatal exposure to winter infections during the first trimester may contribute to the emergence of ADHD (Sadock and Sadock, 2007).

**Brain Damage**

Sadock and Sadock, 2007 have reported that some children affected by ADHD had subtle damage to the CNS and brain development during their fetal and perinatal periods. The hypothesized brain damage may potentially be associated with circulatory, toxic, metabolic, mechanical, or physical insult to the brain during early infancy cause by infection, inflammation and trauma. Children with ADHD exhibit nonfocal (soft) neurological signs at higher rates than those in the general population.

**Psychosocial Factors**

Psychosocial factors have also been reported (Sadock and Sadock, 2007) to play a significant role in ADHD. Children in institutions are frequently overactive and have poor attention spans. These signs result from prolonged emotional deprivation and they disappear when deprivational factors are removed, such as through adoption or placement in a Foster home. Stressful psychic events, disruption of family equilibrium, and other anxiety-inducing factors contribute to the initiation or perpetuation of ADHD. Predisposing factors may include the child’s temperament, genetic-familial factors, and the demands of society to adhere to a routinized way of behaving and performing. Socioeconomic status does not seem to be a predisposing factor (Sadock and Sadock, 2007).
Associated Factors

Children with ADHD may have areas of impairment that are not part of the key symptoms of hyperactivity, impulsivity and inattention. These associated areas include behavioral, cognitive, affective and social spheres (Hechtman, 2005).

Behavioral

Generally, children with ADHD do better in one-to-one settings with an adult than in group settings with peers. They need clear and immediate consequences and reinforcements and a good deal of supervision.

Children with ADHD often lack persistence. They start projects without finishing them. They begin a game or activity, become “bored” quickly, and leave it impulsively. They often have problems with delayed gratification and do not persist if gratification is long incoming. ADHD children may show very variable performance on academic or other tasks. This variable performance leads to frustration and a fragile sense of self.

Cognitive

It has been shown (Hechtman, 2005) that people with ADHD have an impaired sense of time and thus have problems in time-dependent tasks and tests. A poor sense of time results in problems with planning, waiting and playing. Short-term memory may also be affected. Impulsive cognitive styles also affect cognitive functioning (Hechtman, 2005).

Emotional

ADHD is often associated with dysregulation of affect, resulting in temper outbursts, mood lability, and reactivity. Moods can change dramatically for no apparent environmental reason, and they can be explosive, intense and infections. The reaction of others and the consequences are often not well understood by the individual with ADHD, who has moved on to something else and does not see what all the fuss about (Hechtman, 2005).

Social

Children with ADHD often have problems with peers, siblings, parents, and teachers. Individuals with ADHD have problems in reading accurately the social cues; they tend to misinterpret social situations, and they often react inappropriately. Children
with ADHD are often described as bossy, intrusive and insensitive to the needs of other people. They have problems cooperating with other children, respecting social hierarchies, and following rules. They may experience rejection and teasing and become verbally and physically aggressive. These children may overreact to situations. They are unable to let go of an argument or stay out of trouble with other children. They often do better alone with an adult or playing with children younger or older than themselves. These social problems may have significant long-term impact on development and self-esteem and thus need to be addressed (Hechtman, 2005).

**Cognitive Function**

Cognitive deficits, particularly impairments in attention and executive functions, are considered to be a core part of attention-deficit/hyperactivity disorder (ADHD) (Barkley, 1997; Douglas, 1972) and are thought to play a major role in the difficult adaptation of ADHD children. Many studies (Barkley, 1997; Barkley, Grodzinsky, & DuPaul, 1992; Denckla, 1991; Grodzinsky & Diamond, 1992; Pennington, Grosisser, and Welsh, 1993; Pennington and Ozonoff, 1996; Seidman, 1997; Seidman, Benedict, et al., 1995; Seidman, Biederman, et al., 1995; Seidman, Biederman, Faraone, Weber, Mennin & Jones, 1997; Seidman, Biederman, Faraone, Weber, Mennin and Jones, 1997; Seidman, Biederman, Faraone, Weber & Ouellette, 1997) have demonstrated that children with ADHD exhibit subaverage or relatively weak performance on various tasks of vigilance, verbal learning, memory, and executive functions such as set shifting, planning and organization, complex problem solving and response inhibition. These dysfunctions have been demonstrated to persist into late adolescence (Fischer, Barkley, Edelbrock, & Smallish, 1990; Seidman et al., 1997) and adulthood (Seidman, Biederman, Weber, Hatch, & Faraone, 1998) and have been found in non-referred siblings of ADHD patients who have been diagnosed with ADHD (Seidman, Biederman, Monuteaux, Weber, & Faraone, 2000). It is pertinent here to note that the executive function deficits have been shown to be independent of psychiatric comorbidity associated with ADHD (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Seidman et al., 1998, 2000).

A major obstacle, however, in identifying the underlying neuropsychology of ADHD pertains to the overlap between ADHD and various kinds of learning disabilities
The literature on ADHD has consistently documented that a substantial minority of children with ADHD also have LDs, such as reading or arithmetic disability (Cantwell & Satterfield, 1978; Lambert & Sandoval, 1980; Levine, Busch, & Aufseeser, 1982). Rates vary depending on the definition and type of learning disabilities (LD), with estimates ranging from 10% to more than 90% (Semrud-Clickeman et al., 1992), although a rate of approximately 30% using both reading and arithmetic as comorbid learning disabilities has been more realistically suggested (Faraone, Biederman, Monuteaux, & Seidman, 2001). Learning disabilities, when combined with ADHD, have a specific role in school failure (Faraone et al., 2001). Because learning disabilities (Without ADHD) can also manifest neuropsychological deficits in attention and in components of memory (Benezra and Douglas, 1988), more work is needed to further evaluate whether neuropsychological deficits in ADHD children with comorbid learning disabilities are due to ADHD or to learning disabilities (Geidman, Biederman, Monuteaux, Doyle and Faraon, 2001).

Learning disabilities are neuropsychological disorders characterized by specific processing problems. For example, dyslexia (reading disability) is particularly characterized by specific impairments in single word reading, reading fluency, and reading comprehension, usually resulting from deficient phonological processing (Pennington et al., 1993). Although in our work, we documented that executive function deficits in ADHD youth remained significant after statistically controlling for the presence of LD (Seidman, 1997), questions remain as to the nature of association between ADHD, learning disability and executive deficits. For example, in a pilot study of ADHD boys, we found that youth diagnosed with ADHD and learning disabilities (ADHD + LD) performed significantly worse than those with ADHD but without learning disabilities (ADHD − LD) on the Rey-osterrieth complex figure organization score (a measure of executive functions) and on rapid naming on the stroop test (Seidman, Beiderman, et al., 1995). However, because our results were based on a small sample we could not address specific types of learning disabilities such as those associated with arithmetic or reading, which were lumped together. Therefore, an
understanding of the role of the specific learning disabilities combined with ADHD will be important for clarifying the nature of neuropsychological dysfunction in ADHD.

Several hypothetical relations can be tentatively formulated in conceptualizing the complex relationship between ADHD and learning disabilities. One possibility is that the comorbidity of learning disability with in ADHD represents a qualitatively distinct condition, as suggested by family studies relevant to genetic transmission of the disorders (Faraone, Biederman, Krifcher Lehman, Keenan, et al., 1993). There is also support for this model from a number of studies that demonstrate that reading disability and ADHD are characterized by separate deficits, namely phonological processing deficits in the former and executive function deficits in the latter (Pennington et al., 1993; Shaywitz et al., 1995). This leads to the hypothesis that persons with ADHD + LD would not have worse executive function deficits than persons with ADHD – LD (Seidman, Biederman, Monuteaux, Doyle & Faraone, 2001).

Another alternative hypothesis is that persons with ADHD and comorbid learning disability have more severe executive deficits than persons with ADHD without learning disability (Seidman, Biederman, et al., 1995), because of the additive effect of combining two cognitive disorders that both include attentional and memory dysfunctions. There is some support for this hypothesis. A number of studies have compared ADHD children with and without accompanying reading disabilities (RD) on a range of neuropsychological measures. August and Garfinkel, (1990) reported that their combined ADHD + RD group performed significantly worse than the ADHD group (which was also impaired relative to normal controls) on a range of measures in the areas of attention, vocabulary, degraded word recognition and memory for letter sequences. A similar pattern of findings emerged in a study of memory functioning (katarina, Hall, Wong, & Keys, 1992), in which both ADHD and ADHD + LD groups displayed subaverage recall of sequential, ordered auditory and visual information, with the comorbid group showing greater difficulty. Tarnowski, Prinz, and Nay, (1986) found that the ADHD + LD groups was significantly worse on perceptual discrimination on the continuous performance tests (CPT; Weintraub & Mesulam, 1985). In a large recent study, Willcutt and Colleagues (2001) found that ADHD + RD individuals were more impaired on virtually all measures
of neuropsychological function compared with persons with ADHD without RD. However, not all studies have found ADHD children with learning problems to be more impaired on measures of memory, attention, and visual-motor functioning (Halperin, Gittelman, Klein, & Rudel, 1984). Because these results were obtained in studies composed mainly of preadolescent, elementary school boys (ages 6-12), questions remain regarding the relationship of ADHD and learning disability in adolescents. Moreover, almost all research attention has been devoted to studying the impact of comorbid RD, whereas the relevance of comorbid arithmetic disability (AD) has been neglected. We could find no published articles addressing the specific role of AD and ADHD on neuropsychological function, nor had prior studies evaluated the neuropsychological consequences associated with combined RD, AD and ADHD. These results strongly suggest that further analysis of specific learning disabilities associated with ADHD is an important aim.

**Executive Function**

Executive functions are neurocognitive processes that maintain an appropriate problem solving set to attain a future goal (Welsh and Pennington, 1988). In a simplified model, executive functions (EFs) represent “top-down” cognitive inputs that facilitate decision making by maintaining information about possible choices in working memory and interacting this knowledge with information about the current context to identify the optimal action for the situation. Executive functions involve multiple distributed neural networks that include the thalamus, basal ganglia and prefrontal cortex (Casey, in Press; Fuster, 1997; Middleton and Strick 2001, 2002; Pennington, 2002).

Researchers have proposed that symptoms of attention-deficit/hyperactivity disorder (ADHD) arise from a primary deficit in a specific executive function domain such as response inhibition or working memory or a more general weakness in executive control (Barkley 1997; Castellanos and Tannock, 2002). Nearly 10 years ago, Pennington and Ozonoff, (1996) completed a meta-analytic review that systematically examined 18 studies of the neuropsychologic correlates of ADHD. They concluded that ADHD is associated with specific weaknesses in at least a subset of executive function (EF) domains. Many of these samples were small, however, and for most executive function
(EF) measures, only a handful of studies were available. In contrast, a literature search conducted in March 2004 found more than 100 papers between 1980 and 2004 that compared executive function (EF) performance in groups with and without ADHD. This extensive new literature suggests that an updated mete-analysis and new researchers are warranted to examine the implications of these new data for the executive function hypothesis (Willcutt, Doyle, Nigg, Faraone, Pennington, 2005).

Attention Deficit Hyperactivity Disorder (ADHD) and its Comorbidities ADHD has been found to have several comorbid problems and some of these are mentioned below:

**Comorbid Problems**

**Tic Disorder**

Some of the comorbid problems like tic disorder are very common. Children with ADHD and Tourette’s syndrome, or tic disorder, can benefit significantly from stimulations. However, rates of tic exacerbation range from 10 to 36 percent. Rates of stimulant discontinuation because of tic exacerbation range from 0 to 15 percent of greatest concern is that, for a small number of children (0.1 percent) tics do decrease after medication is discontinued. Therefore, careful titration and monitoring of stimulant medication are recommended in these comorbid circumstances (Hechtman, 2005).

**Seizure Disorder**

The co-occurrence of seizure disorder and ADHD is common. The fact that stimulant can theoretically lower seizure threshold have resulted in concern about the use of EEG epileptiform activity, incidence of seizures, or significant interaction with levels of anticonvulsants (Hechtman, 2005). This suggests that stimulants can be use safely in children with seizures and ADHD, provided that the seizure disorder is adequately treated and careful titration and monitoring are in place.

**Aggression and Conduct Disorder**

Hechtman, (2005) reported several controlled studies showing dose-dependent stimulant improvement in ADHD and aggressive symptoms (Physical and non-physical) at home and at school in children with ADHD. Stimulants may also reduce negative social interactions and covert antisocial behavior (stealing and vandalism but not cheating). The short and long term consequences of these reductions are important, as
continued conduct disorder and antisocial behavior predisposes to later drug and alcohol abuse and generally more negative outcome.

**Anxiety Disorder**

Some studies (Hechtman, 2005) have suggested that children with ADHD and comorbid anxiety and depression disorders are less responsive and experience more side effects to stimulant medication than children without this comorbidity. It has also been suggested that children with ADHD and comorbid depression benefit less from stimulant medication than children with ADHD without this comorbidity. Some open label studies (Hechtman, 2005) report that adding SSRIs to stimulant medication for children with ADHD and depression improves their response. More controlled studies are needed to establish the safety and usefulness of this combination. It has also been suggested that children with mania and ADHD should first be put on mood stabilizers, and then stimulants should be introduced very slowly.

**Management of ADHD**

Many clinicians believe that multimodal treatment, using multiple treatments in combination is the most effective way to address the clinical management needs of a child with ADHD. This requires the combined efforts of multi-disciplinary team comprising of a paediatrician, clinical psychologist, child psychologist, special educator and rehabilitation psychologist. Treatment plans need to be individualized according to the spectrum of target symptoms, nature and degree of associated problem, parents and teacher’s motivation and family’s strengths and weaknesses. As the child grows older the treatment plans need to be modified to the changing symptoms, family, and environmental conditions (American Academy of Child and Adolescent Psychiatry, 1997). Although, a large number variety of new medications have arrived on the scene for use in such multi modal intervention, relatively fewer advances have been made in parent training interventions (PI) (Manchanda, 2010).

**Counselling**

As ADHD is one of the major and critical problem in children and medication based treatment though common has been criticized for its side effects It has been suggested that behavioural management approaches including counselling be tried
independently or with medication. Parents teacher and children need to be provided and comprehensive information and guidance after the diagnosis of ADHD is confirmed. Parent training is an effective treatment for the reduction of parent child conflict, child defiance, related disruptive behavior and symptoms in clinically referred children diagnosed with ADHD. Parental concerns doubts and misconceptions about the disorder need to be addressed.

The physician may need to address feelings of guilt, anger, denial, and grief reactions in a sympathetic manner and be supportive of parents' efforts to pursue treatment. School based intervention can also be helpful to improve the basic knowledge among educators about the nature, causes, course and treatments of ADHD. It also helps to increase home and school collaboration, so as to improve the plan of management (Manchanda, 2010).

**Family Therapy**

Family therapy is indicated when parents due to marital conflicts dysfunctional family pattern and psychopathology are unable to carry out a behavioral management programme successfully at home (Barkley, 1997). It includes addressing family dysfunction due to the difficulties of raising an ADHD child, marital problems, increasing family members ability to tolerate frustrations, creating an effective parenting team, helping family members learn self control and gain a sense of mastery and competence and matimizing family’s resources. Such an intervention can be helpful in addressing the management of these children.

**Cognitive-Behavioral Theory**

Cognitive-behavioral theory attempts to modify dysfunctional assumtional assumptions and maladaptive behavior via “self-talk” and self-regulation. Programs were designed to teach children techniques of problem solving, dealing with anger and frustration, persistence and social skills. These programs were not very successful. There were no group differences in measures of self-control, attention, impulsivity, aggression, self-concept and social competence at posttreatment or follow-up. Abikoff’s critical review also indicated that cognitive-behavioral therapy for children with ADHD showed little benefit once treatment was discontinued. There may be several reasons for this lack
of efficacy like inadequate focus on generalization in studies involved, immature verbal executive control in ADHD children gives this training, self-instruction may be inadequate to deal with the severity of impulsivity and affective instability in ADHD, self-interaction and cognitive approaches do not provide training and practice in specific social skills. Thus there is a need for direct and specific behavioural intervention.

**Social Skill Training**

Poor social functioning is one of the most disabling associated feature of ADHD in children. Impaired social functioning and the accompanying peer and adult rejection significantly affect self-esteem and influence long-term outcomes. However, social impairment is very difficult to treat effectively. Social skills programs are usually carried out in small groups with the advantage of providing opportunities for peer modelling and practicing skills with peers. Social skills training combines both cognitive-behavioral and behavioral interventions. Techniques such as modelling, didactic instruction, symbolic play with puppet, in vivo practicing role play, and behavioral rehearsal may be used. Positive reinforcement, self-management and reinforced self-evaluation help reduce negative social interaction and increase positive social behavior. Over the years, Emphasis has been placed on generalization techniques using parents and teachers (Hechtman, 2005)

Skill training program can also be effectively used for the management of ADHD for example F. Frankel’s (c/f Hechtman, 2005) program included 12 weekly session that taught skills such as conversational skills, group entry, handling teasing and rejection, negotiating skill for changing activities and praising others. Parents were taught coaching strategies to use with their child in social interactions with peer at home. Benefits were seen parent and teacher ratings. Other program have used a motivational system to reduce performance problem: increase awareness and understanding of verbal and non-verbal social cues; and promote generalization by involving parents and teachers in the treatment used at home, school and the playground, six specific modules were used.

In addition token reinforcement system may also be used for social skill group, home and school. Teacher included “getting along with peers” in the daily report card. This and other peer interactions were rewarded by the parents at home.
Behavior Management

Behavior management refers to the technique of systematically analyzing the child’s appropriate and inappropriate behavior and designing contingencies that increase the former and minimize the later. Identification of behavioural deficits and excesses is the initial step in such interventions. Parents and teachers are taught techniques such as positive reinforcement, token economies, time out, punishments, response cost, contracts, extinction procedures, grounding, over correction to manage the child’s behaviour.

To start a behavior management programme, the parents are asked to describe in detail the finite actions which constitute the problem behavior along with us frequency, intensity, antecedent events that lead to the problem and the consequences following the behavior. The next step may be to decide the rewards and punishments which are to be introduced in the programme. Rewards may include points, stars, praise, punishments may include time out, loss of privileges, doing extra household chores, and not being allowed to do a favourite activity. The rewards and punishments should occur frequently, be salient to the child, and occur as close in time as possible to the targeted behavior, to make the programme effective. Parents should be consistent across time and situation. Token economies refer to a system wherein a child gains points for performing the desired behavior and loses a point for not complying with the conditions of the contract. This system is often used for adolescents (Martin and Pears).

Research indicated that behavioral intervention programs improve behavior, social skill, and academic performance of children. The major weakness of behavior therapy are lack of maintenance of improvement over time and the failure of change to generalize situations other than the ones in which training occurred (Abromowitz, 1994).

Psychoeducation

Psychoeducation can refer to educating the child and family about ADHD and its possible etiology, presentation, treatments, side effects, and prognosis. The educational process may also address issues of comorbidity and deficits the child experiences, as well as stresses on the child, parents and family as a whole. This aspect of treatment is crucial for laying the ground work. For a trusting respectful relationship between the child, family and therapist (Hechtman, 2005).
Psychoeducational interventions also refer to intervention at school designed to improve school behavior, academic productivity, and achievement. These interventions, after management strategies, which are proactive and meant to prevent undesirable behavior by optimizing conditions for appropriate adaptive behavior. Such intervention can include reduction in task demands, making tasks more stimulating (e.g., via computers), and providing students with choices related to academic work, resulting in increased academic engagement. Classroom interventions also include consequent-oriented programs (e.g., token reinforcement and response cost to improve behavior). A daily report card, listing desired behaviors and academic productivity, is completed by the teacher at school and rewarded by parents at home. In older children and adolescents, this can be replaced by contingency contracting, which still has to have tasks that are readily attainable and rewards that are meaningful to the adolescent and delivered with relatively short delays (within the day). Generally, these programs are effective when they are carried out. However, efficacy decreases when they are discontinued (Hechtman, 2005).

**Individual Psychotherapy**

Though there is a dearth of evaluating well-controlled studies have evaluated the efficacy of individual psychotherapy for children with ADHD yet some scattered efforts have been made. It is unlikely to be effective for ADHD symptoms. However, individual therapy can help the child understand what ADHD is and how the symptoms may be affecting his or her functioning. Issues of poor self-esteem and a sense of isolation and alienation can also be dealt with in therapy. Psychotherapy can explore the child’s feelings about and understanding of stimulant medication, its side effects, and what it can and cannot do. Some individual therapy are quite structured, with the particular areas to be addressed clearly delineated; others are more open-ended. Generally, being able to confide worries and vulnerabilities to a knowledgeable, non-judgmental, supportive adult who provides an optimistic view that change is possible may result in improvement of secondary symptoms such as low self-esteem, anxiety, and depression. These secondary symptoms are often comorbid with ADHD. Individual therapy with the child can also establish the foundation of long-term, although at times intermittent, therapeutic
relationship, which contributes to better long-term treatment of the child with ADHD and his or her family in this chronic, lifelong condition (Hechtman, 2005).

**Parental Training in Behavior Therapy**

Parents are the person with whom the child spends maximum time. If parents are trained and given same special training in handling and managing ADHD children then these children may be managed in a better way parents of children with ADHD often have difficulty in effectively managing their child's behavior. Parent training in an intervention that teaches parents how to implement a contingency management behavioral program. Training involves providing parents with an overview of social learning and behavior management principles. Strategies of behavioral management, such as identifying, target behaviors, instituting (with the child's input) a meaningful reward system, contingency attention, time-out, and response cost, are all taught. The intervention can be carried out with individual parents or groups of parents. Parent groups are more efficient and provide added group acceptance and support, but techniques and strategies learned in the group often need to be reinforced in individual parent sessions to be effectively used at home. Parental training is all the more important as it is the parents with whom the child come in contact very early and spend maximum time with them.

There is an important question as to how the training be given, what should be the process and what should be the size of the group? It can be suggested that parent groups should be relatively small, with no more than eight sets of parents per group. Training sessions may usually held weekly for 8 to 20 weeks. Parents may be given reading material and assigned homework, which is documented in behavioral and reinforcement charts to chronicle their efforts, experiences, successes, and failures. The more active the parents' participation, the better the results. Parents with ADHD may have problems with organizational skills, paying attention to details, following outlines, and being consistent. They have great difficulty carrying out a behavioral program unless they, too, are treated (Hechtman, 2005).

Parent training has been shown (Hechtman, 2005) to be effective but less so than stimulant medication. However, parent training is helpful in improving home functioning and the overall home environment. It seems somewhat more effective in younger (e.g.,
preschoolers) than in older children. Reports from parents indicate that parent training increases their sense of parental efficacy tends to decrease when the acute intervention is discontinued. Therefore, William Pelham advocates a program of maintenance and relapse prevention via continued intermittent contact.

Children with Attention Deficit Hyperactivity Disorder (ADHD) need guidance and understanding from their parents and teachers to reach their full potential and to succeed in school. Before a child is diagnosed, frustration, blame and anger may have built up within a family. Parents and children may need special help to overcome bad feelings. Mental health professionals can educate parents about ADHD and how it impacts a family. They also will help the child and his or her parents develop new skills, attitudes, and ways of relating to each other.

Parenting skills training helps parents learn how to use a system of rewards and consequences to change a child’s behavior. Parents are taught to give immediate and positive feedback for behaviors they want encourage, and ignore or redirect behaviors they want discourage. In some cases, the use of “times-outs” may be used when the child’s behavior gets out of control. In a time out, the child is removed from the upsetting situation and sits alone for a short time to calm down.¹

Parents are also encouraged to share a pleasant or relaxing activity with the child, to notice and point out what the child does well, and to praise the child’s strengths and abilities. They may be learn to structure situations in more positive ways. For example, they may restrict the number of playmates to one or two, so that their child does not become over stimulated or, if the child has trouble completing tasks, parents can help their child divide large tasks into smaller, more manageable steps. Also, parents may benefit from learning stress management techniques to increase their own ability to deal with frustration, so that they can respond calmly to their child’s behavior.¹

Sometimes, the whole family may need therapy. Therapists can help family member find better way to handle destructive behaviors and encourage behavior changes. Finally, support groups help parents and families connect with others who have similar problem and concerns. Groups often meet regularly to share frustrations and successes, to
exchange information about recommended specialists and strategies and to talk with experts.¹

Overactive hyperkinetic child with poor attention span is a source of frustration to parents and sibling. His teacher may dub him as lazy and disobedient. Help may be sought from a Clinical psychologist, child counsellor, child psychologist or a paediatrician. Handling and managing such problem by a team is all the more effective.