EVALUATION OF BRAIN ELECTRICAL ACTIVITY MAPPING (BEAM) IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVE DISORDER (ADHD)

THESIS

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

,, قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ ,,

صدق الله العظيم
سورة البقرة ــ آية ٣٢

Errata

Page	Paragraph	Line	Wrong	Correction
13	4	6	aud	and
24	1	9	. hyperkinetec	hyperkinetic
39	3	5	at age seven	at age of seven
51	4	2	different	are different
61	3	4	he	the
71	4	7	when	.When
86	4	5 & 8	DBH	DBM
89	2	5	50 males	56 males
108	Fig. 4	1	51.4%	54.3%
			48.6%	45.7%
111	Tab.15	2	37.1%	59.3%
			low SES	middle SES
130	Fig. 17	index box	Consang.	Non consang.
			Non consang.	Consang.
147	2	3	that	than
	4	13	"missing line"	"families no enough time could be spent to children's medical and"
151	2	8	As far we	As far as we
154	1	2	fist	first
156	1	1	score	same
	4	4	above below	above and below
160	5 & 6	1	higier	higher
200	5	2	1977	1997

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List Of Abbreviations

AAD	Arousal Adjustment Deficit
AD	Anxiety Disorder
ADD	Attention Deficit Disorder
ADD-H	Attention Deficit Disorder with Hyperactivity
ADDH	Attention Deficit Disorder with Hyperactivity
ADD-NoH	Attention Deficit Disorder without Hyperactivity
ADHD	Attention Deficit Hyperactivity Disorder
ADHD-AD	Attention Deficit Hyperactivity Disorder (Inattentive type)
ADHD-HI	Attention Deficit Hyperactivity Disorder (Hyperactive - Impulsive type)
APA	American Psychiatric Association
ATRS	Abbreviated Teacher Rating Score
Aud EP	Auditory Evoked Potential
BEAM	Brain Electrical Activity Mapping
CAT	Computerized Axial Tomography
CC	Corpus Callosum
CD	Conduct Disorder
CEEG	Computerized EEG
CME	Computerized mapping of EEG
CNS	Central Nervous System
CNV	Contingent Negative Variation
CSF	Cerebro Spinal Fluid
CT	Computerized Tomography
CTEEG	Computerized Tomography of EEG
DBH	Dopamine –Beta Hydroxylase enzyme
DBM	Dynamic Brain Mapping
DMP-1	Diagnostic Manual of Psychiatric Disorders
DSM-II	Diagnostic and Statistical Manual – Second Edition
DSM-III	Diagnostic and Statistical Manual – Third Edition
DSM-IV	Diagnostic and Statistical Manual – Fourth Edition
DZ	Dizygotic
EEG	Electro Encephalo Graph
EMG	Electro myoGraph
EP	Evoked Potential
ERP	Event Related Potential
FAS	Fetal Alcohol Syndrome

FFT	Fast Fourier Transformation Algorithm
GSA	Grid Sector Analysis
HSD	Honesty Significant Difference
HVA	Homo Vanilic Acid
5-HIAA	5-Hydroxy-Indol Acetic Acid
5-HT	5- Hydroxyl Tryptamine
ICD-9	International Classification of Diseases – 9th Edition
ICD-10	International Classification of Diseases – 10th Edition
IQ	Intelligence Quotient
MAO	Mono Amine Oxidase
MBD	Minimal Brain Dysfunction
MFF	Maching Familiar Figures Test
MHPG	3-Methyl 4 –Hydroxyl Phenyl Glycol
MRI	Magnetic Resonance Imaging
MZ	Monozygotic
NIMH	National Institute of Mental Health
NMR	Neuro Magnetic Resonance
ODD	Oppositional Defiant Disorder
PET	Positron Emission Tomography
QEEG	Quantitative EEG
SD	Standard Deviation
SES	Socio Economic Status
SPM	Statistical Probability mapping
TAT	Thematic Appreciation Test
TS	Tourette's Syndrome
UK	United Kingdom
US	United States
USA	United States of America
WISC-R	Wechsler – Intelligence Scale for Children – Revised

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Introduction

Introduction

Children with attention deficit and hyperkinetic disorder (ADHD) were described for three decades as having minimal brain damage syndrome. By restricting the definition to children of average intelligence, the condition was distinguished from mental retardation syndromes and emphasized the integral nature of learning failures in the presence of adequate intelligence (*Conners, 1969*). The major diagnostic criteria are increased motoric behavior (restlessness) and an elevated frequency of gross motor movements, inattention, failure to complete tasks and impulsivity which is characterized by acting quickly without thinking of the consequences (*Okasha, 1980, 1988; American Psychiatric Association, 1987; and Conners, 1993*).

Few attempts have been made to understand the brain mechanisms involved and the cerebral structures affected by the this disorder (*Lou, et al, 1989; and Satterfield, et al, 1990*). Electroencephalogram brain mapping makes it easier to discern topographic differences in electro-encephalograms (EEGs). Also it can help in communication of EEG features and their localization (*Duffy, 1984; and American Psychiatric Assocation, 1997*). Because these children frequently have no frank neurological signs or diseases, the EEG brain mapping gained importance as an objective measure of possible brain dysfunction . EEG examination is often made with the assumption that an abnormal electrical pattern is a firm evidence of physical pathology. However, any clinical use of EEG brain mapping must be a direct extension of routine EEG testing (*American Psychiatric Association, 1993; and Chabot, et al, 1996*).

Aim of the work

The aim of this work is to find out :

1-The value of EEG and BEAM findings in the diagnosis of children with ADHD.

2-The prevalence rate of ADHD in school aged children and the factors affecting it.

3-The way through which we can help to minimize the incidence of ADHD in school children .

Literature Review

Literature Review

A- Attention Deficit Hyperactivity Disorder

I. Introduction

Children who present with inattention, impulsivity, and hyperactivity constitute a large portion of the behavior problems seen in pediatric patients ,*(Rostain, 1991)*. They are certainly one of the most time consuming problems in current pediatric practice ,*(Shaywitz and Shaywitz, 1984)*.

Activity level was one of the features found by *Thomas and Chess ,(1977)*, to distinguish between the behavioral styles of the children they studied. Some children are so active that they present major challenges to those caring for, and attempting to educate them. Some of these represent a normal distribution, but others may be suffering from a specific disorder, though the nature of this is not fully understood ,*(Barker, 1988)*.

"Hyperactivity" is a shorthand term for a cluster of complaints about childrens' behavior such as restlessness, inattentiveness, excitability, overactivity, impulsiveness, fidgetiness, disruptiveness and distractibility being the most prominent. Some regard hyperactivity as the cardinal feature of a widespread psychiatric disorder; some consider it as non-specific difficulties that are a part of conduct disorder in general; others as manifestations of a personality style that should not be seen as

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pathological or as a handicap to development. The controversy has been confused by the lack of agreement on diagnostic criteria,(Taylor, 1985).

In fact the other term "Overactivity" is used to mean an excessive quantity of movement. This is evidently a very nonspecific phenomenon; it might simply reflect high energy or agitation, and is not necessarily to be seen as evidence for any abnormality of development. While the term "hyperactivity" will be used in the description of a behavioral style of inattentiveness and impulsiveness, restlessness, of which overactivity is only one part of it. (Taylor, 1985).While such children may have been previously diagnosed as having Minimal Brain Dysfunction (MBD), or Hyperactive Child Syndrome, current evidence suggests that Attention Deficit Disorder (ADD), is a more appropriate and descriptive term,(Shaywitz aud Shaywitz, 1984).

Clements,(1966),stated that MBD is a learning and behavioral disability associated with deviation of function of the central nervous system in children with normal intelligence. He cited the most commonly noted characteristics of a child with MBD, as hyperactivity, perceptual motor impairment, emotional liability, general coordination deficits, disorders of attention, impulsivity and disorder of speech and hearing.

History of the disease:
A description compatible with the current knowledge of ADD appears in the German physician Heinrich Hoffmann's children's story "der Struwwelpeter", published in 1854, one of whose main characters ,(Der Zappelphilip), could not sit still at the table and showed many other features characteristic of ADD ,(Clements, 1966).

Children described as "restless, irritable, disobedient, emotionally quite unstable" were next reported by Hohman, (1922), who examined children after recovery from the acute phase of encephalitis. Kasanin ,(1929), and Biau ,(1936),

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described a similar picture in children following recovery from head injuries.

The autonomy of ADD as a syndrome was not suggested until 1937, when *Bradley* accidentally discovered the therapeutic usefulness of dextroamphetamine in calming children. *Bradley, (1957)*, had originally used the drug for its sympathomimetic effects to treat the headaches of children who had undergone pneumoencephalograms performed on almost all patients entering his hospital. To his surprise, the drug also calmed the behavior of numerous children who displayed those symptoms currently associated with ADD, thus focusing attention on a possible common etiology of the disorder. However, follow-up studies of these same children *(Bradley, 1957, Laufer and Denhoff ,1957)*, suggested that the syndrome of ADD constituted a separate clinical entity, leading to a more detailed investigation of its etiology, course and treatment.

Apart from these trends in medical research, a number of social researchers focused attention on the ADD child. Beginning in the late 1950's, public education services for children with behavior and learning difficulties began in North America and Europe *(Dunn, 1973)*.

Within the past decade, significant advances have been made in understanding ADD, developments in the diagnosis and management of this most common of all pediatric neuropsychiatric disorders. Perhaps the most important development has not been technological but rather conceptual. Systems of classifications incorporated both the establishment of categories *(taxonomy)*, and the development of rules for identification *(diagnosis)*, *(Shaywitz and Shaywitz, 1984)*.

Ross and Ross, *(1976)*, traced the origin of MBD to the early 1900's in which observations were made of brain-damaged adults and children who exhibited hyperactive, distractible and unmanageable behavior patterns. Similar behaviors that were observed in children without demonstrable brain damage were assumed to reflect underlying neuropathology, thus the term minimal brain damage or dysfunction was proposed.

Routh , (1978), stated that subsequent researches had provided empirical support for the validity of MBD as a medical syndrome, researches had focused on establishing the validity of hyperactivity as a behavioral syndrome by specifying the nature of behavioral problems shared by hyperactive children.

II. Classifications

The "Hyperkinetic Syndrome of Childhood" is the term used in _International Classification of Disease ,(9th Revision), (ICD-9), (WHO, 1979),_ for a condition of short attention span, distractibility and disinhibited, poorly organized,and poorly regulated extreme overactivity. _ICD-9_ subdivides hyperkinetic disorders into:

a) Simple disturbance of activity and attention in which the above symptoms are not accompanied by disturbance of conduct or delayed development of specific skills.

b)Hyperkinesis with developmental delay. In these cases, there are associated delays in the development of speech, motor skills, reading or other specific skills.

c)Hyperkinetic conduct disorder, in which there is an associated "marked conduct disturbance" but no developmental delay.

d)"Others" and "unspecified" categories, which are not defined. _(Barker, 1988)._

"Hyperkinetic Reaction of Childhood" is the term used in _Diagnostic Manual of Psychiatric Disorders ,(DMP-1, 1979),_ and this disorder is characterized by overactivity, restlessness, distractibility, especially in young children. This behavior usually diminishes in adolescence. If this behavior is caused by organic brain damage, it should be diagnosed under Non-Psychiatric Brain Syndrome ,_(Egyptian Psychiatric Association, 1979)._

The third edition of the _Diagnostic and Statistical Manual ,(DSM-III), ,(American Psychiatric Association, 1980), - APA -_ has replaced hyperactivity with "Attention Deficit Disorder" _(ADD),_ as the cardinal condition to which hyperactivity is a qualifier, so that one may diagnose ADD with and without hyperactivity ,_(Taylor, 1985)._

According to _DSM-III,_ three major subtypes have been identified within this overall clinical syndrome:

1),Attention Deficit Disorder with Hyperactivity ,(ADDH),characterized by inattention, impulsivity, and hyperactivity.

2),Attention Deficit Disorder without Hyperactivity ,(ADDNoH),
characterized by inattention and impulsivity but no hyperactivity.

3),Attention Deficit Disorder residual type, the term used to describe adolescents and young adults who at a younger age satisfied diagnostic criteria for ADDH, but who no longer exhibit significant hyperactivity ,*(Shaywitz and Shaywitz, 1984)*.

Diagnostic Criteria According to *DSM-III*

Inattention:

Operationally diagnosed when at least four of the following characterize the child:

1 Needs a calm, quiet atmosphere or is unable to work or concentrate.
2. Frequently asks to have things repeated.
3 Easily distracted.
4. Confuses details.
5 .Does not finish what he starts.
6. Hears but does not seem to listen.
7. Difficulty concentrating unless in a one-to-one structured situation.

Impulsivity:

Operationally diagnosed when at least three of the following occur:

1. Calls out in class, makes noises in class.
2. Is extremely excitable.
3 Has trouble waiting his turn .
4. Talks excessively.
5. Disrupts other children.

Hyperactivity:
Operationally diagnosed when at least three of the following characteristics are present:
1. Climbed onto cabinets and furniture.
2 .Was always "on the go", would run rather than walk.
3. Fidgets or squirms.
4. Does a thing in a loud and noisy way.
5 .Must always be doing something or he / she fidgets.
Other diagnostic criteria include:
1. Onset before age of 7 years.
2. Duration of at least 6 months ,*(Shaywitz aud Shaywitz, 1984)*.

The *American Psychiatric Association ,(APA), (1980),* distinguished attention deficit disorders with and without hyperactivity. The validity and practical usefulness of this distinction has, however, been questioned and in *DSM-III*-R ,*(Revised), ,(1987)*, they found only two categories:
* **Attention Deficit Hyperactivity Disorder ,(ADHD).**
* **Undifferentiated Attention Deficit Disorder.**
  Prior and Sanson ,(1986), suggested that even if one overlooks the problems of defining attention deficit ADDH lacks a clearly distinguishable symptomatology, i.e. sharing symptoms with conduct disorders and learning disabilities. Attention deficits are also found in autism, developmental delay, and epileptics on medication, and schizophrenics.
  The tenth edition of the International Classification of Diseases ,(ICD-10), was used to identify psychiatric problems in a provincial child-guidance clinical population. Categorization by degree of hyperactivity produced groups that differed in terms of variables that were independent of the diagnostic criteria. *ICD-10* was found to be a short, precise and manageable schedule which facilitated reliable categorization of a group of children with mixed disorders of conduct and hyperkinesis ,*(Yapa and Hague, 1991)*.
ICD-10 ,(1987), classification considers "Hyperkinetic disorder" as a group of disorders characterized by:
  a) An early onset.

b), The combination of overactive, poorly modulated behavior with marked inattention and lack of persistent task involvement.

c), Pervasiveness over situations and persistence over time of these behavioral characteristics.

In recent years, the use of the diagnostic term "Attention Deficit Disorder" for these syndromes has been promoted. It had not been used in *ICD-10* because:

1), It implies a knowledge of psychological processes that is not yet available.

2), It suggests the inclusion of anxious preoccupied or dreamy apathetic children whose problems are probably different in kind .However, it is clear that from the point of view of behavior, problems of inattention constitute a central feature of these hyperkinetic syndromes.

Cardinal features are impaired attention and overactivity. Both are necessary for the diagnosis, moreover, they should be evident in more than one situation ,(e.g. home, classroom, and clinic).

The associated features are not sufficient or necessary for the diagnosis but help to sustain it; disinhibition in social relationship; recklessness in situations involving some danger; and impulsive flouting of social rules ,(as shown by interrupting others' activities, or prematurely blurting out answers to questions before they have been completed or difficulty in waiting turns), are all characteristic of children with this disorder ,(ICD-10, 1987).

Symptoms of conduct disorder are neither exclusion nor inclusion criteria for the main diagnosis, but their presence or absence constitutes the basis for the main subdivision of the disorder. Hyperkinetic disorder is diagnosed with priority over conduct disorder when its criteria are met. However, milder degrees of overactivity and inattention are common in conduct disorder.

ICD-10, classifies "Hyperkinetic disorder" as:

Simple disturbance of activity and attention.

McMahon,(1984), stated that the ADD-H is compatible to the Hyperkinetic Reaction of Childhood in that both presume that the disorder is best described as consisting of an essential configuration of symptoms that includes: hyperactivity, impulsivity, concentration problems, and a variety of associated problems among which are aggressive behavior, poor peer relations, low frustration tolerance and non-responsiveness to discipline.

Nelson ,et al ,(1983), stated that the term ADD designates the central disturbances of children who had been labeled as suffering from hyperactivity, hyperkinesis, and minimal brain damage or minimal brain dysfunction. The word "hyperactivity" as a primary designation for this syndrome has been misleading objective measure. Many children with hyperactive syndrome have learning disabilities, on the other hand many children with learning disabilities do not have hyperactivity.

Rutter and Hersov ,(1987), stated that in spite of the lack of proven specificity, the concept of impaired attention has become central in modern descriptions of hyperactivity.

In *1993,* the *APA* introduced the *DSM-IV* with the diagnostic criteria of ADHD either with inattention or with hyperactivity-impulsivity. Both to be present before age of seven and in two or more different locations ,(home, school or work), and leading to clinical significant impairment ,(in social academic or occupational functioning), and not occurring as a part of other psychiatric disorders.

The authors of the <u>*DSM-IV,(1993),* applied the diagnosis of attention deficit hyperactivity disorder ,(ADHD),</u> to children and adults who consistently display certain characteristic behaviors over a period of time. The most common behaviors fall into two categories: inattention, and hyperactivity-impulsivity.

Inattention is diagnosed if at least 6 of the following symptoms persist for at least 6 months to a degree that the person is maladaptive and inconsistent with developmental level:

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1) Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
2) Often has difficulty sustaining attention in tasks or play activities.
3) Often does not seem to listen to what is being said to him/her.
4) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in workplace ,(not due to oppositional behavior or failure to understand instructions).
5) Often has difficulties organizing tasks and activities.
6) Often avoids, expresses reluctance about, or has difficulties engaging in tasks that require sustained mental effort ,(such as schoolwork or homework).
7) Often loses things necessary for tasks or activities ,(e.g. school assignments, pencils, books, tools or toys).
8) Often easily distracted by extraneous stimuli.
9) Often forgetful in daily activities

Hyperactivity-Impulsivity is diagnosed if at least 5 of the following symptoms have persisted for at least 6 months to a degree that the person is maladaptive and inconsistent with developmental level:

A) **hyperactivity**:
1) Often fidgets with hands or feet ,or squirms in 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 where it is inappropriate ,(in adolescents or adults, may be limited to subjective feelings of restlessness).
4) Often has difficulty playing or engaging in leisure activities quietly.
5) Is always "on the go" or acts as if "driven by a motor".
6), Often talks excessively.

B) **Impulsivity:**
7), Often blurts out answers to questions before the questions have been completed.
8), Often has difficulty waiting in lines or awaiting turn in games or group situations.
9), Often interrupts or intrudes on others ,(e.g. butts into others' conversations or games.),(DSM-IV, 1993).

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The authors of *DSM-IV,(1993)*, stressed on that some of the symptoms should be present before age of seven, and should be present in two or more settings ,(e.g. school, work and home). They stated that there must be clear evidence of clinically significant impairment in social, academic or occupational functioning, and does not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychiatric Disorders, and is not better accounted for by Mood Disorder, Anxiety Disorder, Dissociative Disorder or a Personality Disorder.

Sabatino and Vance ,(1994), stated that the comorbidity among 8 of the 14 *ADHD* descriptors of *DSM-III-R* had rendered it useless in accurately describing the presenting behaviors, and rendering it critically challenged. Data of their work support the changes in *DSM-IV* ,which differentiate predominantly inattentive type from hyperactive-impulsive type disorders, while altering and adding several new behavioral descriptors.

Lahey ,et al ,(1994), stated that the *DSM-IV* identified more impaired girls and preschool children than did *DSM-III-R*. They suggested that a threshold of 5 symptoms would optimize both identification of impaired patients and agreement with physicians.

Erk ,(1995), stated that the revised ADHD criteria in the *DSM-IV* are said to be an improvement over the previous criteria for ADD in terms of reliability, internal consistency, and clarity of association with functional impairment, so helping school counselor to recognize children's difficulties and enhance their outcomes by adjusting counseling services, interventions, and multidimensional treatment should optimize the prognosis for many children.

According to *Baumgaertel ,et al ,(1995)*, using comparative study for the diagnostic criteria of *DSM-III, DSM-III-R,* and *DSM-IV* it was shown that the overall prevalence of attention deficit disorders increased from 9.6% *(DSM-III)*, to 17.8% *(DSM-IV)*, due mostly to new cases identified as ADHD-AD (inattentive type), and to ADHD-HI (hyperactive-impulsive

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type*).   *DSM-IV*   ADHD subtypes   showed significant behavioral, academic, and demographic differences. Inattention in any subtype was associated with academic problems, while perceived behavior problems were associated with more than 80% of cases involving hyperactivity-impulsivity.

Okasha   ,*(1979)*,   defined ADD as a general motor hyperactivity; which may be either a constitutional peculiarity or a developmental delay. It is characterized by:

Slow development of intellect.

Violent motor unrest.

Marked distractibility.

Tireless exploration of their environment, and a tendency to put objects in their mouth and suck, bite or chew them.

Marked aggressiveness to brothers and sisters.

Learning difficulties.

Summary of Classifications of Hyperactivity

ICD-9	DMP-1	DSM-III	DSM-III-R	ICD-10	DSM-IV
International Classification of Diseases	Diagnostic Manual of Psychiatric Disorders	Diagnostic and Statistical Manual	Diagnostic and Statistical Manual	International Classification of Diseases	Diagnostic and Statistical Manual
WHO (1979)	Egyptian Psychiatric Association (1979)	American Psychiatric Association (1980)	American Psychiatric Association (1987)	WHO (1987)	American Psychiatric Association (1993)
"Hyperkinetic Syndrome of Childhood"	"Hyperkinetic Reaction of Childhood"	"Attention Deficit Disorder"	"Attention Deficit Disorder"	"Hyperkinetic Disorder"	"Attention Deficit / Hyperactivity Disorders"
Subdivisions: • Simple disturbance of activity and attention. • Hyperkinesis with developmental delay. • Hyperkinetic conduct disorder. • Unspecified categories.		• Attention Deficit Disorder with Hyperactivity. (ADD-H) • Attention Deficit Disorder without Hyperactivity. (ADD-NoH) • Attention Deficit Disorder residual type.	• Attention Deficit Hyperactivity Disorder. (ADHD) • Undifferentiated Attention Deficit Disorder.	• Simple disturbance of activity and attention. • Hyperkinetic conduct disorder. • Hyperkinetic disorder not otherwise specified.	• Attention Deficit Hyperactivity Disorder (inattentive type). (ADHD) • Attention Deficit Hyperactive Disorder (hyperactive-impulsive type. (ADHD-HI)

Conners, (1969) (Modified)   TABLE NO. 1

III. Epidemiology

a) Prevalence:

      Safer and Allen ,(1976), indicated that hyperactivity is the most common childhood pediatric disability accounting for 10% of children seen in pediatric clinics. *Lambert, ,et al ,(1978),* demonstrated that the prevalence of hyperactive children varied from 1.19% to 13% as function of the social system ,(medical - educational, or family), defining the disorder. They studied a sample of over 5000 school children and identified hyperactive children based on the following criteria:

1) Diagnosed by a physician.
2) Considered hyperactive by parents, and
3) Judged hyperactive by school authorities ,and all children were rated by their teachers on a scale measuring hyperactive behaviors. Only 1.9% of children were found to be hyperactive by agreement of home, school and physician. If identification by any one of the three sources mentioned above were accepted as the criterion of hyperactivity, the estimate would rise to 4.02%. Of children not identified as hyperactive by any of the three sources, 7.75% received teacher ratings equivalent to the hyperactive children identified by all three sources. Therefore, it was concluded that the maximum prevalence rate is above 13% when all possible means to define hyperactivity are employed.

Nelson ,et al, (1983), depending on the definition of ADHD, it is estimated to occur in 5-10% of school age children.

Kaplan and Sadock ,(1985) , stated that the figure more likely to be accepted is that of 3% of prepubertal elementary school children.

      Sandberg ,(1985), stated that hyperactivity is one of the child psychiatric conditions for which international

comparisons have produced something like a 20-fold difference in reported prevalence.

In England the diagnosis of hyperkinetic syndrome according to *ICD-9* formula is applied to only 1-2% of children of normal intelligence. In North America about 40% of those referred to child psychiatrists are given the diagnosis of ADHD which is the term currently preferred by the *APA*. Similar epidemiological surveys carried out on both sides of the Atlantic indicate that whereas the disorder is considered to occur in 4-5% of children in randomly selected populations in the USA, in England two children in the total population of 2199 ten-year olds were given the diagnosis. However, there is one point of agreement; the syndrome is less reported for girls. The prevalence of this disorder will vary greatly according to diagnostic criteria employed, and with the methods of investigation and population studied ,*(Parker, 1988)*.

Prendergast ,et al ,(1988), showed that, to examine the unequal prevalence of hyperactivity in the U.K. and the U.S.A., case histories of 6-11 year old boys were evaluated by British and American research teams as well as British and American clinician panels using both *ICD-9* and *DSM-III* criteria. Interrater agreement was acceptably high only for the specially trained research teams.

ICD-9 generated fewer diagnoses of hyperkinetic syndrome than did *DSM-III* of ADD with hyperactivity. The difference was greatest for U.K. clinicians. Diagnostic scheme and clinician training both contributed to the difference in reported rates.

Cohen ,et al ,(1994), found that the incidence of "pure" ADHD ,*(with no other psychopathology)*, was only 15.8% for children in special education ,*(using teacher rating only)*, while in children referred to a tertiary-care specialty clinic ranged from 11.9% ,*(using parent rating only)*, to 35.5% ,*(using teacher rating only)*.

Kanbayashi ,et al ,(1994), found that the prevalence rate using the 14 behavior items of *DSM-III -R* for ADHD was lower for 10-12 year old boys than for 7-9 year old boys, and for 7-9

year old girls than 4-6 year old girls. Out of the 7.7% of subjects meeting ADHD criteria, 41.5% had been identified by their teachers as having problems symptomatic of ADHD, and one-third had been reported by their parents as having conduct problems and emotional difficulties.

In Egypt, *Okasha ,(1988),* showed an epidemiological study of incidence of *0.5%.*

*The National Institute of Mental Health ,(NIMH), ,(1994),*declared that ADHD affects 3-5% of American Children, nearly 2 million.

b)Age of onset:

Nelson, ,et al ,(1983), stated that it is difficult among 2-4 years old children to identify those who will develop hyperactivity from those who are simply active.

*Kaplan and Sadock ,(1985), ,*stated that onset is usually by the age of 3, but the diagnosis is not generally made until later when the child is in school. The behavioral disturbances must be present for at least 6 months and appear before age of 7 for proper diagnosis.

Parker,(1988), stated the same age of onset for inattention, but hyperactivity may be manifested in infancy or even may be reported by the mother while the child was still in utero. The prevalence of ADHD increased with age, from *5.2%* in those aged 3-4 years, up to 29.2% in those aged 11-12 years *,(Bhatia, et al, 1991).*

c)Sex differences:

DSM-III ,(1980), reported a ratio of from 6:1 to 9:1 between boys and girls, while *Nelson ,et al ,(1983),* stated that the incidence of hyperactivity in boys is 4-6 times that in girls.

Sandberg ,(1985), reported a universal presentation of 3-9 times more in boys than in girls, depending on the age it occurs.

Lambert ,et al ,(1978), showed that boys were 6-8 times more frequently identified ,as hyperactive than were girls.

Rutter and Hersov ,(1987), stated that more boys than girls are affected, the ratio being about 3:1 or 4:1, though *DSM-III-R ,(1987),* noted a ratio of 6:1 to 9:1.

In Egypt, the disorder is ten times more common in boys than in girls *,(Okasha, 1988).*

NIMH,(1997), declared that 2-3 times more boys than girls are affected, at least one child in every classroom in the U.S.A.

d)Socio-economic status:

Paternite and Loney, (1980), noted that socio-economic status *(SES),* was not useful in predicting the presence of primary hyperactive symptoms such as overactivity and inattention. However, SES appears to be related to the security of the secondary symptoms associated with hyperactivity. Higher frequencies of aggression, low self-esteem, and delinquency, differentiate low SES hyperactive children from high SES hyperactives. It may be that a lack of parenting skills in low SES families results in over reaction to and poor management of the hyperactive child in such families.

For example, parental disturbances proved to be the best predictor of aggression as referral for hyperactive children.

Nelson ,et al ,(1983), stated that child-rearing practices and emotional difficulties in parental interactions are possible predisposing factors.

Sandberg ,(1985), stated that it is still necessary to consider the role of psychosocial factors. Hyperactivity and conduct disorder appear on present evidence to be in large part the result of social influences.

Rutter and Hersov ,(1987), showed also that aggressive, non-complaint behaviors are known to be related to family and social factors, to the extent that these behaviors are a part of "hyperactivity". Overactivity and inattention were prominent in children in deprived environments as those growing up in institutions. These disorders were improved for these children who were adopted out of the institution and taken into a normal family.

Parker ,(1980), stated that the family environment in contributing to these disorders are also unclear, but the focussing and sustaining of attention are probably to some extent learned skills. The learning processes are likely to be impaired in disorganized and unstable families, but there has been little research into exactly how this happens.

e) Birth order

Paternite and Loney ,(1980), included birth order in a multiple regression study of hyperactivity. Birth order was not found to be a significant contributing variant. Thus while it is plausible to expect a relationship between birth order and hyperactivity, and while some clinicians seem to note a tendency for firstborns to be more involved, data to support this are lacking.

Walker ,(1983), stated also that while some early authors commented on the tendency for hyperactive children to be firstborns, there have been few studies that have examined data having to do with birth order. Most researchers who have included this variable failed to find any significant relationship between birth order and hyperactivity.

Much of this considerable variation in epidemiology undoubtedly results from sampling populations of different ages, since hyperactivity is common in the school age population, but becomes less common with maturity. Still another explanation for the variation in the prevalence rate can be traced to how strict diagnostic criteria for ADHD were sought. Thus any interpretation of prevalence must take into consideration the age and sex of the population sample as well as the specific diagnostic criteria employed ,*(Shaywitz and Shaywitz, 1984)*.

IV.   Etiology of the Disorder

There have been many efforts to establish the etiology of hyperactivity but no single cause had been expected (*Taylor, 1985*).

1.Genetic Inheritance:

Good evidence now indicates that genetic factors play an important role in a large majority of cases of ADHD. The first suggestion for this relationship emerged from investigations demonstrating an increase incidence of a history of hyperactivity in the parents of hyperactive children compared to controls. Still other studies implicating a genetic influence are those demonstrating a high concordance for hyperactivity in monozygotic compared to dizygotic twins ,*(Shaywitz and Shaywitz ,1984).*

Monozygotic twins . *(MZ)*, showed a high correlation for activity level,while dizygotic twins *(DZ)*, showed no correlation. The results were interpreted as suggesting a substantial heritable component to activity level *(Willerman, 1973).*

Nobody disputes that alcoholism, antisocial personality, and hysteria are usually prevalent among the adult relatives of hyperactive children seen at psychiatric clinics ,*(Cantwell, 1972).*

Safer ,*(1973)*, stated that the siblings' of hyperactive children are also at risk of hyperactivity. The main reason for these family links is not securely established. The strategies of adoption studies and twin studies have been used to try to disentangle genetic from environmental transmission; in addition, hyperactive children and those with other conduct disorders have been compared to consider the specificity of the familial association.

Cunningham ,*et al* ,*(1975)*, used the more powerful strategy of comparing the adopted-away offspring of psychiatrically normal and disturbed parents. Not only did they

find that disorder was more common in the biological children of disturbed parents, but this tendency was particularly strong (and significant) for the children diagnosed as hyperkinetic.

Tizard and Hodges ,(1978), have described and followed the psychological problems of children growing up in institutes. Overactivity and inattention were prominent in those deprived children. These findings are persuasive evidence that the behaviors of hyperactivity can result from the lack of secure and stable family ties. Thus, indeed, it would be just as logical – and just as invalid - to regard hyperactivity as a "Minimal deprivation syndrome". Evidence leading same support for a genetic basis for hyperactivity is found in the fact that parents of hyperactive children have a higher prevalence of psychiatric disorders than those of non-hyperactive children.

Bohman ,et al ,(1982), also found that the biological children of disordered mothers showed higher levels of activity and inattention ,(as well as some other behavior problems), even when they were brought up in other families.

Twin studies support the notion of a genetic component. Activity level in the first year of life is more similar between MZ and DZ twins ,*(Rutter and Hersov, 1987)*.

Results of *Biederman, et al ,(1995)*, supported the validity of the adult diagnosis of ADHD and suggested that the adult form of this disorder may have stronger familial etiological risk factors than its pediatric form.

They supposed a familial nature of adult ADHD and that children of parents with adult ADHD are at higher risk for the disorder than those of parents of childhood-onset ADHD.

Faraone ,et al ,(1994), proved that the 2nd degree relatives of ADHD subjects are at high risk for ADHD. Consistent with the greater prevalence of ADHD among boys compared with girls, grandfathers were at greater risk than grandmothers, and uncles were at greater risk than aunts. Results support the usefulness of ascertaining information from 2nd degree relatives .

Comings and Comings ,(1994), and *Kurlan ,et al, (1994)*, suggested that Tourette's syndrome (T.S.), ADHD,

learning disorders, speech disorders and stuttering are all variant expressions of T.S. and share genes in common.

2. Minimal Brain Dysfunction:

Early, *Clement ,(1966)*, stated that MBD was the underlying cause of such behavior. Following this line of reasoning, extensive studies were conducted, examining everything from prenatal factors to electroencephalographic recording of children who were hyperactive. Unfortunately, much of the research conducted in this area suffered from inadequate research design, and few studies employed appropriate control groups.

Schmitt ,(1975), stated that the concept of MBD, and minimal brain damage, once popular, have now largely been discarded. At one time, "hyperkinetic syndrome" and "MBD" were almost synonymously used by many, but the relationship between minor brain damage abnormalities - as manifested by soft neurological signs - and hyperactivity has not been satisfactorily demonstrated. MBD - as a clinical entity - was probably a myth, and the use of the term is better avoided.

An objection by *Seidel ,et al.,(1975)*, to this theory studied that children with neurological disease or brain injury show no specific tendency to be hyperactive. They do certainly have higher rates of diagnosed hyperkinesis. Equally they have higher rates of underactivity and unduly persistent attention. Their problems are better seen as a nonspecific vulnerability to disorder than as any pathognomonic pattern.

Another objection to the view that hyperactivity is simply a form of MBD stems from research into heterogeneous groups of children referred for psychological help. No association had been demonstrated between measures of overactivity, impulsivity, and inattention on the one hand and neurological damage on the other ,*(Shaffer,et al., 1974; Sandberg ,et al, 1978)*.

The vast majority of children with hyperactivity or attention deficit, broadly defined, showed no evidence of brain

damage as detected by the usual tools of neurology. This is particularly the case in American series from which children with less than normal intelligence have been excluded. English series ,(e.g. that of *Ounsted, 1955)*, often do include many children with evidence of brain damage, epilepsy or low I.Q. simply because of the nature of the population studied ,in *Ounsted's* series, children were attending a specialized epilepsy service. Analysis of the records of children seen at a psychiatric hospital did show that the rare, conduct disorder diagnosis of hyperkinetic syndrome was applied more often to retarded children ,*(Taylor, 1980)*.

Rutter and Hersov ,(1987), stated also that there is no good evidence that hyperactivity is specially associated with brain damage, and most brain damaged children are not hyperactives.

Shaywitz and Shaywitz ,(1984), stated that the majority of children with hyperactivity or attention deficit show no evidence of brain damage as detected by the usual tools of investigation. As, none of these procedures, including computerized tomography ,*(C.T.)*, has provided convincing evidence of any anatomic aberration in children with ADD.

However , magnetic resonance imaging *(MRI)*, studies suggest that compared to non-disabled controls, ADHD children had a smaller corpus callosum, particularly in the region of the genu and splenium, and in the area just anterior to splenium. Interhemispheric fibers in those regions interconnect the left and right frontal, occipital, parietal, and posterior temporal regions. *Hynd, et al, (1991)*, results suggested that fine differences may exist in the brains of children with ADHD and that deviations in normal corticognesis may underline the behavioral manifestations of this disorder.

Carter, et al ,(1995), stated that children with ADHD, showed an asymmetrical performance deficit characterized by a less controlled *(endogenous)*, attentional orienting left visual field targets. They concluded that this asymmetry might be related to diminished right hemispheric frontal-striatal catecholamine activity.

Srandburg ,et al. ,(1996), showed that ADHD children have no longer reaction time than normal children, and concluded that they have a diminished late frontal reaction suggestive of reduced involvement in post-decisional processing.

Johnstone and Barry ,(1996), proposed that ADHD children - relative to controls - utilize an additional cognitive process while processing task-relevant stimuli. This process is more frontally disturbed and may reflect an attentional compensation mechanism in ADHD children.

3. Abnormalities of Neurophysiological Function

Hyperactive children are not under-aroused, in the sense of showing low tonic levels of autonomic nervous system activity ,*(Taylor, 1981)*.

Sattersfield ,et al ,(1994a, b), stated that results from cardiac and electrodermal variables indicated that although basal *(*tonic*)* arousal level appear to be normal, measures of responsivity *(*phasic*)* arousal in hyperactive children are lower than normal.

Hyperactive children are less responsive to stimuli than normal controls ,*(Zahn ,et al, 1975)*. This selective unresponsiveness is similar to that found in adults with psychopathic personality disorder ,*(Hare, 1980)*.

Abnormal EEGs are probably no more common in the hyperactive if they are carefully matched ,*(Werry ,et al, 1972)*, but the hyperactive may show a shift towards lower frequencies and towards less power in the beta but more in the alpha frequencies ,*(Grunewald-Zuberbier. ,et al, 1975)*.

Two areas of uncertainty make it premature to use these interesting findings as biological validation of a common syndrome of hyperactivity. **Firstly**; nobody knows what these deviations of neurophysiology mean. It is possible that they are simply reflections of impaired attention - indeed they have already been considered in this way above. **The second**; is about the specificity of the finding. Is hyporesponsiveness found in hyperactive because it is associated with hyperactivity? Or is it

associated with one of the other clinical features that are found in hyperactive children - such as aggressive behaviors, a relatively low intelligence quotient (I.Q.), learning disorders, and low motivation to do psychological tests? Insufficient processing of information and diminished physiological responsiveness have been constant findings of studies of children with specific learning disorder ,(Sheer, 1976; Fuller, 1977; and John ,et al, 1977).

It is therefore, possible that physiological work validated the concept of cognitive impairment rather than of hyperactivity. Furthermore, research will be needed before psychophysiological measures are established as a useful tool in routine clinical diagnosis ,(Rutter and Hersov, 1987).

Satterfield ,et al ,(1988), found constant abnormal findings in Brain Electrical Activity Mapping (BEAM), of auditory event-related evoked potential (Aud. EP), suggesting an abnormality in information processing in the frontal lobes of children with ADHD. These findings are consistent with the work of Satterfield ,(1990), who found metabolic abnormalities in the frontal lobes of adults who were formerly hyperactive children, and with blood-flow studies of Lou ,(1990), who found abnormalities in the frontal lobes of children with ADHD.

Lastochkina and Puckinskaia ,(1991), noted that children with ADHD, were shown to have the weakening of the interhemispheric correlations by the alpha rhythm together with their potentiation according to the beta rhythm, a decrease of positive interhemispheric asymmetries, and a great number of negative asymmetries. The same conclusion was stated by Malone ,et al ,(1994a).

Giedd ,et al ,(1994), and Quay ,(1997), found that the corpus callosum (C.C.), is affected in two anterior regions, the rostrum and the rostral body and had significantly smaller areas in ADHD patients as measured by using magnetic resonance images (MRI).

Kressling ,et al ,(1994), found antineuronal antibodies to caudate nucleus cells and putamen cells in children

with ADHD, suggesting a mechanism for the altration in the size of components of the basal ganglia seen in MRI.

Chabot and Serfontein,(1996), found two neurophysiological subtypes in quantitative electroencephalogram (QEEG), in ADHD children. The first, showed varying degrees of EEG slowing especially in frontal regions, whereas the second showed an increase in EEG activity, especially in frontal regions. In conjunction with recent MRI, positron emission tomography (PET), and regional cerebral blood-flow studies, these results indicated neurophysiological dysfunction within the cortical and subcortical structures that serve the frontal/striatal system. They suggested either hypo- or hyperarousal of these structures.

Jonkman ,et al ,(1997), hypothesized that in ADHD children in both auditory and visual task, there is a deficit in the activation process that might be caused by disturbance in other aspects of the attention process.

Jones ,et al ,(1994), reported that hyperactive children process information in a more diffuse manner than controls. They suggested that abnormalities in recall strategies rather than information processing, may explain incidental learning disabilities.

Scientists at NIMH hoped to compare the use of glucose and activity level in mild and severe cases of ADHD. They were trying to discover why some medication work better than others in treating ADHD, and if they can increase activity in certain parts of the brain *(NIMH, 1997)*.

4. Hyperactivity as Dysfunction of Arousal:

A number of influential theories and researches have argued that the central features of hyperactivity disorder are related to the physiological arousal characteristics of these children. Most current theories of hyperkinesis can be divided into those that posit either physiological overarousal or underarousal as the central explanatory mechanism. Early researchers tended to favor the overarousal positive. Some influential theoretical position was that hyperactivity resulted from brain damage, while interfered with the child's ability to screen out irrelevant input and to organize relevant stimulation.

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The effect was a flood of stimulation that resulted in excessive and poorly organized activity. An alternative theory, for which exists a certain amount of empirical support, suggests that hyperactivity may be attributable to underarousal rather than overarousal, with activity serving as a stimulus generating function. Major proponents of this position rely upon physiological studies that suggest that hyperactive children are low in various indices of autonomic and cortical arousal. Within this orientation, it has been argued that the excessive and seemingly disorganized motor activity seen clinically is associated with low autonomic excitation and reflects attempts by these children to increase levels of sensory input ,(McMahon, 1984).

Brocke ,et al ,(1987), conducted a study using 35 children with hyperkinetic syndrome and 33 age-matched controls. After a relaxation period, specimens were administered 3 standard performance tasks, including simple reaction time. Under different conditions, a spontaneous EEG was derived precentrally and occipitally. Significant differences in cortical arousal behavior were apparent in terms of hyperkinetic syndrome -specific deviations in adjustment of tonic cortical arousal to varying situation and task requirements ,(arousal adjustment deficit [AAD]). This suggests that an ADD may be a second central form of hyperkinetic syndrome - specific arousal anomaly besides the well-supported deficit in arousability ,(i.e. phasic arousal anomaly).

Magnuson and Klinteberg ,(1987), investigated a representative group of children from a community in Sweden to discover the relationship between early behavioral indicators of a possible underlying vulnerability and for hyperactivity and later social and /or pervasive conduct problems. Measures of sympathetic-adrenal activity from a normal and a stressful situation ,(mental arithmetic test), as well as teacher ratings of school behavior were analyzed for 86 boys when they were aged 10-13 years. Adrenaline excretion was significantly negatively correlated with ratings of aggressiveness, motor restlessness and concentration difficulties and with the sum of the latter two, uses

as an indicator of hyperactive behavior in the stressful situation. The association persisted when aggressiveness was partialed out from hyperactive behavior. Specimen identified as hyperactive differed significantly from nonhyperactive in adrenaline excretion in both situations.

5. Biochemical causes:

a) Serotonin:

There are some studies reporting a decreased platelet concentration of serotonin in hyperactive children compared to the control group *(Bhagauan ,et al, 1975)*. Others reported a decrease in 5-hydroxyindole acetic acid *(5-HIAA)*, - the metabolite of serotonin - levels in whole blood *(Shaywitz. ,et al, 1977)*.

However another study *(Ferguson ,et al, 1981)*, reported no difference between hyperactive and normal children in the level of free and total whole blood serotonin concentrations.

Shetty and Chase ,(1976), measured serotonin metabolites in the cerebro-spinal fluid *(CSF)*, they found no difference between hyperactive and control children. Thus, several measures of peripheral serotonin function, and measures of CSF metabolites that better reflect central function, indicate that seronergic systems may be normal in attention deficit disorder children. *Halperin, et al, (1994)*, suggested a difference in central 5 - hydroxytryptamine *(5-HT)*, between aggressive and nonaggressive children with ADHD.

b) Norepinepherine and Dopamine

The earliest study in which levels of norepinephrine and dopamine metabolites were measured in the urine of ADD children reported no difference from controls. Other studies ,however, suggested alterations of norepinephrine and dopamine systems in ADD *(Raskin ,et al, 1984)*.

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In two studies performed by *Shekin and his associates* *(1977 and 1979)*, baseline levels of urinary homo-vanillic acid *(HVA)*, 3 -methoxy-4-hyroxy-phenylglycol *(MHPG)* *,(metabolites of dopamine)*, were compared in hyperactive and control children. Results indicated that, although levels of urinary HVA did not differ between the two groups, urinary concentrations of MHPG were significantly lower in the clinically diagnosed as ADHD.

The first study that measured basal levels of HVA concentrations in spinal fluid did not reveal any difference in clinically diagnosed children. This study, however, did not use probenecid loading, and all children were not free from medications *(Sleator and Ullmann, 1981)*.

In a study performed by *Shaywitz ,et al ,(1977)*, concentrations of neurotransmitter metabolites were measured in lumbar CSF two hours following oral probenecid administration. Results showed that concentrations of HVA per unit of probenecid were significantly lower in ADHD children than in controls. These results suggested an alteration in brain dopamine in children with ADHD.

Studies on blood from children with ADHD had shown lower serotonin *,(Coleman, 1971)*, or no difference in serotonin *,(Ferguson ,et al, 1981)*, higher mean of dopamine-betahydroxylase *(DBH)*, enzyme in children with ADHD *(Rapoport,el al, 1974)*, and either lower *(Shekin ,et al, 1979)*, or higher *(Khan and Dekermenjian, 1981)*, urinary MHPG. These studies suggested that ADHD may be a heterogeneous illness made up of several subtypes.

Satterfield ,et al ,(1990), suggested that the abnormality of event-related potential *(ERP)* waves that is found in ADHD boys to attend target stimuli may be due in part to insufficient noradrenergic activity normally triggered by attended task-relevant or novel stimuli.

Levy ,(1991), hypothesized that an underlying biochemical abnormality and a disorder of reinforcement is the primary deficit in ADHD children in terms of a disorder of

polysynaptic dopaminergic circuits, between prefrontal and striate centers.

Results of *Malone ,et al,(1994 b)*, suggested a link between ADHD and abnormalities of hemispheric pressing in the form of:

(1) a neurotransmitter imbalance between norepinephrine and dopamine and *(2)* an asymmetric neural control system that links the dopamine pathways to left hemispheric processing and links the noradrenergic pathways to right hemispheric processing. It appears that ADHD may involve a bihemispheric dysfunction characterized by reduced dopamine and excessive noradrenergic functioning. And so favorable medication effects may be mediated by a restoration in the neurotransmitter balance and by increased control over the allocation of attentional resources between hemispheres.

c) Role of Enzymes:

Shekin ,et al ,(1982), suggested an association between low platelet monoamine-oxidase *(MAO)* activity and the behavioral state of hyperactivity, short attention span, and impulsivity. After the hyperactive children were treated with d-amphetamine for 2 weeks, their platelet MAO levels were found to be equal to those of the control children.

The enzyme DBH is released form neurons and can be measured in blood and urine; abnormal levels were noted in children with minor congenital anomalies *(Rapoport ,et al, 1974)*, and in young adults with ADHD *(Spring ,et al, 1997)*. This may prove a useful means of monitoring the disorder. However, the uncertainty of the replicability of these findings, their meaning and their specificity again make it impossible to conclude that the syndrome has been biologically validated (*Taylor, 1985 ; and Rutler and Hersov, 1987).*

Parker ,(1988), showed also that there are biochemical theories concerning the etiology of hyperactivity, which may be associated with perturbations in brain-neurotransmitter function.

6. Other Physical Illness

Some physical illnesses are probably associated with overactivity more than with other patterns of problems, notably temporal lobe epilepsy, but also thyrotoxicosis and Sydenham's chorea. They are however rare causes (*Taylor, 1985*).

ADHD children are often reported to display signs of allergies to various substances and/or atopic symptoms ,(i.e. atopic eczema, hay fever, or asthma). Parental hyperactivity ratings revealed a significantly higher frequency of ADD symptoms in the atopic group, and laboratory tests sensitive to attentional capacity showed poor performance in the atopic children, similar to the hyperactive children. *Roth, et al ,(1991)*, assumes that there is a common predisposing factor for both atopy and ADHD children.

Hagerman and Falkenstein ,(1987), demonstrated a correlation between hyperactivity and recurrent otitis media beyond what is generally expected in large populations. Ninety-four percent of the patients that were diagnosed and medicated for hyperactivity had three or more otitis infections, and 69% had more than 10 infections.

There had been little work on auditory attention in hyperactive children, and *Prior ,et al ,(1984)*, dichotic listening studies have not elicited clear deficits. It is possible that attention processes in the auditory modality are not severely handicapped. Auditory attention may develop earlier and may not be as difficult as a sustained visual task that would seem to require more self-motivated control of attention that a structured, predictable pace auditory-verbal task delivered through headphones *(Prior ,et al, 1984)*.

Lufi and Cohen ,(1985), showed clearly that the child with ADHD is inferior to children with emotional problems in short-term visual memory measured by Wechsler-Intelligence

Scale for Children-Revised *(WISC-R)*, which is an important cognitive ability in reading, writing and arithmetic. Further research is needed to substantiate this interpretation and to determine to what extent coding recall can be a viable tool for diagnosis of ADHD. If it is true that the ADD children tend to mediate visual input specially rather than verbally, this would have a major impact on therapeutic intervention with this group *,(Lufi and Cohen, 1985)*.

Some studies have found an association between the number of minor physical anomalies of hands, feet, head, ears, face, and mouth and behavior or learning problems originating in early childhood *,(Quinn and Rapoport, 1974)*. These anomalies are found in children with Down's syndrome, but also are associated with other genetic defects, and also occur within presumably normal populations *,(Smith, 1970)*.

Ferguson ,et al ,(1981), found a significant correlation between minor physical anomalies and teacher and parent ratings of hyperactivity and conduct problems for a group of 79 elementary school boys.

These studies suggest that, at least for males, there is a congenital contribution to those types of behavior. These relationships were chiefly obtained with "normal" preschool populations, and their clinical usefulless has not been demonstrated *,(Rapaport and Ferguson, 1981)*.

Cuffe ,et al ,(1994), reported cases of ADHD and posttraumatic stress disorder in children. This combination is explained by: *,(1)*, children with ADHD are at higher risk for trauma due to impulsivity and dangerous behavior and due to parents who may have a genetic predisposition for impairment of their own impulse control; *,(2)*, hyperarousal induced by severe trauma and manifested by hypervigilance and poor concentration may impair attention to create an ADHD-like syndrome.

Kaneko ,et al ,(1993), suggested abnormalities in the hypothalamic – pituitary – adrenal axis function in some children with ADHD, especially those exhibiting severe hyperactivity.

Yordanova ,et al ,(1996), stated that according to the amplitude, topography and correlation criteria, tic disorder with

hyperactivity in patients of ADHD does not appear to be a subgroup of hyperactivity but seems more similar to pure tic disorder. *Yordanova ,et al ,(1997),* concluded that in these patients a psychopathological classification concerning the comorbidity of tic disorder and ADHD was only partially supported by the observed pattern of psychophysiological results.

Millichap ,(1997), reported 3 childhood cases of temporal lobe arachnoid cyst in association with ADHD. Cysts appeared to be developmental and not related to trauma or hemorrhage. The relation of arachnoid cyst to headache in these cases is not definitely determined, but a causal association with ADHD is considered plausible because of coincidental learning and language disabilities that might be explained by temporal lobe and sylvian region pathology. This appears to be the first report of the association of ADHD, and temporal fossa arachnoid cyst in children.

Henriksen ,(1990), stated that in ADHD children with epilepsy the subclinical epileptiform discharges could adversely affect the child's performance. Also psychological problems in epilepsy patients as anti-epileptic side effects may exaggerate learning problems.

7. Psychological Causes:

Tizard and Hodges ,(1978), have described and followed the psychological problems of children growing up in institutions. Overactivity and inattention were prominent in these deprived children. These findings are persuasive evidence that the behaviors of hyperactivity can result from the lack of secure and stable family ties.

Sandberg ,et al ,(1980), noted a high rate of depressive feelings in mothers whose children (in normal schools) had been identified by questionnaires as hyperactive.

Clinically, there can be no doubt that some children are presented as overactive by parents intolerant of a normal degree of childish energy. Depression, obsession, and alcoholism

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can have this effect, and of course they can also impair parental coping as well as causing misperception ,*(Taylor, 1985)*.

Abmayr and Day ,*(1994)*, showed that parents of ADHD children reported higher frequencies of sleep disturbances in the retrospective phase of their study.

8. Dietary Factors:

The history of the use of diet in children's behavior problems, specifically hyperactivity, includes a series of dietary therapeutic modalities suggested as successful treatment for children ,*(Varley, 1984)*.

The megavitamins therapy was first popularized by *Linus Pauling ,(1968)*, when he suggested that doses much higher than the recommended daily allowance of vitamin C, the fat-soluble vitamins ,*(A, D, E, K)*, thiamin, and B6 can result in general improvement in the health of children.

In review of clinical trials *Cott ,(1971)*, reported positive outcome in the children with learning and behavior disorders treated by megavitamins. However no differences were found between megavitamin therapy and placebo in a group of children with MBD ,*(Arnold ,et al, 1978)*.

In *1976, The American Academy of Pediatrics* reviewed these recommendations and produced a position paper negating the role of megavitamins on the behavior of children and adults as well.

Food allergies have been offered as a potential explanation of symptoms similar to those found in children with ADHD, namely, inattentiveness, impulsivity, and hyperactivity. Several substances, including orange juice and most frequently milk, have been suggested ,*(Kittler, 1970)*.

Feingold ,(1975a), suggested that both naturally and artificially occurring salicylates such as those found in many different food products and food additives and food colorings negatively affect the behavior of children. He reported that as many as *50%* of children diagnosed as hyperactive will

respond dramatically to his elimination diet. He initially theorized that these substances produced their negative effect through allergic mechanisms; more recently some form of direct toxicity has been postulated. The Feingold diet is complicated and usually requires the family to change its dietary practices as a unit *,(Varley, 1984)*.

Feingold had observed that if a child deviates from his elimination diet *,(i.e. one free of food containing synthetic flavors, colors, and salicylates)*, ingestion of even minute amount of the prescribed food *,(e.g. a cookie or soft drink)*, "causes a recurrence of the complete behavioral pattern within 2-4 hours which persists for 1-4 days" *,(Feingold, 1975b)*.

The diet must be vigorously maintained, and forbids many of the delicacies of childhood foods. Soft drinks, sweets, ice cream, sausage, hamburger, breakfast cereal and fish fingers are all prescribed. The result can be a whole one diet, however, it is not easy to maintain and tends to isolate children from their fellows, and it can sometimes appear to a child to be a prolonged punishment. These disadvantages would matter little if the effect was clear, but it is not *(National Institute of Health, 1982)*.

Trials of the experimental diet against a control diet suggest that the effect is small *,(Conners ,et al, 1976)*, or absent *,(Harley ,et al, 1978b)*. Nevertheless, some children do respond well to the diet, when such responders are then challenged with test doses of additives and also with an indistinguishable placebo, they do not get worse *(Mattes and Gittelman, 1981)*.

Parental and teacher ratings, classroom behavior observation and neurophysiological test scores obtained during baseline, placebo, and challenge conditions after strict elimination Feingold diet for 11 weeks, were not found to be adversely affected by the artificial color challenge materials *,(Harley ,et al,1978a)*.

Very high doses of additives may lead to poor performance on a paired associated learning task *,(Swanson and Kinsbourne, 1980)*, and these may be a transient, fleeting effect

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upon behavior ,*(Goyette ,et al, 1978)*, but no major effect on hyperactivity has been shown in group studies.

On the other hand, while the evidence argued against Feingold's hypothesis as being a pervasive cause of hyperactivity, challenge tests with food dyes demonstrate that their ingestion can cause behavioral symptoms characteristic of hyperactivity, and suggest that the challenge dose of dyes tested in previous studies were too low ,*(Swanson and Kinsbourne, 1980)*. In addition, controlled studies by *Trites ,et al. ,(1980)*, have provided inconsistent evidence of specific food allergies being a basis for hyperactivity, and such positive results as do exist implicate only the broad hyperactivity - conduct disorder entity.

The initial claims that *50%* of children with hyperactivity will improve if they are placed on a special diet free of food colorings have not been substantiated by several large prospective investigations. After an initial period of enthusiasm for diet, it is only rarely that a sustained benefit attributable to the special diet is observed ,*(Shaywitz and Shaywitz, 1984)*.

Wiess ,(1982), reviewed the literature and reinterpreted the conclusions of several studies, concluding that Feingold, at least to an extent was correct. What does emerge, however, from several well controlled studies, is a picture which suggests that in the population of children diagnosed with ADHD, there is evidence of behavioral improvement on the Feingold diet in perhaps as many as 5-10% of children ,*(Varley, 1984)*.

Egger ,et al ,(1985), concluded, "the suggestion that diet may contribute to behavior disorders in children must be taken seriously."

Later , sugar has been the substance invoked as creating behavioral problems in children. It is felt that, in particular, sugar present in the refined state of sucrose is likely to produce behavioral toxicity. It is unclear whether the suggested behavioral toxicity is supposed to represent some form of reactive hypoglycemia, or whether there may be some direct toxic effect of sugar on the behavior of children ,*(Varley, 1984)*.

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Prinz ,et al ,(1980), found that children with "hyperactivity" were found to have a positive correlation of restlessness and destructive aggressive behavior and percentage consumption of sugar products in diet. There was no attempt to change the behavior of children through dietary manipulations.

In a large study in New York, 261 children referred to a clinic for behavioral disorders, 74% were found to have abnormal glucose tolerance. "Low flat curves" accounted for *50%* of the abnormal results *,(Langseth and Dowd, 1978).*

Many parents of ADHD children believe that hypoglycemia plays a role in their child's symptoms. However, there is no evidence to support such a notion. Similarly there does not seem to be any relationship between hypoglycemia and symptoms of ADHD *,(Shaywitz and Shaywitz, 1984).*

However no study demonstrates that controlling sucrose intake significantly affect the behavior of children *,(Varley,1984).*

Colquhon ,(1994), found that Feingold diet program improves not only the hyperactivity of children with ADHD, but also their general health especially when adding nutrients such as vitamins, minerals, and essential fatty acids.

Greenblatt ,et al ,(1994), stated that the link between folate and neurotransmitter metabolism is of potential relevance to neuropsychiatric disorders occurring in childhood, changing synaptic relations among neurotransmitter systems are presumed to be associated with the age-related emergence of specific behaviors such as autism, ADHD, and Tourette syndrome.

9. Perinatal causes:

Morrison and Stewart ,(1971), first identified a possible relationship between hyperactivity and alcoholism, finding that a higher proportion of fathers *,(20%),* and mothers *,(5%),* of hyperactive children were alcoholics, compared to 10% of fathers and none of mothers of the controls.

There is an increasing awareness about possible negative effects of alcohol exposure on the human fetus. In *1973, Jones and Smith* suggested the name "Fetal Alcohol Syndrome" (FAS), for a characteristic pattern of abnormalities found in the offspring of alcoholic mothers. These abnormalities consist of varying degrees of prenatal and postnatal growth and mental retardation and a number of congenital anomalies most frequently of the craniofacial area, heart, and limbs.

Interest in focusing not only on FAS, but also on children with less obvious forms of FAS, i.e. those without congenital anomalies but with learning disabilities and hyperactivity. Links had been observed between parental alcoholism and both learning disabilities and hyperactivity in children, *(Shaywitz, 1978)*.

Nichols and Chen, (1981), found that maternal smoking or proteinuria during pregnancy had some relation *to* hyperactivity or low academic achievement. But any suspected abnormality in the neonatal period only increased the likelihood of ADHD at age of seven from 2 to 5 percent. In contrast, W*erner and Smith, (1977),* showed that poor social and emotional support systems increased the likelihood of pathology by 200% to 400%.

Pasamanick and Knoblock ,(1960), suggested that prenatal and perinatal risk factors, such as bleeding during pregnancy or low birth weight, showed significant relationship with learning and behavioral problems when the child was of school age.

The results of *Fiana and Joelsson ,(1979),* suggested that besides other etiological factors, maturity at birth as well as trauma and possibly even period of asphyxia during delivery, play a significant role for the occurrence of ADHD.

It ·was found that among "prematurely delivered children", the frequency of ADHD was 20% and was similarly distributed between children delivered breech and in vertex presentation. Among " term delivered children", the average frequency of ADHD in children born in breech presentation was 14%, while in those born in vertex presentation the corresponding figure was 2%. It might therefore be concluded that with correct

management of the delivery, especially in breech presentation, the obstetrician could contribute considerably to the prevention of ADHD ,*(Fiana and Joelsson, 1979)*.

Rapoport and Ferguson ,(1981), concluded that there are some *(*extremely weak*)* relationships between childhood behavioral disturbances and these factors, but the relationship appears to be non-specific with regard to behavioral profile.

Njiokiktjien ,et al ,(1994), found that the size of the corpus callosum *(CC)*, varies due to the likely presence of genetic influences or of adverse perinatal events. Children with learning disabilities and with perinatal adverse events had a smaller CC than the control group, suggesting CC damage, and so inhibit the interhemispheric function and inhibit the establishment of cerebral dominance. This may contribute to the pathophysiological mechanisms that give rise to ADHD.

McIntosh ,et al ,(1995), stated that by using the maternal perinatal information, prediction of children with ADHD could elevate from *52%* to 67% of the cases in their study.

Researchers at *NIMH ,(1997)*, stated that a mother's use of cigarettes, alcohol or other drugs during pregnancy may have damaging effects on the fetus's developing brain or may distort the developing nerve cells. FAS is an example, due to heavy alcohol use during pregnancy, leading to children with hyperactivity, inattention, and impulsivity.

They also found that drugs such as cocaine - including the smokable form known as crack - seem to affect the normal development of brain receptors ,*(*that transmit signals from skin, eyes, and ears and control responses*)*, leading to ADHD.

10. Lead Exposure

NIMH ,(1997), stated that toxins in the environment might also disrupt brain development or brain processes, which may lead to ADHD. Lead is one of such toxins

that are found in dust, soil, and flaking paint in areas where leaded gasoline and paint were once used. It is also present in some water pipes. Some animal studies suggested that children exposed to lead my develop symptoms associated with ADHD, but only a few cases have actually been found.

Lin-Fu,(1973b), found that in young animals nutritional inadequacies may potentiate the toxic effect of lead, that is of particular importance since children at risk for lead poisoning are often also at risk for inadequate nutrition. There is a considerable vulnerability of the immature organs of the fetus and young children to the toxic effect of lead. In children, the appearance of clinical toxicity is at somewhat lower blood levels than those described in adults.

Therefore, infants and young children are particularly susceptible to lead toxicity because of their developing central nervous system, smaller body mass, increased intestinal absorption rate, reduced elimination rate, tendency to put objects and dirt in their mouths, *(Rockway ,et al, 1984).*

Clinically, increased lead burden in children includes gastrointestinal symptoms ,*(*abdominal pain, diarrhea, vomiting, constipation, and loss of appetite*),* and CNS symptoms ,*(*clumsiness, irritability, increase in sleepiness, drowsiness, convulsions and headaches*).* The main non-specific nature of these complaints casts doubt on the validity of attributing them to an increased lead burden ,*(Pueschel ,et al, 1972).* The mean I.Q. of these children was 86, with standard deviation of 15 ,*(*the majority are in the range of low normal intelligence*).*

Sudden onset of encephalopathy without previous symptoms is not infrequent in older children and unusual in adults. On the other hand, the blue line that is an indicator of lead exposure in adults is not seen in children. Peripheral nerve palsies that are characteristic of adult plumbism are rarely encountered in children. Severe colic with board-like rigidity of abdomen, typical of lead poisoning in adults, is seldom seen in children who tend to have vague and less acute abdominal pains.

Lead is not only much more toxic to young and pre-adolescents than to adults, but also the effects produced by the metal are general in the young and local in the adults*(Lin-Fu,1972)*.

The early signs of lead poisoning among children are frequent crying for no apparent reason, fearfulness, loss of affection, refusal to play, inattention and developmental regression generally precede the onset of the more classic manifestations of encephalopathy. Many children are considered to have behavioral problems before the diagnosis of lead poisoning is entertained. It is possible that many who do not progress to the stage of frank encephalopathy are never diagnosed and treated, and eventually appear in schools with learning disabilities, hyperkinetic syndrome, and other behavioral problems *,(Lin-Fu, 1973a)*.

A large number of lead-exposed children had normal intelligence but failed in one or more tests related to some areas. The most significant difference between exposed and non-exposed groups was found in motor and behavioral areas *,(Burde and Choate, 1972)*.

According to *Scanlon ,(1971),* the mean blood lead values of rural children was 12.5 µg % whereas that of urban children was 25.1 µg % , this difference is in agreement with findings in adults. *Maskovac and Goldstein ,(1988),* stated that, there is no threshold for adverse effects from the universal exposure to inorganic lead.The mean blood level of lead in the U.S. has ranged from 9.2 to 16.0 µg % *,(Bertoni and Sprenkle, 1991)*.

11. Metal Imbalance:

Barlaw and Sidani ,(1986), examined a hair sample of hyperactive children for 20 elements and found that only Zinc, Lead, and Cadmium showed significant difference, namely, low

zinc in hyperactive males and females, and raised lead and cadmium in hyperactive males.

While persons exhibiting a zinc deficiency are more susceptible to cadmium and possibly lead toxicity, hyperactive children showed some degree of zinc deficiency , *(Bunday and Colguhoun, 1981)*.

The incidence of reading disorders and hyperactivity are relatively more frequent than other disorders in outcomes of pregnancy with hypertension , *(Brophy, 1986)*, while zinc deficiency in pregnancy and pre-eclampsia may contribute to production of the hyperactive syndrome in offspring. Low zinc occurs consistently in severe pre-eclampsia , *(Hagn and Funchs, 1974; Brophy ,et al , 1985)*.

Collipp ,et al ,(1982), found a significantly higher level of manganese in hair of hyperactive children than the control group. *Barlow ,(1983)*, confirmed the same finding and related it to the possible use of infant formulas, many of which do contain high manganese levels , *(*up to one hundred times the level found in human milk*)*. These findings suggest that the high concentrations of manganese in infant formula may not be as safe as had been assumed , *(Cawte, 1985)*.

Barlaw ,(1983), and *Howard ,(1984)*, reported a small increase in serum aluminum levels in hyperactive and memory loss-related problems, and suggested that aluminum may be a factor in hyperactivity.

Reviewing all this literature for the etiology of restlessness, hyperactivity, agitation, akathisia, fidgetiness, and jitteriness, *Sachdev and Kruk ,(1996)*, suggested that human and animal studies should investigate the pathophysiology. So that intervention can be based on the underlying mechanisms.

V. Clinical Picture of the disorder

In the fourth edition of the Diagnostic and Statistical Manual of the American Psychiatric Association ,*(DSM-IV)* ,*(1993)*, Attention deficit/hyperactivity disorder has been reconstructed to have a regular and specific base of diagnosis ,(based on specific criteria, either with inattention or together with hyperactivity-impulisivity complex.Previously, *DSM-III* ,*(1980)*, created the ADHD to take the place of the hyperkinetic reaction of childhood used in *DSM-II*. The change in *DSM-III* was based on the argument that developmentally inappropriate inattention is virtually always present and often prominent in children described under the variety of diagnostic headings including " Hyperactive child syndrome", "Minimal Cerebral Dysfunction", and other similar tables. Further it is argued that the change in emphasis is appropriate because the excessive motor activity in diagnosed individuals often diminishes in adolescence, while the attention difficulties often persist.

McMahon ,*(1984)*, stated that in *DSM-III* the new ADD with hyperactivity is comparable to the old hyperkinetic reaction of childhood in that both presume that the disorder is best described as consisting of an essential configuration of symptoms that includes hyperactivity, impulsivity, attention concentration problems, among which are aggressive behavior, poor peer relationship, low frustration tolerance and non-responsiveness to discipline.

Frick ,et al, (1994), described *DSM-IV* field trials for the disruptive behavior disorders conducted to provide data to be used in making decisions in *DSM-III*-R criteria for oppositional defiant disorder ,*(ODD)*, conduct disorder ,*(CD)*, and ADHD. The results of this study supported the inclination of more restricted definitions of "lying" and "truancy" to increase their association with a CD diagnosis and supported the elimination of "swearing" in the ODD criteria.

Sabatino and Vance ,(1994), supported the changes in *DSM-IV,* which differentiate the predominantly inattentive type from hyperactive-impulsive type disorders, while altering and adding several new behavioral descriptors. They found that nearly one-third of cases diagnosed as ADHD ,(according to DSM-IV), were classified with mental disorders ,(according to *DSM-III*-R). The comorbidity among ADHD symptoms, other mental disorders, and learning disabilities has rendered 8 of the 14 ADHD symptoms useless in accurately describing the presenting behavior. ADHD as a single entity using the 14 *DSM-III*-R behavioral descriptors is critically challenged.

Baumgartel ,et al ,(1995), studied the same population using different DSM subtypes and found that the overall prevalence for ADHD increased from 9.6% *(DSM-III),* to 17.8% *(DSM-IV),* due mostly to new cases identified as ADHD-AD (inattentive type), and to ADHD-HI (hyperactive-impulsive type). Inattention in any subtype was associated with academic problems, perceived behavior problems were associated with more than 80% of cases involving hyperactivity-impulsivity. *DSM-IV* ADHD subtypes showed significant behavioral, academic, and demographic differences.

1. Hyperactivity

Juliano ,(1974), Barkly and Ullman ,(1975), stated that several studies have found a significant difference between diagnosed hyperactives and normal controls. The difference is quantitative, not just qualitative. Even when the difference falls short of statistical significance, the trend is generally in the same direction.

Rosenthal and Allen ,(1978), stated that a substantial amount of research interest has been devoted to two alternative constructs - attention and arousal. They have indicated that activity, arousal and attention are interrelated constructs that focus on three separate dimensions of human functioning. Activity is an overt behavioral construct, arousal ordinarily refers

to quantity and quality of cortical and autonomic activation, whereas attention is a cognitive construct that is considered to involve a number of components including alertness, selectivity and persistence.

Firestone and Martin ,(1979), stated that it is not enough to know that hyperactive children move more than normal controls in most situations, we also need information on whether they are different from children with other kinds of psychiatric problems, particularly conduct disorder or not. The evidence here is much more scanty. Children broadly defined as hyperactive move only slightly more than children with conduct disorder.

Porrino ,et al ,(1983), have extended the findings into natural settings by continuous recording of activity for 24 hour periods. They found that hyperactive children move more than normal controls in all situations, whether structured or free and even during sleep.

Walker ,(1983), stated also that there often very strong situational variables in hyperactive children's behavior. Thus some children are hyperactive in group situations, but are relatively calm and easy to control in one-to-one situations, while other children demonstrate exactly the opposite pattern. Many hyperactive children display a great deal of overactivity in novel situations, while others appear to be more active in situations that are familiar to them. Often the child who is completely unmanageable in a group situation that he or she is familiar with such as a school classroom , will be extremely docile-cooperative and definitely not hyperactive in a one-to-one situation with a pediatrician or psychologist in his or her office. This leads the unwary clinician to deny that the child is hyperactive.

Sandberg ,(1985), stated that it is quite commonly assumed that the central behavioral characteristics of a hyperactive child should more or less appear regardless of the situation he or she is in. This may be far from the case, one compelling interpretation of the finding that parents, teachers and clinicians tend not to identify the same children as "hyperactive" is that children in fact often exhibit different behaviors in different

situations. If we assume that the parent and teacher reports in these studies are reasonably accurate, a marked situational effect is indicated: the children who are "hyperactive" at home tend not to be similarly regarded at school, and vice versa. Whatever the reason for such a low level of agreement may be, they have led some to suggest that much of the hyperactive behavior is "socially caused". It may represent an adaptation to a demanding situation or be a consequence of the influences of a particular social setting.

Sahakian ,*(1986)*, stated that hyperactivity in young children is manifested by gross motor activity, such as excessive running or climbing. The child is often described as being on the go, "running like a motor", and having difficulty sitting still. Older children and adolescents may be extremely restless and fidgety. Often it is the quality of the motor behavior that distinguishes this disorder from ordinary overactivity is that hyperactivity tends to be haphazard, poorly organized and not goal directed. In situations in which high levels of motor activity is expected, such as on the playground, the hyperactivity seen in children with this disorder may not be obvious.

Rutter and Hersov ,(1987), stated that hyperactivity has been defined socially. It is a deviation from what is expected of children by their care takers. The first step in considering its validity is therefore to determine whether "hyperactive" children can be distinguished by objective tests: are they truly hyperactive? Many physical measures of activity exist that record some component of movement, for example: ,*(1)*, by mechanical recording with an actometer or stabilimeter in contact with the child; ,*(2)*, by electronic mats sensitive to the pressure of a child moving on them; or ,*(3)*, by ultrasonic or photoelectric system in which child's movement interrupts a beam. Several studies have found a significant difference between diagnosed hyperactives and normal controls.

2-Inattention:

Douglas ,(1980), summarized a program of research studying attentional deficits in hyperactive children. On tasks such as experimental measures of sustained attention, hyperactive children are found to respond more slowly and make more errors of commission than normal children. Hyperactives also perform poorly on tasks that require inhibiting their responses, such as measures of cognitive impulsivity and reaction time tasks. The response of hyperactive children to these experimental tasks seem to differentiate them from normal as well as children from other clinical populations.

Douglas ,(1983), has described also experimental work on hyperactive children in detail . One thing seems clear, "attention" is not a single cognitive process, but describes main functions which are only loosely related . These include the ability: to resist distraction, to maintain one's performance on long tasks to focus intensely on specific stimuli, and to explore complex stimuli in a planned and efficient way.

McMahon ,(1984), stated that attention deficit is intimately linked with interest, motivation and ability factors. A child with a different or deficient learning history cannot be expected to be highly attentive to tasks that are either difficult or uninteresting due to lack of familiarity or to other factors

Berry ,et al ,(1985), stated that ADHD is defined as age inappropriate inattention and impulsivity . Diagnosis depends upon demonstration of attention deficits rather than hyperactivity

Kaplan and Sadock ,(1985), showed also that coupled with hyperactivity is a short attention span. The child is unable ,without a great and conscious effort, to inhibit his response to any stimulus that comes along, regardless of appropriate meaning or significance. In addition, the child seems incapable of attending to more than one stimulus at a time. Such child is incapable of attending to any stimulus for more than 10 seconds. They added that, attentional problems are shown , at home by a failure to follow through on parental requests and instructions and

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by the ability to stick to activities, including play for periods of time appropriate for the child's age.

Rutter and Hersov, (1987), stated that inspite of lack proven specificity, the concept of impaired attention has become central in modern descriptions of hyperactivity. The ninth revision of the International Classification of Diseases defines "the hyperkinetic syndrome of childhood" as disorder in which the essential features are short attentional span and distractibility. The third revision of DSM of the American Psychiatric Association has replaced hyperactivity with "attentional deficit disorder" as the cardinal condition to which hyperactivity is a qualifier, so that one can diagnose ADD with and without hyperactivity . One likely reason for this preference for the concept of ADD is that it refers not only to a complaint about behavior, but also to the impairment of an important psychological process. The central processing of information from the outside world requires an orderly process of sampling stimuli. One could not analyze everything all the time without drowning in information ;selectiveness is essential to efficiency. Strategies for selecting certain kinds of information ,and concentrating on them at the expense of others, must be an important part of normal functioning .

3. Impulsivity:

Lewis, (1974), stated that the idea of impulsiveness in children exists in at least two distinct forms. The first, refers to the uncontrolled behavior of children who are heedless of consequence and pursue the pleasure of the moment with the inhibition of prudence. In this sense, it overlaps with aspects of the adult psychopathic character. The second sense refers to a style of problem-solving activity in which one leaps rapidly to a conclusion in a situation of high uncertainty, and is therefore often wrong. The first meaning is behavioral or moral, the second is cognitive. The first behavioral, sense of impulsiveness has been associated with

hyperactivity by a number of experimental studies. Indeed, one should expect this to be true, simply by definition (it is necessary part of the DSM-III description). The second, cognitive meaning of impulsiveness has been operationalized by the matching familiar figures test. A difficult test of matching is scored by the proportion of responses that are inaccurate and emitted rapidly. Hyperactives certainly make more errors on this area than normal individuals *Douglas and Peters, (1979)*.

Kaplan, et al, (1980), showed that impulsivity and inability to delay gratification are other phenomena related to ADHD. The child is often accident-prone. In school, he may rapidly attack a task and do only the first two questions. The child is seldom able to maintain visual fixation independently of manipulation, and he finds it hard to solve a problem of visually before taking it manually. The child is often explosively irritable and sensitive to minor stimuli. He is frequently emotionally labile, easy set off to laughter and to tears, and his mood and performance are apt to be variable and unpredictable.

Lahey, et al, (1980), stated that the characteristics that hyperactive children exhibit (overactivity, impulsivity, inattention . . . etc.) predispose them to numerous conflicts with authority and peers. These conflicts and the resulting consequences would be expected to prove frustrating for these youngsters, and predispose them to anger and hostile outbursts.

Walker, (1983), showed also that these children will tend to have increasing difficulties as they grow older and will suffer much more maladjustment from their overactive behavior than children who do not develop aggressive behavior along with the hyperactivity.

Rapport, et al, (1985), stated also that the impulsivity component of the disorder is in need of regulation because its effects interfere with school performance and persist into adulthood. As they grow older, their impulsivity impairs social functioning. They added also that, the impulsivity characteristic of children with ADHD with hyperactivity has been empirically verified by several investigators using Kagan's Matching Familiar Figures test (MFF), a primary index

of children's cognitive tempo. Children are shown a stimulus picture and are required to select the one match from six similar pictures. The entire test consists of 12 such tasks and yields two primary measures: error rate and latency (duration of time between the presentation of the stimulus and the child's initial response), the former of which has been shown to be a more reliable measure in diagnosing attention deficit disorder with hyperactivity and assessing behavior.

Zentall ,et al, (1994) studied 2 performance measures (accuracy and speed) and 3 behavioral measures (vocalizations, head movements, and button movements) and found that ADHD subjects had significantly lower problem-solving scores in specific math concepts and slower computational performance. Poorer performance of math facts also co-occurred with greater activity. Candidates with ADHD were observed to be more frequently (1) looking away from the task, (2) being physically active, and (3) vocalizing.
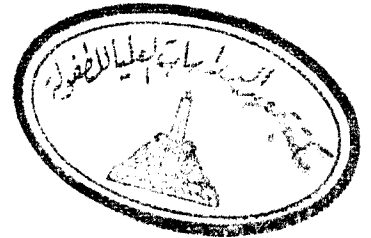
4.Antisocial behavior and Aggression:

American writers on hyperactivity have agreed that aggression and defiance are frequent symptoms. They are reported in around 75% of diagnosed hyperactive children (Cantwell, 1978; and Barkly, 1981).

Stewart, et al, (1980), stated that hyperactivity and antisocial behavior are different kinds of problems, but they are often present together.

Loney ,et al, (1981), found that the social outcome for hyperactive children was rather strongly determined by the extent to which they were also aggressive. Hyperactive children have a very large number of relatives showing features of antisocial personality.

Stewart ,et al, (1981), used clinically gathered data as the basis for research diagnosis of pure hyperactivity, unsocialized aggression,

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and mixed hyperactivity and aggression. They found that the mixed quite closely resembled those with unsocialized aggression only. By contrast, the rather small group of children with pure hyperactivity appeared to be a distinct group; they had lower I.Q levels and an earlier onset of problems.

Taylor ,(1983), stated that overactivity and restlessness are commonly noted in children diagnosed as conduct disorder in Britain. Clinically significant hyperactivity was present in 82% of a group of conduct disordered children. He also described a series of heterogeneous cases subsummed into conduct disorder in Britain; both found separate dimensions of attentive restlessness and of defiance. Defiance and aggression were associated with adverse family relationships; hyperactivity was not. Hyperactivity, by contrast, was more closely linked to developmental delays in sensory motor coordination, language, and impaired performance on cognitive tasks.

Parker, (1988), stated that hyperactives report more malaise and have impaired social skills, compared with the controls. Some 20% or more display the features of "antisocial personality disorder".

Satterfield ,et al, (1994), stressed that clinicians and parents should be alerted to the fact that minor antisocial features in ADHD boys, such as defiance, signal an increased risk for later serious antisocial behavior. However, they should not be misled by the absence of defiance in ADHD boys to think that these children are not at risk for serious antisocial behavior (arrests for serious criminal behavior) later in life.

5. Cognitive and learning disabilities:

Friebergs and Douglas, (1969), administered a concept learning task to hyperactive and control children under continuous and intermittent reinforcement conditions. They found no difference between hyperactive and control specimen under continuous reinforcement, but rather dramatically poorer performance by

hyperactive specimen under the partial reinforcement conditions. The authors concluded that hyperactive children suffer no basic deficit in fundamental cognitive operations involved in concept formation processes; however, the adequacy of performance of hyperactive children was highly dependent upon continuous reinforcement despite an experimental situation that had a number of features likely to enhance interest and focus attention.

Campbell ,et al ,(1971),also suggested that hyperactive children have a more impulsive cognitive problem-solving style than normal individuals. Children who score high in impulsivity measures, have been found to be more distractible, less attentive, more physically active and have been found to experience a variety of learning problems.

Safer and Allen, (1976), stated that 70-80% of hyperactive children are learning disabled, and 30-40% of learning disabled children are hyperactive.

Cantwell, (1977), showed that three hypothetical mechanisms, each with some empirical support, have been proposed to explain these learning problems, (a) neurological impairment causes both behavioral syndrome and cognitive disabilities, (b) overactivity interferes with attention and the acquisition of information, and (c) hyperkinetic children make decisions too quickly.

Lambert and Sandoval ,(1980), found 42.6% of a sample of hyperactive students achieving sufficiently below grade level that they could be classified as learning disabled. Among a random control group 11.3% were classified as learning disabled according to the same criteria. They also stated that some studies have indicated discrepancies between verbal and performance scores for hyperactive children, usually with hyperactive children scoring higher on performance subtests of intellectual measures than on the verbal portions. They found significant differences between verbal and performance I.Q. scores occurring in over 50% of a group of hyperactive children; however, the occurrence of a significant verbal/performance I.Q. difference was also found in 30% of children

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in a random control group.

Nelson, et al, (1983), stated that in children with ADHD with hyperactivity there is high incidence of learning disabilities in reading, mathematics, spelling and handwriting. Academic performance may lag by 1-2 years, and are less than would be expected form their measured intelligence.

Walker, (1983), stated that the relationship between intelligence and hyperactivity remains a controversial issue. Studies done with younger children fail to find difference in intellectual ability, while studies done with children at older ages find significant differences, with hyperactive children scoring lower. This would suggest that the hyperactive children may be performing lower on the tests as a result of their failure to profit from educational experiences, no doubt as an effect of the hyperactive behavior rather than a cause of it.

McMahon , (1984), stated that it is well established that hyperactive children underachieve academically to a significant degree, but the basis for that underachievement is not well understood. Although certainly arousal and attention deficit may well have a significant contributory influence, the question remains as to whether other factors such as cognitive capacity or learning style also may play a role.

Berry, et al,(1985), stated in their study that boys and girls with ADHD did not differ significantly on the performance scale, but girls obtained a lower mean verbal I.Q. score than boys, and a lower mean full I.Q. score. Lower scores for the girls with ADHD with hyperactivity reflect the poorer performance of the girls with the disorder. The children with ADHD without hyperactivity showed no significant sex differences on the I.Q. measures.

Aydin, et al, (1987), investigated the relationship between contingent negative variation (C.N.V.); an evoked potential related to cognitive functions such as attention, intelligence ,behavioral problems, and school achievement in 36 children with ADHD with hyperactivity and 21 normal children (aged 9-12 years). CNVs were obtained for all of the normal group, and for 55.5% of the ADHD

group. CNV amplitudes were significantly lower for the ADHD group. There was a negative correlation between CNV amplitude and hyperactivity scores and a positive correlation between CNV amplitude and school achievement. While both groups were within the normal and upper normal levels of I.Q., Wechsler Intelligence Scale for children scores were significantly lower for the ADHD group than for the normal group.

Satterfield, et al, (1994), stated that auditory and visual stimuli amplitude to attended target stimuli were significantly reduced in ADHD children. No between-group differences were found for responses to nonattended stimuli. Both amplitude and latency abnormalities indicate that ADHD boys suffer from deficient preferential processing of attended stimuli.

Milisch and Lorch, (1994), found that ADHD children can sustain attention for a relatively long time at the same level as with that of control group children, but a difference exists between ADHD and control children in terms of story comprehension and recall.

Fee, et al, (1994), found that there were no significant differences between the normal I.Q. ADHD children and the mentally retarded ADHD group on all but one of the factors. Mentally retarded children in general were found to be more anxious than their normal peers, while normal I.Q. ADHD children were rated higher than the other groups on the asocial factor.

VI. Diagnosis of the disorder.

Sleater and Ullman, (1981), stated that the child who comes to the physician with a presumptive diagnosis of hyperactivity couldn't be diagnosed accurately, nor can the outcome be predicted by his behavior in the clinic. Only 20% of hyperactive patients show hyperactive behavior during clinic examination.

This problem can be solved if the physician has confidence in medical history information from the parents through using Conner's Abbreviated Teacher Rating Scale (ATRS), as a reliable aid in the diagnostic process of hyperactive children *(Sleater and Ullman, 1981).*

Nelson, et al, (1983), stated that it is difficult among 2-4 year olds to identify those who will develop hyperactivity from those who are simply active. The latter learn during the preschool period to master motor output, to maintain attention and concentration, and to modulate social behavior in preparation for school. To assess accurately the term "hyperactive" the physician needs a description of behavior, this will clarify the expectations of parents and reveal their levels of tolerance for motor activity such as running, playing, shouting and so on. The physician must assess the psychosocial structure within which judgement of "normal hyperactive" or "deviant" is made. As they enter the nursery or elementary school, their teacher's report demonstrates that they are uncontrollable, cannot sit still, inattentive, won't concentrate, won't follow instructions, etc.

Kaplan and Sadock, (1985), stated that the history is of great importance. It may give clues to prenatal (including genetic), natal and postnatal factors that may have affected the central nervous system structures or function. They added that by history, observation or report, the child should have shown excessive motor ability. It may be less obvious in structured situations than in understructured or the reverse. Inattention and distractibility represent other essential features.

Rutter and Hersov, (1987), stated that diagnosis of hyperactivity

based on a single person's description has rather little value and its predictive validity is low. The diagnosis is useful when based upon pervasive hyperactivity of sufficient severity to affect development. Children identified in this way are likely to show cognitive immaturity. The diagnostic formulation and assessment should start from the recognition that hyperactivity is present, not stop with it.

Lambert and Hartsough, (1987), stated that behavior ratings to teachers have been the most common source of evidence about the target symptoms of a high activity level, inattention, impulsivity, unsocialized behavior and conduct problems. DSM-III reinforced the cardinal importance of inattentiveness and impulsivity in the diagnosis of the condition. By defining conduct disorder as either aggressive or nonaggressive, DSM-III further challenged investigators and clinicians to be specific in making diagnosis of children by requiring specification of the evidence for attention deficit disorder, hyperactivity, and aggressive and nonaggressive conduct disorder. Other investigators have pursued a different strategy and have asked parents to complete a teacher rating scale. Even though behavior ratings by parents are not commonly used, parents reports of their children's behavior have become the primary source of information used by the independent practitioners for a symptomatic picture of the child. Recent approaches of defining hyperactive children as being situationally hyperactive (i.e. in only one sitting, home or school) or pervasively hyperactive provide an impetus for re-examination of parent ratings of hyperactivity as important sources for providing a comprehensive picture of children's functioning. The DSM-III suggests that when the reports of teachers and parents conflict, primary consideration should be given to the teacher's reports because of greater familiarity with age appropriate norms.

a) Neurological and physical examination:

Nelson, et al, (1983), stated that physical examination does not generally contribute to the diagnosis, although there is an increased

frequency of minor congenital anomalies in children with behavioral difficulties, retardation or neurologic disease. The reported anomalies include fine hair, malformed ears, epicanthal folds, high arched palates and single palmer creases. Children with the disorder are generally believed to show increased numbers of "soft" neurologic signs such as mixed hand performance, impaired balance astereognosis, dysadiadochokinesia, and problems in fine motor coordination. The most useful diagnostic tools are observation in the classroom and psychoeducational testing.

Kaplan and Sadock ,(1985), stated that a neurological examination might also show visual or auditory perceptual impairment problems with coordination and a variety of soft signs.

b) Psychological and psychoeducational testing:

Nelson ,et al, (1983), stated that it is uncertain whether hyperactive children with ADHD have significantly lower I.Q. scores than children who do not have ADHD when appropriately matched for age, school grade level and socioeconomic status. Some hyperactive children have verbal scores more than 10 points higher than performance scores on Wechseler intelligence scale, and lower scores on the attention/concentration subset. Projective tests such as the Thematic Appreciation Test (TAT) and Rorschach are not helpful in the diagnosis of ADHD, though they may provide information on psychodynamics and on emotional strength and weakness.

Kaplan and Sadock, (1985) ,stated that the mental status examination might show a secondarily depressed mood, but no thought disturbance.

c) Laboratory and other studies:

Nelson ,et al ,(1983), stated that no laboratory studies established the diagnosis of ADHD. Children with hyperactivity are reported to have increased amounts of slow waves in the electroencephalogram (EEG), without evidence of progressive neurological disease or epilepsy.

Kaplan and Sadock,(1985), showed that the value of an EEG in the evaluation of a child with hyperkinetic syndrome remains in dispute. A major reason for obtaining an EEG is to recognize the child with frequent bilaterally synchronous discharges resulting in short absence spells. Such a child may react in school with hyperactivity out of sheer frustration. The child with an unrecognized temporal lobe seizure focus can present a secondary behavior disorder. In these instances, several features of the hyperkinetic syndrome are often present. Identification of the focus requires an EEG obtained in drowsiness and in sleep.

Jeffry, et al,(1986), stated that examination of the relationship between neurological abnormalities and stimulant medication efficacy in 80 pervasively hyperactive children (6-12 years old) - 39 were administered methylphenidate hydrochloride (20-60 mg/day) for 4 weeks, 41 received a placebo. Treatment outcome measures included teacher and psychiatric ratings. No evidence was found to indicate that clinical neurological status (i.e. EEG abnormalities and neurological soft signs) was predictive of drug responsivity.

No doubt that the most successful use of brain mapping is in the field of psycho-pharmacology. *Itil , et al , (1974),* was probably the first to introduce the term quantitative pharmaco-EEG, to describe the application of computer assisted – EEG in the study of the prediction of therapy outcome in psychiatry, especially drug therapy . *Fink , et al , (1974),* suggested 2 main methodological approaches , one involving frequency , and the other concentrating on amplitude analysis across frequency .

Using dynamic brain mapping , *Itil, (1985),* reported that the slow component power reduction was maximum in frontal (temporal zone), while the. alpha power increase was most prominent in the parietal and occipital zones , when methylphenidate (Ritalin) was used in hyperactive children. Absolute power of combined delta –theta activities remained unchanged , that of combined alpha bands was attenuated over the left occipital , and occipitotemporal and parietal regions , while that of combined beta activities decreased significantly over the right frontal region .Relative power remained generally

unchanged in three combined frequency bands (delta- theta , alpha , and beta activities). He also stated that the centroid of the combined delta –theta activities remained unchanged. No change was seen in regards to the deviation of the delta –theta centroid. The alpha centroid was significantly accelerated over the left and right occipital , parietal , and occipitotemporal regions. The centroid of the total beta activity remained unchanged , while its deviation increased minimally over the left fronto-temporal region .

Seban , et al , (1984), found only , subtle changes in the form of attenuation of total power and of alpha and beta powers, relative power showed similar alterations. The most obvious findings were , the acceleration of the alpha centroid and the dominant frequency.

Friedman, et al , (1986), studied the effect of imipramine on evoked potentials in patients with affective disorders .They found that although diminished attention to external stimuli , might well be expected from the clinical symptoms of depression , attention deficits are less known in affective disorders. .They stated that evoked potential (EP) amplitude tends to be larger with selective attention. Studies examining EP correlation of the continuous performance test confirmed the pattern seen in a variety of paradigms. *Saletu , et al , (1986) ,* showed that patients with depression had smaller amplitudes , but stimulus intensity on task were important modifying variables, while antidepressants generally have decreased EP amplitudes , but they have usually been studied at posterior leads in normal volunteers with acute imipramin doses. However, it should be noted that antidepressants differing in effects on serotonin and norepinephrine , the best example of which is the marked differences in EP effects between imipramin and amitryptyline. *Buchbaum , et al , (1987),* studied the effects of imipramin on EP, and submitted to continuous performance test in patients with affective disorder fulfilling DSM-III criteria. They focused on the maintenance of attention and its electrophysiological correlates in parietal and frontal regions .They stated that imipramin is a strong inhibitor of serotonin re-uptake , a potent inhibitor of neuronal noradrenanline re-uptake , and has a lower affinity as serotonin S2 receptors antagonists than other anti-depressants. So serotonin S2 receptors , should be activated by high synaptic serotonin concentration , resulting in serotonin re-uptake

inhibition. As the post synaptic inhibition is the response to serotonin at the serotonin S2 receptors , so the lower amplitude EP , observed in parietal and frontal cortex is the observation seen with imipramine. In the occipital cortex , it appears to increase the amplitude level , and selective attention effects were found to be greater than with normal controls. Also it showed enlargement differences in P200 between target and non-target stimuli in comparison to placebo (*Buchbaum , et al , 1986*).

Shetty , (1971), designed an experiment to study the alpha rhythm in the EEG of hyperactive children by administering methylphenidate intravenously during EEG examination. In a statistically significant proportion of children who later showed good response to CNS stimulant therapy , the alpha energy increased after the drug injection. However, studies of alpha rhythm have suggested that hyperkinetic children who respond best to stimulants have high CNS arousal .

Studies have reported that , 35-50% of hyperkinetic children have abnormal EEG , (*Werry , et al , 1972; and Satterfield , 1973*) with an increase in slow wave activity being the most common finding. However there are no EEG abnormalities specific to the syndrome .

Positron Emission Tomography (PET) provides a unique opportunity to examine metabolic factors in vivo. *Zametkin , et al ,(1990),* examined cerebral glucose metabolism in ADHD children and suggested global reductions in glucose metabolism in ADHD with largest reductions in premotor and superior prefrontal cortex (anterior attentional brain regions) .

Zeitlin ,(1997), invented 3 clinical tests that can easily diagnose ADHD in the office ; the first is the 1,2 reversal test , by asking the child to count from 1 to 30 then from 30 to 1 , ADHD cases cannot continue. The second is that the child cannot follow appropriate rules when he is asked to follow , for example, walking on a definite line several times. The third is the fruit yogurt test , by asking the child to eat only the fruit parts from the yogurt , 80-90% of ADHD cases fail to finish this work, due to the loss of concentration and coordination.

VII -Differential diagnosis

Age appropriate overactivity is seen in some active children, but it does not have the hazard and poorly organized quality characteristic of behavior of children with ADD *(DSM-III, 1980)*.

Kaplan and Sadock, (1985), stated that anxiety, most likely in the form of an overanxious disorder, must be considered. Anxiety may accompany ADD as a secondary reaction: and, by itself, anxiety may manifest overactivity and distractibility. It may present evidence of fearful expectations, disturbing dreams, difficulty in tolerating new and stressful situations and autonomic overactivity on psychological testing.

They added that an entity for possible differentiation is that of depression. As with anxiety, there is a problem as to whether depression is primary or secondary to the problems created by the psychic consequences of having ADHD. The literature dealing with the topic uses the terms "primary depression", "pure depression, secondary depression", and "masked depression. These terms are presumably equivalent in the current classification to "major depressive disorder", "chronic depressive or hypomanic disorder", and "adjustment disorder with depressed mood."

Welner, (1978), showed that diagnostic assumption based on responses to medication or concepts of a masked depression manifested by overactivity, aggressiveness, school phobia, delinquency, psychosomatic disorders, and hypochondrasis, were thought to be invalid, as compared with the ascertainment of criteria in children of specific diagnostic factors. The criteria used for diagnosis of depression included a dysphoric mood and a number of the following: anorexia or weight loss (without medication), sleep problems, loss of energy, agitation or retardation, loss of interest in

activities, impaired ability to think or concentrate, recurrent thoughts about death and suicide - all persistently present and not transient. The criteria were exceedingly rare in prepubertal children but were more easily found in adolescence, suggesting that depressive symptoms, when seen, are secondary to the overactivity and the ADHD rather than primary.

Kaplan and Sadock , (1985), added also that similar considerations apply to the various forms of conduct disorder that may be confused with the ADHD, or quite commonly, be associated with or secondary to those disorders. In undersocialized conduct disorder, aggressive type, the poor frustration tolerance, irritability and reckless impulsiveness may be confused with ADD-H. However, although children with conduct disorder may be anxious, show a lack of concern for others and of guilt, and accept no responsibility, they recognize the requirements of society and aggressively act out a pervasive hostility. The undersocialized conduct disorder, unaggressive type, although after found in conjunction with ADHD, is not likely confused with it.

Florence, et al,(1987) studied the relationship between ADD-H, conduct disorder (CD), and anxiety disorder (AD) in 158 (5-10 year old children). The sample diagnosed as having severe or moderate ADD-H were found to be younger at referral, and to have a lower I.Q. than the sample with CD and AD. When age, I.Q., social class and sex were controlled, the sample with severe ADD-H were found to perform significantly worse than other diagnostic groups. It is suggested that children with severe ADD-H form a distinct group, and those with mild ADD-H overlap, symptomatically and on tests of vigilance with children with conduct disorder.

Cocoress, et al,(1987), stated that ADHD residual type has been reported in abusers of cocaine. The same reported by *Khantzian, (1983),* and *Gatwin, et al ,(1985).* The diagnostic criteria for bipolar and cyclothymic disorders include physical restlessness, racing thoughts, distractibility, and mood instability. The disorders are also characterized by periods of fatigue, low self-esteem, apathy, poor

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concentration, and depression. The differential diagnosis issue involving ADHD, bipolar illness, and cocaine addiction has been considered by others *(Khantzian, et al, 1984)*. Although these three disorders rarely appear in the same individual, they are important differential diagnoses when considering any one illness with the above symptoms complexes. They added that cocaine ability to deplete dopamine in an already dopamine compromised individual usually results in an ADHD clinical picture. The ADHD may be exacerbated or reviewed by the cocaine-induced dopamine depletion. Dopamine deficiency secondary to cocaine abuse can induce a temporary ADHD even in individuals without a history of ADHD.

The clinical feature characteristic of hyperkinetic syndrome may be present in severe and profound mental retardation. They could be differentiated by clinical examination and investigations.

Epilepsy must be considered in the differential diagnosis. The epileptic child can be differentiated from the hyperkinetic one by proper history, clinical and neurological examination, EEG and psychological testing *(Okasha, 1979)*.

Learning disability is a deviation in learning process which is associated with an educationally significant discrepancy between apparent intellectual capacity and actual performance and academic tasks. A hyperkinetic child may suffer from learning difficulties due to short attention span and distractibility. The differential diagnosis of a hyperkinetic child from that with learning disability depends on careful pediatric and developmental history, physical and neurological examination and psychological tests *(Heinicke, 1972)*.

Organic brain damage must be taken into consideration in the differential diagnosis of hyperactivity. It could be differentiated by psychological tests, clinical examination, EEG and other investigations *(Walker, 1983)*.

Richards, (1994), stated that ADHD should be differentiated from other disorders particularly dyslexia.

De-Mesquita and Gilliam, (1994), stated that it is important to differentiate ADHD from childhood depression, anxiety disorders, and conduct disorders. Also cases with substance abuse, uncomplicated bereavement, posttraumatic stress disorder, and adjustment disorder has to be differentiated from ADHD

VIII. Treatment.

a) Pharmacotherapy:

From the standpoint of many who are in charge of managing children's behavior; medication is the treatment of choice for hyperactive children.   Currently the most popular medications are stimulants;   dextroamphetamine   (Dexedrine),   methylphenidate (Ritalin), and pemoline (Cylert).

Cantwell and Carlson,(1978), summarized that stimulants are usually prescribed at the lowest possible dose and titrated upward until a positive response is obtained or until side-effects are noted. Methylphenidate and the amphetamines are usually given one to three times daily in dosage ranging from $0.3 - 1$ mg/kg and $0.15 - 0.50$ mg/kg respectively. For pemoline - a longer acting drug - a dosage of $0.5 - 2.0$ mg/kg is administered once a day.

Major tranquilizers, such as chloropromazine (Tharazine) and thiroidazine   (Melleril),   tricyclic   antidepressants   as imipramine (Tofranil), and sedatives such as diphenhydramine (Benadryl) have been prescribed for hyperactive children who do not respond to stimulants *(Fish, 1975; and Gittelman-Klein,et al , 1987).*

Walker , (1983), showed that there is general agreement on several points regarding the treatment of hyperactive children with stimulant medications.

First, a proportion of hyperactive children responds positively to the medication. *Barkley , (1990),* reported that

approximately 5% of these children are judged improved when treated with medications.

Second, there is no evidence to indicate that actual academic achievement or performance increases with these medications. Thus the child becomes more manageable, but does not appear to profit educationally beyond that.

Third, numerous researchers have expressed concern about using such medications over long periods of time during the developmental years may have serious consequences that are not yet recognized or fully understood.

One **final** point has to do with the mechanism of activity of the stimulant drug. These drugs are often described as having a paradoxical effect in hyperactive children since the medication is a stimulant, but reduces activity level possibly because inhibitory centers of the central nervous system are more likely affected than other centers , though this is purely speculative and the actual mechanism of action is unknown *(Weingartner, et al , 1980).*

Nelson , et al,(1983), stated that the therapeutic range of dose of stimulant medication may be rather narrow. Too small a dose has no effect; too large a dose will produce a jittery and agitated or socially withdrawn child. Some children develop with chronic medication a pale, shallow, drawn, glassy-eyed appearance; it is not thought to represent a significant disturbance, but can be distressing to child and parents. If these effects do not abate with decrease in dose, another medication may be tried. Children who not respond show little or no change in behavior with increasing doses. Parents or teachers may report that the child is worse. Before a child is judged to be unresponsive, a full therapeutic trial should be undertaken, so long as side effects are not serious.

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Nelson, et al, (1983), stated also that the effect of magnesium pemoline develops more slowly and lasts about 12 hours. Three to four weeks are required to determine its efficacy. About 1-2% of treated children may show changes in the liver functions, accordingly, pretreatment studies and the monitoring of liver functions every 2 months are required. Side effects of pemoline include increased nervousness and jitteriness.

Carolyn , et al , (1984), stated that administration of methylphenidate improved subjects' performance on oral reading measures. The findings provide evidence in support of a two factor theory of ADHD. In general the short-term benefits of stimulant therapy are attributable to improved attentional control during testing rather than to gain in learning achievement, and longitudinal studies of stimulant therapy provide little empirical support for the effectiveness of stimulant treatment on academic achievement.

Safer, et al, (1972), reported that these medications appeared to result in growth deficits in children taking them regularly. However, later results failed to support this, particularly if drug holidays are employed.

Kaplan and Sadock,(1985), stated that the medication probably most used now is methylphenidate (Ritaline). Recommended dosage for children 6 years of age and older begins with 5 mg daily before breakfast and lunch, increasing by 5-10 mg weekly. Daily dosage above 60 mg is not recommended, also discontinuation of the medication is favorable if no effect has been achieved within a month. Of course adverse effects call for the reduction of the dose or discontinuation of the medication. They stated also that another central nervous system stimulant is pemoline (Cylert). It is supplied in tables of three strengths 18.75, 37.5, (both chewable and non-chewable) and 75 mg. It is not recommended for children under 6 years. The medication is inherently long acting, which affects

the dosage recommendation. It should begin as a single morning dose of 37.5 mg daily and increase by 18.75 mg weekly until the desired effects are obtained. The maximum recommended daily dose is 115 mg. There is an abuse potential with pemoline, but less than that of amphetamine. There is no abuse potential with imipramine hydrochloride (Tofranil). These drugs work by blocking the reuptake of catecholamines.

Rapport, et al, (1985), found in their study a fundamental linear relationship between reduction in impulsivity and increasing dose of methylphenidate in children with ADHD. They added that methylphenidate is presently the most widely studied and prescribed medication for children with ADHD in the USA. Pediatricians typically prescribe this medication using the titration method of increasing dosage until favorable reports are obtained by the parents and/or teachers. Unfortunately, both of these sources tend to base their judgements on the child's social behavior, which may not correspond with improvements in learning and impulsivity.

Zamitkin ,et al, (1986), treated eight 4-12 year old children with ADHD with methylphenidate (maximum 0.7 mg/kg/day) and promethazine (maximum 2 mg/kg/day). No improvement occurred with promethazine, while behavioral deterioration in four children, necessitated discontinuation of the drug in two cases.

Pliszka, (1987), compared and summarized significant drug effects in 5 studies examining the use of stimulants with the use of imipramine and other tricyclics in the treatment of ADHD. Although both stimulants and antidepressants ameliorated ADHD symptoms, the bulk of the literature suggests that over all, stimulants are superior to imipramine in the treatment of ADHD. There may be a subgroup of children with ADHD who respond better to imipramine, and this group may be characterized by higher level of anxiety or depression or both.

Burd , et al,(1987), showed that data are presented on 12 patients

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(aged 5-12 years) in whom phenobarbital, when used as an anticonvulsant medication, either precipitated hyperactivity or permanently exacerbated existing hyperactivity. Discontinuation of the medication resulted in a decrease in hyperactivity in all patients. None of the patients returned to a pre-drug level of activity. The effect of phenobarbital on the reticular system is discussed. It is argued that ascertaining whether hyperactivity has been caused or exacerbated by exposure to phenobarbital may be important in the differential diagnosis of hyperactivity.

Parker, (1988), added that tranquilizers such as chloropromazine and haloperidol may also reduce activity levels, but often at the cost of sedating the child. A recent trial suggests that clonidine, an unrelated drug, may be of value.

Wilens, et al, (1994), described the use of low to moderate doses of clonidine at bedtime for the amelioration of sleep disturbances with ADHD. Results showed reduced sleep latency, less sleep restlessness, increased number of hours slept, and improved morning awakening. Also, many children had fewer ADHD-like symptoms on the next day.

Steingard, et al, (1994) ,found that clonazepam when used as an adjunctive treatment together with colonidine in children with ADHD with tic disorder, reduced and treated tics.

Gay and Ryan, (1994), reported the development of brief, episodic, movement-induced dystonia 10 days after initiation of methylphenidate treatment (20 mg, twice per day) in ADHD children. They suggested that the stimulant triggered the onset of a genetically determined disorder.

Barrickman, et al, (1995) ,reported that methylphenidate and bupropion both reduced significantly greater and equivalent

improvement on different rating scales in children with ADHD.

Gadow, et al ,(1995), stated that treating children with ADHD with methylphenidate 0.3mg/kg twice daily for 2 weeks, resulted in marked reduction of hyperactive, disruptive and aggressive behavior. They also reported increased frequency of motor tics.

Wilenes, et al ,(1995), suggested that increasingly, multiple agents are being used for the treatment of psychiatric disorders in children with ADHD. The need to use combinations of medications has risen out of the often less than satisfactory responses to single agents.

b) Psychotherapy:

Nelson, et al ,(1983), stated that there is no conclusive evidence that psychotherapy is primarily beneficial in the ADHD, but individual and family therapy will be indicated when it is complicated by depression, social withdrawal or negativism and chronic family conflicts. The child with ADHD with hyperactivity often has a learning disability, and may need any of the special educational approaches prescribed for learning disabilities. Special classes frequently use operant conditioning for behavior modification; when carefully planned and carried out by teachers, such approaches can help these children. It is essential that physician and school personnel maintain close communication about the child's progress.

Kaplan and Sadock, (1985) ,showed that the use of medication is infrequently sufficient to meet the needs of the children and their families. He needs to understand that perfection is not the goal and that he has an equal right with all other human beings of being occasionally disagreeable, unpredictable, and difficult. Parents need to be helped not to focus everything on medications, to recognize that they have a child, not a pill. When the child not only is allowed, but helped, to structure his environment, his anxiety diminishes. Thus, his parents and teachers need to set up a predictable structure of reward and punishment following a behavior therapy model. Parents also need to be helped to recognize that, despite their child's deficiencies in

some areas, he faces the normal tasks of maturation and attendant problems, including the need to form a normal flexible super ego.

Ruman, (1988), presented the case of a seven-year-old boy whose hyperactivity was of psychogenic origin. A behavior management approach combined with a positive teacher-child interaction resulted in better child behavior than did a regimen of methylphenidate.

Calhoun, et al, (1994), stated that because effects may be adverse and repercussions are associated with drug therapy, many researchers endorse alternative controls for children with ADHD, including intervention in the family and parental training, ecological control, group relaxation training, and hypnotherapy.

c)Behavioral therapy:

Behavioral interventions are regarded as the principal alternatives to treatment with stimulant medication. Behavioral techniques studied with hyperactive children include contingency management, cognitive behavioral modification and relaxation biofeedback *(Meichenbauni and Goodman, 1971).* The authors have accomplished an interesting approach which involved the concept of cognitive self-control, by teaching the child to maintain a steady stream of self-instructions that serve to bring his behavior under internal control and keep the behavior task-oriented. These cognitively oriented behavioral techniques appear to represent useful and exciting approaches to the management of hyperactivity in children.

O'Leary, et al, (1976), stated that parents were responsible for rewarding the child's daily behavior at school. After a ten week treatment period, children treated with the reward program were significantly more improved than those in a control group. The authors noted that ratings of hyperactivity were decreased to a level comparable in magnitude to changes reported with stimulant drugs.

Braud, (1978), stated that another interesting behavioral treatment for hyperactivity is the employment of relaxation training or

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biofeedback. Several studies demonstrated that, with training in deep muscle relaxation, either using standard relaxation procedure or relaxation training assisted by biofeedback, hyperactive children can develop sufficient control over their behavior to reduce greatly their hyperactivity. In intensive biofeedback the child was brought to the clinic for three successive days and maintained in the clinic from 8:00 Am. until 5:00 Pm. Every hour, the child was given 15 min. of biofeedback training employing tangible rewards and reinforcers for meeting criteria set and revised during the three days of training. The remaining time of each hour was devoted to free play and other relatively unstructured activities. Children were able to learn, through this massed practice biofeedback, sufficient self-control that they were able to return to their classrooms and perform successfully.

Wolraich, et al, (1978), examined the effect of relatively low dosage of Ritalin alone, behavior modification alone, and the combination of both. In this study, drug treatment was more effective during individual classwork, while behavioral modification was more effective during group work. Academic achievement also significantly improved with behavior modification. However, the authors noted that in some situations a combination of the two might prove more effective than either separately.

Gittellman , et al, (1980), compared the effects of Ritalin alone, Ritalin plus behavior modification, and behavior with placebo pills. All three treatments resulted in significant improvement . The combination of Ritalin and behavior modification produced the greatest improvement. Similar results were reported by *Pelham, et al, (1980).*

It would appear that medication might be employed in the early stages of treatment in order to bring the activity level under control and make the child more manageable for other programs. The medication might then be reduced as the child progresses in the treatment program. Along with the medication behavioral treatments involving training in cognitive self-control as well as relaxation or biofeedback training, along with reinforcement for on-task behavior,

would be an important component of the treatment regimen *(Gittelman, et al, 1980).*

Barkley (1981a & 1981b), has presented a behavioral analysis of parent-child interactions with hyperactive children. He presents a guide for training the parent to utilize behavioral techniques that focus on improving compliance and rule-governed behavior.

Etscheidt and Ayllon, (1987), evaluated the efficacy of contingent exercise in reducing hyperactive behavior and enhancing academic performance in a 13-year-old hyperactive boy whose lack of academic progress was attributed to hyperactivity and general distractibility. Contingent exercise consisted of providing the child with an opportunity to engage in a high level of movement whenever he failed to meet academic criteria. Results indicate reduction in hyperactive behavior during reading and arithmetic as well as gain in academic performance.

Henry, (1987), stated that six 4-10 year-olds stabilized on psychostimulant medication and diagnosed as having ADHD, underwent a treatment program consisting of symbolic modeling and parent training. Symbolic modeling, which required the children to view videotaped sequences of the same sex and age equivalent models complying with parental commands, was of limited effectiveness in bringing about better compliance. Phase-I, of parent training, which taught mothers appropriate attending, ignoring and rewarding behaviors, was more effective than symbolic modeling alone. Phase - II of parent training, which incorporated a time-out procedure for noncompliance, was most effective. Follow up questionnaire data obtained maintenance of treatment effects.

Zigler and Holden, (1988), proposed a child and family therapy model for children with learning disabilities and ADD-H, who are vulnerable to increase psychological difficulties in 3 areas; self-esteem, self-control, and frustration tolerance. Within a psychotherapeutic-educational framework, parents and children are taught to distinguish the impact of the learning disability form children's emotional reactions and other aspects of their personalities.

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All in all, the treatment methods for hyperactivity are largely the same as for the majority of child psychiatric disorders which, as we know, tend to predominantly involve disturbances of conduct (i.e. noncompliance, defiance, antisocial behavior and aggression). Seldom is one form of treatment adequate on its own. A combination of psychological treatments is often needed. These include behavior modification techniques, family therapy, counseling of parents and teachers and in small minority of cases, individual psychotherapy. Changes in the child's environment may be at least as important as such therapeutic techniques. Many of the children also need specialized help for their educational problems. Stimulant drugs, though in some instances, capable of producing instant and dramatic therapeutic effects, should seldom be seen as a treatment of first choice. They can, however, be useful in the treatment of certain hyperactive children often in combination with other treatment techniques. The choice of the treatment method should be a result of careful clinical assessment of the child and his/her entire living situation. A referral to a child psychiatric facility is therefore likely to be necessary for such an assessment to be carried out as well as for the provision and adequate monitoring of the possible treatment.

Maretti, et al, (1994), assessed the impact of a community-based program for children with ADHD and reviewed the effectiveness of the program for 203 children. The response program is based on the view that attachment issues are central in understanding and providing care for children. The program begins with a 30-day residential stay during which a multidisciplinary team works with the child and the community to come to a full understanding of the developmental and family history, the nature of the problems, and the functioning of the immediate and wider social community. Specific scales were used to evaluate and monitor presence and severity of symptoms. Caretakers reported significantly reduced levels of ADHD, conduct disorder, and oppositional defiant disorders at all follow-up intervals.

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IX. Course and Prognosis

Minde, et al,(1972), had reported a careful 5-year follow-up into adolescence. The cases were found still to be impulsive, distractible, and disorganized. Many had failed in their school grades, their self-image was poor, and one quarter were delinquent.

Loney, et al, (1978), stated that within a group of hyperactive children, several factors determine the prognosis. Aggressive behavior in childhood and family stress predict antisocial behavior in adolescence; lower I.Q. or lower achievement in childhood predict later school failure in the same studies. These factors would of course have similar predictive value in the normally active.

Schachar, et al,(1981), showed that the poor outcome of the disordered was very largely determined by the presence or absence of overactivity.

Weiss , (1983), reviewed the follow-up studies from the early school years. The one review concludes that they fail to support the notion of a hyperactivity syndrome, the other that they validate it. The uncertainty comes from the lack of the uniformity in the children selected for study, and the lack of clear description of them which would allow clinicians and investigators what kind of disorder is being followed.

Walker, (1983), stated that as these children approached puberty, the hyperactivity would decrease. In fact some speculated hormonal changes in the body at .puberty might result in hyperactivity disappearing. More recent studies suggest that this is not the case for many hyperactive children. As some children advance in age, they learn to control their behavior better, however, for a large number of them attention difficulties and impulsivity remain prominent characteristics, and they continue to experience difficulty throughout their life span. Hyperactivity does not disappear at puberty particularly

in those who have developed aggressiveness with their hyperactivity. Children who suffer from poor self-concept tend to suffer numerous social consequences such as failure in school, increased delinquency, underemployment and marital interpersonal difficulties. On the other hand, those youngsters who do increase their self-control, develop a relatively adequate self-concept and do not develop hostile or aggressive behavior, and manage to adapt to social situations in a much more adequate manner.

Berry, et al, (1985), stated that the clinical course of ADHD is influenced by maturational factors differently in girls than boys, so that this attention deficit becomes evident at a later age. The early emergence of social difficulties reported for these girls suggests, however, that symptoms of the disorder may be present even in the preschool period, and the risk of underidentification may be greatest for attention disturbed girls not demonstrating hyperactive and impulsive behaviors. The girls with ADHD with/without hyperactivity were more likely to suffer peer rejection than their male counterparts. The peer relations are a sensitive predictor of later psychiatric disturbances and a prognosticator of long-term outcome in emotionally disturbed children. Community or school-based studies using uniform criteria are needed to establish the true prevalence of ADD-H in the general population. Furthermore, with the current interest in the uniqueness of ADHD as a clinical disorder distinguishable from other behavioral disorders of childhood, studies are needed to establish criteria specific for accurate diagnosis as well as early identification and development of effective treatment.

Rutter and Hersov, (1987), stated that newborn babies differ in their levels of activity, alertness, and their response to stimuli; but the differences are neither stable nor consistent. Stability from the first days of life is not to be expected. However, continuities in activity and attention are complex and dependent upon the child's psychological environment. By the age of 3 years, individual differences become somewhat consistent. At this age locomotor overactivity is quite a

common complaint, and it is a strong predictor of antisocial behavior in late childhood.

Parker, (1988), also stated that hyperactivity tends to lessen with increasing age, but the other care symptoms of the disorder which are inappropriate restlessness, attentional difficulties and impulsivity - still present though they may be less marked. By reaching adult life about one-third become "indistinguishable" from normal adults. But in all studies a higher percentage of hyperactives have been found to have a history of antisocial behavior than control subjects. Drug addiction or alcoholism has not been identified as adult outcomes. Hyperactives report more malaise and have impaired social skills, compared with controls. Some 20% or more display the features of antisocial personality disorder, as it was defined in DSM-III. Most have lower status jobs than control subjects, and employers rate their work performance as inferior.

Rapport, et al, (1994), reported that large proportions of children treated with methylphenidate significantly improved or normalized classroom functioning, however, a large subset of them failed to show improved academic functioning. Those failing to respond at lower dose levels have a good chance of improving at an increased dose.

Handen, et al, (1995), found that both independent play and restricted academic tasks may serve as appropriate clinic-based means of assessing medication efficacy in children with ADHD.

Jonkman, et al , (1997), stated that in ADHD children, methylphenidate ameliorated some, but not all, deficits and also improved processing where no differences with normal children were present.

B-Brain Electrical Activity Mapping (BEAM)

I) Introduction:

The classical EEG amplifies and records the brain electrical activity, but this technique is comparatively insensitive. The introduction of the computer for EEG analysis allowed more precise localization of the brain electrical activity than it has been possible by the older EEG technique *(Duffy, 1984)*.

Among the techniques used in the field of the computerized EEG (CEEG) are detection of transients and power spectral analysis.

Brain electrical activity mapping (BEAM) is one of the most promising techniques. It is the topographic analysis of the EEG recording where lines of the EEG recording are converted into color codes image on an anatomical map of the head. These images are made in such a way that we can show the electrical activity of the brain in real time. Moreover, these images reflect the electrophysiological status of the brain, which allows studying the functional aspect of the brain in contrast with the static anatomical images of the NMR and the CAT scan *(Duffy, 1984)*. And this represents a different window towards studying the brain.

The CEEG was found helpful in the field of neuropsychiatric disorders, as it has a role for evaluating the patients suffering from cerebro-vascular stroke *(Nagata, 1984)*, dementia *(Duffy, 1984)*, dyslexia *(Duffy, 1980)*, focal neurological disorders and brain tumors *(Duffy, 1980)*, and epilepsy *(Lombroso, 1982)*. While in psychiatric disorders it was found helpful for evaluating patients suffering from schizophrenia *(Morstyn, 1983)*, also it has a role in assessment of psychotrophic drug action.

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It has generally been found that visual inspection of the EEG tracing is a complex process. The EEGer through idiosyncratic impressionistic and highly subjective technique tries to reduce the polygraphic recording into few interpretative statements. In this respect, EEGer tries to detect any transients and describe changes that take place in the background. Transients when they have enough energy to stand in clear contrast with the background can easily be detected by the unaided visual inspection. Examination of the background is more complex, the amount, spatial and temporal distribution of the various frequency bands must be assessed, as the human ability to assimilate the large amount of information contained within the EEG background is limited. The clinical utility of the EEG has apparently fallen short of our expectations *(Lombroso 1980; Aminoff 1981; and Duffy, 1984)*. Although the EEG is a very powerful window on the brain function visual inspection of the EEGs of functional disorders such as schizophrenia failed to detect any diagnostically helpful information. The frequent within normal reporting of the EEGs of those patients is not because the EEG is inherently intensive, but because the visual inspection is incapable of detecting subtle, though diagnostically and clinically useful information. Application of computer for analysis of the EEG signals has clearly enhanced the clinical utility of the EEG, through two main objectives:

  * Detection of transients.
  * Power spectral analysis.   *(Duffy, 1984)*

Computer detection of transients was disappointing because of the following reasons:

  1.   Absence of generally agreed parameters that describe the EEG transient events in a mathematical and quantitative form.

  2.   Computer assisted differentiation between transients and artifacts was most difficult, false detection was made more often than the transients were missed*(Aminoff,1981)*.

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Aminoff, (1981), concluded that computer detection of transients has not reached the degree of accuracy to obviate the needs for visual inspection, although the computer might help in this request, the final decision must be left to the EEGer.

The second objective is the power spectral analysis. Although the name appears daunting, it is nothing but a name coined by electrical engineers for frequency analysis. The EEG signal (epoch) is broken down into groups based on frequency. Power here is a function of frequency [the squared amplitude / cycle / second within a given range and is expressed as microvolt / cycle / second (μVHz)]. Power spectral analysis is the most commonly used technique for EEG quantification. The following steps are used:

1. An epoch (EEG segment) for suitable duration is chosen and visually inspected for artifact.

2. The EEG signal (epoch) is amplified and prepared for sampling by the computer where the signal passes through filters, which attenuate the frequencies below and above the desired range.

3. The EEG signal is then digitized in which the analog EEG signal is sampled by the computer and converted to a digital representation and stored in the computer memory.

4. Spectral analysis where the power (spectral energy, electrical activity) in a given frequency range is determined, the most commonly used algorithm is the Fast Fourier Transform algorithm (FFT). The frequency analysis is frequently called Fourier analysis *(Gotman,et al , 1975).*

5. Results of spectral analysis are represented in:

A) Graphical forms:

1) A histogram; where only frequency and power are considered in this presentation.

2) Three dimensional display; where time is added to the previous parameters. This method is particularly helpful for obtaining an overall view of the changes in the EEG spectra for monitoring purposes in the operating room.

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B)   Topographic forms:

This form shows the topographic spatial distribution of spectral energy, so the process called brain electrical activity mapping was introduced (Am*inoff, 1981)*.

II) BEAM Recording :

Brain electrical activity mapping (BEAM) is a name given to the topographic display of the spectral energy in a given frequency range *(Lombroso, et al,1981)*.

A variety of names was given to this methodology for example *Duffy, et al,(1979) and (1984),* called this technique brain electrical activity mapping (BEAM); *Gaches, (1985), Etevonon, (1986), and Sebban, et al, (1984) and (1986),* called this technique EEG cartography; *Nagata, et al, (1982) and (1984)* called it computerized mapping of the EEG (CME); *Pidoux, et al, (1980),* called it computerized topography of the EEG (CTEEG).   All are different names for one and the same methodology.

Although BEAM can be traced as far back as *Grey Walter, (1943),* who built a low frequency analyzer and a new toposcopic display system, the recent utilization of microprocessors specialized in EEG frequency analysis has far advanced the clinical utility of EEG mapping which appears like a Diaspora and there is growing usage of EEG mapping among classical electroencephalographists. Moreover, EEG mapping has been correlated by *Duffy, et al,(1979), (1981), and (1984), Etevonon ,(1986), Gashes, (1985), Nagata, et al,(1982), Morstyn, et al,(1983) and Buchsbaum , et al,(1982),* with other imaging techniques such as computed tomography scans, emission tomography and local cerebral circulation.

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While producing the EEG map six issues confront the EEG researcher *(Etevonon, 1986):*

1.   Choice of number of electrodes [4-8-12-16-20-32-48-58].
2.   Position of electrodes.
3.   Choice of reference [common average reference monopolar-bipolar].
4.   Choice of spectral parameter.
5.   Choice of interpolation algorithm.
6.   Choice of statistics prior to EEG map·

The **first** issue is choice of the number of electrodes. It is generally known that too many electrodes are better than too little *(Etevonon, 1986).* *Duffy and his school, (1979),* are using 20 electrodes for their beam methodology. *Buchsbaum, et al, (1982), Etevonon, (1986), Sebban, et al,(1984) and (1986), Pidoux, (1983), and Gaches, (1985),* are using 16 electrodes. *Itil, (1985),* used only 8 electrodes for his dynamic brain mapping methodology. Anyhow, it is generally agreed that the number of the electrodes used depends upon the research goals*(Etevonon, 1986).*

The **second** issue is the position of the electrodes over the scalp which is crucial for a meaningful topography of the computed EEG maps. Generally, the 10-20 system is chosen as a basic grid for the EEG electrode location *(Etevonon, 1986).*

The **third** issue is the choice of the electrode reference. The best known choice is the common average reference rather than the monopolar technique which present lower amplitudes near the monopolar positioning of the reference *(Etevonon, 1986).*

The **fourth** ·issue is the choice of spectral parameters to be mapped. In spite of the fact that any of the spectral parameters could be mapped, the most commonly used are the relative power and the absolute power in the classical frequency bands. However, the choice depends on the goals of the researcher *(Etevonon, 1986).*

The **fifth** issue is the choice of the interpolation algorithm. During EEG mapping, the spectral energy is only calculated at

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the electrode sites which are placed according to the 10-20 system. As the EEG maps must represent the real power values from the whole surface of the scalp, then the spaces between the electrode sites on the EEG maps must be filled with numerical power values close to the real values. Filling the spaces between the electrode sites on the EEG map is called "interpolation" *(Etevonon, 1986)*. A variety of interpolation algorithms were proposed, such as *Buchsbaum, et al, (1982) and Duffy, et al,(1982),* that are applying a weighting function to inverse to the distance of the proximal experimental measured EEG values (linear interpolation). *Duffy, et al (1982)* used triangular linear interpolation (3 points), while *Buchsbaum, et al (1982)* used quadrilateral (4 points) interpolation.

Nuwer, (1985), concluded that EEG maps represent the most straight-forward way to represent the electrical data. He commented that the EEG maps are an inherently insufficient means of representing the electrical data, because a typical EEG map represents only a few points of real data, while 99% of the display are just interpolation between the few real data points.

The **sixth** issue is the choice of the statistics. *Duffy,et al,(1981, 1982, 1984 and 1986),* introduced and improved two statistical methods:

1) Significance probability mapping (SPM): Its main goal is to make the visible data characteristic that otherwise might remain obscured. Two types are known:

 a) t-statistic SPM: used to statistically delineate regional difference between a group of subjects, by using the student's t-test statistics.

 b) Z-statistic SPM: used to statistically delineate abnormal region during clinical evaluation of the BEAM images. It defines regions in which brain electrical activity from an individual subject differs statistically from that of a reference population.

2) Grid sector analysis (GSA): Its main role is to develop numerical measures of the degree of abnormality *(Duffy , et al,1981).*

Duffy, et al,(1984), concluded that the abnormality in the BEAM image

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is best detected by the SPM in which the images are compared to those of the normal control subjects by z-transform or student's t-test and the results are converted into an image of differences.

III) Description of the Normal EEG Maps:

During the study of EEG maps, the neurophysiologist is frequently confronted with the problem of defining the normal EEG map. *Sebban, (1986),* counted the points to be taken in consideration as follows:

1. The spectral parameter mapped i.e. absolute power, relative power or alpha/theta ratio.
2. The topographic distribution of the spectral energy in the frequency band represented on the map.
3. Effects of the eye opening on the power values.

Sebban, (1986), stated that normal EEG maps representing the absolute energy on the classical frequency bands, have the following criteria:

1. The distribution of the power values is rather diffuse. No focci are present
2. For all frequency bands, the maximum power values are topographically located posteriorly forming a crown in the parieto-occipital region.
3. The distribution of the spectral energy in the EEG map shows good degree of symmetry, but slight asymmetry might be present with spectral energy more on the right side than on the left side especially in the occipito-parietal region which should be regarded as a normal physiological finding.
4. With eye opening, the spectral energy for all frequency bands tends to decrease *(Sebban , 1984,and 1986).*
5. High voltage delta activity might be present in the frontal and pre-rolandic area especially on the left side which represents artifact due to eye blinking or eye opening.

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Therefore, it is necessary to determine by comparing with conventional EEG interpretation whether these high voltage data foci in the frontal region represent rhythmic slow activities due to eye movements and/ or blinks or genuine focal slow components *(Duffy , et al , ,1982,and1984.).*

Regarding the relative power EEG maps, *Pidoux, (1983),* reported that the topographic distribution of the relative power values are different from those of the absolute power values in the following points :

1 Relative delta and theta power values are maximum in the frontal region.
2. Alpha-relative power is maximum in the occipital region.
3. Beta-relative power is maximum in the rolandic and temporal zone.
4. Some degree of asymmetry is present, the intensity of the relative power is more on the right side than on the left side, and may be represented by a much wider area on this side. *Pidoux (1983).*

IV) BEAM in ADHD:

Latachkina and Puchiskaia , (1991) , examined 33 children with a normal mentality for EEG , and distinguished 2 different interhemispheric relations , depending on EEG types .Children with hyperactivity had weak interhemispheric correlations by the alpha rhythm together with their potentiation according to the beta rhythm and with a decrease of positive interhemispheric asymmetries .

Turgay , et al , (1992), compared EEG , quantitative EEG (QEEG) and Dynamic Brain Mapping (DBM) among 50 children with ADHD and stated that whereas routine EEG reported the prevalence of borderline and pathological findings , QEEG and DBM , were found to be more sensitive to identify abnormalities associated with seizures , drug effects and other primary EEG abnormalities .They concluded that , QEEG and DBM are valuable in clinical

practice and research and exceeding the data provided by routine EEG .

Ackerman , et al , (1994) , examined 100 ADHD cases by measuring EEG power spectra using bilateral temporal and parietal sites , and 4 midline sites . They found greater low beta band difference than the controls , with greater power on the right site in delta and alpha bands .They concluded that the combination of greater low beta and less theta power significantly predicted better progress .

Ackerman , et al , (1995), compared EEG spectra in poor vs adequate readers and found that adequate readers had higher beta values than poor readers indicating the lesser engagement of poor readers in the task .

Chabot , et al , (1996), stated that pretreatment QEEG could be utilized to distinguish ADHD children who responded to dextroamphetanine from those who responded to methylphenidate , with a high level of accuracy .They also stated that QEEG should be considered as a general utilization basis for the initial evaluation of children with ADHD and / or learning disorder .

Kuperman , et al , (1996), examined 40 ADHD children based on DSM – III –R criteria by spectral EEG and found that ADHD subjects had increased beta band relative percent power . They stated that interhemispheric asymmetries in QEEG using ERP findings may prove useful in differentiating specific ADHD subtypes namely ADHD and undifferentiated ADHD .

DeFrance , et al , (1996) , compared EEG spectral difference in children according to DSM-IV criteria and found that ERP components (P250 , P350 , P500,)differed markedly between ADHD – AD and ADHD-HI subtypes .

Chabot and Serfontein, (1996),compared QEEG in 407 ADHD children & 310 normal children and found that 90% sensitivity and specificity to distinguish normal children from those with ADHD .They defined 2 subtypes : the first was varying degrees of EEG sl owing especially in frontal regions

while the second was an increase in EEG activity especially in frontal region.

Subjects and Methods

Subjects and Methods

I -Subjects :

Seventy hyperactive children were chosen from patients attending the Pediatric Neuropsychiatric Outpatient Clinic , Ain Shams Univ. Hospital in the period from January 1st 1994 to the end of December 1997.

The group composed of 56 males and 14 females with a sex ratio of 4:1 and ages ranged from 6.1 ± 1.8 y .

a)Criteria for inclusion :
   1-Hyperactivity
   2-Inattention
   3-Imulsivity
   4-Duration of illness not less than 1 year
   5-Onset before the age of 7 years
b)Criteria for exclusion :
   Any recognizable mental , genetic , endocrinal or metabolic psychological disorder .
c)Medications :
   Patients on medications continued their medicines , while for those not receiving any treatment , specific medication was prescribed .

II- Methods :

All cases were subjected to the following :

1- Full medical , social and behavioral history taking from patients.
2- Complete thorough physical and neurological examinations . Using the general neuropsychiatric examination sheet , stressing on SES , consanguinity ,

perinatal history , family size , birth order and family breakage .

3- ADHD rating score :
   Using the Abbreviated Teacher Rating Score (ATRS) *(Conners,1969).*

4- Intelligence Test (IQ):
   Using Good -Enough –Harris Drawing Intelligence Test *(Harris , 1963).*

5- Socio-economic status scoring system (SES):
   Using *Park and Park classification (1979).*

6- Brain Electrical Activity Mapping (BEAM) using Nicolet BEAM-II machine (1990),

III- Statistical Methods :

All statistical analysis was done using an IBM-PC computer Statgraphic Statistical Package, Version 3, 1988.

The statistical methods can be summarized as follows:
$$\text{(Castle, 1977 and Allam, 1993).}$$

1- **The mean:** It is the measure of central tendency. It is represented by the symbol $\bar{x}$ (called x-bar), where Σ is sum, x is individual variables, and N is number of values.
$$\bar{x}=\Sigma x/N$$

2- **The standard deviation (S.D.):** It is a measure of variance of individual data around its arithmetic mean.

$$S.D=((x^2 -(x/N)^2)/(N-1))^{1/2}$$

3- **Chi-square test (x^2):** It is used to find the difference between proportions.

$$X^2=\Sigma \ [(O-E)^2/E]$$

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Where :

$X^2 =$ Chi-square

O = observed values.

E = expected values.

Then probability of being by chance "p" is calculated. The difference between proportion is considered statistically significant, if the "P" value is 0.05 or less. A probability of 0.001 or less indicated that the difference is highly significant.

4- Student's t-test: To test significance of difference between the means.

Paired t-test =

$(x_1 - x_2) / \{sqrt [(SD_1)^2 / N1 + (SD_2)^2 / N_2]\}$,

where:

x_1 = mean of 1st group.

x_2 = mean of 2nd group.

SD_1 = standard deviation of data in the 1st group.

SD_2 = standard deviation of data in the 2nd group.

N1 = Number of cases in the 1st group.

N_2 = Number of cases in the 2nd group.

The difference was considered significant if: the "p" value is 0.05 or less. This means that the observed difference is real and not a matter of chance. A probability of 0.001 indicates that the difference is highly significant.

5-Tukey's Honestly Significant Test (HSD) : To test all the possible double comparison among equally sized samples.

$$HSD = \frac{\overline{X}_A - \overline{X}_B}{\sqrt{\dfrac{m}{n}}}$$

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Where :

$$\overline{X_A} = \text{mean of group to be compared}$$
$$\overline{X_B} = \text{the smallest mean}$$
$$M = \text{mean of sum of squares of groups}$$
$$N = \text{Number of cases in each group.}$$

The procedures used were :

• <u>ATRS of Conners</u>

Sleater and Ullman ,(1981), have used the convenient Conners Abbreviated Teacher Rating Scale **(ATRS Score)** extensively. The range of possible scores on this scale is zero for no hyperactive behaviors to 30 for the extreme of all the ten listed behaviors (Table2).

The mean score for randomly selected subjects was 4 (according to *Sleater and Ullman, 1981),* and the mean score for children who had been diagnosed hyperactive was 22. Following a common practice, the hyperactivity "cut off point" was set at 2 SD above the normal mean. In their study there was 14 (45%). These norms, or minor variations, are now widely used in research *(Sleater and Ullman, 1981).*

Mothers and/or fathers were asked the ten questions of ATRS in colloquial Arabic, to answer each of them by either No, Sometimes, Much, or Very Much. The activities were scored as follows:

Zero degrees for No.
One degree for Sometimes.
Two degrees for Much.
Three degrees for Very Much. *(Conners, 1969).*

Conners Abbreviated Teacher Rating Scale (ATRS).

Observation		Degree of activity			
		No	Sometimes	Much	Very Much
1	Restless or overactive	-----	-----	-----	-----
2	Excitable, impulsive	-----	-----	-----	-----
3	Disturbs other children	-----	-----	-----	-----
4	Fails to finish things he starts, short attention span	-----	-----	-----	-----
5	Constantly fidgeting	-----	-----	-----	-----
6	Inattentive, easily distracted	-----	-----	-----	-----
7	Demands must be met immediately — easily frustrated	-----	-----	-----	-----
8	Cries often and easily	-----	-----	-----	-----
9	Mood changes quickly and drastically	-----	-----	-----	-----
10	Temper outbursts, explosive and unpredictable behavior	-----	-----	-----	-----
Subtotal Score		x 0	x 1	X 2	x 3
Total Score			----- 30		

(Conners, 1969)

Table 2.

• <u>IQ test of Good –Enough – Harris Drawing Intelligence Test</u>

This test was applied to all children in a group manner about 5 children at a time. The following rules were fulfilled during the proceeding of this test:

1) Before beginning, all pictures of men and women were kept away, so that children could not look at any picture during the test.

2) Every child was given a pencil, eraser and paper. The papers were labeled with the child's name and age.

3) The following instructions were given in colloquial Arabic to the children: "Draw a picture for a man, making the best picture that you can."

4) Some children needed encouragement to continue the test. During this encouragement, comment on the children's pictures was avoided.

5) There was no time limit, but most children spent 10-15 minutes to complete the picture.

6) The drawing was scored for completeness (for 73 items), e.g. the presence of head, neck, eyes, arms, hands, fingers and so on, and not for beauty.

7) Scores were converted to I.Q. from special tables according to age taking in consideration that:

I.Q.= (mental age / chronological age) x 100.

8) Scores of 100 or more were considered above average IQ ,while scores less than 100 were considered below average IQ.(*Harris, 1963)*

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• <u>SES classification of Park and Park</u>

<u>Socioeconomic standard (SES)</u> was estimated for each subject according to *Park and Park classification , (1979)*. This classification depends on giving scores to the educational level and the occupation of both parents:

1.Educational level scoring:

Illiterate	1
Primary Education	2
Preparatory Education	4
Secondary Education	5
University Graduate	7

2. Occupational level scoring:

No occupation or unskilled worker	1
Industrial worker	2
Skilled worker	4
Semi-professional worker	5
Professional	7

3. Socioeconomic standard is classified as:

Scores less than or equal to 8	= Low
Scores between 9-18	=Middle
Scores between 19-28	= High

(Park and Park , 1979)

• <u>Brain Electrical Activity Mapping (BEAM)</u>

Subjects were instructed to wash head and hair the day before the test, adding no oils or shampoo and insuring clean and dry hair before starting. Routine EEG and BEAM tests were performed simultaneously during a single test sitting. The EEG was obtained using a 25 channel GRASS polysomnograph machine. BEAM tests were performed on a NICOLET BEAM-II computer through a program named BEAM. Brain mapping was carried out through the following steps while patient was sitting:

1. A 20-electrode cap (according to the International 10-20 system) is fixed over the scalp of the patient. Electrodes have holes that allow a special gel to be introduced by using a special blunt needle and a 5-ml syringe, insuring good electrode contact to the skin. After filling all the 20 electrode holes with the gel, impedance is measured by an impedancemeter keeping it below 5 KOhms. Subjects and machines were kept in soundproof, semi-darkened room with controlled lights. Two reference electrodes were connected to both ears, 2 neck electrodes, and 4 facial electrodes are connected to detect any artifact that may arise from eye, head and neck movements. Subjects were instructed to avoid any facial, limb or eye movements. Special care is taken to minimize the effect of eye movement and / or blinking, which constitute a major problem in computer analysis of EEG. *Morstyn, et al,(1983),* mentioned that eye movement and eye blinking are monitored off line during EEG recording by reducing their frequency and extent through the following maneuvers:

a) Using fixed target to limit eye movement.
b) Using large recording session to obtain sufficient segments free

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AMPLIFIER

POLYGRAPH

EMG

MAGNETIC TAPE
Protocol instructions
are delivered by earphones.

The signal is delivered
to the polygraph and
the 25 channel recorder simultaneously.

25 CHANNEL RECORDER

The signal is marked and
recorded.

SPECTRAL ANALYSIS

FOURIER ANALYSIS

MICRO_COMPUTER
Storage and map
representation.

The digitalized signal is converted into frecuency
histograms.

Fig1 Schematic diagram showing the method of recording BEAMfindings

of artifacts.

c) Allowing frequent rest breaks for subjects to rest eyes.

d) Instructing the subject to blink suppresses blinking.

2. Raw EEG data and BEAM signals were recorded for the same time at eyes-opened and eyes-closed for a suitable recording time segment - each of 2 seconds duration at end of which recording was stopped, and subject was disconnected from the machines.

3. Raw EEG data was carefully inspected on the computer screen allowing to exclude any segment containing artifact.

4. Data was then treated by:

a) Filtration of waves from 1.5 to 30 Hz to remove artifacts due to eye movement that usually contaminate the slow delta range below 1.5 Hz, and to remove EMG signals due to muscle movements that contaminate frequency at about 30 Hz.

b) The remaining EEG was analyzed by a spectrum analyzer using the real time Fast Fourier Transform algorithm (FFT) which yields a histogram of power values for each frequency and for each electrode.

5. Map presentation:

a) Single values are obtained from each electrode representing the amount of energy in a given frequency band.

b) The calculated voltage values are plotted on the head-shaped array.

c) The space in between electrode sites on the head-shaped array is filled by interpolation. The power at a specific point is determined by the power values at the 3 or 4 nearest electrodes according to the interpolation algorithm.

d) The system then calculates for each point on the map a value interpolated between all the 16 original values, which are an inverse square ratio of the distance. Each value being calculated is related to one of the 12 colors of the scale. Red color was used to designate the highest percent of power areas, the blue for the lowest percent power areas. The power values run through the colors of the rainbow from white to red to

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reddish-yellow to yellow to yellowish-green to green to greenish-blue to blue *(Lambroso, et al, 1980, 1982).*

Buschbaum, et al, (1982), mentioned that the advantages of color EEG maps are:

i) Colors ADHD an authentic touch to the EEG map.

ii) Colors give more power levels.

6. Data analysis: The net result of BEAM data analysis for each of the following frequency bands (Delta 0.0-0.3 Hz, Theta *3.0-7.5* Hz, Alpha 7.5-13.5 Hz, Beta-1 13.5-18.5 Hz, Beta-2 *18.5-30* Hz) will be expressed in two forms:

a) Digitally: and is expressed as:

i) Relative power (percent power of each frequency band to total EEG power) of each of the delta, theta, alpha, and beta frequency bands.

ii) Power ratio index =

(% Power delta + % Power theta) / (% Power alpha + % Power beta).

b) Topographically: As the topographic mapping of the spectrally analyzed EEG data *(Nicolet, 1990).*

Results

Results

In the present piece of work , BEAM was studied on 70 children , who were diagnosed as hyperactive by both clinical observation and using the ATRS of *Conners , (1969).* No control group was chosen as the BEAM-II computer machine had an in-built control to which the analyzed patient's results are compared to find out after processing whether there is any abnormality or not , and locating its position and its frequency on the SPM .

The results obtained are shown in tables (3 – 46) and figures (2 – 25) .These results were statistically analyzed according to the student's (t)test and correlation between different parameters were done by calculation of the correlation co-efficient (r) and the Chi-square test (X^2) .

Table 3 shows the collective raw data obtained from the 70 cases of the study .Qualitative data includes :sex , consanguinity , perinatal history , diagnosis , SES , family breakage and BEAM findings .Quantitative data includes :age,order of birth , family size , ADHD score and IQ.

No	SEX	AGE	BIR. ORD.	FAM SIZE	CONSANG.	PERI H	ADHD SCORE	DIAGN.	IQ	SES	BRK. FAM	BEAM
1	M	6.1	2	4	0	N	16	P	86	M	N	N
2	M	9.2	1	1	0	N	16	P	83.5	H	P	N
3	M	11.2	2	3	2	N	21	P	97	M	N	N
4	M	9	1	3	3	N	20	P	89	M	N	N
5	F	9.2	1	2	0	N	18	P	111	M	N	N
6	M	8.1	2	3	2	P	18	P	94	M	P	S
7	M	10.3	1	3	1	N	16	P	89.5	M	P	N
8	M	6.3	1	4	0	N	21	C	115	L	N	C
9	M	6.2	1	3	3	N	19	P	87.5	M	N	S
10	M	9.3	1	2	0	N	20	P	118.5	H	P	N
11	M	11.3	1	4	0	O	22	C	102	M	N	S
12	F	6.5	1	3	2	N	18	P	81	L	N	N
13	F	9.1	3	5	0	N	16	P	83	M	N	N
14	M	8.1	1	3	3	P	18	P	83	M	P	N
15	M	11.1	1	4	0	N	15	P	121	M	N	N
16	M	12.2	1	5	2	O	23	C	84.5	M	N	S
17	M	8.3	1	4	0	N	14	P	90	M	P	N
18	M	7.2	1	3	0	N	16	P	110.5	M	N	N
19	F	7.2	1	2	0	N	17	P	104.5	H	N	N
20	M	11.1	1	5	0	P	22	P	91	L	N	N
21	M	9.2	1	3	1	N	17	P	108.5	M	N	N
22	M	8.7	1	3	2	N	21	O	87	M	N	S
23	M	10.1	2	4	1	O	19	P	88.5	M	P	N
24	F	11.2	1	2	0	N	16	P	104.5	M	N	N
25	M	6.5	1	3	3	N	15	P	106	M	N	N
26	M	10	1	5	0	O	21	C	110	M	N	C
27	F	6.2	1	2	0	P	16	P	111.5	H	N	N
28	M	10.1	1	4	0	N	20	O	81	M	P	S
29	F	7.7	1	3	0	O	18	P	85	L	N	N
30	M	11.8	2	3	0	N	21	O	82	M	N	N
31	M	8.1	3	5	0	N	16	P	104.5	M	N	N
32	M	10	1	6	1	P	17	P	87	L	N	N
33	M	8.7	1	4	0	N	15	P	87	M	P	N
34	M	10.4	1	2	2	O	20	O	111	H	N	F
35	M	12.3	1	3	0	N	22	O	84	M	P	N
36	M	9.2	2	3	3	O	21	C	124	M	N	S
37	M	6.1	1	3	1	N	20	O	82	L	P	N
38	M	11.2	2	3	0	N	22	O	110.5	M	P	N
39	M	7.5	1	2	0	P	20	P	83	M	N	N
40	M	10.3	1	5	2	P	19	P	112.5	L	N	N
41	M	12.3	1	4	0	N	24	O	95	M	P	C
42	F	10.2	1	3	0	N	16	P	123.5	H	N	N
43	M	11.8	1	4	0	N	15	P'	104	M	N	N
44	F	10.8	2	7	0	O	27	O	89.5	L	N	N
45	M	9.7	1	3	0	N	17	P	108.5	M	P	N
46	M	11.5	2	4	2	N	19	O	109.5	L	N	N
47	M	9.2	5	7	0	O	16	P	82	M	P	N
48	M	7.3	1	3	0	N	20	P	128.5	M	N	N
49	M	10.5	3	5	3	N	21	O	110.5	M	N	S
50	M	8.4	2	2	0	P	15	P	109	M	N	N
51	F	10.3	4	7	1	O	19	P	83	L	N	N
52	M	12.2	2	5	0	N	22	P	110	M	P	N
53	M	9.5	1	2	0	O	18	O	86	H	P	N
54	F	8.1	1	1	0	N	23	O	109	H	N	F
55	M	8.8	1	4	0	N	16	P	107	L	N	N
56	M	10.1	1	4	0	N	19	O	107	M	N	C
57	M	11.7	2	5	3	P	23	P	83	L	P	N
58	f	6.9	1	3	2	N	21	O	106	M	N	F
59		9.7	1	3	0	P	19	O	110	M	P	N
60			2	4	0	N	17	P	84	M	N	C
61	M		1	5	1	N	20	P	105.5	L	N	N
62	M	.5	1	3	0	N	18	P	106	H	N	N
63	M	11.4	3	5	2	N	19	P	103.5	M	P	N
64	M	12	1	4	2	P	22	O	88	L	N	N
65	M	11.9	2	2	0	N	19	P	110.5	M	N	N
66	M	7.4	1	3	0	N	21	O	88.5	M	N	N
67	F	11.8	1	6	1	N	20	O	85	M	P	C
68	M	10.7	1	2	0	O	21	C	114	H	N	N
69	M	8.6	1	3	0	N	22	O	88	M	P	F
70	M	9.7	1	5	1	N	22	P	89.5	L	N	N

TABLE 3
RAW COLLECTIVE DATA

(101)

Fig 2
A pie chart showing the percentage frequency of sex among ADHD cases

MALE
80.0%

56

14

FEMALE
20.0%

SEX

(102)
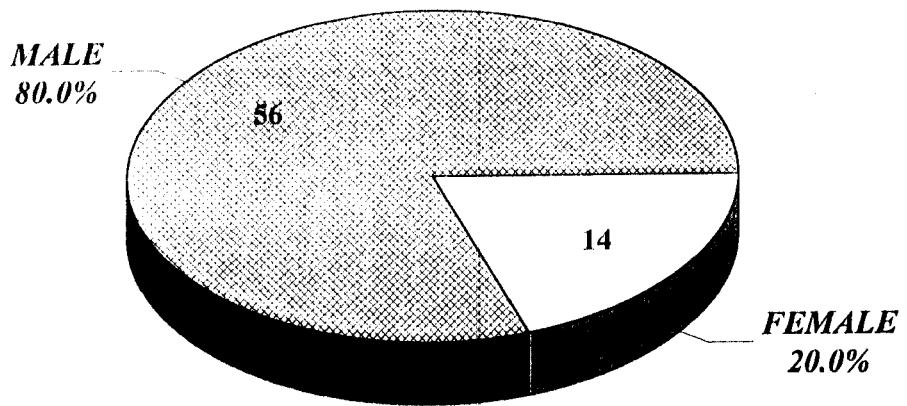
TABLE 4
SEX RATIOS AND PERCENTAGE FREQUENCY OF BOTH SEXES AMONG DIFFERENT TYPES OF ADHD

VARIABLE / SEX	Diagnosis							
	1ry (n=44)			2ry (n=26)				
						Total		
	No	%	Ratio	C	O	No	%	Ratio
Male (n=56)	34/56	60.7	3.4	6	16	22/56	39.3	5.5
Female (n=14)	10/14	71.4	1	0	4	4/14	18.6	1
X2	1.7							
p	0.42 (NS)							

This table shows that males are predominantly
affected more than females in all types of ADHD , and
the whole ratio of male to female is 4:1.Also in both
sexes , the incidence of primary ADHD is significantly
higher than that o f secondary type

TABLE 5
MEAN VALUES OF IQ LEVELS AMONG MALES VS FEMALES AND SEX RATIOS

VARIABLE / SEX	IQ				
	<100 (n=35)		100- (n=35)		Total (n=70)
	No	Ratio	No	Ratio	Mean+/-SD
Male (n=56)	28	4	28	4	98.8+/-12.9
Female (n=14)	7	1	7	1	97.5+/-14.4
X2	0.09				
p	0.76 (NS)				

This table shows that there is no significant sex
difference as regards mean IQ levels in the studied
cases of ADHD (P > 0.05) , and the sex ratio
is 4:1 to the side of males in both above and below
average IQ groups

(103)

TABLE 6
MEAN VALUES OF ADHD SCORE IN MALE VS FEMALE PATIENTS

VARIABLE / SEX	ADHD score				
	10-<20 (n=38)		20-30 (n=32)		Total (n=70)
	No	Ratio	No	Ratio	Mean+/-SD
Male (n=56)	27	2.5	29	9.7	19.2+/-2.6
Female (n=14)	11	1	3	1	18.6+/-3.1
X2	3.03				
p	0.08 (NS)				

This table shows that there is no significant sex difference as regards mean ADHD score (P> 0.05)

TABLE 7
PERCNTAGE FREQUENCY O F BEAM FINDINGS IN MALE VS FEMALE ADHD PATIENTS

VARIABLE / SEX	BEAM findings								
	Normal (n=52)			Abnormal (n=18)					
							Total		
	No	%	Ratio	S	F	C	No	%	Ratio
Male (n=56)	41/56	73.2	3	8	3	4	15/56	26.8	5
Female (n=14)	11/14	78.6	1	0	1	2	3/14	21.4	1
X2 .	2.77								
p	0.42 (NS)								

This table shows that there is no significant sex difference as regards the percentage frequency of both normal and abnormal BEAM findings in ADHD studied cases (P>0.05)

(104)

Fig 3
A pie chart showing the percentage frequency of birth order among ADHD cases

1st
71.4%

50

6

14

3rd OR >
8.6%

2nd
20.0%
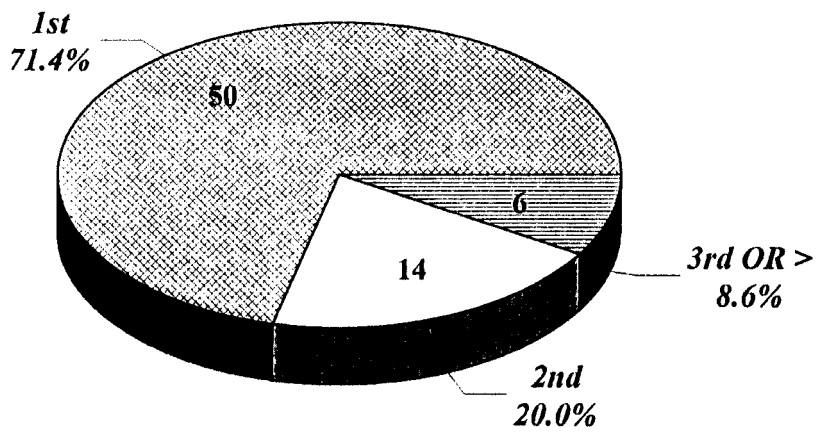
BIRTH ORDER

TABLE 8
BIRTH ORDER AND PERCENTAGE FREQUENCY OF NORMAL VS ABNORMAL PERINATAL HISTORY

VARIABLE	PERINATAL HISTORY					
	NORMAL (n=47)		ABNORMAL (n=23)			
					Total	
B.O.	No	%	P	O	No	%
1st (n=50)	35/50	70	8	7	15/50	30
2nd (n=14)	8/14	57.1	3	3	6/14	42.9
3rd OR > (n=6)	4/6	66.7	0	2	2/6	33.3
X2	2.03					
p	0.36(NS)					

This table shows that though first ordered child is more commonly affected (71.4%) than others , there is no significant difference as regards the birth order and percentage frequency of abnormal perinatal history in all birth ordered children (P>0.05)

TABLE 9
PERCETAGE FREQUENCY OF BIRTH ORDER IN PRIMARY VS SECONDARY TYPES OF ADHD

VARIABLE	DIAGNOSIS					
	1ry (n=44)		2ry (n=26)			
					Total	
B.O.	No	%	C	O	No	%
1st (n=50)	30/44	68.1	5	15	20/26	76.9
2nd (n=14)	9/44	20.5	1	4	5/26	19.3
3rd OR > (n=6)	5/44	11.4	0	1	1/26	3.8
X2	1.26					
p	0.53(NS)					

This table shows that there is no significant birth order difference as regards diagnosis in the studied ADHD cases (P>0.05)

(106)

TABLE 10
PERCENTAGE FREQUENCY OF BIRTH ORDER IN ABOVE VS BELOW AVERAGE IQ GROUPS

VARIABLE B.O.	I.Q.					
	<100 (n=35)			100- (n=35)		
	No	%	Mean+/-SD	No	%	Mean+/-SD
1st (n=50)	24/50	48	86.5+/-3.4	26/50	52	110.6+/-6.4
2nd (n=14)	8/14	57.1	88+/-5.4	6/14	42.9	112.3+/-5.8
3rd OR > (n=6)	3/6	50	82.7+/-0.6	3/6	50	106.2+/-3.8
X2	0.37					
p	0.83 (NS)					

This table shows that there is no significant birth order difference as regards IQ in the studied cases (P>0.05)

TABLE 11
PERCENTAGE FREQUENCY AND MEAN VALUES OF ADHD SCORES IN DIFFERENT BIRTH ORDERS

VARIABLE B.O.	ADHD score					
	10-<20 (n=38)			20-30 (n=32)		
	No	%	Mean+/-SD	No	%	Mean+/-SD
1st (n=50)	26/38	68.4	16.8+/-1.5	24/32	75	21.2+/-1.2
2nd (n=14)	7/38	18.4	17.6+/-1.6	7/32	21.9	22.4+/-2.1
3rd OR > (n=6)	5/38	13.2	17.2+/-1.6	1/32	3.1	21
X2	2.25					
p	0.32 (NS)					

This table shows that there is no significant birth order difference as regards both moderately and severely affected ADHD cases (P>0.05)

(1?7)

Fig 4
A pie chart showing the percentage frequency of family
size distribution among ADHD cases

SMALL
54.3%

38

32

LARGE
45.7%

FAMILY SIZE
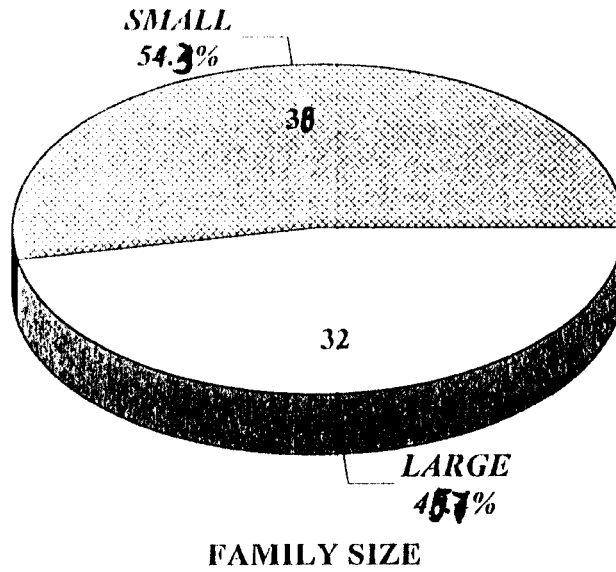
TABLE 12
PERCENTAGE FREQUENY AND MEAN IQ
LEVELS IN SMALL VS LARGE SIZED FAMILIES

VARIABLE F.S.	I.Q.					
	<100 (n=35)			100- (n=35)		
	No	%	Mean+/-SD	No	%	Mean+/-SD
SMALL <=3 (n=38)	17/38	44.7	86.5+/-4.3	21/38	55.3	111.7+/-6.6
LARGE >3 (n=32)	18/32	56.3	86.5+/-3.7	14/32	43.7	108.7+/-5.1
X2	0.52					
p	0.47 (NS) r=-0.26 (S)					

This table shows that there is no significant difference
in the mean IQ levels among small vs large sized
families . Yet there is a weak negative correlation
between mean IQ and family size by using regression
analysis test at P>0.05 (r = - 0.26)

(108)

TABLE 13
FAMILY SIZE AND PERCENTAGE FREQUENCY OF DIFFERENT TYPES OF ADHD

VARIABLE F.S.	Diagnosis					
	1ry (n=44)		2ry (n=26)			
					Total	
	No	%	C	O	No	%
SMALL <=3 (n=38)	24/38	63.2	2	12	14/38	36.8
LARGE >3 (n=32)	20/32	62.5	4	8	12/32	37.5
X2	0.04					
p	0.84 (NS)					

This table shows that there is no significant difference
as regards different types of ADHD in small vs large
sized families (P> 0.05)

TABLE 14
PERCENTAGE FREQUENCY AND MEAN VALUES OF ADHD SCORE IN SMALL VS LARGE SIZED FAMILIES

VARIABLE F.S.	ADHD score					
	10-<20 (n=38)			20-30 (n=32)		
	No	%	Mean+/-SD	No	%	Mean+/-SD
SMALL <=3 (n=38)	21/38	55.3	17.1+/-1.3	17/38	44.7	20.9+/-0.9
LARGE >3 (n=32)	17/32	53.1	16.9+/-1.8	15/32	46.9	22+/-1.8
X2	0.004					
p	0.95 (NS)					

This table shows that there is no significant difference
as regards percentage frequency and mean levels of
ADHD score in small vs large sized families
(P> 0.05)

(109)

Fig 5
A pie chart showing the percentage frequency of
consanguinity distribution among ADHD cases

POSITIVE
38.6%

27

43

NEGATIVE
61.4%

CONSANGUINITY

(110)
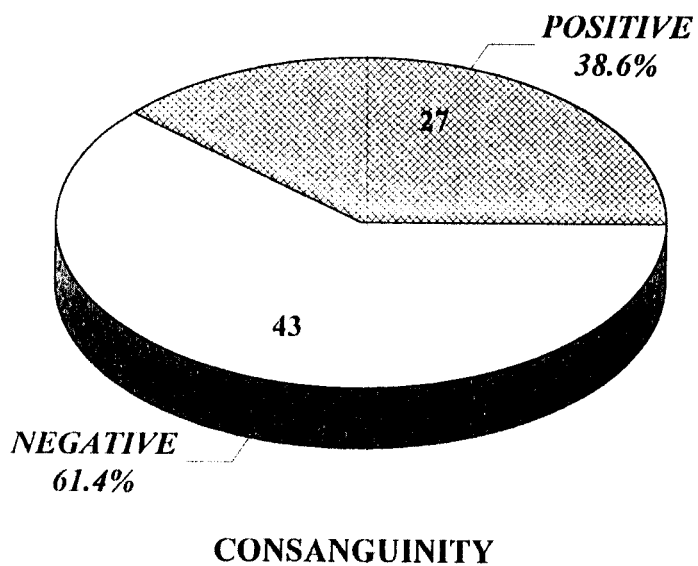
TABLE 15
PERCENTAGE FREQUENCY OF CONSANGUINEOUS MARRIAGE AMONG DIFFERENT SES IN ADHD PATIENTS

VARIABLE	SES					
	L (n=15)		M (n=45)		H (n=10)	
CONSANG.	No	%	No	%	No	%
NON C. (n=43)	5/43	11.6	29/43	67.4	9/43	20.9
CONS. (n=27)	10/27	37.1	16/27	59.3	1/27	3.7
X2	8.62					
p	0.01 (S)					

This table shows that the significant higher consanguineous rate (59.3%) is among the ~~low~~ middle SES group   (P=0.01)

Percentage frequency of consanguineous & nonconsanguineous marriages in low,middle & high SES levels

Legend: LOW(n=15), MIDDLE(n=45), HIGH(n=10)
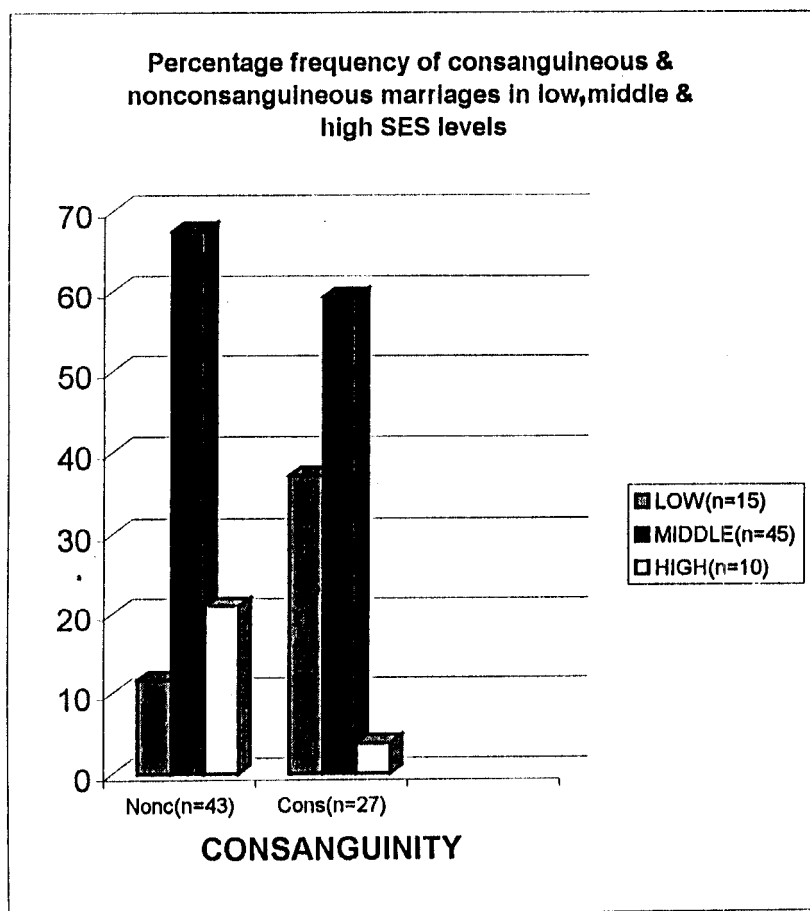
CONSANGUINITY

Fig.

(1)

TABLE 16
PERCENTAGE FREQUENCY OF CONSANGUINITY IN DIFFERENT TYPES OF ADHD PATIENTS

VARIABLE	Diagnosis					
	1ry (n=44)		2ry (n=26)			
					Total	
CONSANG.	No	%	C	O	No	%
NON C. (n=43)	27/44	61.4	4	12	16/26	61.5
CONS. (n=27)	17/44	38.6	2	8	10/26	38.5
X2	0.09					
p	0.95 (NS)					

This table shows that there is no significant consanguinity difference between primary and secondary types of ADHD and between different types of ADHD .Meanwhile the incidence of consanguineous marriage is higher (38.6%) in ADHD cases than in the general population (P>0.05)

TABLE 17
CONSANGUINITY RATE AMONG BELOW VS ABOVE AVERAGE IQ GROUPS

VARIABLE	I.Q.					
	<100 (n=35)			100- (n=35)		
CONSANG.	No	%	Mean+/-SD	No	%	Mean+/-SD
NON C. (n=43)	18/35	51.4	86+/-3.7	25/35	71.4	110.8+/-6.4
CONS. (n=27)	17/35	48.6	86.9+/-4.2	10/35	28.6	109.7+/-5.8
X2	2.17					
p	0.14 (NS)					

This table shows that in the below average IQ group the consanguinity rate is higher (48.6%) than that in the above average IQ group , in the ADHD cases as a whole , (38.6%), and in the general population as well .This difference is statistically insignificant (P>0.05)

(112)

TABLE 18
PERCENTAGE FREQUENCY AND MEAN VALUES OF ADHD SCORE IN CONSAGUIEOUS VS NONCONSANGUINEOUS ADHD CASES

VARIABLE / CONSANG.	ADHD score					
	10-<20 (n=38)			20-30 (n=32)		
	No	%	Mean+/-SD	No	%	Mean+/-SD
NON C. (n=43)	25/38	65.8	16.6+/-1.4	18/32	56.3	21.7+/-1.7
CONS. (n=27)	13/38	34.2	17.9+/-1.3	14/32	43.7	21.1+/-1.1
X2	0.33					
p	0.57 (NS)					

This table shows that although the consanguinity rate is of no significant difference in relation to moderate vs severe ADHD , the percentage frequency o fADHD is higher in consanguineous cases than in the general population (P>0.05)

(113)

Fig 7
A pie chart showing the percentage frequency of family
breakage among ADHD cases

POSITIVE
31.4%

22

48

NEGATIVE
68.6%
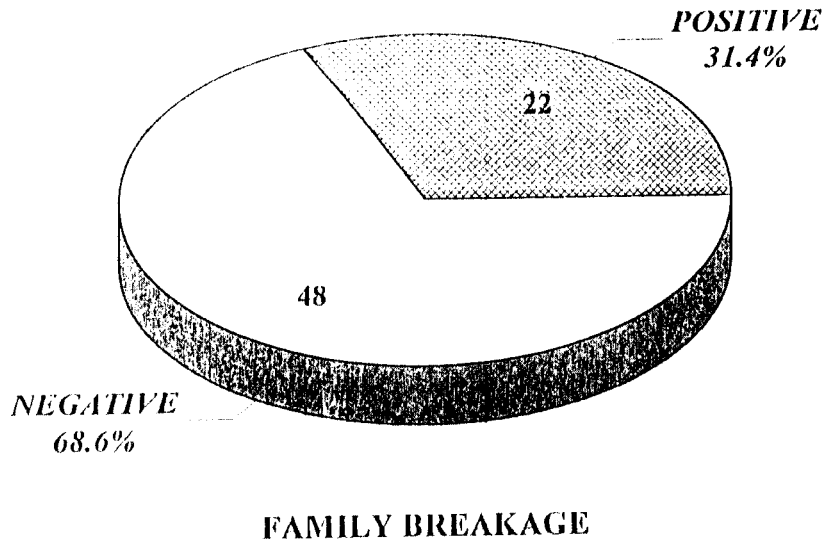
FAMILY BREAKAGE

TABLE 19
PERCENTAGE FREQUENCY OF FAMILY
BREAKAGE IN PRIMARY VS SECONDARY
TYPES OF ADHD

VARIABLE	Diagnosis					
	1ry (n=44)		2ry (n=26)			
					Total	
F.B.	No	%	C	O	No	%
NEGATIVE (n=48)	31/44	70.5	6	11	17/26	65.4
POSITIVE (n=22)	13/44	29.5	0	9	9/26	34.6
X2	4.53					
p	0.1 (NS)					

This table shows that the percentage frequency of
family breakage is insignificant statistically as regards
primary vs secondary ADHD . The family breakage
rate is higher (34.6%) in secondary ADHD cases than
in both the whole ADHD cases and the general
population (P<0.05)

(114)

TABLE 20
PERCENTAGE FREQUENCY OF FAMILY
BREAKAGE IN BELOW VS ABOVE AVERAGE IQ
GROUPS

VARIABLE	IQ						
	<100 (n=35)			100- (n=35)			Total (n=70)
F.B.	No	%	Mean+/-SD	No	%	Mean+/-SD	Mean+/-SD
NEGATIVE (n=48)	19/35	39.6	86.6+/-3.8	29/35	60.4	110.6+/-6.5	101.1+/-13.1
POSITIVE (n=22)	16/35	72.7	86.3+/-4.2	6/35	27.3	110.2+/-4.8	92.8+/-11.7
X2	5.36						
p	0.02 (S)						

This table shows that while 72.7% of below average
IQ cases are from broken families only 39.6% are from
unbroken families .This is of statistical significance
(P<0.05)

Fig 8

(115)
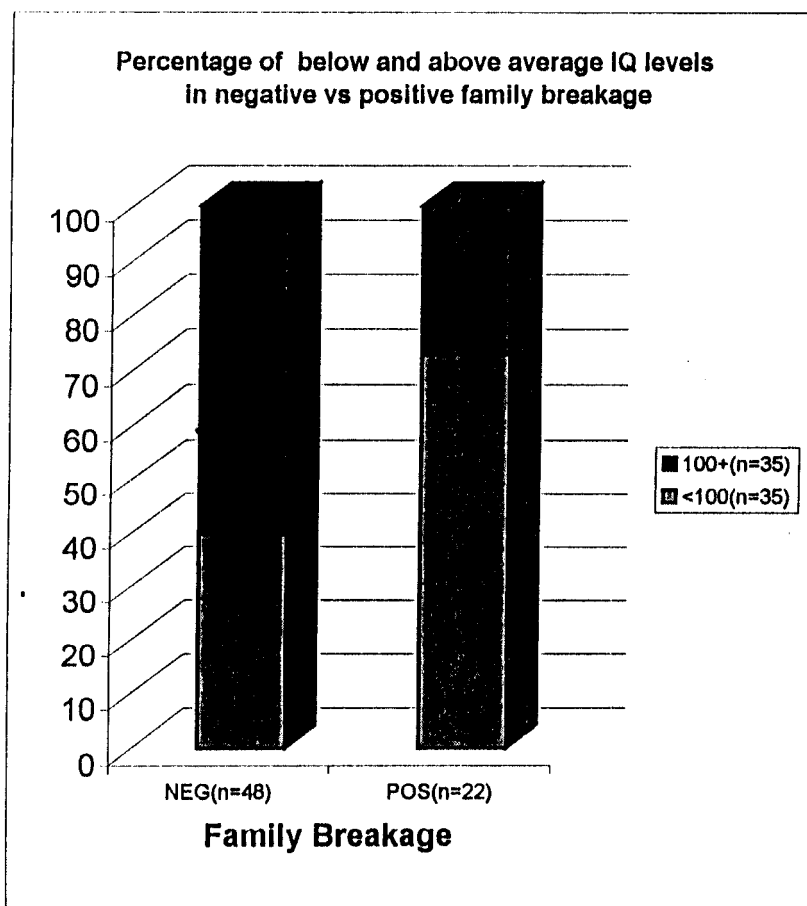
TABLE 21
PERCENTAGE FREQUENCY OF FAMILY
BREAKAGE IN MODERATE VS SEVERE ADHD
SCORE

VARIABLE / F.B.	ADHD score						
	10-<20 (n=38)			20-30 (n=32)			Total (n=70)
	No	%	Mean+/-SD	No	%	Mean+/-SD	Mean+/-SD
NEGATIVE (n=48)	26/48	54.2	17+/-1.4	22/48	45.8	21.4+/-1.5	19+/-2.7
POSITIVE (n=22)	12/22	54.5	17.1+/-1.7	10/22	45.5	21.5+/-1.4	19.1+/-2.7
X2	0.5						
p	0.82 (NS)						

This table shows that there is no significant family
breakage difference as regards to different grades of
ADHD scores in ADHD cases (P> 0.05)

TABLE 22
PERCENTAGE FREQUENCY OF FAMILY
BREAKAGE IN CONSANGUINEOUS VS
NONCONSANGUINEOUS ADHD PATIENTS

VARIABLE / F.B.	CONSANGUINITY			
	P (n=27)		N (n=43)	
	No	%	No	%
NEGATIVE (n=48)	19/27	70.4	29/43	67.5
POSITIVE (n=22)	8/27	29.6	14/43	32.5
X2	0.06			
p	0.79 (NS)			

This table shows that there is no significant family
breakage difference between consanguineous and
nonconsanguineous ADHD cases   (P> 0.05)

(116)

Fig 9
A pie chart showing the percentage frequency of
different SES levels among ADHD cases

LOW
21.4%

15

45

10
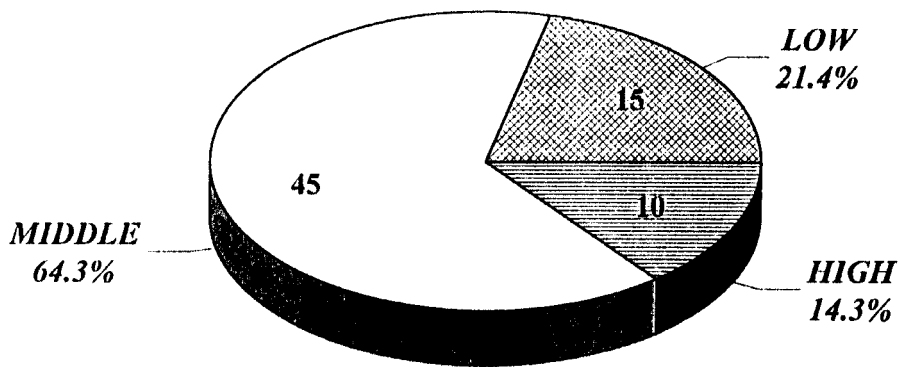
MIDDLE
64.3%

HIGH
14.3%

SOCIO-ECONOMIC STATE

TABLE 23
PERCENTAGE FREQUENCY OF FAMILY
BREAKAGE IN RELATION TO SES

VARIABLE	F. BREAKAGE			
	N (n=48)		P (n=22)	
SES	No	%	No	%
LOW (n=15)	13/15	86.7	2/15	13.3
MIDDLE (n=45)	28/45	62.2	17/45	37.8
HIGH (n=10)	7/10	70	3/10	30
X2	8.62			
p	0.01 (S)			

This table shows that there is a significant family
breakage difference in relation to SES level , being
highest (37.8%) in the middle SES and lowest (13.3%)
in the low SES   (P< 0.01)

Fig 10

(118)
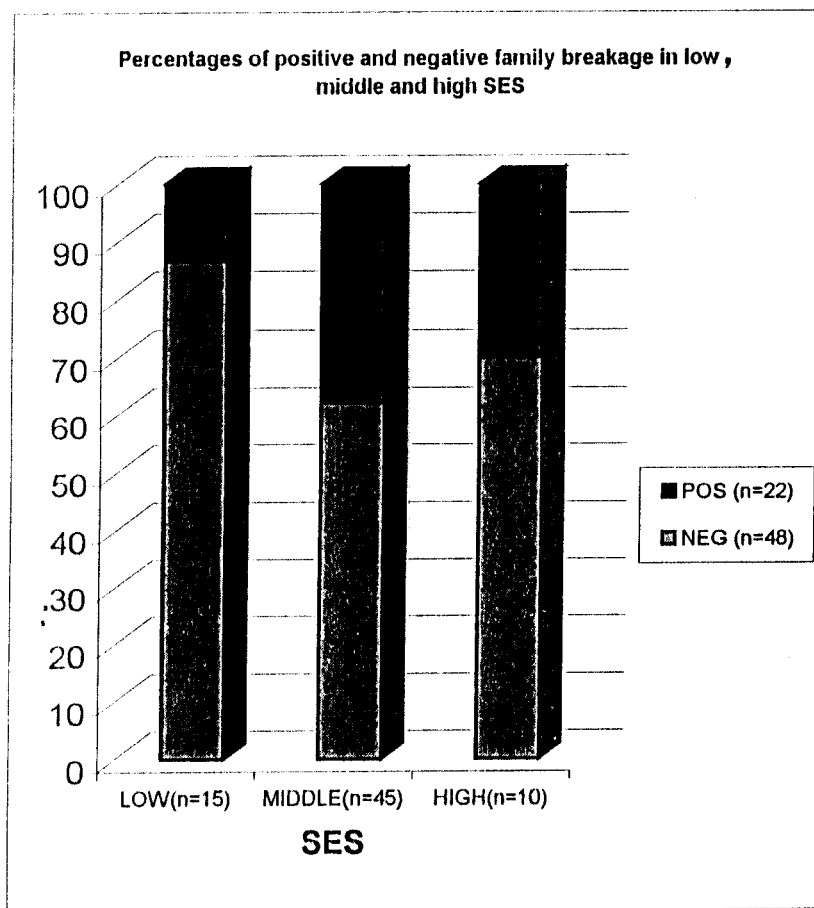
TABLE 24
PERCENTAGE FREQUENCY OF FAMILY
SIZE IN RELATION TO SES IN ADHD PATIENTS

VARIABLE	F. SIZE				
	3 OR < (n=38)		>3(n=32)		Total (n=70)
SES	No	%	No	%	Mean+/- SD
LOW (n=15)	3/15	20	12/15	80	4.7+/-1.3
MIDDLE (n=45)	25/45	55.6	20/45	44.4	3.6+/-1.1
HIGH (n=10)	10/10	100	0	0	2+/-0.7
X2	15.56				
p	0.00041 (HS)				

This table shows that in the low SES level ,most of ADHD patients are from large sized families (80%) .On the other hand ADHD patients in high SES levels are totally from small sized families. This finding is of high statistical significance (P<0.001)

Percentage frequency of different family sizes in low , middle & high SES

Fig 11

(119)
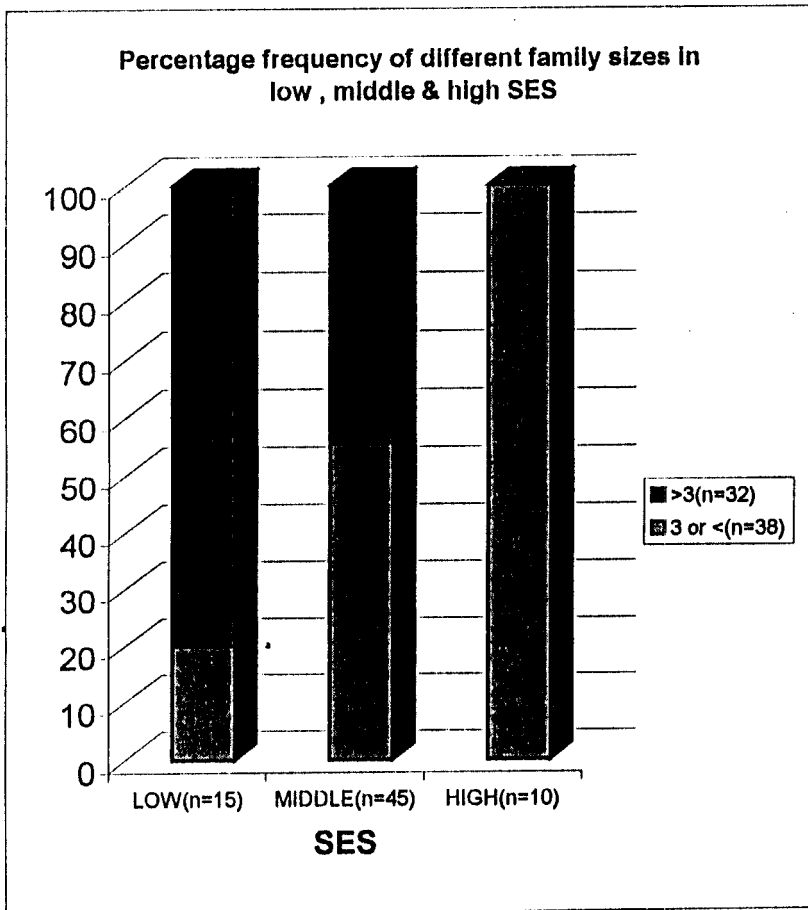
TABLE 25
PERCENTAGE FREQUENCY OF NORMAL VS ABNORMAL PERINATAL HISTORY IN RELATION TO SES

VARIABLE	PERINATAL HISTORY					
	NORMAL (n=47)		ABNORMAL (n=23)			
					Total	
SES	No	%	P	O	No	%
LOW (n=15)	7/47	14.9	5	3	8/23	34.8
MIDDLE (n=45)	34/47	72.3	5	6	11/23	47.8
HIGH (n=10)	6/47	12.8	1	3	4/23	17.4
X2	6.68					
p	0.15(NS)					

This table shows that there is a high percentage frequency of abnormal perinatal history in low SES level .On the other hand it is low in high SES levels. However the difference is statistically insignificant (P>0.05)

TABLE 26
PERCENTAGE FREQUENCY OF PRIMARY AND SECONDARY ADHD VS SES

VARIABLE	DIAGNOSIS					
	1ry (n=44)		2ry (n=26)			
					Total	
SES	No	%	C	O	No	%
LOW (n=15)	10/15	66.7	1	4	5/15	33.3
MIDDLE (n=45)	28/45	62.2	4	13	17/45	37.8
HIGH (n=10)	6/10	60	1	3	4/10	40
X2	0.17					
p	0.99(NS)					

This table shows that there is no statistical significance of primary and secondary ADHD in relation to SES level (P> 0.05)

(120)

TABLE 27
PERCENTAGE FREQUENCY AND MEAN VALUES OF ADHD SCORE IN VARIOUS LEVELS OF SES

VARIABLE	ADHD score				
	10-<20 (n=38)		20-30 (n=32)		Total (n=70)
SES	No	%	No	%	Mean+/-SD
LOW (n=15)	7/15	46.7	8/15	53.3	20.2+/-2.8
MIDDLE (n=45)	25/45	55.6	20/45	44.4	18.8+/-2.6
HIGH (n=10)	6/10	60	4/10	40	18.5+/-2.4
X2	0.51				
p	0.77 (NS)				

This table shows that there is a higher incidence (53.3%) of high ADHD score (severe ADHD) in low SES level , than in other SES levels . However this difference is statistically insignificant . (P> 0.05)

(121)

TABLE 28
PERCENTAGE FREQUENCY AND MEAN VALUES OF IQ IN VARIOUS SES LEVELS

VARIABLE	I.Q.				
	<100 (n=35)		100- (n=35)		Total (n=70)
SES	No	%	No	%	Mean+/- SD
LOW (n=15)	10/15	66.7	5/15	33.3	93.9+/-12.2
MIDDLE (n=45)	24/45	53.3	21/45	46.7	98.2+/-12.9
HIGH (n=10)	1/10	10	9/10	90	106.8+/-12.9
X2	8.26				
p	0.01 (S)				

This table shows that there is a statistically significant higher incidence rate of ADHD with below average IQ in the low SES than in other SES levels (P< 0.01)

Percetages of below & above average IQ levels in low , middle & high SES
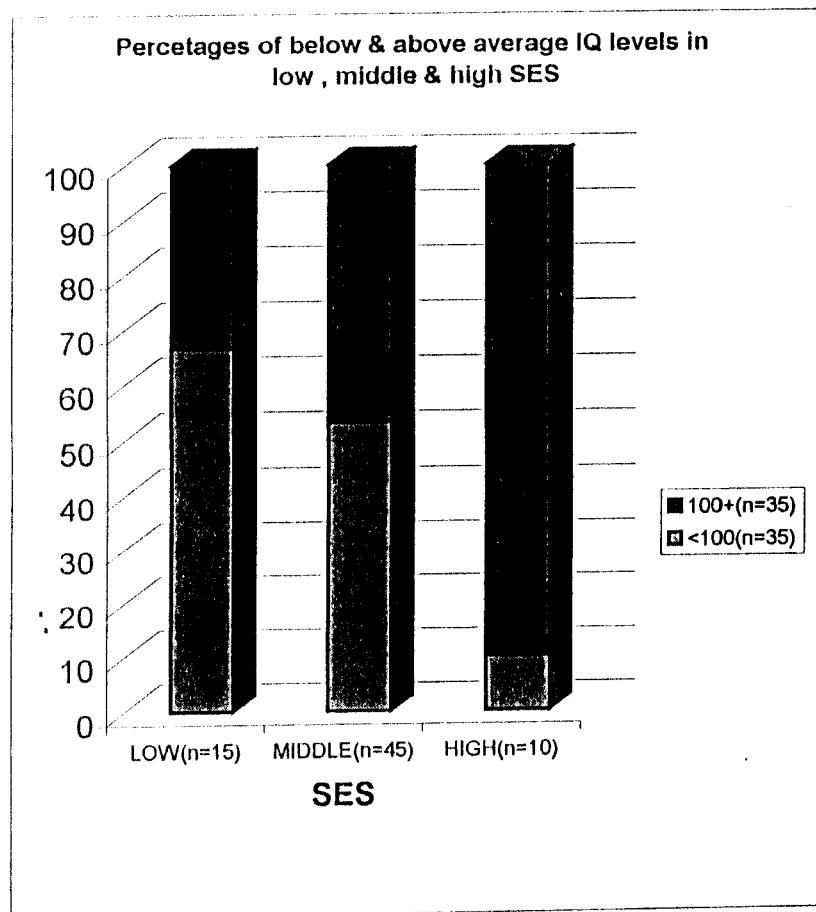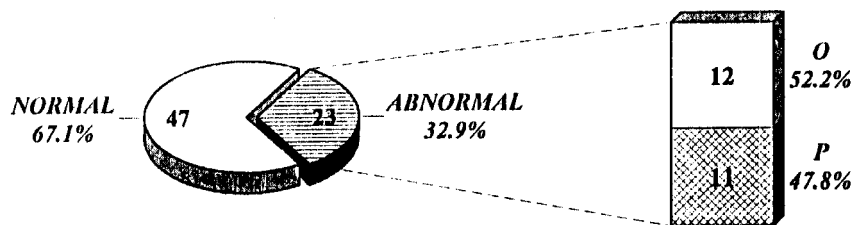
SES

Legend: 100+(n=35), <100(n=35)

Fig 12

(122)

Fig 13
A pie chart showing the percentage frequency of
perinatal history , with a segmented bar showing the
percentage of different abnormal perinatal histories
among ADHD cases

PERINATAL HISTORY

(123)

TABLE 29
PERCENTAGE FREQUENCY OF PERINATAL HISTORY IN SMALL VS LARG SIZED FAMILIES

VARIABLE		F. SIZE				
		3 OR < (n=38)		>3(n=32)		Total (n=70)
PERINATAL H.		No	%	No	%	Mean+/- SD
NORMAL (n=47)		27/38	71.1	20/32	62.5	3.4+/-1.1
ABNORMAL (n=23)	P (n=11)	6		5		3.6+/-1.4
	O (n=12)	5		7		4.3+/-1.9
	TOTAL	11/38	28.9	12/32	37.5	3.8+/-1.6
X2		0.25				
p		0.61 (NS)				

This table shows that in the group of large sized families (37.5%) of ADHD cases have positive abnormal perinatal history, while in small sized families only 28.9% of cases have positive abnormal perinatal histories .However this difference is insignificant statistically (P> 0.05)

TABLE 30
PERCENTAGE FREQUENCY OF PERINATAL HISTORY IN CONSANGUIEOUS VS NONCONSANGUINEOUS ADHD PATIENTS

VARIABLE		CONSANGUINITY			
		N (n=43)		P (n=27)	
PERINATAL H.		No	%	No	%
NORMAL (n=47)		31/43	72.1	16/27	59.3
ABNORMAL (n=23)	P (n=11)	5		6	
	O (n=12)	7		5	
	TOTAL	12/43	27.9	11/27	40.7
X2		1.64			
p		0.44 (NS)			

· This table shows that in the group of consanguineous marriage, 40.7% of cases have abnormal perinatal history, compared 27.9% in the nonconsanguineous group, yet this difference is not of statistical significance (P>0.05)

(124)

TABLE 31
PERCENTAGE FREQUENCY OF PERINATAL HISTORY IN PRIMARY VS SECONDARY ADHD PATIENTS

VARIABLE			DIAGNOSIS			
			1ry (n=44)		2ry (n=26)	
PERINATAL H.			No	%	No	%
NORMAL (n=47)			38/44	86.4	9/26	34.6
ABNORMAL (n=23)	P (n=11)		2		9	
	O (n=12)		4		8	
	TOTAL		6/44	13.6	17/26	65.4
X2			17.56			
p			0.0000027 (HS)			

This table shows that in the group of abnormal neonatal history ,while 65.4% of cases have secondary ADHD , only 13.6% have pure ADHD . Also 34.6% of the irrelevant perinatal history cases are secondary ADHD , compared to 86.4% who are primary ADHD . Both differences are of high statistical significance   (P<0.01)
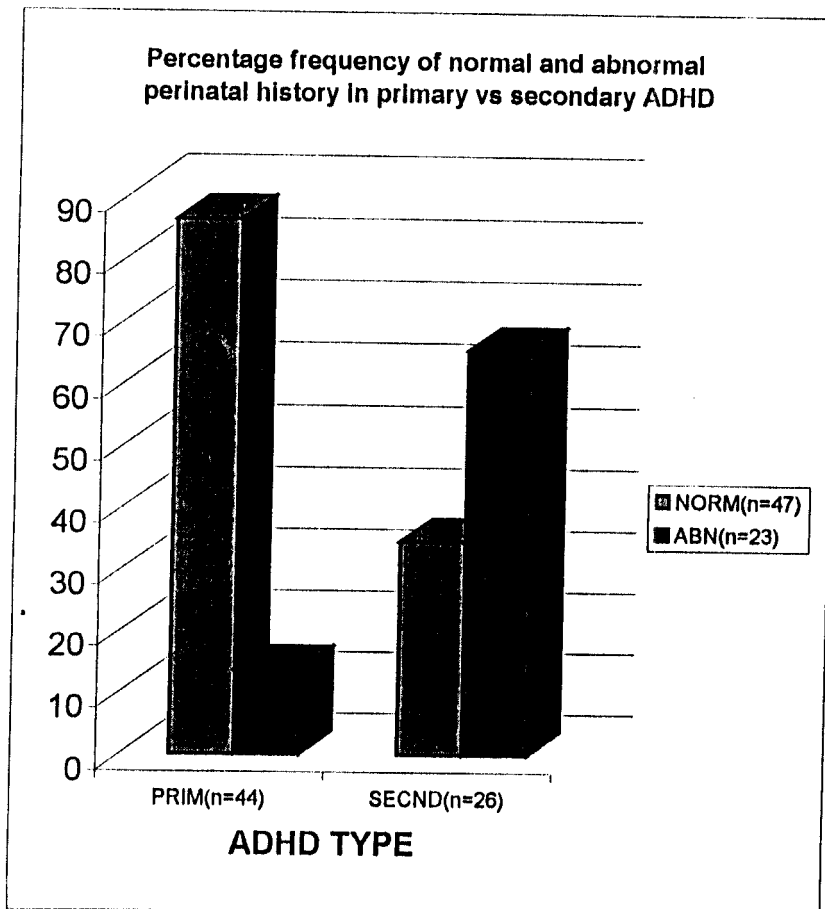

Fig 14

(125)

TABLE 32
PERCENTAGE FREQUENCY & MEAN VALUES OF IQ LEVELS IN NORMAL VS ABNORMAL PERINATAL HISTORY

VARIABLE		I.Q.				
		<100 (n=35)		100- (n=35)		Total (n=70)
PERINATAL H.		No	%	No	%	Mean+/- SD
NORMAL (n=47)		21/35	60	26/35	74.3	99.6+/-13
ABNORMAL (n=23)	P (n=11)	7		4		95.6+/-12.5
	O (n=12)	7		5		96.6+/-14.7
	TOTAL	14/35	40	9/35	25.7	96.1+/-13.6
X2		1.03				
p		0.3 (NS)				

This table shows that there is a higher percentage frequency (40%) of abnormal perinatal history , in below than in above average IQ groups (25.7%) .This difference is not of statistical significance   (P>0.05)

TABLE 33
PERCENTAGE FREQUENCY AND MEAN VALUES OF ADHD SCORE IN NORMAL VS ABNORMAL PERINATAL HISTORY

VARIABLE		ADHD score				
		10-<20 (n=38)		20-30 (n=32)		Total (n=70)
PERINATAL H.		No	%	No	%	Mean+/- SD
NORMAL (n=47)		26/38	68.4	21/32	65.6	18.7+/-2.6
ABNORMAL (n=23)	P (n=11)	7		4		19+/-2.6
	O (n=12)	5		7		20.4+/-2.8
	TOTAL	12/38	31.6	11/32	34.4	19.6+/-2.7
X2		0.0001				
p		0.99 (NS)				

This table shows that there is no statistical significant perinatal history difference as regards ADHD score (P> 0.05)

(126)

TABLE 34
BEAM FINDINGS & PERCENTAGE FREQUENCY OF NORMAL VS ABNORMAL PERINATAL HISTORY

VARIABLE		BEAM			
		NORMAL (n=52)		ABNORMAL (n=18)	
PERINATAL H.		No	%	No	%
NORMAL (n=47)		40/52	76.9	7/18	38.9
ABNORMAL (n=23)	P (n=11)	7		2	
	O (n=12)	5		9	
	TOTAL	12/52	23.1	11/18	61.1
X2		7.12			
p		0.00075 (HS)			

This table shows that 61.1% of cases who showed abnormal BEAM findings are in the group of abnormal perinatal history ,while only 23.1% of cases have irrelevant perinatal history .This difference is of statistical significance (P< 0.05)
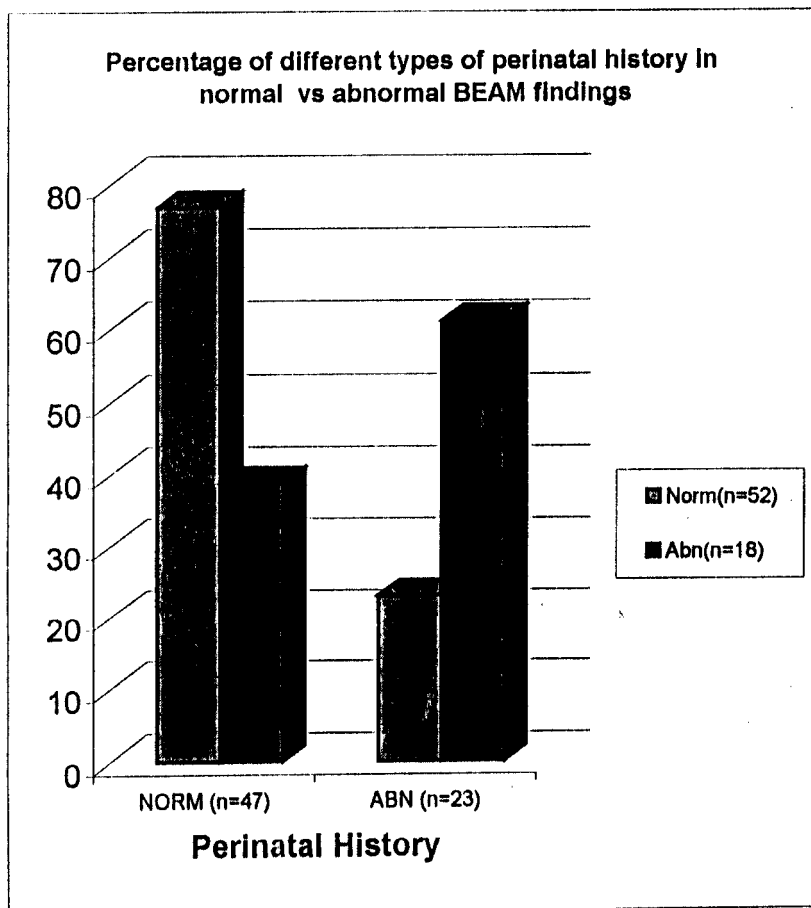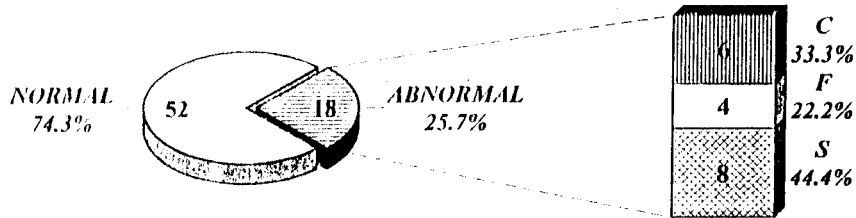

Percentage of different types of perinatal history in normal vs abnormal BEAM findings

Fig 15

(127)

Fig 16
A pie chart showing the percentage frequency of
BEAM findings , with a segmented bar showing the
percentage of different types of abnormal BEAM
findings among ADHD cases

NORMAL
74.3%

52

18

ABNORMAL
25.7%

C
33.3%

F
22.2%

S
44.4%

BEAM FINDINGS

TABLE 35
PERCENTAGE FREQUENCY AND MEAN VALUES OF AGE IN NORMAL VS ABNORMAL BEAM FINDINGS

VARIABLE		AGE				
		6-<9 (n=25)		9- (n=45)		Total (n=70)
BEAM		No	%	No	%	Mean+/-SD
NORMAL (n=52)		17/25	68	35/45	77.8	9.6+/-1.8
ABNORMAL (n=18)	S (n=8)	3		5		9.5+/-1.9
	F (n=4)	3		1		8.5+/-1.5
	C (n=6)	2		4		9.9+/-2.2
	TOTAL	8/25	32	10/45	22.2	9.3+/-1.9
X2		0.37				
p		0.54 (NS)				

This table shows that 32% of cases below th e age of 9
have abnormal BEAM findings compared to only
22.2% in the above 9 years group .Yet this difference
is of no statistical significance (P> 0.05)

(128)

TABLE 36
PERCENTAGE FREQUENCY OF BIRTH ORDER IN NORMAL VS ABNORMAL BEAM FINDINGS

VARIABLE / BEAM		B.O.					
		1st (n=50)		2nd (n=14)		3rd or > (n=6)	
		No	%	No	%	No	%
NORMAL (n=52)		36/52	69.2	11/52	21.2	5/52	9.6
ABNORMAL (n=18)	S (n=8)	5		2		1	
	F (n=4)	4		0		0	
	C (n=6)	5		1		0	
	TOTAL	14/18	77.8	3/18	16.7	1/18	5.5
X2		0.52					
p		0.76 (NS)					

This table shows that firstborns commonly have abnormal BEAM findings (77.8%) yet there is an insignificant birth order difference in relation to BEAM findings (P> 0.05)

TABLE 37
PERCENTAGE FREQUENCY AND MEAN VALUES OF FAMILY SIZE IN NORMAL VS ABNORMAL BEAM FINDINGS

VARIABLE / BEAM		F. SIZE				
		3 OR < (n=38)		>3(n=32)		Total (n=70)
		No	%	No	%	Mean+/-SD
NORMAL (n=52)		30/38	78.9	22/32	68.7	3.6+/-1.4
ABNORMAL (n=18)	S (n=8)	4		4		3.8+/-0.9
	F (n=4)	4		0		2.3+/-1
	C (n=6)	0		6		4.5+/-0.8
	TOTAL	8/38	21.1	10/32	31.3	3.5+/-0.9
X2		0.48				
p		0.48 (NS)				

This table shows that in large sized families , 31.3% of cases showed abnormal BEAM findings compared to 21.1% of cases from small sized families . .This difference is of no statistical significance (P> 0.05)

(129)

TABLE 38
PERCENTAGE FREQUENCY OF BEAM FINDINGS IN CONSANGUINEOUS VS NONCONSANGUIEOUS ADHD PATIENTS

VARIABLE		CONSANGUINITY			
		N (n=43)		P (n=27)	
BEAM		No	%	No	%
NORMAL (n=52)		34/43	79.1	18/27	66.7
ABNORMAL (n=18)	S (n=8)	2		6	
	F (n=4)	3		1	
	C (n=6)	4		2	
	TOTAL	9/43	20.9	9/27	33.3
X2		4.71			
p		0.03 (S)			

This table shows that 33.3% of consanguineous cases showed abnormal BEAM findings compared to only 20.9% of non consanguineous cases .This difference is of statistical significance (P< 0.05)
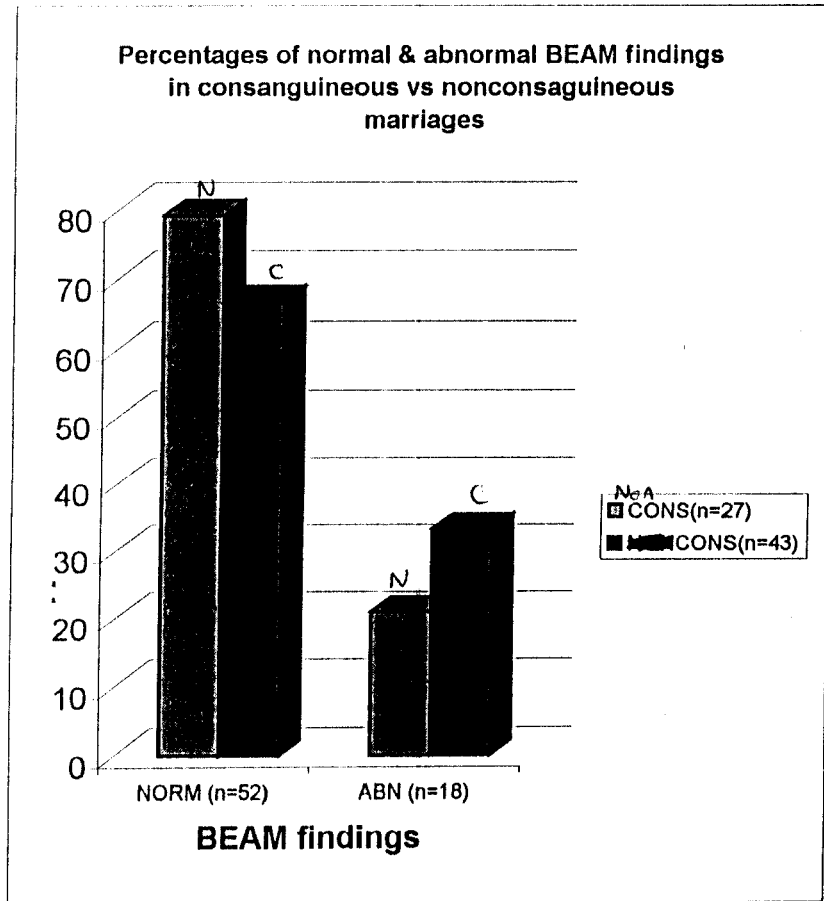

Fig 17

(130)

TABLE 39
PERCENTAGE FREQUENCY AND SES OF
NORMAL VS ABNORMAL BEAM FINDINGS

VARIABLE		SES					
		L (n=15)		M (n=45)		H (n=10)	
BEAM		No	%	No	%	No	%
NORMAL (n=52)		6/15	40	38/45	84.4	8/10	80
ABNORMAL (n=18)	S (n=8)	6		2		0	
	F (n=4)	1		1		2	
	C (n=6)	2		4		0	
	TOTAL	9/15	60	7/45	15.6	2/10	20
X2		11.8					
p		0.00026 (S)					

This table shows that 60% of low SES cases showed abnormal BEAM findings in relation to other SES this difference is of statistical significance (P< 0.01)
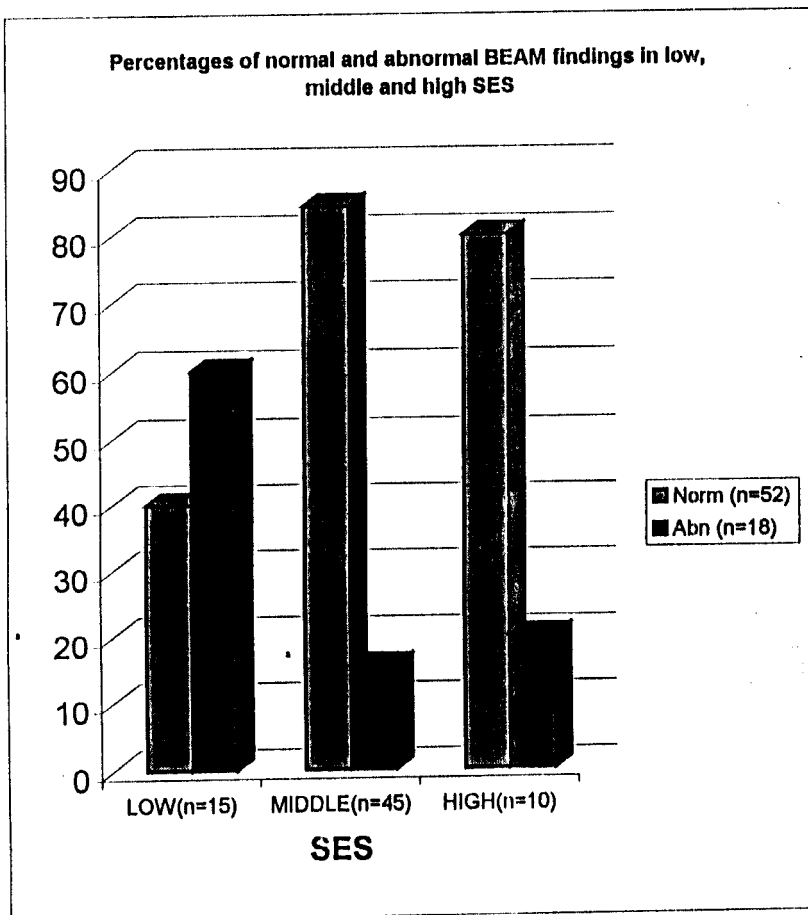

Fig 18

(131)

TABLE 40
PERCENTAGE FREQUENCY OF DIFFERENT TYPES OF BEAM FINDINGS IN PRIMARY VS SECONDARY ADHD CASES

VARIABLE		DIAGNOSIS			
		1ry (n=44)		2ry (n=26)	
BEAM		No	%	No	%
NORMAL (n=52)		41/44	93.2	11/26	42.3
ABNORMAL (n=18)	S (n=8)	2		6	
	F (n=4)	0		4	
	C (n=6)	1		5	
	TOTAL	3/44	6.8	15/26	57.7
X2		33.5			
p		0.00000082 (HS)			

This table shows that 57.7% of cases of the secondary ADHD showed abnormal BEAM findings compared to only 6.8% of the primary ADHD cases. Both differences are of high statistical significance (P< 0.0001)
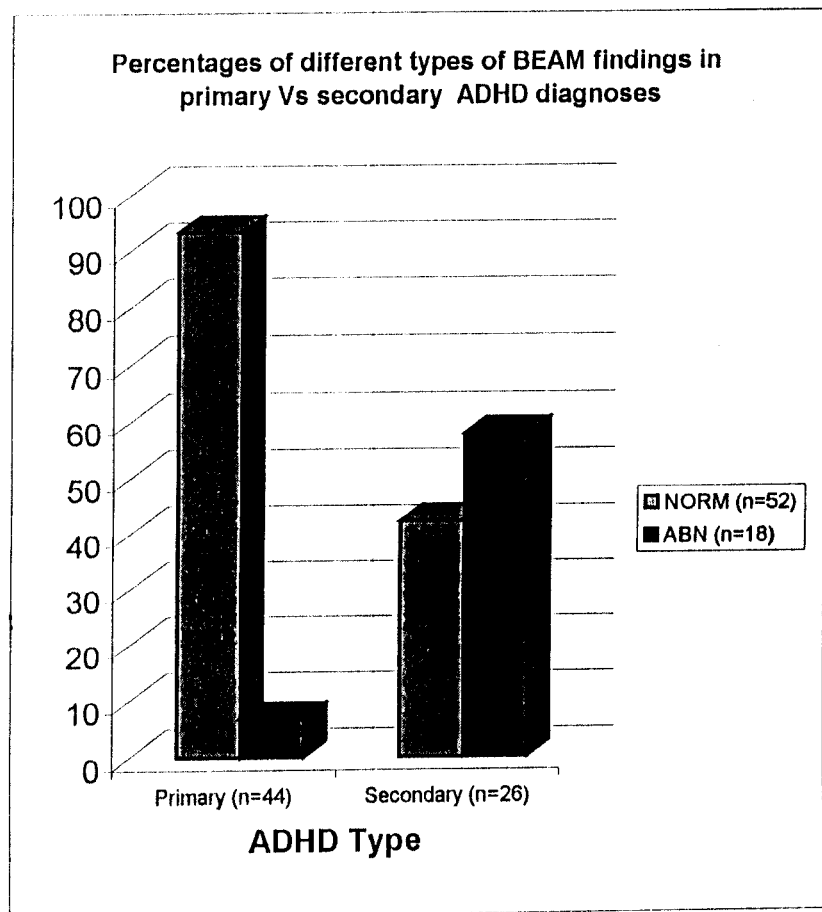

Percentages of different types of BEAM findings in primary Vs secondary ADHD diagnoses

Fig 19

(132)

TABLE 41
PERCENTAGE FREQUENCY OF BEAM
FINDINGS IN BELOW VS ABOVE AVERAGE IQ

VARIABLE		I.Q.				
		<100 (n=35)		100- (n=35)		Total (n=70)
BEAM		No	%	No	%	Mean+/-SD
NORMAL (n=52)		26/35	74.3	26/35	74.3	98.3+/-13.3
ABNORMAL (n=18)	S (n=8)	5		3		96.3+/-14.9
	F (n=4)	1		3		103.5+/-10.5
	C (n=6)	3		3		99.3+/-13.3
	TOTAL	9/35	25.7	9/35	25.7	99.7+/-12.9
X2		0.07				
p		0.78 (NS)				

This table shows that there is no significant IQ difference as regards BEAM findings in ADHD cases under study (P > 0.05)

(133)

TABLE 42
PERCENTAGE FREQUENCY AND MEAN
VALUES OF BEAM FINDINGS IN MODERATELY
VS SEVERELY AFFECTED ADHD CASES

VARIABLE		ADHD score				
		10-<20 (n=38)		20-30 (n=32)		Total (n=70)
BEAM		No	%	No	%	Mean+/-SD
NORMAL (n=52)		34/52	65.4	18/52	34.6	18.5+/-2.7
ABNORMAL (n=18)	S (n=8)	2		6		20.6+/-1.6
	F (n=4)	0		4		21.5+/-1.3
	C (n=6)	2		4		20.3+/-2.3
	TOTAL	4/18	22.2	14/18	77.8	20.8+/-1.7
X2		8.37				
p		0.00038 (S)				

This table shows that 77.8% of cases of abnormal
BEAM findings are in the group of high ADHD scores
Compared to only 22.2% in thegroup of moderate
ADHD affection .On the other hand 65.4% of normal
beam finding cases are in the moderately affected
ADHD group compared to only 34.6% in the severely
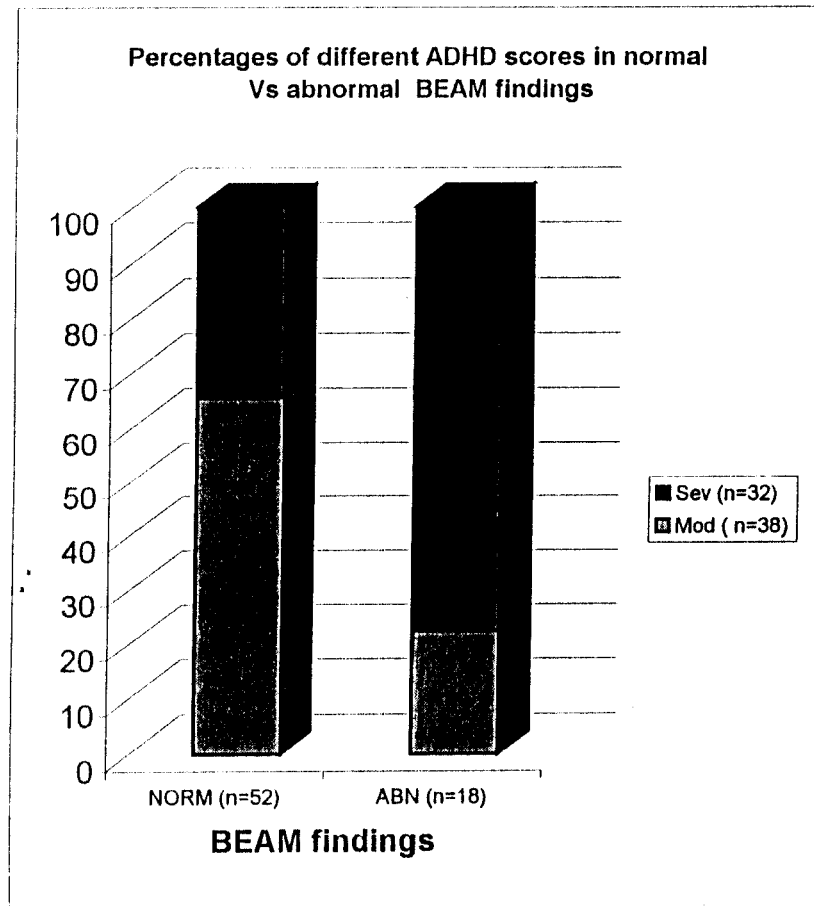affected group .Both   differences are of statistical
significance (P< 0.01)

Fig 20

(134)

Fig 21
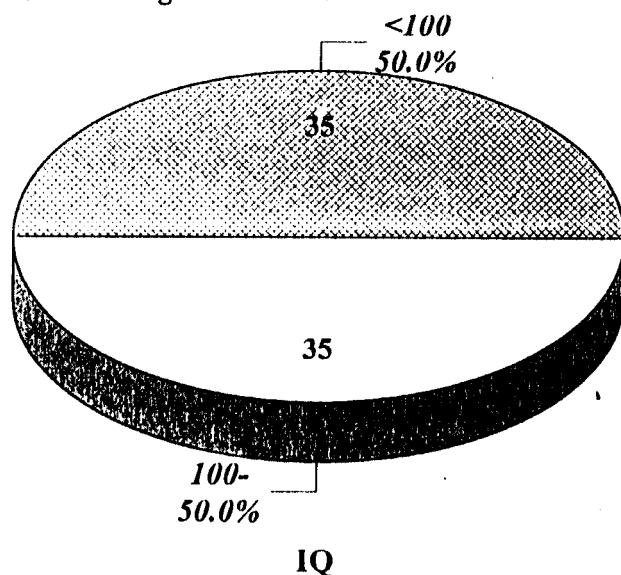A pie chart showing the percentage frequency of
different IQ levels among ADHD cases

IQ

TABLE 43
PERCENTAGE FREQUENCY AND MEAN
VALUES OF IQ IN PRIMARY VS SECONDARY
ADHD PATIENTS

VARIABLE	Diagnosis					
	1ry (n=44)		2ry (n=26)			
					Total	
I.Q.	No	%	C	O	No	%
<100 (n=35)	22/35	62.9	1	12	13/35	37.1
100- (n=35)	22/35	62.9	5	8	13/35	37.1
MEAN	98.5		108.3	95.5	101.9	
SD	13.2		13.7	11.9	12.8	
X2	0.06					
p	0.8 (NS)					

This table shows that although there is no significant IQ
difference as regards diagnosis , there is a significant
statistical difference among both types of secondary
ADHD .

(135)

TABLE 44
PERCENTAGE FREQUENCY OF ADHD SCORE IN BELOW VS ABOVE AVERAGE IQ

VARIABLE	I.Q.			
	<100 (n=35)		100- (n=35)	
ADHD score	No	%	No	%
10-<20 (n=38)	12/35	43.3	26/35	74.3
20-30 (n=32)	23/35	65.7	9/35	25.7
X2	9.7			
p	0.0018 (S)			

This table shows that 65.7% of ADHD cases showed high ADHD score and are in the group of below average IQ. This difference is of statistical significance (P< 0.01)

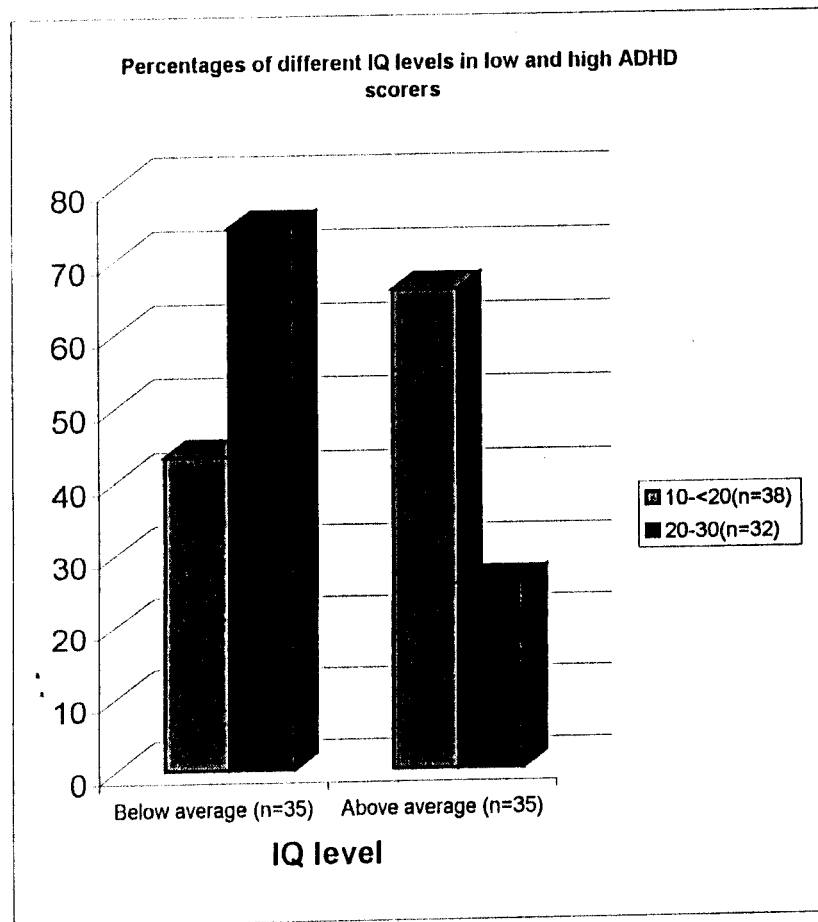Fig 22

(136)

ADHD SCORE vs. IQ
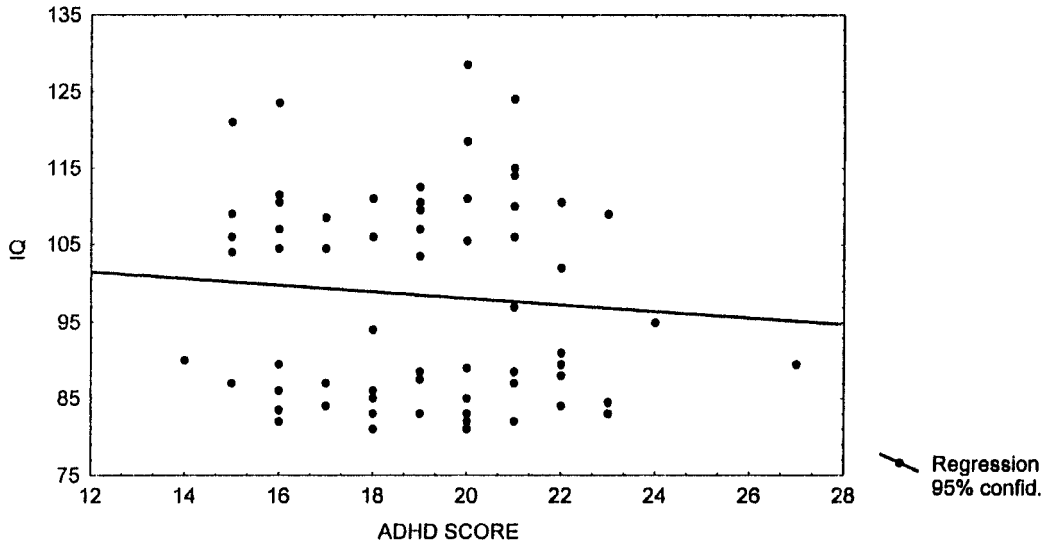IQ = 106.42 - .4162 * ADHD SCORE
Correlation: r = -.0843

Fig 23
A scatter graph showing the relation between IQ and
ADHD score

Fig 24
A pie chart showing the percentage frequency of
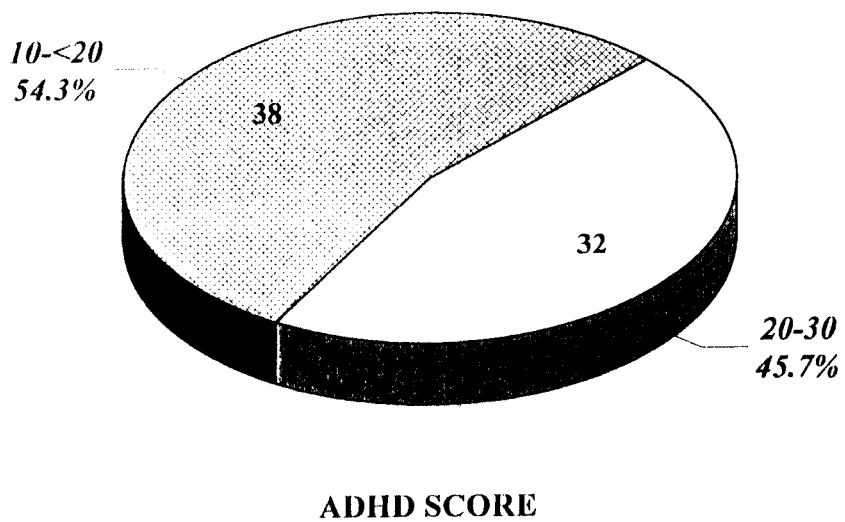different ADHD scores among ADHD cases

10-<20
54.3%

38

32

20-30
45.7%

ADHD SCORE

(138)

TABLE 45
PERCENTAGE FREQUENCY OF ADHD SCORES AND MEAN VALUES IN PRIMARY VS SECONDARY ADHD PATIENTS

VARIABLE	Diagnosis					
	1ry (n=44)		2ry (n=26)			
					Total	
ADHD score	No	%	C	O	No	%
10-<20 (n=38)	34/38	89.5	0	4	4/38	10.5
20-30 (n=32)	10/32	31.3	6	16	22/32	68.6
X2	22.79					
p	0.0000018 (HS)					

This table shows that 68.6% of cases with severe (high) ADHD score , have secondary,while only 10.5% of cases have moderate ADHD .This difference is of high statistical significance (P< 0.00001)

Fig 25

(139)
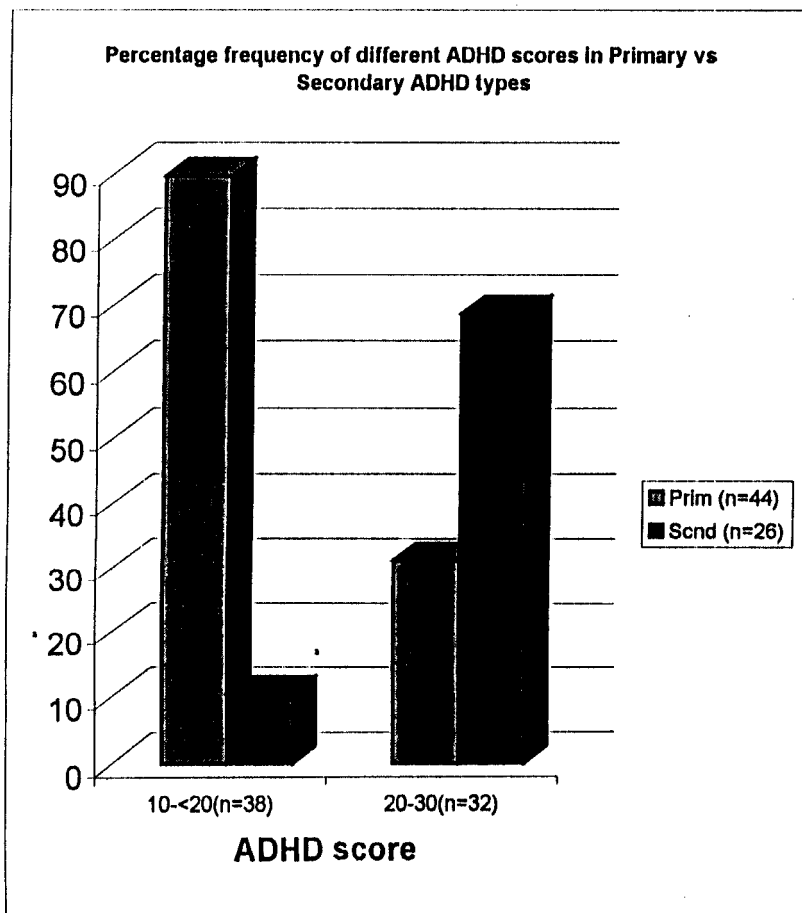
TABLE 46
PERCENTAGE FREQUENCY OF AGE IN
MODERATE VS SEVERE ADHD AFFECTION

VARIABLE	AGE			
	6-<9 (n=25)		9- (n=45)	
ADHD score	No	%	No	%
10-<20 (n=38)	16/38	42.1	22/38	57.9
20-30 (n=32)	9/32	28.1	23/32	71.9
X2	0.93			
p	0.33 (NS)			

This table shows that 71.9% of cases with severe ADHD (high score) are in the group of above 9 years ,compared to only 28.1% of cases below 9 years.This difference is of no statistical significance (P>0.05)

(140)

Discussion

Discussion

Attention deficit hyperactivity disorder (ADHD) is a heterogeneous condition of debatable etiology, which affects mostly boys. The symptoms of ADHD may be identifiable at a very young age and may persist into adolescence and young adulthood *(Golden, 1991)*.

Children who present with inattention, impulsivity and hyperactivity constitute a large portion of the behavior problems seen in pediatric patients ADHD is associated with various other childhood conditions that need to be investigated systematically *(Rostain, 1991)*. In many instances the symptoms of ADHD signal the onset of severe psychopathology and indicate comorbidity *(Mandoki ,et al , 1991)*.

The most widely used definition of ADHD is provided by *the Diagnostic and Statistical Manual (4th edition) DSM-IV by the American Psychiatric Association,(1993)*,which provided 2 diagnostic categories ;inattention and hyperactivity, as well as impulsivity .

Techniques used in the clinical evaluation of ADHD are grouped into 3 areas: rating scales, instruments that evaluate the specific aspects of ADHD (impulsivity , inattention , distractibility), and psychometric techniques such as measures of intelligence , academic achievement , memory or processing .

Many of these techniques formerly have been considered as laboratory measures *(Cantwell and Barker, 1987)* , but their clinical use has increased dramatically over the last several years *(Kelly and Aylward, 1992)*.

The original Conners Teacher Rating Scale *(Conners,1969)* consists of 39 items , the revised version contains 28*(Conners,1973)* .There is also a 10-item Conners Abbreviated Teacher Rating Scale (ATRS), (Hyperactivity Index),(ADHD score)-(Table–1), which appears to be sensitive to both hyperactivity and inattention *(Kelly and Aylward, 1992)*.

Parent check-lists appear most liberal with respect to indicating attention problems whereas computerized assessment is most

conservative, teacher rating scales fall in the middle *(Cohen, et al ,1989)*. Hence the last method is preferred in our work.

Office diagnosis of hyperactivity can present a problem to the pediatrician because only about 20% of potential patients show hyperactive behavior during office examination. Eighty percent of children ultimately accepted as hyperactive on the basis of home and school reports showed no sign of hyperactivity in the office . The physician can therefore have confidence in information about the child's history provided by the parents by using the ADHD score as a reliable aid in the diagnostic process with hyperacive children (*Sleater and Ullman , 1981*). In the present work the child was deemed hyperactive if he achieved a score equal to or greater than 45% on the score of (50% ± 5) whicl the lowest accepted score below which the patient was excluded (14 the ATRS scale).

Children with a low IQ or those who were psychotic ,grossly brain-damaged or markedly antisocial were excluded from the present sample.

1)Incidence and Sex Ratios:

According to the data presented in this work , it was found that while in the pure (primary) ADHD cases the male to female ratio 3.4:1 compared to 5.5:1 in secondary (with associated symptoms) cases .The total male to female ratio was 4:1 .Regarding IQ levels, it was found that the ratio is kept the same in both below and above average IQ ,namely 4:1 .As for ADHD score, in the moderately affected cases (below 20) the ratio was 2.5:1 , on the other hand in severe high score , the sex ratio was 9.7:1 male to female.

The male to female ratio in this study varied with different variables .Abnormal BEAM findings were found to be present 5 fold in males than in females

The mean IQ of male cases was 98.8 ± 12.9 compared to 97.5 ± 14.4 for females . This difference was insignificant statistically .Regarding ADHD score , the mean value for

males was 19.2 ± 2.6 on the Conners ARTS ,compared to 18.6 ± 3.1 in females .This difference was statistically insignificant .

The frequency rate of ADHD in our cases was found to be 5.04% of our Pediatric Psychoneurology Clinic attendants.

These results go along with studies of *Lahey ,et al,(1984),*[incidence 1-2% and sex ratio 4-5:1] ;*Harold,et al,(1984),* [incidence 2-20% and sex ratio 3-5:1] ; *Olweya,(1988),* [incidence 4% and sex ratio 4:1] ;*Gad ,(1990),* [incidence 4.5% and sex ratio 4:1] ;*Shaheen,(1990),* [incidence 0.5-1% and sex ratio 4:1] ;*Anderson ,(1990)* , [incidence in New Zealand 6.7% and sex ratio 5:1] ;*Cartwright,(1990),* [incidence among black children in South Africa 2.7% and sex ratio 2.4:1];*Sauceda, and Vega ,(1990),* [incidence in Mexico 5.2% and sex ratio 2:1] ;*Salem ,(1991),* [incidence 2.4% and sex ratio 1.8:1];*DSM-IV,(1993)* ,[incidence 3-5% and sex ratio 3-5:1] ;*NIMH,(1997),* [incidence 3-5% and sex ratio 2-3:1] ;and *El.-Baz and Ghanem ,(1995),* [incidence 6.4% and sex ratio 5:1] .Contrarily , our results disagreed with studies of *Okasha ,(1988),* [incidence 0.5% and sex ratio 10:1] ; and *Luk , (1990),* [incidence among Chinese children 5.8% and sex ratio 9:1].

The variation in incidence and sex ratio between our study and others may be due to either ethnic or methodological criteria for ADHD diagnosis .However ,the sex difference per se can be explained on genetic basis.

2)Birth Order:
In the present work it was found that 71.4% of the ADHD cases were firstborns ,compared to 20% who were second birth ordered and only 8.6% of cases were more than second birth ordered .Inspite of that , the difference was not statistically significant compared to other variables .

Our results go along with the results found by *Paternite and Loney (1980), Walker (1983), Shaywitz and Shaywitz,(1984)* and *Hamouda (1984),* who noted a tendency for firstborns to be more involved . Results of this work also agreed with the results *of Olweya ,(1988),*who found that the first child represented

49% of cases , compared to 35.5% for the second child , and *Afaf* *,(1991)* , who found a tendency for firstborns to be more affected .On the other hand , *Baker , (1971)*, and *Wolman ,(1977)* , stated that being a member of a large sized family , enables the child to share with others , especially parents' and sibs' affection , so , reacting to stress with a minimal reaction .

Our findings and the findings of others could be explained by the fact that the elder child is always a precious child , especially if it is an only child , thus it is always forgiven for its misbehavior , and at the same time , parents are afraid in their approach to management .

Surprisingly   ,though firstborns are commonly affected by ADHD ,abnormal perinatal history was commonly observed among second birth ordered children (42.9%).This finding might be due to the high medical and psychological antenatal care given to the firstborn .However the genetic background accounts for the high incidence of ADHD in firstborns.

Regarding **the type of ADHD**, it was revealed in this work that 76.9% of cases diagnosed as secondary ADHD and 68.1% diagnosed as primary ADHD, were firstborns , while the second birth ordered children constituted   20.5% of primary and 19.3% of secondary ADHD , therefore these findings showed no significant difference among different birth orders in relation to the type of ADHD .

Regarding birth order and **IQ levels** , no significant relations could be observed (P>0.05).

Regarding **ADHD** **score** in relation to different birth orders , it was found that 68.4% and 75% of moderate and severe ADHD- affected   cases were firstborns with mean ADHD values of 16.8 ± 1.5 and 21.2 ± 1.2 ,respectively .Also ,18.4% and 21.9% of moderate and severe affection were second birth ordered .Both were of insignificant statistical difference .To the best of our knowledge, no available results in the literature were comparable to our findings .

3)Family Size :

In this study we found that 51.4% of ADHD cases were from small sized families (3 or less), compared to 48.6% from large sized families (greater than 3) .However this difference was not statistically significant (P>0.05).

With regards to the relation between family size and **the type of diagnosis**, it was revealed that 63.2% and 62.5% of primary ADHD were of small and large sized families respectively. Meanwhile, secondary ADHD constituted 36.8% and 37.5% of small and large sized families respectively (P>0.05).

Regarding **IQ levels** and family size, there was a negative correlation (r =- 026) between the 2 parameters .Also we found that , 55.3% of ADHD cases of small sized families had above average IQ with a mean value of 111.7 ± 6.6, compared to 43.7% of ADHD cases of large sized families with a mean IQ value of 108.7 ± 5.1 .Yet ,all these differences were insignificant statistically (P>0.05).

Similarly , **ADHD score** was statistically insignificant among those cases of small vs. large sized families (P>0.05).

So, it can be stated that better child care can be attained in small sized families .

Our results agreed with the results of *Robins ,et al,(1966),* who suggested that large family size and poverty are correlated with hyperkinesia. Also , *Ibtehal ,(1983),* found that the majority of hyperkinetic children came from large sized families .The same was found by *Taylor ,et al,(1986),* and *Olweya, (1988) ,*who found that children from large sized families tended to have a lower level of IQ and were twice as likely to develop behavioral disorders . *Shaheen ,(1990); Afaf,(1991);* and *El-Baz and Ghanem ,(1995),* found that most cases came from large sized families .

We can explain the prevalence of ADHD not on birth order basis, but on a family size basis. The large size of Egyptian families as compared to other societies ,puts the child ,whether he is first , second , or last , at risk .However, *Baker ,(1971);* and *Wolman*

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,*(1977)*,suggested that in large sized families the child is more able to share parents' and sibs' affection , so , reacting to stress minimally .

4)Consanguinity:

According to the data presented in this work , it was found in this study that 38.6% of ADHD cases were the product of consanguineous marriage .This is statistically highly significant in comparison to the consanguineous rate among general population 28.96% in Egypt *(Hafez ,et al ,1983)*.

Meanwhile, the study revealed that ADHD cases of consanguineous marriage were 37.1% and 59.3% among low and middle SES respectively. On the contrary , ADHD cases of non-consanguineous marriage constituted a bigger proportion among high SES (20.9%) in comparison to 3.7% among ADHD cases of consanguineous marriage .All these data were statistically significant (P<0.01).This finding shows the rule of consanguinity (genetic basis) in the etiogenesis of ADHD .At the same time, we cannot deny the rule low SES among consanguineous families .

However ,the study demonstrated that consanguinity had no significant effect whether on the type of ADHD , IQ level , or the ADHD score (P> 0.05)

5)Family Breakage :

Concerning the results of this work , it was found that 31.4% of ADHD cases had a degree of family breakage either in the form of parental death ,separation , divorce , severe parental conflicts , or travelling to work abroad .This was compared to 68.6% of cases that had no family breakage. This rate was higher than that in the general Egyptian population (12%) found by *El-Sherbini,et al,(1981)*, and *Olweya (1988)* .In Western societies, *Sugar ,(1970)*, and *Smart,(1978)* , found it to be 33.3%, and in the USA, *Fine ,(1980)*, found a rate of 40%.The low percentage figure in our community indicated more stability of the families .The higher rate of family breakage in ADHD cases indicated that these children lived in a disturbed home that added another factor to a predisposed child to increase morbidity and severity of cases .Also, these high figures in

ADHD cases can correlate between environment as a major precipitating factor and the occurrence of this disorder .Our results supported the results of *Biller,(1970), Andry ,(1971),Steinhauer and Grant ,(1983),Connel , (1985), Dorothy ,et al ,(1985),*and *Olweya,(1988).*

Concerning our results , the relation between family breakage and **consanguinity** showed that family breakage was more in non- consanguineous marriages (32.5%) than in consanguineous ones (29.6%). Inspite of being a statistically insignificant difference this can be explained by the fact that consanguineous families had more bonds to tie the family and prevent its breakage .

Regarding **diagnosis** , our results demonstrated that 34.6% of secondary ADHD cases were from broken families , compared to 29.5% in primary cases. This insignificant statistical difference can be explained by the assumption that the disturbance in the home atmosphere in these families contributed one more factor so that more of the psychiatric disorders can be expressed in predisposed subjects. No relevant data could be found in literature as regards the relation between family breakage and each of consanguinity , diagnosis and ADHD score .

As regards **IQ** , our results showed that 72.2% of below average IQ cases were from broken families with a mean value of 86.3 ± 4.2 , compared to 27.3% of the above average IQ cases with a mean value of 110 ± 4.8 .Whereas , with negative family breakage 39.3% of cases were below average IQ with a mean value of 86.6 ± 3.8, compared to 60.4% of above average IQ with a mean value of 110.6 ± 6.5. The difference was of statistical significance (P<0.05).Also , the general mean value of the negative and positive family breakage were 101.1 ± 13.1 and 92.8 ± 11.7 respectively .This also was found · to be a significant difference .From this finding we concluded that family breakage had a proven effect in lowering the IQ of the ADHD patient .This can be explained by the fact that in broken families no enough time could be spent to children's medical and psychological care with delaying mental growth. This finding goes along with the results of *Olweya ,(1988)* ,who found delayed milestones in ADHD cases with parents conflicts compared to the

control group .Also it agreed with the results *Minde ,et al,(1971)*, and *Cantwell,(1977)*.

With respect to **ADHD score** , our results demonstrated that there was no significant statistical difference between ADHD score in negative vs. positive family breakage in ADHD patients .From this finding and the finding before , it is clear that , whereas the IQ was highly affected by the family breakage , the ADHD score was not at all. This can be explained by the fact that IQ is a developmental process which is affected by environmental changes , while ADHD score is a measure of the severity of the disorder which is genetically based and environmental changes alter it minimally .

6)Socio-Economic Status (SES):

According to the data presented in this work it was found that 21.4% of the studied ADHD cases were from low SES families , according to *Park and Park classification , (1979)* ,which was the only available standardized method that could be found. Meanwhile ,64.3% and 14.3% of cases were from middle and high SES families respectively .

As regards **family size** it was found that , 80% of cases from low SES families were from large families (more than 3) and all the cases in the high SES families were from small families (3 or less). The difference was of high statistical significance, meaning that the combination of large family size and low SES or small family size and high SES had a great effect in provoking the disorder in genetically predisposed subjects .This finding coincided with the results of *Rutter, et al ,(1970)* and *(1975); McConville ,(1983); Armanious ,(1985); Sandberg , (1985);* and *Rutter and Hersov ,(1987).*Our results also were found to be in accordance with the results of *Olweya ,(1988),* who stated that the rate of the disorder increases as one descends the social class ladder .This can be explained by the higher chance of anxiety due to multiple stresses and frustrations upon subjects of the lower social class .Only *Kaplan and Sadock ,(1985),*denied the effect of SES to be a predisposing factor for ADHD .

Regarding **family breakage** our results showed that 37.8% and 13.3% of middle and low SES families had positive family breakage .This was found to be a statistically significant difference (P<0.01).The maximum family breakage rate was found in the middle SES .This can be attributed to ambitions beyond the ability of the family's supporter, so instigating great conflicts in the workplace and/or at home .This was in accordance with *Mohamed ,(1990)*, who found that large family size , low SES and family breakage are the main factors in the precipitation and development of behavioral disorders in children .

As regards **perinatal history** , our results revealed that 17.4% of cases with abnormal perinatal history were in the high SES , compared to 47.8% and 34.8%, in the middle and low SES respectively .This higher rate of abnormal perinatal history in middle and lower SES ,inspite of its statistical insignificance indicated the higher perinatal care given to both mother and child in high SES .As hard as we tried, we found no comparable results in the available literature with respect to this point.

Considering **the type of ADHD diagnosis** , it was found that , primary ADHD incidence was almost double that of secondary type in all SES yet the difference was statistically insignificant .This result agreed with the results of *Ayden ,et al ,(1987),* and disagreed with the results found by *Paternite and Loney,(1980);* and *Gad ,(1990),* who found a higher incidence of associated symptoms in low SES . The difference in our results and others could be attributed to different criteria of diagnosis of both ADHD and SES .

As for **IQ** , our results declared that 66.7% of ADHD cases of low SES ,were of below average IQ, compared to 33.3% of above average IQ , and with a total mean of 93.9 ± 12.2 .Meanwhile , 90% of cases of high SES were of above average IQ , compared to 10% of below average IQ, and with a total mean of 106.8 ± 12.9.This maximum incidence of below average IQ in low SES and of above average IQ in high SES , which had a statistical significance (P<0.01), can be explained by the psychosocial distresses associated commonly with the low social classes .These findings support the results found by *Douglas ,(1956); Opell, et al, (1968); Okasha,(1980);*

Olweya,(1988), and *Gad,(1990).* Also, the mean IQ level of affected children of low SES was lower than that of those of middle SES , and that in the middle was lower than that in high SES. This could be explained by the fact that raising the SES allowed for more psychological and developmental reaction with the environment leading to elevation of IQ. Also, these results agreed with the results found by *Ayden , et al, (1987),* who found that marked mean IQ difference between different SES .

Considering **ADHD score** , our results demonstrated that , 53.3% of low SES patients were of high score (severe ADHD), and 46.7% were of moderate score with a total mean of 20.2 ± 2.8, compared to 40% of high SES cases were of high score , and 60% of moderate score with a total mean of 18.5 ± 2.4 .This demonstrated that even with the insignificant statistical difference , the lower SES ,the more severe the disorder .This also coincided with the results found by *Ayden ,et al ,(1987),* and *Gad ,(1990),* who found a higher incidence of severely affected cases in lower SES .

7)Perinatal History :

According to the data announced in this work , it was found that, while 67.1% of cases had normal perinatal history , only 32.9%, had an abnormal perinatal history in the form of preterm , 15.7%, birth trauma 2.9 %, neonatal jaundice ,11.4%, and neonatal fever 2.9% .For reliable statistical analysis the group of abnormal perinatal history was subdivided into preterm cases (P) and others (O), which included , trauma , jaundice , and fever .While these results agreed with the results found by *,Illingworth, (1975),* who correlated the occurrence of toxemia of pregnancy or anoxia at birth with overactivity and defective concentration ,yet , *Olweya ,(1988)* , found no correlation .Meanwhile , our results supported the results found by *Rolls ,(1983),* and *Lou ,(1990),* who found high incidence of adverse antenatal and perinatal events in ADHD cases .

Regarding **family size** , the results of this work demonstrated that , whereas abnormal perinatal history was positive in 37.5% of large sized families , yet ,it affected only 28.9% of small

sized families .Although this was an insignificant difference, it indicated that perinatal problems were more prone to occur in large sized families .This can be explained by the decreased medical and psychological care given to children of large families .

As regards **consanguinity** , our results declared that 40% and 27.9% of consanguineous and non- consanguineous ADHD cases respectively had abnormal perinatal histories .Inspite of its statistical insignificance , this difference pointed to more abnormal perinatal problems happened to ADHD cases of consanguineous parents .This could be explained by the genetic basis of the disorder and the higher incidence of perinatal problems in hereditary conditions .As far [as] we know , no literature could be traced that commented on the relation between perinatal history and family size, consanguinity , and ADHD score in ADHD cases .

Regarding **diagnosis,** the researcher found that 65.4% of secondary ADHD had abnormal perinatal history , compared to only 13.6% , of primary cases .Also , it was found that 39.1% of abnormal perinatal history were preterms in secondary ADHD , compared to 8.7%in primary cases .Also , 34.8% of abnormal perinatal history were other problems than preterm in secondary ADHD cases , compared to 17.4% in primary cases .These differences were all of high statistical significance (P<0.001), meaning that abnormal perinatal history had adverse effects on the associated symptoms of ADHD and that prematurity was responsible for more than 50% of secondary cases .This can be explained by the fact that abnormal perinatal history especially prematurity is the main cause of perinatal brain anoxia , that leads to cerebral dysfunction which is a suspected etiology for the disorder and its associated symptoms .These results correlated with the results of *Rolls ,(1983)* and *Lou, (1990)* , who suggested· a role of antenatal and perinatal factors in their retrospective studies of ADHD patients .

As regards **IQ** , our results revealed that , 40% of the below average IQ cases had abnormal perinatal histories compared to 25.7% of the above average IQ cases and with an overall mean IQ value of 96.1 ± 13.6 .Although this difference was statistically insignificant , it showed that perinatal history abnormality had an effect in lowering the IQ level in ADHD cases .This can be explained

by the fact that brain anoxia due to abnormal perinatal history lead to cerebral dysfunction with its subsequent IQ reduction .This was found to be in accordance with *Skov , et al , (1984),* who found that ischemia during the first hours of life was a critical determinant for the development of cognitive disorders, by measuring cerebral blood flow in ADHD cases .

Regarding **ADHD score** , our results showed that 34.4% of severely affected cases (high score) had abnormal perinatal history ,compared to 31.6% of moderately affected cases , with a mean value of 19.6 ± 2.7. This indicated that abnormal perinatal history had more adverse effects in severe ADHD cases than in moderate cases. Even for the general mean ADHD score for normal perinatal history (18.7 ± 2.6) , it was found to be lower than that for abnormal perinatal history. Both findings were of no statistical significance .

As for **BEAM findings** , it was demonstrated that 61.1% of cases with abnormal BEAM findings had abnormal perinatal history , whereas 23.1% of normal BEAM findings had abnormal perinatal histories and 38.9% of abnormal BEAM findings had normal perinatal histories. It was also found that 76.9% with normal BEAM findings had normal perinatal histories .This was found to be of significant statistical difference (P<0.01).It can be concluded that abnormal perinatal events had a considerable effect on positive BEAM findings in ADHD cases. Also this can be explained by the cerebral dysfunction that resulted form brain anoxia or destruction at early life . Also it was found that prematurity had a minor effect in comparison with other causes of abnormal perinatal history for the appearance of positive BEAM findings. This data was in accordance with *Furguson, (1984);* and *El-Baz and Ghanem,*(1995), who found positive perinatal history in the form of neonatal hyperbilirubinemia in their BEAM positive tracings , denoting a reflection of a sort of cortical immaturity or an underlying brain damage .

8) BEAM Findings:

According to the data presented in this work , we found that 74.3% of the studied ADHD cases had normal BEAM maps , and 25.7% had abnormal beam findings, in the form of either

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slow (delta and theta) wave abnormalities , fast (alpha and beta) wave abnormality , or combined slow and fast together .Abnormal slow waves were found in 11.4% of cases , while abnormal fast waves constituted 5.7% of cases and combined anomalies were found in 8.6% of cases .So the slow wave anomaly was the most common one. This was found to agree with *Grunwald-Zuberbier ,et al ,(1975)* , who showed a shift towards lower frequencies and towards less power in beta but more in alpha frequencies in hyperactive children .*Duffy , and McAnulty , (1990)* , showed that there were no differences in EEG changes between ADHD patients , and patients with learning disabilities. While *Turgay ,et al , (1992)*, found that , BEAM was more sensitive than EEG in detecting abnormalities in ADHD especially when compared to age and gender ,*but Valdizan and Andreu ,(1993),* found no difference in sensitivity and specificity in ADHD cases according to DSM-III-R .Also *Ackerman ,et al,(1994)* found greater low beta and less theta power , which was in accordance with the results of this work , the same was found by *Okasha and Raafat, (1994); Ackerman , et al , (1995) ;Chabot ,et al , (1996); Linden and Radojevic,(1996); Kuperman,et al , (1996); DeFrance,et al , (1996)*;and *Chabot and Serfontein, (1996)*.

Regarding the relation between BEAM findings in ADHD cases and **Age** , it was found that 77.8% and 68% of cases above and below age of 9 years old , respectively had normal beam findings , with a general mean age value of 9.6 ± 1.8 y, compared to 22.2% above 9 y and 32% below 9y with a general mean of 9.3 ± 1.9 y had abnormal BEAM findings .This showed that age had no effect on BEAM findings , as the difference was statistically insignificant between the 2 groups .There was a higher incidence (64.3%) of the disorder in older children, which can be attributed to the normal development period of adolescence and pre-adolescence normally associated with an increase in overactivity and violence .This was found to go along with the results obtained by *Hamouda ,(1984); Berry ,(1985); Olweya ,(1988); Shaheen, (1990);Afaf,(1991);* and *El-Baz and Ghanem ,(1995).*

Regarding **birth order** , our results showed that 77.8% of abnormal BEAM findings were firstborns followed by second ordered children (16.7%), and the least the third or more birth

ordered (5.5%). Compared to normal BEAM findings , 69.2% were fist ordered followed by 21.2% , second ordered , and the least was the third or more birth ordered (9.6%) .This difference was found to be statistically insignificant (P>0.05).The genetic predisposition of the disorder , is an accepted explanation for this finding .None of the available data concerned with the relation between birth order and EEG and BEAM findings . The same was found regarding family size , consanguinity , and SES .

Regarding **family size** , our results showed that 31.3% of ADHD cases of large sized families had abnormal BEAM findings , compared to only 21.1% from small sized families .This finding raises the question of whether familial and social factors had effects on the appearance of abnormal BEAM findings in ADHD cases or not .But inspite of its statistical insignificance , this has to be investigated in specific research , especially as no research was done regarding this correlation .

As regards **consanguinity,** we found that 33.3% of consanguineous ADHD cases had abnormal BEAM findings ,compared to 20.9% in non-consanguineous patients .This difference being statistically significant (P<0.05) , can correlate between consanguinity and positive BEAM findings . This can be explained as ,, the genetic predisposition of ADHD was accentuated by consanguinity , leading to more accompanying manifestations and more brain dysfunction that could be elicited as positive BEAM findings .

Regarding **SES** our data demonstrated that 60% of low SES ADHD patients had abnormal BEAM findings , compared to 15.6% and 20% , of middle and high SES respectively. The difference was statistically significant (P<0.05) , showing that low SES , comprised a powerful stress to the predisposed ADHD patients and more provocation of the disorder and associated symptoms .

As regards to **diagnosis** , our results revealed that 57.7% of secondary ADHD cases had abnormal BEAM findings ,compared to 6.8% in primary cases. Also, for all types of BEAM anomalies , it was found to be higher in secondary than in primary cases. The difference was of high statistical significance .This could mean that in secondary ADHD there were more associated symptoms

due to more brain dysfunction ,that appeared in the form of abnormal BEAM findings .This finding , supported the results of *Lastochkina and Puchinskaia ,(1991)* ,who found that hyperkinetic children shown to have different types of EEG rhythms according to different hemispheric asymmetries .

Regarding **IQ**, our results declared that IQ level had no effect on BEAM findings at all , as equal frequency percentage of BEAM findings were found in both below and above average IQ. Also , no significant difference was found among the mean values of IQ in relation to the presence or absence of abnormal BEAM findings .This was in accordance with *El-Baz and Ghanem,(1995),* but it disagreed with the results found by *Linden , et al , (1996),* who found a significant increase of IQ in ADHD cases as compared to the control group in relation to the EEG biofeedback .Also it contradicted the results found by *Robaey ,et al ,(1995),* who showed that IQ was correlated with EEG changes in hyperactive children .They suggested that intelligence did not rely on the same changes and that intelligence forms may not be referred to the same use of the same processes in hyperactive and normal children. For finding an explanation for that difference between these results and the results of this work further researches utilizing improved data collection and analysis with stringent control groups and sample sizes are recommended to support and replicate either of the 2 points of view .

Regarding **ADHD score** , our results revealed that 77.8% of abnormal BEAM findings belonged to the severe type of ADHD compared to 22.2% of moderate affection and with a general mean value of 20.8 $\pm$ 1.7 and that 34.6% of normal BEAM findings belonged to the severe type and 65.4% to the moderate type with a general mean of 18.5 $\pm$ 2.7. As the difference was statistically significant ($P<0.05$) , it could be understood that the more severe the disorder (according to Conner's ATRS), the greater the probability of finding abnormality in the BEAM examination .This can be explained by the fact that with increase of ADHD score (severity of the disorder) more brain dysfunction was produced and so we are more liable to find BEAM abnormality. This was found to agree with the results found by *Lubar , et al,(1995),* who found significant improvement of the score after neuro feedback training which can be an appropriate

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treatment for children with ADHD. The ~~score~~ ^{same} was found by Linden ,et al , (1996).

9)ADHD Score:

According to the data presented in this work , it was found that 54.3% of cases had moderate affection (score below 20) and 45.7% had severe (score of 20 or more). The ATRS of Conners was studied in ADHD cases in relation to age , and it was found that 71.9% of cases were above an age of 9 years ;28.1% were below 9 years .Although this was an insignificant statistical difference when compared to moderately affected cases (57.9% and 42.1% for above and below 9 years respectively),it indicated that whether moderate or severe , it was more prevalent in ages above 9 years .This was found to agree with the results of *Olweya,(1988); Gad ,(1991);* and *Kanbayashi , (1994)* . This distribution can be explained by the normal development of increase in activity associated with preadolescent and adolescent development periods .

Regarding **diagnosis** , the results of this work showed that 68.7% of severe ADHD cases had secondary diagnosis (associated symptoms), compared to 31.3% of pure type and that 89.5% and 10.5% of moderately affected cases had primary and secondary ADHD diagnosis respectively. The difference was statistically highly significant (P<0.001).A high correlation was proved between the severity of the disorder and the associated symptoms. This can be explained by considering that brain dysfunction was a common cause of both severity and associated symptoms .

This was in accordance with the results of *Maurice ,et al , (1985),* who used ATRS to screen ADHD in the general population .They found high scores in combined symptoms. Also, our results agreed with *Olweya, (1988);Gad , (1990); Afaf,(1991); Golden (1991)* and *Morcos , (1993),* who found a strong association with learning disabilities (scholastic achievement).

Concerning **IQ,** our results revealed that within the high score group (severe), 65.7% and 25.7% were of below and above average IQ respectively , compared to 43.3% and 74.3% of moderately affected cases who were of above^v below average IQ respectively. As these differences proved to be statistically significant

(P<0.01) they indicated that IQ and ADHD score were negatively correlated .This can be explained on the neurological basis of both IQ and ADHD score .These findings agreed with the results of *Ayden, et al , (1987), Olweya , (1988), Gad ,(1990), and Afaf,(1991).*

10) I.Q. :

According to the data presented in this work , it was revealed that the mean IQ level for primary ADHD cases was 98.5 ±13.2 compared to 101.9 ± 12.8 for secondary ADHD .Also , it was found that there was no effect of IQ level on the type of diagnosis whether primary or secondary , as the difference was statistically insignificant (P>0.05). This finding agreed with the results found by *Ayden , et al , (1987), Gad,(1990), Skalofske , (1994),* and *El-Baz and Ghanem, (1995).*

Recommendation

Recommendation

From this study we recommend the following :

1- Screening the measures for raising the SES levels , so, preventing family breakage.

2- Parental and school staff education on how to deal with hyperactive children (mental hygiene education) using TV , radio , video and the press .

3- Promotion of antenatal , natal and postnatal care for both mother and baby , physically and psychologically .

4- Screening for ADHD cases in school aged children , especially in those with multiple risk factors that include : consanguinity , low SES , broken families , low IQ , abnormal perinatal history , especially prematurity , associated symptoms with ADHD , and high ADHD score .

5- BEAM is a mandatory investigational procedure test for confirming the diagnosis in high risk group .

6- Further researches to be completed in this study field:

a)Evaluation of ADHD problem in the general population.

b)Comparative study of ADHD in rural and urban areas .

b)Effect of TV , video , and other media on ADHD .

Summary and Conclusion

Summary and Conclusion

Attention Deficit Hyperactivity Disorder (ADHD), is a multifactorial disorder that constitutes a definite problem to both family and community. This work aimed at studying this disorder among Egyptian children; according to the Diagnostic and Statistical Manual (4th edition) (DSM-IV) criteria, using a new investigative modality namely Brain Electrical Activity Mapping (BEAM) to find out its efficacy in diagnosing the disorder. Further, the study extended to identify demographic factors as well as others that may interplay with this problem.

The subjects of this study were recruited from those children diagnosed and followed-up in the Neuropsychiatry Clinic of Children's Hospital of Ain Shams University; in the period from 1 st of January 1994 to the end of December 1997. They were 70 patients out of 1390 attendants, giving a frequency rate of 5.04 %. They were 56 males and 14 females. Their ages (in decimal) ranged between 6.1 and 12.3 years , with a mean age of $9.5 \pm 1.8y$. This sample was subjected to detailed history taking and thorough physical and neurological examination , ADHD scoring using Conner's Abbreviated Teacher Rating Score (ATRS) (1969) , I.Q. estimation using Good-Enough-Harris Drawing Intelligence Test (Harris, 1963), Socio -Economic Status level determination using Park and Park classification , (1979) , and BEAM Test using BEAM- II machine of Nicolet (Nicolet BEAM- II , 1990).

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This study showed the Following results:

I - Sex: The male sex was significantly dominating the female sex with a ratio of 4 : 1 , however , there were no significant sex difference as regards to I.Q. level , ADHD score and BEAM findings.

II - Birth order: First born child was the most frequently affected one (71.4%) with statistical significant difference, P-value (P<0.01). Yet, birth order had no significant effect on each of ; Perinatal history , type of ADHD, I.Q. level, and ADHD score (P>0.05).

III- Family Size: Higher rate of ADHD was found in small families but the difference was statically insignificant. Meanwhile, family size had no significant effect on each of ; type of ADHD, I.Q. level, and ADHD score (P>0.05).

IV- Consanguinity: A higher statistically significant consanguinity rate was found in ADHD cases than in general population , specifically for middle and low SES, while consanguinity had no significant effect on type of ADHD , I.Q level , and ADHD Score .

V - Family Breakage: ADHD cases had a higher family breakage rate than in Egyptian general population. Family breakage had a statistically significant effect in lowering I.Q. level , (P<0.05) , but had no effect on consanguinity, type of ADHD , and ADHD score (P>0.05).

VI- SES: ADHD had higher incidence in middle SES (64.2%) , it was found to be of statistical significance in : large sized families of low SES (P<0.001), broken families of middle SES (P<0.01), and below average I.Q. in low and middle SES(P<0.01). SES had no

significant effect on perinatal history , type of ADHD , and ADHD score (P>0.05).

VII - Perinatal history :   A statistically significant abnormal ~inatal history was found in 32.9% of ADHD cases , of which maturity constituted 47.8% (p<0.05). Whereas, abnormal perinatal ~ory had high statistically significant effect on both: secondary type ~ ADHD (65.5%) of which prematurity constituted 39.1% (P<0.001); and abnormal BEAM findings (61.1%) of which prematurity constituted only 8.7%(P< 0.01),yet perinatal history had no significant effect on each of family size, consanguinity, I.Q. level, and ADHD score (P>0.05).

VIII- BEAM findings :   Abnormal BEAM findings were found in 25.7% with slow, fast and combined waves with a ratio of 2:1.5:1 respectively. Abnormal BEAM findings had a statistical significance among cases of consanguineous marriage (33.3%) (P<0.05), low SES (60%)(P<0.01), secondary ADHD type (57.7%) (P< 0.0001) and high ADHD score (77.8%) (P<0.01), on the other hand there were no significant BEAM changes in relation to age, birth order , family size, and I.Q level (P>0.05).

IX - ADHD score: While, 45.7 % of ADHD cases had high ADHD score (severe), there was a high statistically significant effect of high score on secondary ADHD type (P<0.0001), but had no effect on age (p>0.05).

X- I.Q. level: I.Q. level had a statistically significant effect on high ADHD score (P<0.05), but had insignificant effect on the type of ADHD (P>0.05).

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Arabic Summary

مستخلص الرسالة

يعتبر اضطراب نقص الانتباه وزيادة النشاط من المشاكل المقلقة للأسرة والمجتمع .
ويهدف هذا البحث لدراسة هذا الاضطراب فى الأطفال المصريين باستخدام أحدث الأساليب فى التشخيص والبحث و باستخدام وسيلة حديثة هي مسح النشاط الكهربي للمخ وكذلك دراسة العوامل المختلفة آلتي تؤثر فى هذا الاضطراب .

وقد وقع الاختيار على مرضي عيادة الأمراض النفسية والعصبية بمستشفي الأطفال
ـ جامعة عين شمس فى الفترة من أول يناير ١٩٩٤ وحتى نهاية ديسمبر ١٩٩٧ وكان عدد الأطفال المصابين بالاضطراب سبعين طفلاً من بين ١٣٩٠ مريضاً متردداً على العيادة (٥٦ ذكراً و ١٤ أنثي وتتراوح أعمارهم ما بين ١و٦ سنة ٣و١٢ سنة). وقد تم استعراض التاريخ المرضي للأطفال السبعين مع فحص طبي إكلينيكي وعصبي كامل كما تم قياس درجة الاضطراب ومعامل الذكاء باستخدام اختبار رسم الرجل وأيضاً تحديد المستوي الاجتماعي والاقتصادي للأسرة وكذلك عمل رسم خريطة للنشاط الكهربي للمخ .

وقد أظهر البحث النتائج التالية :-

١- **نوع الجنس** :- يسود انتشار المرض فى الذكور أكثر من الإناث بنسبة ٤ : ١ مع عدم وجود دلالة إحصائية بالنسبة لكل من معامل الذكاء ودرجة الاضطراب وخريطة النشاط الكهربي ما بين الجنسين .

٢- **الترتيب فى الأسرة** :- الطفل الأول أكثر عرضة للإصابة وذو دلالة إحصائية . كما أن الترتيب ليس له دلالة إحصائية مع كل من تاريخ ما حول الولادة ، نوع الاضطراب ، معامل الذكاء وقياس درجة الاضطراب .

٣- **حجم الأسرة** :- وجد أن معدل الإصابة أكثر فى الأسر الصغيرة ولكن بدون دلالة إحصائية وكذلك فإن حجم الأسرة ليس له دلالة إحصائية على كل من نوع الاضطراب ، ومعامل الذكاء ، قياس درجة الاضطراب .

٤- **درجة التزاوج** :- معدل درجة التزاوج وجد أنه أعلى فى الأطفال المصابين وذلك بمغزى إحصائي عالي عنة في المجتمع وخاصة للمستوي الاجتماعي والاقتصادي المتوسط والمنخفض ، بينما لا تؤثر درجة التزاوج على نوع الاضطراب ومعامل الذكاء وقياس درجة الاضطراب .

٥- *التفكك الأسري* :- التفكك الأسري له دلالة إحصائية عالية فى حالات الاضطراب عنها فى المجتمع المصري كما أنه توجد دلالة إحصائية بأن التفكك الأسري يخفض معامل الذكاء فى الأطفال المصابين بالاضطراب ولكنه ليس له أي تأثير على كل من درجة التزاوج ، نوع الاضطراب وقياس درجة الاضطراب .

٦- *المستوى الاجتماعي والاقتصادي* :- إن معدل الإصابة بالاضطراب أعلى فى المستويات الوسطى فى السلم الاجتماعي كما وجدت دلالة إحصائية لكل من الأسر كبيرة الحجم فى المجتمعات المنخفضة ، الأسر المفككة فى المجتمعات المتوسطة ، انخفاض معامل الذكاء فى المجتمعات المنخفضة والمتوسطة بينما لا توجد دلالة إحصائية لكل من تاريخ ما حول الولادة ، نوع الاضطراب وقياس درجة الاضطراب .

٧- *تاريخ ما حول الولادة* :- وجد أن ٣٢,٩% من الحالات لها مشاكل غير عاديـة عند الولادة وتمثل الولادة المبكرة (المبتسرة) نسبة كبيرة منها . كما أن تاريخ ما حـول الولادة له تأثير ذو دلالة إحصائية على كل من نوع الاضطراب الثانوي ووجود خلل فى مسح النشاط الكهربي للمخ . حيث تمثل الولادة المبكرة نسبة عالية من الأولــى ونسـبة منخفضة من الثانية . وعلى العكس فليس لتاريخ ما حول الولادة تأثير متميز علـى كـل من حجم الأسرة ، درجة التزاوج ، معامل الذكاء وقياس درجة الاضطراب .

٨- *خريطة النشاط الكهربي* :- وجد أن ٢٥,٧% من الحالات بها نشاط غير طبيعـي فى صورة موجات بطيئة وسريعة ومختلطة بنسبة ٢ : ١,٥ : ١ على التوالــي . وقـد وجدت دلالة إحصائية مع كل من حالات زواج الأقارب وانخفاض المستوي الاجتمـاعي والاقتصادي والنوع الثانوي من الاضطراب والدرجات العالية من الاضطـراب بينمـا ليست لها دلالة إحصائية مع كل من السن ، الترتيب فى الأسرة وحجم الأسرة ومعـامل الذكاء .

٩- *قياس درجة الاضطراب* :- وجد أن ارتفاع درجة الاضطراب له تأثير ذو دلالـة إحصائية عالية على النوع الثانوي من الاضطراب بينما ليس له تأثير على سن الطفل .

١٠- *معامل الذكاء* :- وجد أن معامل الذكاء له تأثير ذو دلالة إحصائية علـى درجـات الاضطراب العالية ولكنة ليس له تأثير على نوع الاضطراب.

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عنوان الرسالة : تقييم مسح النشاط الكهربي للمخ في الأطفال المصابين باضطراب نقص الانتباه وزيادة النشاط
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٣١ / ٣ / ١٩٩٣م

الدراسات العليا

أجيزت الرسالة بتاريخ ٢٩/٨ / ١٩٩٨م

ختم الإجازة

موافقة مجلس الكلية
١٩٩٨/ ٩ / ١٩

موافقة مجلس الجامعة
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سنة التخرج : ١٩٧٦

سنة المنح : ١٩٩٨

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

,, قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ ,,

صدق الله العظيم

سورة البقرة -- آية ٣٢

جامعة عين شمس

معهد الدراسات العليا للطفولة

قسم الدراسات الطبية

تقييم مسح النشاط الكهربي للمخ فى الأطفال المصابين باضطراب نقص الانتباه وزيادة النشاط

رسالة مقدمة من

الطبيب / محمد أشرف محمد كمال الدين عكاشة

ماجستير طب الأطفال

توطئه للحصول على درجة الدكتوراه فى دراسات الطفولة

(من قسم الدراسات الطبية).

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١٩٩٨