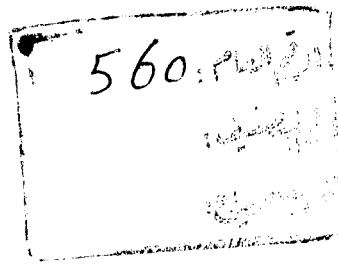




Medical Studies Department

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# Positive Psychological Effects of "Lamotrigine" on Maladaptive Behaviour in Non-epileptic Mentally Retarded Children

## T H E S I S

Submitted for fulfillment of M.Sc. degree  
in childhood studies

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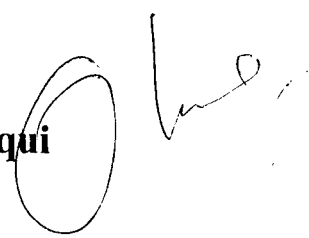
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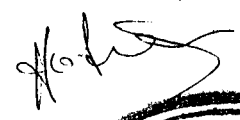
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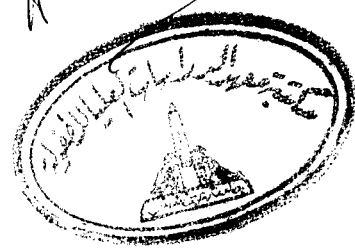
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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

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

صدق الله العظيم

البقرة الآية ٢٢



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# Abbreviations:

- AAMD: American Association for Mental Deficiency
- AAMR: American Association for Mental Retardation
- ADHD: Attention Deficit Hyperactivity Disorder
- AED: Antiepileptic drug
- Age Eq: Age Equivalent
- AIDS: Acquired Immuno-Deficiency Syndrome
- Arc: Association for Retarded Citizens
- CA: Chronological Age
- CNS: Central Nervous System
- DQ: Developmental Quotient
- ECG: Electrocardiography
- EEG: Elctroencephalography
- IQ: Intelligence Quotient
- IBM: International Business Machine
- LMG: Lamotrigine
- M.R: Mental Retardation or Mentally Retarded
- OCD: Obsessive Compulsive Disorder
- PKU: Phenylketonuria
- S.D: Standard Deviation
- SQ: Social Quotient
- SPSS: Statistical Package for Social Sciences
- TCA: Tricyclic Antidepressants
- Vineland ABS: Vineland Adaptive Behaviour Scale
- WHO: World Health Organization
- WISC: Wechsler Intelligence Scale for Children





# Abstract

The aim of this study was to evaluate the possible positive effects of Lamotrigine drug on maladaptive behaviour of non-epileptic mentally retarded children in relation to presence or absence of EEG changes.

This study included 60 non-epileptic mentally retarded children, selected from psychiatric outpatients of Bani-Sweif Psychiatric Hospital; 36 males and 24 females, with mild to moderate intellectual limitations and all belonged to middle socio-economic class. 30 patients had abnormal EEG (Study group) and the other 30 had normal EEG (Control Group). The main common characteristic of those patients was a persistent pattern of socially dysfunctional disruptive behaviour. All children who actually continued treatment in both groups were given Lamotrigine over a period of 6 months.

This 15-months study (including only 6 months of actual continuous treatment of the subjects with Lamotrigine drug) demonstrated that **Lamotrigine** produced both statistically and clinically significant improvements in **56.7%** of total. Lamotrigine was effective in controlling stubbornness, hyperactivity, aggression, impulsivity, negativism and social withdrawal and also was concomitantly effective in improving alertness and mood in the studied children. Better improvement was significantly observable in patients with abnormal EEG (**63.3%** improved) more than in patients with normal EEG (**50%** improved). This may favour the use of Lamotrigine in treating maladaptive behaviour in non-epileptic mentally retarded children especially for those ones having abnormal EEG records.

However, in a new therapeutic area in which no adequate placebo-controlled studies have been conducted, it is difficult to be certain of the clinical significance of the observed results. We may need further studies on larger population to confirm our results.

**Key words:** Adaptive behaviour – Al-Shakhs Scale for the Socio-economic Level of the Family - Antiepileptic drugs - Bani-Sweif - Lamotrigine – Maladaptive behaviour - Mental retardation – Mentally retarded - Middle socio-economic class - Positive psychological effects - Non-epileptic – Vineland Adaptive Behaviour Scale – Wechsler Intelligence Scale for Children.



# Introduction



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## Positive psychological effects of “Lamotrigine” on maladaptive behaviour in non-epileptic mentally retarded children

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### ***\*Introduction :***

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**“No man's intellect can be judged by the size of his hat”.**

This old saying still sounds correct, though it contradicts the opinion of William Thomson, who at the turn of the nineteenth century, observed that "the brains of most idiots and of half-witted persons are usually smaller and weigh less than the average of normal brains, while many men distinguished for their mental powers have had large and heavy brains. But the exceptions are very numerous both ways (Sadock & Sadock, 2000). The relationship between brain size and IQ has been greatly played down in most recent psychology textbooks. The recorded correlations, even when statistically significant, are small in magnitude, suggesting that about one per cent of the variability in intelligence is associated with head size. Van Valen's attempt in 1974 to establish that the 'real' association is higher than that observed is invalid, since it does not really take account of the sampling errors in the estimates of other correlations (Borthwick et al, 1979).

**Mental retardation** is a universal problem found in every race, religion, culture and economic background (Shapiro, 1996). It affects approximately **1% of the population** worldwide (Kolevzon & Simeon, 2002). Different studies have reported different rates depending on definitions used, methods of ascertainment, and population studied (APA, 1994). Many have reasoned that mental retardation is as frequent as nearer to a **3% prevalence rate** (Sadock & Sadock, 2000). Accordingly; there is expectedly about 60 to 180 million people out of our present six billion world population diagnosable as mentally retarded.

Persons with mental retardation have increased risks of co-morbid psychiatric or behavioural dysfunction (Sadock & Sadock, 2000). Mentally retarded children frequently show one or more item of **mal-adaptive behaviour** that interrupt the

process of their rehabilitation and learning and necessitate comprehensive psychiatric attention along with family and social support (Carr, 1999).

There is a wide range of such maladaptive behaviour. It can be just simple & tolerable by other family members e.g. mild irritability or occasional stubbornness or might be severe and very noisy e.g. poor frustration tolerance, persistent hyperactivity, impulsivity, destructive or aggressive behaviour or disinhibited & embarrassing talks & acts (Sadock & Sadock, 2000).

Early diagnosis of psychiatric disorders in children with mental retardation leads to early treatment. Medications are one part of overall treatment and management of children with mental retardation (Janicki et al, 1999).

Particularly, hyperactivity & aggression infrequently show good response to conventional anti-psychotic drugs (e.g. thioridazine or haloperidol), yet their resultant disabling adverse effects (e.g. over-sedation, overweight & extrapyramidal manifestations) put beforehand limitations for their use both in adults or children (Gelder et al, 2000). However, when prescribed appropriately (e.g., for psychotic disorders or for severe behavioural disturbances that fail to respond to less restrictive treatment modalities), antipsychotic drugs may have significant beneficial effects (Rately, 1991).

Alternatively, Antiepileptic drugs (AEDs) have been used for the same purpose with promising results especially the extensively studied and widespread used Carbamazepine & Valproic acid which have been proved effective in adults in controlling violent and aggressive behaviour and also in stabilization of mood for manic and hypomanic states. Newer antiepileptics e.g. **lamotrigine** & Gabapentin, proved effective in treatment of epilepsy in developmentally disabled children, with less adverse effects especially on cognition and behaviour (Rutecki & Gidal, 2002).

Lamotrigine, a relatively new but more safe antiepileptic drug, 1<sup>st</sup> marketed as antiepileptic in 1994, has recently been approved also as a mood stabilizer. It also showed promising effects on modification of behaviour of persons with borderline personality disorder (Goldberg, 1997). Furthermore, **Lamotrigine** recently showed favourable psychological effects when used in treatment of some mentally retarded children suffering from epilepsy (Mikati, 2003).

# AIM OF STUDY





## **\* Aim of Study \***

### **Hypothesis of the study:**

The question arisen was: "Can lamotrigine ameliorate & soothe those maladaptive behavioural symptoms (like: mood changes, hyperactivity, impulsivity or aggressive behaviour) of the non-epileptic mentally retarded children regardless of presence or absence of significant EEG changes?". Improvement of such maladaptive behaviour can have its positive impact on mentally retarded children and also on their families by:-

- Helping them to be more quite, more safe & in a better mood without adding impairment to their attention & cognitive functions.
- Reducing part of negative psychological factors that disturb their academic teaching allowing for better achievement.
- Partial relief of stress caused by disorganized behaviour of the mentally retarded on their caregivers.

So, this study *aimed at*:

**"Evaluation of possible positive effects of Lamotrigine on maladaptive behavioural symptoms associating many cases of non-epileptic mentally retarded children, with particular concern to presence or absence of EEG changes in those children".**



# Literature Review



# \*Literature Review:

## **Historical Perspective:** (of service delivery for the **mentally retarded**)

The history of mental retardation dates back to the **beginning of man's time on earth**. The idea of mental retardation can be found as far back in history as the therapeutic **papyri of Thebes (Luxor), Egypt** (around 1500 B.C). Although somewhat vague due to difficulties in translation, these documents clearly referred to disabilities of the mind and body due to brain damage (Scheerenberger, 1983). The concept of mental illness in Pharonic Egypt was monistic and it was attributed to bodily etiology and treated physically and psychotherapeutically (magico-religious) (Okasha and Maj, 2001).

Persons with mental retardation have long been of interest to their extended societies. This interest has ranged from overly positive to overly negative attitudes, from the French expression "les enfants du bon Dieu" (= God's children) to Martin Luther's exclamation that "The Devil sits where their souls should be" (Sadock & Sadock, 2000).

The plight of individuals with developmental disabilities has been dependent on the customs and beliefs of the era and on the culture or locale (Scheerenberger, 1983). The societies' different attitudes towards the mentally disabled across successive ages can be traced in the following hints:

- **In the beginning**, the handicapped were considered as human rubbish, of no-benefit human creatures (Halawa, 1995), and their societies isolated them and gave them humiliating names like: mad, foolish or sons of the devils (Alquraity, 1996). The treatment applied was by whipping, torturing, depriving from food or by opening a burr hole in the skull of the patient to allow the devil escape out (Melaika, 1997) or to release noxious humours out (Puri, 1996).
- **In ancient Greece and Rome (B.C)**, *infanticide* was a common practice. In old **Sparta**, for example, neonates were examined by a state council of inspectors. If they suspected that the child was defective, he or she was thrown from a cliff to its death, (Scheerenberger, 1983) thrown into River Ortas (Alquraity, 1996), or burnt (Halawa, 1995).  
**Hippocrates** (أبقراط), (460-370 B.C.) introduced the idea that 'the brain is the centre of mental activity, and its sickness is the cause of mental illness'

(Melaika, 1997). He ascribed the reasons for mental illness to the change in the equilibrium of the four body 'humours' that were thought to constitute man's body at that time (Puri, 1996). The modern concept that mental health is influenced by the fluctuations of body chemicals resembled in some ways the 2500-year-old Hippocratic concept (Sadock & Sadock, 2000). Hippocrates proposed a number of therapeutic methods including music, but the destiny of such ideas was to be buried, and magic and superstition returned to spread again (Melaika, 1997). **Plato** (أفلاطون), (428-348 B.C.) in his "Ideal Republic" postulated that: "Intelligent and thoughtful people ought to rule, passionate people should be chosen to defend the state, and **dull and spiritless** individuals, lacking reason and passion, should be given the menial chores of agriculture and industry" (Sadock & Sadock, 2000). His view was to isolate and expel the mentally-handicapped out of the country (Alquraity, 1996). Although **Aristotle** (سقراط) (384-322 B.C.) labelled emotions and postulated that man has the choice to be drawn to positive experiences and to avoid painful ones, (Puri, 1996), he advocated the deaf are unable to learn because they can not speak or understand what is going around them (Alquraity, 1996).

- **By the second century A.D.** individuals with disabilities, including children, who lived in the **Roman Empire** were frequently sold to be used for entertainment or amusement (Scheerenberger, 1983). Roman treatments were more punitive, advocating whipping or ducking to purge the body of ghosts (Puri, 1996). Yet, in fact, all of the **early religious leaders; Confucius, Buddha, Jesus, and Mohammed** advocated human treatment for the mentally retarded, developmentally disabled, or infirmed (Scheerenberger, 1983).
- **The dawning of Christianity** led to a decline in these barbaric practices and a movement started towards care for the "less fortunate" (Scheerenberger, 1983). The Christian religion adopted mercy and care for the handicapped by establishing shelters for them and fulfilling their essential needs of food, drink and clothes (Alquraity, 1996). But, later in the middle ages, the Christian church in the West took over speculation on mental illness and its management. Equating insanity with **alienation** produced the extremes of charity and cruelty to those afflicted (Puri, 1996).
- **During the middle ages (476 - 1799 A.D.)**, the care of individuals with mental retardation varied greatly. Although more human practices evolved, i.e., decreases in infanticide and the establishment of foundling homes, many

children were sold into slavery, abandoned, or left out in the cold (Scheerenberger, 1983).

- **The forgiving Islamic legislation** came to settle the principles of equality, socialization, respect and the right of every body to live with own capabilities regardless of any discrimination attributable to his/her disability, ethnicity, colour or sex. Moslems should be proud that they had paid early attention and care for the mentally retarded -before scientists and educators had done in modern history- at the time of middle ages in Europe where the mentally-ill were treated cruelly with a belief that they were possessed by evil spirits. **Al-Razy**, died in 923, **Ibn-Sina**, died aged 58 years in 1038, and **Al-Kindi**, excluded the role of devils in causing mental illness or other handicaps (Alquraity, 1996; Mohmalat, 2000). Islamic psychiatry in the middle ages used hospital treatment for the mentally ill. Revered as messengers from God, mentally ill people were housed in commodious buildings in some of the big cities of the Middle East (Puri, 1996). During his ruling of 'The Islamic Empire', Prince **Al-Waleed Ibn-Abdul Malik** established the first worldwide institute for the mentally retarded in Damascus in year 707-A.D (Al-Quraity, 1996).
- **Egypt, Before about 700 years**, had the advance of establishing the famous **Hospital** (called Bimaristan) of **Kalaaoon** in Cairo in 1284 A.D, that included, for the first time, a department for treating mental illnesses side-by-side with departments of surgery, medicine, Gynecology and ophthalmology (Abdel-Wahab, 1994). Two features were striking: the care of mental patients in a general hospital, and the involvement of community in the welfare of patients (Baasher, 1975).
- In the **age of renaissance in Europe**, the mentally handicapped and mental patients again were subject to violence, torturing by fire and imprisonment away from the normal society with the claim that they had been controlled by evil spirits (Alquraity, 1996). During that age, there was a few benefactors of the insane. Indeed art and literature suggested that the prevailing attitude at that time was that of ridicule (insane seen as buffoons) or fear (ill seen as possessed by demons) (Puri, 1996). Magic and superstition spread widely to the extent of treating King George-III by the most senior doctors in England at that time by the same cruel methods, the thing that led to parliamentary investigations and appeals for reformation (Melaika, 1997).

- **In the 17<sup>th</sup> century**, medical writers, philosophers and anatomists searched for a physical site for psychological and spiritual entities. There was still a strong belief in demonic possession. From the 17<sup>th</sup> century onwards, **institutions for the insane** did exist. However many accounts refer to unpleasant conditions and treatment (Puri, 1996). The mental patients in Bedlam Hospital, for instance, were hand-cuffed and tied by chains to the walls, and were a source of entertainment for London's public who paid a few money to watch the '*show*' (Cashdan, 1972).
- **From the 18<sup>th</sup> century** there, arose physicians such as **Philippe Pinel** in France, **Tuke** in England and **Chiarugi** in Italy who advocated kinder treatments and removal of chains (Puri, 1996). **Philippe Pinel** (1745–1826) led a reformation move in France (Melaika, 1997). He is credited with revolutionizing the institutional care of the mentally ill when he **liberated over 50 patients from chains and dungeons** at the Bicetre hospital in Paris in 1793. His subsequent publication of "Traite Medico-Philosophique Sur L'alienation Mentale" (1801) outlined a humane approach to the care of these patients. He described how (through an asylum regimen of education, reasoning, and persuasion) many symptoms of insanity could be alleviated. He called this regimen the "moral treatment of insanity"; a philosophical movement that held that: with humane care and close patient-staff interaction, patients could be restored to function. **Non-restraint** was accepted at the first (1841) meeting of the Association of Medical Officers of Hospitals for the Insane (Sadock & Sadock, 2000).
- **In the beginning of the 19<sup>th</sup> century**, the work of the French physician **Jean-Marc-Gaspard Itard** (1775–1838) was a cornerstone event in the evolution of the care and treatment of the mentally retarded (Scheerenberger, 1983). He was hired in 1800 by the Director of the National Institutes for Deaf-Mutes in France to work with a boy he named "Victor", the wild child. A hunter found "Victor" living with animals in Aveyron woods in South France. Victor had apparently lived his whole life in the woods and, after being captured and escaping several times he was captured once again at about age 12, and sent to an orphanage, found to be deaf and mute, and moved to the National Institute for Deaf-Mutes where he lived there till died at age of 40 (Biasini et al, 1998). Itard developed a broad educational program for Victor to develop his senses, intellect, emotions and social interaction. After 5 years of training, Victor continued to have significant difficulties in language and social interaction though he acquired more skills and knowledge than many of Itard's contemporaries believed possible. Itard's educational approach became widely accepted and used in the education of



the deaf. Near the end of his life, Itard had the opportunity to supervise the work of his student, the French educator, Edouard Seguin (Scheerenberger, 1983).

- **In modern history**, the positive concern to deliver a better service to mentally retarded persons began in the **mid-1800s (middle of 19<sup>th</sup> century)** when **Edouard Seguin**, (1812-1880) developed a comprehensive approach, (known as the physiological view of education) to the education of a group of children with mental retardation (Sadock & Sadock, 2000). His curriculum extended from developing basic self-care skills to vocational education (Biasini et al, 1998). He focused education for children's development in three areas: physical activity (including exercises and movement to "awaken" the child's body), intelligence (educating the senses), and will (a "moral education" akin to what one might call socialization). Children with mental retardation were taught to handle objects, discriminate musical and environmental sounds, taste and perform tongue movements for speaking, and visually discriminate forms, colours, and sizes. Such visual discriminations eventually led to drawing and writing (Sadock & Sadock, 2000). Seguin established **the first school for training the mentally retarded** in France in 1837 before his immigration to America in 1948 (Melaika, 1997). His view was that "*children with mental retardation could be trained successfully*" and that fits well with the spirit of the recent times. Throughout, Seguin wanted children with mental retardation to take their rightful place in the societies of their day (Sadock & Sadock, 2000). In 1850, Seguin moved to the United States and became a driving force in the education of individuals with mental retardation. In 1876, he founded what would become later, the American Association on Mental Retardation (Biasini et al, 1998).
- Such optimism helped create the early **residential training schools** for mentally retarded children, first begun in Paris in 1838 by Seguin, then in Massachusetts in 1848 by Samuel Gridley Howe (the first public facility in USA) and by Hervey Wilbur (the first private facility in USA) (Sadock & Sadock, 2000).
- After that, the efforts of pioneers in special education continued; like the physician **Maria Montsouri** in Italy, **De krouliet** in Belgium and **Alfred Binet** in France who put **the first intelligence test** in year 1905 aimed to isolate the mentally retarded children apart from the normal children in the Governmental schools. Their efforts delivered a group of methods and programs for teaching and training the mentally retarded (Alquraity, 1996).

- So, **schooling** was the other major service for persons with mental retardation. "The move to school all American children" began in the **mid-1800s**, and teachers and administrators soon discovered that a subset of children were having difficulty performing school lessons. As a result, **classes for problem children** began in many cities and towns in the late 1800s (Sadock & Sadock, 2000).
- **The late 1800s (= Late 19<sup>th</sup> century)** saw the creation of many such **training schools** which were originally small and homelike, often housing maximum of 10 residents. Gradually, however, these residential schools became larger and less focused on education than on custodial care. Many residents could not return back home. In addition, the isolated placement of most training schools allowed for the segregation, overcrowding, and abuses. Such isolated placements also fostered a change in goals: from an original focus on temporary residence to encourage permanent **custodial institutions** designed to keep persons with mental retardation away from society (Sadock & Sadock, 2000). Mentally retarded people have been subjected to unnecessary institutionalization and, as a result of **the eugenics movement**; they have been also subject to *involuntary sterilization* (Lagasse, 2003).
- The same interest kept on till the **middle of the 20<sup>th</sup> century** stressing on collecting the handicapped children according to their degrees and isolating them into independent schools or establishments away from normal children's schools to teach them according to special curriculae by special teachers and specialized trainers (Alquraity, 1996).
- **At the end of the 2<sup>nd</sup> world war**, there was a great desire for a social change, one of its aspects was the belief that every one has the 'right to health' or at least the right to receive adequate medical care regardless of ability to pay. This resulted in the creation of the National Health Service in the United Kingdom in 1948 and the Social Security System in France, together with similar developments in other countries. The social perspective (which was one of the basic principles underlying these developments), initiated major institutional changes in psychiatry. They were the result of a number of factors including the necessity to give to the whole population an easy access to psychiatric care, and also the belief that social elements played an important role in the etiology of mental disorders and that they could greatly contribute to the healing process, with the aim of progressively reintegrating the patient in the community. The most spectacular aspect of the new policy was the decline in the asylum system (Gelder et al, 2000).

- **Since the mid-1960s**, a move toward **deinstitutionalization** has arisen from many sources. First, the overcrowding and neglect common in many large institutions came to light in various exposés during the late 1960s as reported in some magazines and some television reports. (Sadock & Sadock, 2000) The deinstitutionalization movement of the 1970s reflected a concern for the **civil rights** of mentally retarded (Lagasse, 2003).
- The second most important force was the philosophy of "**normalization**," the idea that individuals with mental retardation were entitled to a more normal lifestyle. **In 1972, Wolf Wolfensberger** extended the idea of normalization to the service delivery system itself, calling on all services for persons with mental retardation (schools, residences, and other services) to be as normative as possible, by having normal rhythm to the day (school or work at day time, leisure-time and sleep), weekends, and vacations or holidays each year. Consequently, parent and professional advocacy groups fought hard to close or at least decrease the size of large residential institutions, to keep in them only the most severely and profoundly impaired individuals, especially those with severe behaviour problems or physical disabilities. In USA, the overall total institutional population of such patients has decreased by almost two thirds during 30 years, from 1967 to 1994 (Sadock & Sadock, 2000).
- By **1968, Lloyd Dunn** declared that most children with mental retardation could be "**mainstreamed**" in classes with non-retarded age-mates; he questioned the need for segregated special education classes for most children with mental retardation. Consequently, educational techniques had also been advanced sufficiently to allow the effective schooling of most children with retardation alongside other, typical children (Sadock & Sadock, 2000).
- **The post-1970 era** has witnessed the strong influence of mainstreaming, community living, and normalization. On the whole, such movements have proven beneficial, as persons with mental retardation increasingly take their rightful place within modern society. Many professionals do, however, question whether normalization has sometimes gone too far. Not every child with mental retardation may be able to be schooled optimally with typically developing age-mates, nor might every adult be able to live independently in the community. (Sadock & Sadock, 2000). Very few of the mentally retarded are now institutionalized; most now live independently, with their families, or in group homes (Lagasse, 2003).

# **\*DEFINITION of MENTAL RETARDATION:**

## **\*Definitions across history:**

The struggle to define and classify mental retardation is long-lived. The condition referred to now as mental retardation has been known by many different names (Payne & Patton, 1981). As a result of the conflicting views and definitions of mental retardation, a growing number of labels used to refer to individuals with mental retardation (Heber, 1961). The psychological heritage included a lot of terms that had been long used to denote the phenomenon of mental retardation as a whole, (e.g.: Oligophrenia, Mental deficiency, mental subnormality, mental dullness, mental backwardness, mental retardation, mental handicap, ...) or to describe a subcategory of mental retardation, e.g.: feeble-mindedness or morons, imbeciles and idiots (Alquraity, 1996; Ibrahim, 2000). Moreover, in a number of countries mental retardation is defined by intelligence quotient or mental age for legal purposes (Grossman, 1973).

- **The oldest of these terms is idiocy**, the usage of which can be traced back at least to the **thirteenth century**; “**an idiot** is one who hath no understanding from his nativity” Though the word is now used as an insult, it is derived from the Latin word **idiota**, meaning an ignorant person and from the Greek word **idiotos**, meaning unfit for public life. It was used to refer to individuals with mental retardation of all levels well into the 20th Century (Payne and Patton, 1981).
- **Toward the end of the 17<sup>th</sup> century, in 1690, John Locke** was the first to distinguish between mental retardation and mental illness; “Herein seems to lie the difference between **idiots and madmen**, that madmen put wrong ideas together and reason from them, but **idiots** make very few or no propositions and reason scarce at all” (Doll, 1962).
- **Jean Esquirol, in 1838**, was the first medical writer to differentiated mental retardation from mental illness. Esquirol, in 1845, noted that intellectual disability is not a disease in itself, but the developmental consequence of some pathogenic process. He penned a new definition, describing mental retardation as a disorder of development instead of being a mental disease, and his definition has been maintained in all modern definitions (which require an onset during childhood or adolescence). Moreover, Esquirol proposed several levels of mental retardation. A few years later, **Wilbur**

(1852) defined MR primarily by deficits in social or moral reasoning. Later, **William Ireland** (In 1898), could classify idiocy into ten categories on the basis of etiology including “genetous, microcephalic, eclampsic, epileptic, hydrocephalic, paralytic, cretinism, traumatic, inflammatory, and idiocy by deprivation.” (Sadock & Sadock, 2000).

- The introduction of the **IQ test** (by **Alfred Binet in 1905** in France) was followed in **1910**, by an early classification scheme (proposed by the American Association on Mental Deficiency -AAMD), that referred to individuals with mental retardation as **feeble-minded**, meaning that their development was halted at an early age or was in some way inadequate making it difficult to keep pace with peers and manage their daily lives independently. **Three levels of impairment** were identified:
- **idiot**, individuals whose development is arrested at the level of a 2 year old; with I.Q between 50-70
  - **imbecile**, individuals whose development is equivalent to that of a 2 to 7 year old at maturity; with I.Q between 25-50
  - **moron**, individuals whose mental development is equivalent to that of a 7 to 12 year old at maturity with I.Q between 0-25
- (Biasini et al, 1998)

So, mentally retarded people have been *officially* referred to as: *the “idiots”* and as *the “feble-minded”*. Over the next 30 years, the definitions of mental retardation focused on **one of three aspects of development**: the inability to learn to perform common acts, deficits or delays in social development / competence, or low IQ (Yepsen, 1941).

- **Edgar Doll**, in 1935, based on his pioneering work at the Vineland Training School in Vineland, New Jersey, proposed that the concept of mental retardation referred to **six criteria**: “social & vocational incompetence, mentally subnormal in relation to normal age group, developmentally arrested early or since birth, persistently retarded till age of maturity, is of constitutional origin whether hereditary or acquired, and the condition is *essentially incurable*” (Ibrahim, 2000). Edgar Doll was the first to develop a formal definition and measure of **adaptive behaviour** in the year 1935. Two decades later, the American Association on Mental Retardation (AAMR) officially included “deficits in adaptive behaviour” in its definition of mental retardation. Since then, “deficits in adaptive behaviour” have been formally included in all definitions of mental retardation (Sadock & Sadock, 2000).

- **Alfred Tredgold, in 1952** defined mental handicap as “a state in which the mind can not reach the level of normal development or can not complete that development.” due to defect in the central nervous system because of an organic brain insult that has a significant effect on intelligence. **Jervis, in 1952** defined mental handicap as “a state of arrest or lack of development of mind due to a disease or an injury before adolescence or due to genetic factors during embryonic period.” **Christine Ingram, in 1953** defined the child with mental handicap as “**Slow learner**” with **IQ of 50-70**, and described him to be unable to achieve academically. But, it has been clear that this definition is valid only for one category of the mentally handicapped & it does not explain the cause or time of the handicap, so it is not valid for identification, diagnosis or treatment of such handicap. **Sarason, 1953** defined mental handicap as “a state of social incompetence associated with defect in central nervous system. **Benoit, 1959** tried a more inclusive definition, so he defined mental handicapping as “a state of defective intellectual functions due to internal or external factors to the individual that lead to weakness in the capability of the nervous system, defect in the general ability of development and defect in ability in adaptation.” (Ibrahim, 2000).

The years after, witnessed a change in emphasis from a genetic or constitutional focus to a desire for a function-based definition that became increasingly popular and acceptable.

- **Heber, in 1962** postulated a multi-dimensional definition of mental handicap. He put certain criteria for identifying the mentally-handicapped person including: “*sub-average general intellectual functioning which originates in the developmental period and is associated with impairment in adaptive behaviour*” (Ibrahim, 2000).
- **That definition had been adopted later in 1959 by AAMD** (The American Association on Mental Deficiency) who declared it as a procedural definition of mental handicapping (Ibrahim, 2000). Accordingly, mentally handicapped had been classified into 5 categories. So, a **five level classification scheme** was introduced replacing the previous three level system which had acquired a very negative connotation. The generic terms of *borderline* (IQ 67-83), *mild* (IQ 50-66), *moderate* (IQ 3-49), *severe* (16-32), *and profound* (IQ <16) were adopted. (Biasini et al, 1998) Although this definition included the three components of: low IQ (<85), impaired adaptive behaviour, and

origination before age 16, only IQ and age of onset were *measurable* with the existing psychometric techniques. Deficits in adaptive behaviour were generally based on subjective interpretations by individual evaluators even though the Vineland Social Maturity Scale was available (Sheerenberger, 1983). Due to concern about the over or misidentification of mental retardation, particularly in minority populations, the definition was **revised in 1973** eliminating the borderline classification from the interpretation of significant, sub-average, general intellectual functioning. The upper IQ boundary changed from <85 to < 70. This change significantly reduced the number of individuals who were previously identified as mentally retarded impacting the eligibility criteria for special school services and governmental supports. Many children who might have benefited from special assistance were now ineligible for such help (Grossman, 1973). A **1977 revision** modified the upper IQ limit to (70-75) to account for measurement error (Biasini et al, 1998).

- **The most recent change in the definition** of mental retardation was adopted in 1992 by the American Association on Mental Retardation stating that: "Mental retardation refers to substantial limitations in present functioning. It is characterized by significantly sub-average intellectual functioning, existing concurrently with related limitations in two or more of the following applicable **(10) adaptive skill areas**: communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work. Mental retardation manifests before age 18" (AAMR, 1992). On the surface, this latest definition does not appear much different than its recent predecessors. However, the focus on the functional status of the individual with mental retardation is much more delineated and critical in this definition. There is also a focus on the impact of environmental influences on adaptive skills development that was absent in previous definitions. Finally, this revision eliminated the severity level classification scheme in favour of one that addresses *the type and intensity of support needed*: intermittent, limited, extensive, or pervasive. Practically, a child under age 18 must have an IQ  $\leq 75$  and deficits in at least 2 of the adaptive behaviour domains indicated in the definition to obtain a diagnosis of mental retardation (Biasini et al, 1998). The new definition of AAMR does not view mental retardation as an inherent characteristic of people, but as an interaction between individuals and their environments. With this assumption, the new definition eliminates traditional nosology based on four level of cognitive impairment (i.e., mild, moderate, severe and profound) and instead proposes **four levels of environmental supports** (intermittent, limited,

extensive and pervasive) **across the 10 different adaptive domains**. Thus, instead of giving a person a diagnosis of "moderate mental retardation," the new definition specifies that a person has "intermittent needs for supports in health and safety, limited needs for supports in self-care, and so on, across the 10 domains". Critics argue that this system is unwieldy, is more pertinent for practice than diagnosis, and represents a giant step backward for research as it leaves researchers without a meaningful way to classify subjects. Two other features of the new definition have been hotly debated. The new definition extends the IQ criterion from "70 and below" to "70 or 75 and below," opening up the possibility for a more lenient IQ cut-off point of 75. Many critics thus predict that if followed, the new definition will increase the size of the population with mental retardation, including increases in the overrepresentation of several minority groups (Sadock & Sadock, 2000).

### **\*Does IQ remain unchanged by time!**

William White (1919) observed that "**feeble-mindedness**", is a relative affair when expressed in the behaviour of an individual, and a "conduct" which would be considered normal under certain conditions is possibly considered defective, under others (Sadock & Sadock, 2000). **Goddard**, the research director at the Vineland Training School (in New Jersey, USA), was the first to use Binet and Simon's intelligence quotient (IQ) tests in the United States. After testing Vineland residents over several years, Goddard concluded that "the vast majority of feeble-minded children are not changing and are **not improving** in their intelligence levels," a finding that another leading worker, Walter Fernald, called "the most significant and the most discouraging that we have ever known." (Sadock & Sadock, 2000). **Edgar Doll**, in 1941 proposed that the concept of mental retardation referred to 6 essential criteria including the condition is essentially **incurable**" (Doll, 1941).

As reasoned by many researchers, the prevalence rate of mental retardation of 3% considers IQ the sole criterion for mental retardation. That is, persons with an IQ below 70 are considered to have mental retardation, those with an IQ of 70 or above do not. The 3% prevalence rate of mental retardation assumes that "**an individual's IQ remains relatively stable over time**". This assumption seems justified in some ways, unjustified in others. Indeed, across the entire IQ distribution, a median correlation of .77 has been shown between testings at age 4 and age 12. However, correlations between IQ scores during infancy and later IQ scores are essentially zero. That is, with the exception of infants with whose IQ scores are very-low, any baby's IQ score (e.g., "developmental quotient"; DQ, on



the Bayley Scale) has **little relation to that child's IQ during later childhood or adulthood**. Further, **different groups may show different trajectories of IQ with development**. Children with Down syndrome show their highest IQ (or DQ.) scores during the first year of life, then decline in IQ over the early and middle childhood years. Boys with fragile X syndrome also decline in IQ, but their declines seem to begin at approximately 10 to 15 years. Conversely, children with cerebral palsy (half of whom have mental retardation) remain remarkably stable in their IQ scores over time, much like groups with mixed or nonspecific etiologies of mental retardation (Sadock & Sadock, 2000). Also, persons with mental retardation have increased risks of co-morbid psychiatric or behavioural dysfunction. The course of mental retardation is influenced by the course of underlying general medical conditions and by environmental factors (e.g. educational and other opportunities, environmental stimulation, and appropriateness of management). If an underlying general medical condition is static, the course is more likely to be variable and to depend on environmental factors. **Mental retardation is not necessarily a lifelong disorder**. Individuals who had Mild Mental Retardation earlier in their lives manifested by failure in academic learning tasks may, with appropriate training and opportunities develop good adaptive skills in other domains and may no longer have the level of impairment required for a diagnosis of mental retardation (APA, 1994). In many ways, administering IQ tests to persons with mental retardation is quite challenging, both in terms of the testing situation itself and in the choice of an appropriate IQ test (Sadock & Sadock, 2000).

It is not strange for IQ of an individual to change within 50 points up or down due to gross environmental changes, changes in the family structure or in domestic circumstances, fostering by an alternate family, severe prolonged illness or preventive or therapeutic programs. It can be said that: in general, the intelligence of a child in a deprived environment is likely to decrease, while that of a child in a rich-resources environment is likely to increase (Melaika, 1997).

Thus, central to the struggle with how to conceptualize and understand mental retardation is the idea that something more than cognitive deficits alone or low IQ scores alone is involved. Over the years, the American Psychiatric Association (APA) adopted definitions of mental retardation from the AAMR (American Association on Mental Retardation) who officially in 1950's included "*deficits in adaptive behaviour*" in its definition of mental retardation. Since then, "*deficits in adaptive behaviour*" have been formally included (along with low IQ) in all definitions of mental retardation presented by APA till the fourth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) in 1996. Both DSM-IV and ICD-10 (the 10th revision of the International Statistical Classification of Diseases and Related Health Problems) in their diagnostic criteria

for mental retardation specify an **IQ of 70 or less** along with **deficits in adaptive behaviour. IQ scores** are presumably derived from “standardized intelligence tests” that meet appropriate psychometric criteria for reliability and validity. (Sadock & Sadock, 2000) Moreover, an IQ score may involve a measurement error of approximately 5 points, depending on the testing instrument (APA, 1994).

Though all known intelligence tests are generally acceptable, diagnosticians should shy away from tests that tap a single domain (e.g., receptive vocabulary) in favour of more extensive batteries such as the Wechsler or Kaufman tests, as these rely on performance across multiple cognitive domains. Nevertheless, these tests are best used for screening or research purposes only and should not be used alone to diagnose mental retardation. So, **to receive a diagnosis of mental retardation, individuals must be brought to the attention of professionals, tested, and found to meet definitional criteria** (Sadock & Sadock, 2000).

## ***\*Adaptive Behaviour:***

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Adaptive behaviour is an inherently developmental and social construct. Adaptive behaviour is the collection of conceptual, social, and practical skills people have learned so they can function in their lives. Significant limitations impact a person's daily life and affect the ability to respond to the environment." (AAMR, 2002).

Though meanings vary, adaptive behaviour can be typically **viewed as** the performance of behaviours required for personal and social sufficiency. It is the person's effectiveness in meeting the standards expected for his or her age by his or her cultural group as demands for social adaptation are defined by expectations from others i.e. from one's family, society, and culture (Sadock & Sadock, 2000). Adaptive functioning refers to how effectively individuals cope with common life demands and how well they meet the standards of personal independence expected of someone in their particular age group, socio-cultural background, and community setting (APA, 1994). Areas of Adaptive skills are those daily living skills needed to live, work and play in the community. They include: self-care, self-direction, home living, communication, social skills, leisure, health and safety, functional academics (reading, writing, basic math), community use and work (The Arc, 1998).

Adaptive behaviour changes as children grow into adolescence and adulthood. Also, adaptive skills typically change across various settings; one's adaptive performance on the job or at school may differ from one's performance with friends or at home. If individuals with mental retardation can perform certain behaviours but for any reason do not routinely do so, then they necessarily have "compromised adaptive functioning" (Sadock & Sadock, 2000). Problems in adaptation are more likely to improve with remedial efforts than is the cognitive IQ, which tends to remain a more stable attribute (APA, 1994).

Measurements of adaptive behaviour need to have a developmental orientation, to be socially and culturally sensitive, and to represent the many settings in which people live, work, and play. Adaptive behaviour is measured by typical, everyday performance, not ability (Sadock & Sadock, 2000). Adaptive skills are assessed in the person's typical environment across all aspects of an individual's life. A person with limits in intellectual functioning

who does not have limits in adaptive skill areas may not be diagnosed as having mental retardation (The Arc,1998).

Many scales are acceptable measures of adaptive behaviour in persons with mental retardation, yet the Vineland Adaptive Behaviour Scale (Appendix-I,II,III) probably enjoys the most widespread use. Based on his pioneering work at the Vineland Training School in Vineland, New Jersey, Edgar Doll, in 1935, was the first to develop a formal definition and measure of adaptive behaviour. Though many still feel that adaptive behaviour does not belong in the definition of mental retardation, virtually all workers agree that adaptive skills are critical to the long-term adjustment and success of people with mental retardation. Different studies, however, find different correlations between cognitive and adaptive behaviour, strong correlations are especially observed in persons with moderate-to-profound mental retardation but weak correlations are found between cognitive and adaptive behaviour in persons with mild mental retardation. Thus, to resolves at least some of the controversy about the relative importance of these two constructs, **I.Q. may be considered as an upper limit or ceiling to adaptive accomplishments** (Sadock & Sadock, 2000).

# **\*Epidemiology of Mental Retardation:**

## **\*Prevalence:**

Mental retardation is a universal problem that knows no boundaries. It cuts across the lines of racial, ethnic, educational, social and economic backgrounds. It can occur in any family as well (The Arc, 1998). Mental retardation is 10 times more common than cerebral palsy. It affects 25 times as many people as blindness and 28 times more than neural tube defects such as spina bifida (Batshaw, 1997).

Over the past 50 years the prevalence and incidence of mental retardation have been affected by changes in the definition of mental retardation, improvements in medical care and technology, societal attitudes regarding the acceptance and treatment of an individual with mental retardation, and the expansion of educational services to children with disabilities (Biasini et al, 1998).

### **1% OR 3% Prevalence Rate?:**

According to some estimates, approximately **1% of the population** has mental retardation. This one percent figure is cited by DSM-IV and is roughly the percentage found in most prevalence studies. Yet this widely cited 1% figure hides a variety of controversies within mental retardation. In particular, many have reasoned that mental retardation is as frequent as nearer to a **3% prevalence rate** as concluded by most workers of the 1960's and 1970's. (Sadock & Sadock, 2000) Different studies have reported different rates depending on: definitions used, methods of ascertainment, and population studied (APA, 1994).

In Egypt, Farrag reported that about 3% of the Egyptian population suffers from mental retardation taking in consideration that other disorders with similar symptoms to mental retardation, like autism, learning disabilities, and Asperger disorder, were not included (Farrag, 1995).

Given a definition that features an I.Q. below 70 and deficits in adaptive behaviour, the prevalence rate is probably below 3% but above 1% (Sadock & Sadock, 2000). This 3% of total population is **not fixed in all societies**; it increases with decreased economic and cultural levels in society to reach up to **7%** in areas crowded with poor people. In a study done by the Research centre in Arizona University (1982), they concluded that the prevalence of mental handicap in the

white community of high economic level did not exceed 3 %, while it reached double this percentage (6%) amongst **immigrants** from Mexico working in American cotton fields. Also, in a study by The Supreme Corporation for Care of the Handicapped in **Egypt** (1985), the prevalence of mentally handicapped in high and above average socioeconomic population was (3-3.3)% and reached **up to (7)%** in some districts with high-density and poor socioeconomic population (Ibrahim, 2000).

A more striking characteristic, however, is that more people with 'mild mental retardation' come from **minority groups and low socioeconomic backgrounds** than would be expected from their percentages in the general population. This over-representation of minority groups has been used to criticize I.Q. tests and to highlight the importance of both environmental-cultural and genetic influences on mental retardation (Sadock & Sadock, 2000).

Mental Retardation is more common among males, with a **male-to-female ratio of approximately 1.5 : 1**. (APA, 1994) In **Egypt**, Okasha et al (1983) reported a male : female ratio of **2 : 1** (Farrag, 1995) Male predominance may be the result of the culturally determined higher premium on male children in the society with parents being usually more inclined to report intellectual retardation in male children as compared to females. Additionally, relatively low emphasis on the education of girls (especially in rural areas) could also be responsible to some extent (Okasha et al, 1983).

### **\*Familial pattern:**

It should be stressed that *familial does not necessarily mean genetic* and there is plenty of room for the impact of socio-economic factors in cases of mild reduction of intelligence. (Price, 1982) Because of its heterogeneous etiology, no familial pattern is applicable to mental retardation as a general category. (APA, 1994) In mental retardation caused by genetic and chromosomal abnormalities, there are characteristic inheritance patterns for various lesions (e.g., trisomy 21 and metabolic diseases). In mild retardation, there is no familial pattern, although it may be more common in some families (Shaner, 1997).

## ***\*Etiology of Mental Retardation:***

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Mental retardation is a symptom, a syndrome, a condition, and a source of pain and bewilderment to many families. Mental retardation is a challenge and a potential source of stress to the family of an individual with this disorder. From identification through treatment or education, families struggle with questions about **cause and prognosis**, as well as guilt, a sense of loss, and disillusionment about the future (Scheerenberger, 1983). Whilst some of the cases of mental handicap which are ascribed to unknown causes will doubtless prove to be due to the operation of single genetic or environmental factors, most of the unclassified cases are likely to be produced by multiple factors, often representing both unfavourable environmental and heredity interacting in the course of development and maturation (Price, 1982).

Etiological factors may be primarily biological or primarily psychosocial or some combination of both. In approximately **30-40%** of individuals seen in clinical settings, **no clear etiology** for the mental retardation can be determined despite extensive evaluation efforts (APA, 1994). **Most cases of mild mental retardation are idiopathic** in origin (Shaner, 1997).

With more advances in medicine generally and in molecular genetics in particular, new causes of mental retardation or the genetic causes of formerly unspecified syndromes are identified each year. Recently, the **AAMR** offered an admittedly partial listing that enumerated **over 350 causes of mental retardation**. **John Opitz** counted **over 750 genetic causes** of intellectual disability alone. **Eleanor Feldman** noted that **some 95 mental retardation syndromes have been linked to the X chromosome**; the most common of which are: Down syndrome, fragile X syndrome and fetal alcohol syndrome. As clinicians approach the cause of mental retardation in a particular patient, it is helpful to work from a broad framework initially. For example, an initial distinction might be drawn between **congenital and acquired** causes. For the latter, the timing of the insult that led to retardation may be further broken down into **perinatal or postnatal** causes. More severe presentations of mental retardation (Severe and profound mental retardation with visible congenital anomalies) are more likely to have an organic cause, particularly an identifiable chromosomal abnormality, and are more likely to be institutionalized (Sadock & Sadock, 2000).

## **\*Predisposing factors:**

The major predisposing factors include:

- **Heredity** (approximately 5 %): including inborn errors of metabolism inherited mostly through autosomal recessive mechanisms (e.g., Tay-Sachs disease), other single-gene abnormalities with Mendelian inheritance and variable expression (e.g. tuberous sclerosis) and chromosomal aberrations (e.g., translocation Down's syndrome, fragile X syndrome).
- **Early alterations of embryonic development** (approximately 30%): including chromosomal changes (e.g., Down's syndrome due to trisomy 21) or prenatal damage due to toxins (e.g., maternal alcohol consumption, infections).
- **Pregnancy and perinatal problems** (approximately 10%): including prematurity, fetal malnutrition, hypoxia, trauma, viral and other infections (e.g. rubella, cytomegalovirus).
- **Early general medical conditions** acquired in infancy or childhood (approximately 5%): including infections (CNS infections e.g., herpes virus), traumas, anoxia and exposure to toxins.
- **Environmental influences and other mental disorders** (approximately 15-20%): These factors include deprivation of nurturance of social, linguistic, and of other stimulation. Some known risk factors for mental retardation are more common in lower socioeconomic environments, including: intra-uterine exposure to toxins and infection, poor prenatal care and postnatal exposure to heavy metals and physical trauma. Also severe mental disorders constitute a predisposing factor for mental retardation (e.g. Autistic Disorder).

(APA, 1994).



# **\*DIAGNOSIS of MENTAL RETARDATION:**

The diagnostic criteria for Mental Retardation **do not include an exclusion criterion**. Therefore, the diagnosis should be made whenever the diagnostic criteria are met regardless of and in addition to the presence of another disorder. Other than the results of psychological and adaptive behaviour tests that are necessary for the diagnosis of mental retardation, there are no laboratory findings that are uniquely associated with mental retardation. Diagnostic laboratory findings may be associated with a specific accompanying general medical condition (e.g. chromosomal findings in various genetic conditions, high blood phenylalanine in phenylketonuria or abnormalities on central nervous system imaging) (APA, 1994).

## **\*DSM-IV Diagnostic Criteria for Mental Retardation:**

### **\*Essential Characters:**

#### **A. Significantly sub-average intellectual functioning:**

An IQ of approximately 70 or below on an individually administered IQ test. (for infants, a clinical judgement of significantly sub-average intellectual functioning).

#### **B. Concurrent deficits or impairments in present adaptive functioning:**

(i.e., the person's effectiveness in meeting standards expected for his or her age by his or her cultural group) in at least two of the following areas: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health and safety.

#### **C. The onset is before age 18 years.**

(APA, 1994).

\* **Extent of associated impairment of behaviour:** (This fourth character may be used to specify associated impairment of behaviour, if any):

- No, or minimal, impairment of behaviour
- Significant impairment of behaviour requiring attention or treatment
- Other impairments of behaviour
- Without mention of impairment of behaviour

(WHO, 1993).

\* **Degree of severity:** (This additional Code is used to reflect the level of intellectual impairment):

- **Mild** mental retardation: IQ level (50–55) to approximately 70
- **Moderate** mental retardation: IQ level (35–40) to (50–55)
- **Severe** mental retardation: IQ level (20–25) to (35–40)
- **Profound** mental retardation: IQ level **below** (20 or 25)
- Mental retardation, **severity unspecified:** when there is a strong presumption of mental retardation but the person's intelligence is untestable by standard tests.

(APA, 1994).

### **Levels of Mental Retardation (Degree of severity) in detail:**

At present, the field lacks a classification system that reflects the remarkable diversity, strengths, and competencies of people with mental retardation. Until a viable new scheme is developed, it makes sense to use traditional nosology to describe persons with mental retardation (Sadock & Sadock, 2000).

#### **Mild Mental Retardation:** (IQ, 50-55 to 70)

Mild mental retardation characterizes the largest group of persons with mental retardation, possibly as many as 85% of the total. These individuals appear similar to non-retarded individuals and often blend into the general population in the years before and after formal schooling (Sadock & Sadock, 2000). Many achieve academic skills at the sixth grade level or higher (though at elder age of course, by their late teens); hence such group of mentally retarded is termed “*educable*”. As a group, they typically develop social and communication skills during the preschool years, have minimal impairment in sensori-motor areas and often are not distinguishable from children without mental retardation until a later age (APA, 1994). Their predicament usually becomes apparent in early adolescence, as the social and scholastic demands placed on them increase (Fogel et al, 2001).

As adults, they usually achieve social and vocational skills adequate for minimum self-support. Many of these individuals hold jobs, marry, and raise families-yet at times they may appear slow or need extra help in the form of supervision , guidance & assistance , especially when under unusual social or economic life stress. With appropriate supports, such individuals can usually live successfully in the community, either independently or in supervised settings (APA, 1994). Persons with mild disability usually reside in community settings, unlikely to be institutionalized, manageable by their families and even sometimes

unnoticed to be retarded. Traditionally, individuals with mild mental retardation were thought to show relatively few clear-cut organic causes for their delay. While this may still be the case, recent years have seen an increase in the number of people with genetic syndromes who function in the mild range. Examples include most people with Prader-Willi syndrome and some males and most females with fragile X syndrome. A more striking characteristic, however, is that more people with mild mental retardation come from **minority groups and low socioeconomic backgrounds** than would be expected from their percentages in the general population. This overrepresentation of minority groups has been used to criticize IQ tests and to highlight the importance of both environmental-cultural and genetic influences on mental retardation (Sadock & Sadock, 2000).

### **Moderate Mental Retardation:** (IQ, 35-40 to 50-55)

Moderate mental retardation is seen in approximately 10% of those with mental retardation, including persons with more-impaired cognitive and adaptive functioning (Sadock & Sadock, 2000). Moderate mental retardation is usually recognized early in life, when developmental mile stones such as language are delayed (Fogel et al, 2001). So, individuals with moderate mental retardation typically receive their diagnosis in their preschool years, and some show a clear organic cause for their delay. Persons with Down syndrome often function in this range, as do many adolescents and adults with fragile X syndrome. Individuals with moderate mental retardation used to be referred to as “*trainable*”, yet this outdated term should not be used because it wrongly implies that they can not benefit from educational programs (APA, 1994). Most children with moderate mental retardation require special education services and can achieve academic skills at the second to third grade level only. Supportive services are needed throughout life. With proper supports, many live, work, and thrive in their local communities (Sadock & Sadock, 2000).

Most of these children also, can acquire communication skills during early childhood years. During adolescence, they show difficulties in recognizing social conventions leading to disturbed peer relationships. During adulthood, most of them are able to perform unskilled or semiskilled work under supervision in sheltered workshops or in the general work force. They profit from vocational training & with moderate supervision, can attend to their personal care and adapt well to life in the community, usually in supervised settings. (APA, 1994)

### **Severe Mental Retardation:** (IQ. 20-25 to 35-40)

Severe mental retardation occurs in about 3-4% of persons with mental retardation. Individuals at this level often have one or more organic causes for their delay, and many show concurrent motor, ambulatory, and neurological problems as well as poorly developed communication skills (Sadock & Sadock, 2000).

The deficits present in these individuals are apparent at very young ages and range from severely defective speech to somato-sensory deficits to motor handicaps (Fogel et al, 2001). During their early childhood years, they acquire no or little communicative speech. During the school-age period, they may learn to talk & can be trained in elementary self-care skills. They profit to only a limited extent from instruction in pre-academic subjects, such as familiarity with the alphabet and simple counting, but can master some few skills such as learning sight reading of some "survival" words. In their adult years, they may be able to perform simple tasks in closely supervised settings. Most adapt well to life in the community, in group homes, or with their families, unless they have an associated handicap that requires specialized nursing or other care (APA, 1994).

Most of these persons require close supervision and specialized care throughout their lives. Some individuals learn to perform simple tasks or routines that facilitate their self-care or their ability to perform in a sheltered workshop or preworkshop-type setting (Sadock & Sadock, 2000).

### **Profound Mental Retardation:** (IQ. of 20-25 or below)

Profound mental retardation affects relatively few individuals (1 to 2 %) and involves pervasive deficits in cognitive, motor, and communicative functioning. Most of these individuals have an identifiable neurological condition that accounts for their mental retardation (APA, 1994). The problems present in these individuals are often noticeable at birth, owing to the presence of severe physical malformations and other obvious symptoms of abnormality. As with the severely retarded, gross central nervous system pathology is almost always present (Fogel et al, 2001).

During early childhood years, they display considerable impairments in sensori-motor functioning. Optimal development may occur in a highly structured environment with constant aid & supervision and an individual relation with a trained caregiver. Motor development, self-care and communication skills may improve if appropriate training is provided (APA, 1994). The profoundly retarded show multiple handicaps and severe deficits in adaptive skills and are *very resistant to learning*. (Fogel et al, 2001) In general, most of them require total supervision and care throughout life (Sadock & Sadock, 2000).

### **Mental Retardation, Severity Unspecified:**

The diagnosis of Mental Retardation, Severity Unspecified, should be used when there is a strong presumption of mental retardation but the person cannot be successfully tested by standard intelligence tests. This may be the case when children, adolescents or adults are too impaired or uncooperative to be tested or, with infants, when there is a clinical judgment of significantly sub-average intellectual functioning, but the available tests e.g., the Bayley Scales of Infant Development, Cattell Infant Intelligence Scales and others) do not yield IQ values. In general, the younger the age, the more difficult it is to assess for the presence of mental retardation except in those with profound impairment (APA, 1994). In practice, children under the age of 2 should not be given a diagnosis of mental retardation unless the deficits are relatively severe and/or the child has a condition that is highly correlated with mental retardation e.g., Down syndrome (Biasini, 1990).

### **N.B: Borderline Intellectual Functioning: (I.Q., 71-84)**

This label describes an IQ range that is higher than that for mental retardation (generally 71-84). The executive definition of mental handicapping adopted in 1959 by AAMR {The American Association on Mental Deficiency (Retardation)} had classified mentally handicapped into 5 categories (Ibrahim, 2000) to include for the first time the generic term of *borderline mental retardation* with IQ (67-83) (Biasini et al, 1998). Due to concern about the over or misidentification of mental retardation, particularly in minority populations, the definition was revised in 1973 (Grossman, 1973) eliminating the borderline classification from the interpretation of significant, sub-average, general intellectual functioning. The upper IQ boundary changed from  $<85$  to  $\leq 70$ . This change significantly reduced the number of individuals who were previously identified as mentally retarded (Grossman, 1977). As discussed earlier, an IQ score may involve a measurement error of approximately 5 points, depending on the testing instrument. Thus, it is possible to diagnose mental retardation in individuals with IQ scores between 71 and 75 if they have significant deficits in adaptive behaviour that meet the criteria for mental retardation. Differentiating Mild Mental Retardation from Borderline Intellectual Functioning requires careful consideration of all available information (APA, 1994).

Depending upon the cultural norms and expectations of the individuals being studied, research workers must make their own judgments as to how best to estimate intelligence quotient (IQ.) or mental age according to the bands given below: (Table -1)

(Table-1): Degrees of mental retardation

<b>Severity of MR:</b>	<b>IQ range</b>	<b>Maximum Mental Age Reachable</b>	<b>Percentage of cases</b>
<b>Mild</b>	50-55 to 70	9 to under 12 years	85%
<b>Moderate</b>	35-40 to 50	6 to under 9 years	10%
<b>Severe</b>	20-25 to 35	3 to under 6 years	3.5-4%
<b>Profound</b>	Below 20	Less than 3 years	1-1.5%

From the above, it is clear that the more severe the mental retardation, the earlier it is detected. It is worthy also to mention that severe and profound mental retardation is often associated with premature death (Kolevzon & Simeon, 2002).

## **\*Maladaptive Behaviour and Co-morbidity:**

Persons with mental retardation have increased risks of co-morbid psychiatric or behavioural dysfunction. (Sadock & Sadock, 2000). Some individuals with mental retardation have **significant psychiatric symptoms** that in fact, do not allow a clear distinction between certain psychiatric diagnoses. It may be very difficult to distinguish between an “impulse control disorder not otherwise specified” (perhaps characterized by an individual who engages in impulsive aggressive acts) and an “anxiety disorder not otherwise specified” (perhaps suggested by an individual who strikes out in the context of a stressor that would go unnoticed by most people) (Sadock & Sadock, 2000). A child with developmental or intellectual delay will be slower to grow out of child’s practices, and slower to learn tolerance, adequate communication or postponement of gratification (so, may behave by tantrums in some situations) (Spender et al, 2001).

It is worth noting that, in clinical practice, the **distinction between adjustment reactions & adjustment disorders** is not always clear cut. Achenbach in 1991, pointed to the results of multivariate studies which suggested that “**adjustment disorders with a disturbance of conduct and ‘disruptive behaviour disorders’ fall along a continuum of “externalizing behaviour problems”**. A second distinction is that made between “**Oppositional Defiant Disorder**” and “**Conduct Disorder**” with the former reflecting a less pervasive disturbance than the latter and possibly being a developmental precursor of conduct disorder (Carr, 2002).

# \* **APPROACH to MALADAPTIVE BEHAVIOR:**

## \* **What is “Behaviour”?**

Essentially, **behaviour** is: anything that a person says or does. Some commonly used **synonyms** include "activity," "action," "reaction," "performance," and "response". For a behaviour to be successful, it must occur in an appropriate place at an appropriate time and must be appropriate in its form (Martin & Pear, 1988).

\* **What is “Adaptive Behaviour”?** Adaptive Behaviour has been already explained in details under (Literature review) page 19.

## \* **What is “Mal-adaptive Behaviour”?**

**Synonyms: ( = Behavioural Disturbance = Disruptive Behaviour  
= Problem Behaviour = Challenging Behaviour )**

**Behaviour problems** are the “bete noire” of child psychiatry, at any level of service provision because they are such a common presenting complaint & they may appear defying easy remedies. The key question to consider is whether the behaviour results in dysfunction or not. Behaviour problems that are serious enough to cause some dysfunction are common, **affecting approx. 5% of children** and 10% of adolescents. They are more common in urban than rural areas (Spender et al, 2001).

**Disruptive behaviour disorders** (e.g., conduct disorder, oppositional defiant disorder and disruptive behaviour disorder not otherwise specified) are the most common psychiatric disorders of childhood, **occurring in 4%-9% of pediatric population**. The prevalence of disruptive behaviour disorders is particularly high among children with a below-average IQ. One study reported that behavioural disturbances were **three to four times more common** in children with intellectual limitations than in comparison children of the same age. Disruptive behaviour disorders are associated with sequelae that may result in serious **consequences** for both the child and society, including legal trouble, school suspension, substance abuse, and physical injury. Many children with intellectual limitations and severe behavioural disorders require out-of-home placement. The costs of caring for



individuals with disruptive behaviour disorders, which may include loss of productivity and costs of health care, housing, law enforcement, and security, as well as victim and family costs, are substantial. In 1989, the cost of caring for intellectually disabled persons who exhibited destructive behaviour was approximately 3 billion US dollars (Aman, 2002).

Mentally retarded children frequently show (as part of their permanent dysfunctioning) one or more item of **mal-adaptive behaviour** that interrupt the process of their rehabilitation and learning and necessitate comprehensive psychiatric attention along with family and social support (Carr, 1999). There is a wide range of such maladaptive behaviour. It can be just simple & tolerable by other family members e.g. *mild irritability or occasional stubbornness* or might be severe & very noisy e.g. *poor frustration tolerance, persistent hyperactivity, impulsivity, destructive or aggressive behaviour or disinhibited & embarrassing talks & acts* (Sadock & Sadock, 2000).

Individuals labelled "retarded," have **behaviour problems**, that is, **behavioural deficits** (too little behaviour of a particular type), or **behavioural excesses** (too much behaviour of a particular type), or both behavioural deficits and excesses. (Martin & Pear, 1988) It is most helpful to view **disruptive behaviour disorders** (conduct disorder, Oppositional defiant disorder, ADHD) as existing along a continuum (Spender et al, 2001).

There is an increased, incidence of '**behavioural disturbance**' shown by people with disabilities. More recently the terminology has changed to emphasize the interactional and contextual nature of most behaviours, from "**problem behaviours**" to "**challenging' behaviours**". This then begs the question 'challenging to whom'? Complaints about a person's behaviour will vary between contexts and from person to person. It is also often the case that behaviour is rarely 'challenging' to the persons themselves (although self-injurious behaviour may be an exception). A challenging behaviour seen in interactional terms may be a communication of, for example, boredom, irritation, frustration, anger or joy (Puri et al, 1996).

## **Common challenging behaviours: (Puri et al, 1996)**

### **1) Violence to self or others: (self-injury/aggressive/destructive behaviours)**

- Biting
- Hitting
- Scratching
- Pinching
- Spitting
- Head banging
- Tantrums
- Property damage

### **2) Behaviours out of usual context: (passivity/negativism, run away, disinhibition and somatic symptoms)**

- Passivity and oppositional behaviour
- Shouting
- Running away
- Urination / Defecation / Vomiting
- Undressing
- Masturbation
- Sexual behaviours towards others

### **3) Generally inappropriate behaviours: (stereotypy and stealing)**

- Rocking
- Flapping
- Stealing
- Kleptomania

As with child psychiatry in general, little specificity can be attached to a given symptom. Persons with mental retardation typically are referred for evaluation because of self-injurious, aggressive, impulsive, or hyperactive behaviour. These symptoms lack diagnostic specificity and no diagnostic decision tree can be constructed. It may be more useful to ask a series of questions about the expression of a particular behaviour. If the behaviour is of recent onset, one is more likely to consider an acute medical or psychiatric cause. If the behaviour is highly situational, occurring primarily in the context of the stress of task demands, the likelihood of a psychosis or mood disorder is probably reduced. (Sadock & Sadock, 2000).

Disruptive behaviour disorders are associated with **sequelae** that may result in serious **consequences** for both the child and society, including ***legal trouble, school suspension, substance abuse, and physical injury***. Many children with intellectual limitations and severe behavioural disorders require out-of-home placement. The **costs** of caring for individuals with disruptive behaviour disorders (which may include loss of productivity, costs of health care, housing, law enforcement and security, as well as victim and family costs) are substantial. In 1989, in USA, the cost of caring for intellectually disabled persons who exhibited destructive behaviour was approximately 3 billion US dollars (Aman, 2002). There is a need to overcome behaviour problems and to establish more desirable behaviours (Martin & Pear, 1988).

## ***\*Co-morbidity:***

(Associated disorders with mental retardation)

### **Co-morbid physical, mental & Behavioural disorders:**

Mentally retarded individuals often do not clearly fall into a single diagnostic category. **Co-morbidity is common.** Additionally, some individuals have psychiatric symptoms that significantly interfere with habilitative function but do not allow a clear distinction between certain diagnoses. It may be very difficult to distinguish between an impulse control disorder not otherwise specified (perhaps characterized by an individual who engages in impulsive aggressive acts) and an anxiety disorder not otherwise specified (perhaps suggested by an individual who strikes out in the context of a stressor that would go unnoticed by most people). The clinician should always make a best effort to generate working diagnosis and be prepared to modify it as indicated by data gathered through collateral sources and from increasing familiarity with a particular patient (Sadock & Sadock, 2000).

### **Co-morbid general *medical* conditions:**

*Medical* co-morbidity is the rule in individuals with developmental disabilities, and the importance of identifying and treating underlying medical problems (or refining that treatment) cannot be overstated (Sadock & Sadock, 2000). There are **no specific physical features associated with mental retardation.** When mental retardation is part of a specific syndrome, the clinical features of that syndrome will be present (e.g. the physical features of Down's syndrome). The more severe the mental retardation (especially if it is severe or profound), the greater the likelihood of presence of neurological (e.g. seizures), neuro-muscular, visual, auditory, cardiovascular and other conditions (APA, 1994). Also, the number of associated disorders appears to increase with the level of severity of mental retardation (Baird & Sadovnick, 1985).

The application of the diagnoses of organic mental syndromes and disorders is best approached as if patients do not have mental retardation. The same principle should apply to Axis II personality disorders. The diagnosis of a personality disorder due to a general medical condition is best reserved for individuals whose pre-existing personality was altered in a pathological way by some additional cerebral insult. In essence, this category was reserved for patients whose mental retardation is acquired, usually secondary to trauma experienced in childhood or early adolescence (Sadock & Sadock, 2000). Optimum treatment of associated general

medical conditions may improve the individual's overall level of cognitive and adaptive function (Shaner, 1997).

### **Co-morbid *Mental Disorders*: (Dual Diagnosis)**

Individuals with mental retardation have a prevalence of co-morbid *mental disorders* that is estimated to be **3 to 4 times greater than in the general population**. In some cases, this may result from a shared etiology that is common to mental retardation and the associated mental disorder (e.g., head trauma may result in Mental Retardation and in Personality Change Due to Head Trauma) (APA, 1994).

**One third to two thirds** of mentally retarded patients have concomitant mental disorder (Kaplan & Sadock, 1996). Studies estimating the prevalence of mental health disorders among individuals with mental retardation suggest that between **10 and 40% meet the criteria for a dual diagnosis** of mental retardation and a mental health disorder (Reiss, 1990). Feldman stated that about **40%** of individuals with mental retardation will meet criteria for another psychiatric disorder. He described some contributing factors beyond this high rate including: parental disappointment and rejection, low self esteem, and direct effect of CNS dysfunction (Feldman et al, 2000). Okasha found that the prevalence of psychiatric disorders in mentally retarded patients was estimated by **58%** (Okasha et al, 1983). The range in prevalence rates appears to be due to varying types of population sampling. When case file surveys are conducted, the prevalence rates are consistently around 10%. The use of psychopathology rating scales in institutional or clinic samples produces the much higher 40% prevalence rate (Reiss, 1990). The actual prevalence may lie somewhere in between these two estimates. This may be the case due to the tendency of mental health professionals to consider behaviour disorders in individuals with mental retardation as a symptom of their delayed development (Biasini et al, 1998).

Nevertheless, individuals with mental retardation appear to display the full range of psychopathology evidenced in the general population (Jacobson, 1990). Individuals with 'mild' cognitive limitations are more likely to be given a **dual diagnosis** than children with more significant disabilities (Borthwick et al, 1990).

Appropriate assessment of psychopathology in people with dual diagnosis is important because: a) it can suggest the form of treatment; b) it may ensure access to and funding for special services; and c) it can be used to evaluate subsequent interventions. Brain damage, epilepsy and language disorders are risk factors for psychiatric disorders and are often associated with mental retardation (Sturmeay,

1995). Social isolation, stigmatization, and poor social skills put individuals with mental retardation at further risk for affective disorders. (Reiss & Benson, 1985) Rates of emotional disorders are more prevalent in children with mental retardation than children without mental retardation (Bregman, 1988). As noted previously, epidemiological studies of psychiatric disorders in individuals with mental retardation show that this population experiences higher rates of psychopathology (Corbett, 1985).

All types of mental disorders may be seen, and there is no evidence that the nature of a given mental disorder is different in individuals who have mental retardation. However, the diagnosis of co-morbid mental disorders is often complicated by the fact that the clinical presentation may be modified by the severity of the mental retardation and associated handicaps, e.g. deficits in communication skills may result in an inability to provide an adequate history in a non-verbal person with mental retardation having "Major Depressive Disorder". **The most common associated mental disorders** are Attention Deficit / Hyperactivity Disorder, Mood Disorders, Pervasive Developmental Disorders, Stereotypic Movement Disorder, and Mental Disorders Due to a General Medical Condition (e.g. Dementia Due to Head Trauma) (APA, 1994).

A variety of disorders are associated with mental retardation including speech/language problems, and **behaviour problems** (McLaren & Bryson, 1987), yet, **no specific personality and behavioural features are uniquely associated with mental retardation**. Some individuals with mental retardation are passive, placid, and dependent, whereas others can be aggressive and impulsive (APA, 1994).

**The most common associated mental disorders** with mental retardation are:

**Attention-Deficit/Hyperactivity Disorder (ADHD):**

The rates of attention-deficit/hyperactivity disorder in mental retardation are estimated to be between 9 and 18 %. ADHD is mainly characterised by poor attention, hyperactivity and impulsivity. For persons with mental retardation, the diagnosis of attention-deficit/hyperactivity disorder is qualified as being *excessive for an individual's mental age* (Sadock & Sadock, 2000). Some cases are explained by sensory hypersensitivity, others on constitutional basis. Whatever the cause is, due to restlessness and short attention span, the child finds it difficult to learn and socialize. People around reject or punish the child because of his/her disruptive behaviour (Kaplan, 1983).

### **Impulse-Control Disorders: (Aggression and Self-Injury)**

Aggression and self-injurious behaviour are common in mental retardation and increase as cognitive disability becomes more severe. Every effort should be made to treat the underlying cause of aggression or of self-injurious behaviour, not merely suppress the behaviour. Often clinicians encounter situations in which an individual does not evidence remarkable psychomotor activity or attentional difficulties but may be **unusually impulsive**. In these situations one should entertain the diagnosis of an impulse control disorder not otherwise specified. Such a diagnosis, for example, might be appropriate for an individual who inexplicably strikes out at a peer in the absence of any identifiable environmental stressor (Sadock & Sadock, 2000). There is nothing intrinsic in the nature of mental handicap which predisposes to psychopathology or what used to be called "moral defectiveness", but some of the mentally handicapped, like other sections of the community do on occasion become antisocial. Often offences are minor in character and may often arise from lack of understanding of prohibitions, especially in the more severely handicapped (Price, 1882).

**Aggression** is one of the more serious and upsetting behaviour problems of childhood and adolescence. The term is used to describe a wide variety of behaviours; including tantrums, arguing, bullying, property destruction, biting, hitting, pushing, fighting and cruelty to animals. It is usually the result of frustration and anger, and typically is directed toward caregivers or playmates. (Kaye et al, 2002) Patient's aggression or agitation may suggest a disorder of impulse control rather than reflecting underlying anxiety. (Sadock) Environmental influences e.g. irritability and low frustration tolerance induce aggressive behaviour even in normal persons. It is worth noting that the mentally retarded child is vulnerable to both these aspects (Iskander, 1986).

**Self-injurious behaviour** typically is a chronic, repetitive, and frequently stereotyped behaviour causing trauma. It occurs in the context of specific genetic syndromes (e.g., Lesch-Nyhan syndrome and Smith-Magenis syndrome) but more commonly in persons with unknown or non-specific causes for their mental retardation. Since self-injurious behaviour and aggression are **non-specific symptoms**, one must consider the presence or absence of a variety of factors to reach a presumptive diagnosis: the chronicity of the behaviour, whether it may serve a communicative function, whether it is invariant in topography (e.g., hitting only the right ear, suggesting an ear infection), whether it is situational, whether it occurs in concert with regression from a previous level of function, and whether any associated neurovegetative signs correlate with its onset. **Aggression or self-injurious behaviour** may be seen as behavioural manifestations of dysphoria in persons regardless of developmental level (Sadock & Sadock, 2000).

### **Oppositional Defiant Disorder and Conduct Disorder:**

The DSM-IV diagnosis of oppositional defiant disorder or conduct disorder also requires comparisons with others of similar *mental* age. Further, both diagnoses assume some deliberateness on the part of patients (e.g., disobedience motivated by spite or resentment) (Sadock & Sadock, 2000).

### **Anxiety Disorders**

Specific anxiety disorders (e.g., separation anxiety, overanxious disorder, obsessive-compulsive disorder, panic disorder, and generalized anxiety disorder) rely heavily on an individual's ability to describe the subjective symptoms of anxiety. An individual with mental retardation may not be able to identify subjective anxiety as an underlying cause of distress. Although common, anxiety disorders appear to be underdiagnosed in persons with mental retardation. Variability in prevalence rates, from 1 to 25 %, is attributed to difficulty in making a diagnosis. Yet, some individuals have constellations of signs and symptoms that are best captured in the anxiety disorder spectrum. Patients who are clearly avoidant, who exhibit autonomic arousal in the face of stimuli that most of their peers would not find aversive, and who evince other features of anxiety but cannot articulate their subjective states might be given a diagnosis of anxiety disorder not otherwise specified. Common **symptoms of anxiety in persons with mental retardation** include aggression, agitation, compulsive or repetitive behaviours, self-injury, and insomnia. **Panic** may be expressed as agitation, screaming, crying, or clinging, which might even pass for delusional or paranoid behaviour. **Phobias** also occur in this population and may even be more common in persons with developmental disabilities. Ruth Ryan has noted that persons with developmental disabilities are at **high risk for abuse**, which puts them at a greater risk for **posttraumatic stress disorder** which is an important diagnosis to consider in individuals with mental retardation. When individuals engage in behaviour that appears compulsive or driven and seems ego-alien, the diagnosis of **obsessive-compulsive disorder** not otherwise specified might be considered. Often these patients engage in self-restraint (securing their extremities in their clothing) or cling to their parents or care providers; seemingly to prevent self-injurious behaviours (Sadock & Sadock, 2000).

### **Mood Disorders:**

Mood disorders are not uncommon in persons with mental retardation. Learning problems, social skills deficits, and low self-esteem are often associated with developmental disabilities and represent risk factors for the development of mood disorders. No striking differences exist between the expression of mood disorders in persons functioning in the mild and moderate ranges of mental retardation and



their normally developing peers. Even in profound mental retardation, the diagnosis of mood disorders is fairly straightforward. Generally, a change in mood from baseline is obvious (recent-onset lability, tearfulness, mood elevation, irritability). If it is coupled with changes in interests, activity level, sleep, appetite, or sexual behaviour of sufficient duration and causing sufficient impairment in habilitative function, the diagnoses of **mania** or of **depression** can be made in nonverbal patients (Sadock & Sadock, 2000).

### **Eating Disorders:**

Eating problems are much noticed in young mentally retarded children. These vary from excessive salivation, nausea, vomiting, colics, indigestion to pica, rumination, food fad and anorexia. Food refusal may reflect depression in a child with or without mental handicap (Holt et al, 1988).

Because the diagnostic criteria rely upon subjective experiences, the diagnoses of anorexia nervosa and bulimia are effectively precluded for individuals with severe or profound mental retardation (Sadock & Sadock, 2000). Food refusal or self-induced vomiting would have to be considered **atypical eating disorders** if they occurred in the absence of other diagnosable disorders (e.g., depression or rumination).

**Pica** is perhaps the most common eating disorder among persons with mental retardation. (Sadock & Sadock, 2000) Obesity is much noticed especially in Down syndrome and non-hyperactive mentally retarded children (Chad et al, 1990).

### **Psychosis:**

Patients with developmental disorders are at increased risk for **schizophrenia, bipolar disorder, and other mental illnesses** that may include symptoms of thought disorder and hallucinations. The diagnosis of schizophrenia essentially requires that a patient relate the experience of delusions or hallucinations. As has been suggested by others, the diagnosis of classic schizophrenia is arguably impossible for individuals with profound mental retardation and limited communicative ability. Nonetheless, some individuals display presumptive evidence of response to hallucinations (e.g., striking or shouting at empty space, throwing imaginary peers from furniture) or adopt catatonic postures that can appear to be of psychotic origin. In these cases the diagnosis of psychotic disorder not otherwise specified should be considered if these signs exist in the absence of sufficient evidence to warrant the diagnosis of a supervening mood disorder (Sadock & Sadock, 2000).

## **Other Co-morbid Psychiatric Disorders:**

**Elimination disorders** Since *mental* age of 4 years is required for elimination disorders, the diagnoses of functional **enuresis** or functional **encopresis** are seldom made in the context of severe intellectual disability. In some instances, individuals appear to lose previously acquired skills, (e.g., urinary continence), but such losses typically do not occur in isolation, suggesting alternate diagnoses (e.g., delirium or depression) (Sadock & Sadock, 2000).

**Sleep disorders** ultimately require subjective input by the patient regarding the adequacy of rest, occurrence of nightmares, and so on (Sadock & Sadock, 2000). In a longitudinal study of sleep problems in 200 children with severe mental handicap made by Quine in 1991 sleep problems were extremely common. Epsie and Tweedie in the same year made a study on sleep patterns and problems amongst people with mild mental handicap. They found that 15% of their patients presented with significant sleep problems, particularly intermittent waking. They also found that the sleep problems were associated with a number of child characteristics: poor communication skills, poor academic skills, poor self help skills, incontinence, daytime behavioural problems and epilepsy (Quine, 1991). Given the frequent history of abuse reported for people with mental retardation as a group, one should not overlook the possibility of posttraumatic stress disorder when sleep disturbance is a presenting problem. (Sadock & Sadock, 2000)

**Screening-out Behaviour: (Lack of consideration)** It is avoiding the situations involving intense stimulation, anxiety or frustration, or developing a capacity to turn out the environment and remain unresponsive. Although this helps the child maintain emotional equilibrium, yet if it is carried out in excess, autistic aloofness and withdrawal results (Nashed, 1989).

**Tourette's disorder** The diagnosis of Tourette's disorder is difficult in persons with profound mental retardation. These individuals frequently also display stereotyped or other movements, and it is difficult to distinguish intentional from unintentional movements or sounds or vocal tics from spontaneous, stereotyped, or echolalic vocalizations in individuals frequently incapable of functional speech. The diagnosis of stereotyped movement disorder might be considered in such circumstances (Sadock & Sadock, 2000).

**Somatoform disorders, depersonalization disorders, and sexual disorders** are less frequently diagnosed in the context of mental retardation, though they are certainly not precluded (Sadock & Sadock, 2000).

**Child Abuse:** Abuse is the name given to behaviour, which is felt to be inappropriate, intrusive and damaging, which the recipient feels powerless to stop (Chandy et al, 1996). Abuse may take different forms; it may have been physical, emotional or sexual or a mixture of all three and it can happen to any one regardless of gender, ethnic group, culture or age. Child abuse is against the law (Garnefski & Arends, 1998).

Mentally retarded are vulnerable to exploitation or abuse by others (Sadock & Sadock, 2000). A number of factors may make individuals with disabilities more susceptible to sexual exploitation or abuse than their peers without disabilities such as:

- Physical limitations that make self defence difficult.
- Cognitive limitations that make it difficult for the person to determine if a situation is safe or dangerous.
- Vulnerability to suggestion, because of limited knowledge of sexuality and human relations, including public and private behaviour.
- Lack of information about exploitation and what to do if someone attempts to victimize them.
- Lack of social opportunities that result in loneliness and vulnerability.
- Impulsivity, low self esteem, and poor decision-making skills.

(Rosen, 1984)

### **\*Risk factors for mental co-morbidity (for psychopathology):**

Developmental disability is a significant **risk factor for psychopathology** in general, and this increased risk may derive from both biological vulnerabilities and the environment. A host of explanations, highlighted below, have been put forward to account for this added risk, including:

- Shared etiology: In some cases, this high co-morbidity may result from a shared etiology that is common to mental retardation and the associated mental disorder (e.g., head trauma may result in Mental Retardation and in Personality Change due to head trauma).
- Developmental experiences with which these individuals must contend such as:
  - a) Frustration from parents. Family stress may be heightened by presence of child with developmental disability.
  - b) Perceived rejection from peers.

- c) Repeated failure or difficulty achieving what appears to come naturally to normally developing peers. The recapitulation failure with each developmental stage, and less well developed or perhaps less-supportive peer groups must all take a toll on ego development.
  - d) Risk of reduced opportunities for development and exercise of recreational and occupational skills.
  - e) Vulnerability to exploitation or abuse by others.
- Increased likelihood of loss or separation, particularly in out-of-home placements
  - Communication deficits may predispose to emotional or behavioural disturbance (Lack of communication skills may predispose to disruptive and aggressive behaviours that substitute for communicative language.)
  - Inadequate coping skills.
  - Risk of limited network of social relationships and repertoire of social skills.
  - Adverse effect of disability on self-esteem, possible dysmorphology.
  - Some general medical conditions associated with mental retardation are characterized by certain behavioural symptoms e.g., the intractable self-injurious behaviour associated with Lesch-Nyhan syndrome).
  - The common co-morbidity of mental retardation with physical illness (e.g. epilepsy) may also increase the risk of mental disturbance.
  - The treatments for epilepsy and other medical conditions may carry some behavioural toxicity that can increase the likelihood of diagnosed mental illness. Phenobarbital has been widely reported to increase the risk of motoric hyperactivity and disinhibition in children and in individuals with developmental disorders, and Phenytoin (Dilantin) may cause cognitive toxicity (as can essentially any of the medications used to manage epilepsy).

(APA, 1994 and Sadock, 2000).

# **\*TREATMENT of MENTAL RETARDATION:**

## **\* Prevention:**

In the absence of curative therapy, and with treatment difficulty, prevention plays a particularly important role in mental retardation. Research has identified new causes of mental retardation, new means of early diagnosis, and new ways of prevention. Prenatal diagnosis, newborn screening, dietary supplementation or restriction, hormone replacement, vaccination, and immunotherapy are just some of the techniques that have been applied to prevent mental retardation. Together, these interventions have slightly reduced the overall prevalence of mental retardation, and in some instances have nearly eliminated specific causes. Much remains to be done, including developing better means of early intervention for socio-cultural mental retardation and convincing society of the value of investment in such approaches (Kirk et al., 1993).

Treatment strategies of mental retardation largely focus on preventing intellectual disability and mitigating associated complications (e.g., treating associated mental disorders). **Primary prevention** refers to efforts and actions taken to eliminate or reduce the factors and conditions that lead to the development of the disorders associated with mental retardation (Kaplan and Sadock, 1991). The merits of primary prevention are obvious, and the successes enjoyed with PKU (phenylketonuria) should continue to provide powerful incentive for the ongoing collaborations of basic scientists and clinicians. The impact of more-recent programs is less clear. For example, although folic acid supplementation appears to reduce the risk of neural tube defects significantly, compliance with recommendations to increase dietary folate appears disturbingly negligible. It also appears that the prevalence of trisomy 21 is likely to remain unchanged or increase despite of the availability of prenatal diagnostic programs (Sadock & Sadock, 2000).

Early intervention programs with high-risk infants and children have shown remarkable results in reducing the predicted incidence of subnormal intellectual functioning. Early comprehensive prenatal care and preventive measures prior to and during pregnancy increase a woman's chances of preventing mental retardation. Dietary supplementation of the mother during pregnancy with folic acid reduces the risk of neural tube defects (Alexander, 1998).

During the past 30 years, significant advances in research have prevented many cases of mental retardation. For example, every year in the United States, of mental retardation cases they prevent:

- 250 cases due to **PKU** by newborn screening and dietary treatment;
- 1,000 cases due to **congenital hypothyroidism** thanks to newborn screening and thyroid hormone replacement therapy;
- 1,000 cases by use of anti-Rh immune globulin to prevent **Rh disease** and severe jaundice in newborn infants;
- 5,000 cases caused by **Homophiles influenza** disease (Hib) by using the Hib vaccine;
- 4,000 cases due to **measles** encephalitis thanks to measles vaccine; and
- untold numbers of cases of mental retardation caused by **rubella** during pregnancy thanks to rubella vaccine.

(The Arc, 0000)

Genetic counselling, good prenatal care, and safe environments are important in primary prevention (Shaner, 1997). Removing lead from the environment reduces brain damage in children. Preventive interventions such as child safety seats and bicycle helmets reduce head trauma. Early intervention programs with high-risk infants and children have shown remarkable results in reducing the predicted incidence of subnormal intellectual functioning. Finally, early comprehensive prenatal care and preventive measures prior to and during pregnancy increase a woman's chances of preventing mental retardation. Research continues on new ways to prevent mental retardation, including research on the development and function of the nervous system, a wide variety of fetal treatments, and gene therapy to correct the abnormality produced by defective genes (The Arc, 1993).

Once the disorder or condition associated with mental retardation has been identified, the disorder should be treated so as to shorten the course of the illness (**Secondary prevention**) and to minimize the sequelae or consequent handicaps (**Tertiary prevention**) (Schaffer, 1989). Secondary prevention is aimed at reducing the prevalence of illness by reducing its duration in those who have just developed it. Factors that tend to prolong an episode of illness, including inadequate or inappropriate treatment protocols, can be targeted in secondary prevention efforts. Reduction of disability produced by a disorder, or tertiary prevention, can also be assessed, even if full recovery does not occur (Sadock & Sadock, 2000).

## **\*Intervention methods:**

**The approach to treatment begins with diagnosis** (Sadock & Sadock, 2000). Treatment interventions must always be preceded by appropriate levels of assessment (Puri et al, 1996). In some cases the underlying cause of mental retardation may be particularly important in considering treatments. For example, in mental retardation associated with phenylketonuria, a number of attempts have been made to minimize or to attenuate hyperactivity and impulsivity by dietary modification. Identification of the underlying cause of mental retardation has become increasingly important in considering biological treatments. Likewise, the diagnosis of mental disorders or syndromes in persons with mental retardation will guide and influence treatment strategies. Experience over the past decade clearly shows that mental retardation is a multidisciplinary problem and optimal treatment is multimodal. Typically, a treatment plan includes attention to psychoeducational, psychotherapeutic, and psychopharmacological interventions (Sadock & Sadock, 2000).

There are only four illnesses in mental subnormality which are treatable; cretinism, phenylketonuria, galactosemia and idiopathic hypoglycaemia. Surgical interference may be beneficial in some cases like hydrocephalus (Okasha, 1988).

A variety of treatments have been considered for patients with disruptive behaviour disorders:

**[A] Non-pharmacologic approaches:** These include: Behaviour modification, psychotherapy, and cognitive and social interventions. They are the subject of ongoing trials evaluating short- and long-term effectiveness.

**[B] Pharmacologic approaches:** These include the use of mood stabilizers and antipsychotic agents. Although antipsychotics are often used in the treatment of severe disruptive behaviour disorders, few controlled studies have examined the use of these treatments for this purpose. Furthermore, many studies are limited by the small size of the study group, short duration, and open-label and noncomparative design. The data on treatment of behaviour disorders in patients with intellectual limitations are even fewer.

(Aman, 2002).

## \*Behavioural therapy:

Behavioural therapies are demonstrably effective in managing many difficulties in persons with mental retardation (Sadock & Sadock, 2000). Behaviour guidance and attention to promoting self-esteem may improve long-term emotional adjustment (Shaner, 1997). The laws of learning theory apply as much to retarded children as to others, though they tend to learn more slowly and more behavioural trials may be needed to achieve the same results. Suitable programs can promote toilet training, the extinction of such behaviours as temper tantrums, rocking or head banging, the acquisition of motor and language skills, and other therapeutic aims (Yule and Carr, 1980).

Behavioural approaches can be very effective in reducing psychological and behavioural morbidity, but they **require skilled management** and supervision in order first to be effective and second to avoid being abusive... particularly where aversive stimuli or withdrawal of privileges are concerned (Puri et al, 1996). Typically, a behavioural assessment begins with a functional analysis of behaviour, that is, a detailed examination of the variables that reinforce or maintain particular behaviours. One considers the antecedent events and consequences of behaviour in question and typically tests hypotheses to confirm the results of the behavioural analysis. A behavioural psychologist can best generate and implement a behaviour program based upon a functional analysis of behaviour (Sadock & Sadock, 2000).

**Behaviour modification** is useful for treatment of self-injury, stereotypies, pica, and asocial behaviour. Removing inappropriate attention, changing deviant communication patterns, consistently applying social and practical demands and adding environmental contingencies can effectively reduce the frequency of these behaviours. Specialists may provide educational and developmental training to enhance speech and language, motor, cognitive, social, and occupational functioning; and adaptive skills such as toileting, dressing, grooming, and eating (Dulcan & Martini, 1999).

## \*Psychotherapy:

One should never assume that persons with mental retardation cannot benefit from psychotherapeutic intervention simply because of their impaired intellectual functioning. For example, Anton Dosen has highlighted the use of **psychoanalytic approaches** that focus on developmental theories to improve emotional expression, enhance self-esteem, increase personal independence, and broaden social interactions. Christian Gaedt has similarly advanced the usefulness of ego



psychology (in particular, object-relation theory) in the approach to individuals with mental retardation. In addition to psychoanalytic or developmentally based approaches, **cognitive therapy** may benefit the treatment of depression, and **brief relaxation therapy** may help reduce anxiety even in the context of moderate-to-severe MR. (Sadock & Sadock, 2000) **Developmentally-oriented psychotherapeutic interventions** may be effective to manage crises or to address long-term psychosocial goals (Dulcan & Martini, 1999).

However, all types of **individual therapies** in this population benefit from certain modifications in approach. For example, an active therapeutic stance should be used with concrete, supportive interventions and careful attention to the language abilities and developmental level of the patient. When these types of alterations are made, many patients with mental retardation clearly can benefit. **Group therapy** can be an important part of the treatment program for persons with mental retardation, particularly in the area of social skills building. **Supportive groups** for parents and siblings may also be of particular benefit (Sadock & Sadock, 2000).

**Family therapy:** Many of the problems of retarded children or adolescents are intimately grounded in their family systems. Family therapy approaches are as possible to families containing retarded members as they are to those in which there are normally developing children (Baker, 1988). Family therapy is indicated where there is clear evidence that symptoms are related to family relationships, or where the family themselves are concerned about such issues (Puri et al, 1996).

**Family Reactions:** Over the past two decades, family studies in mental retardation have changed from a predominantly negative to a more balanced perspective. Before the early 1980s, these families were perceived to be *families in crisis*. Mothers were examined for their mourning reactions, couples for divorce, and mothers, fathers, and unaffected siblings for the presence of depression and other forms of psychopathology. The basic findings were that families of children with disabilities may suffer more divorces, parents and siblings are somewhat more prone to depression, and families and individual family members have more difficulty when there is only one parent, when the mother receives little support from the husband, or when the family is of low socioeconomic status. In the early 1980s family researchers began to change their conception from the earlier focus on family pathology to families facing increased stress. Children with mental retardation might add stress to the family system, but this stress could result in negative or positive adaptation. Since families of children with mental retardation vary in their ability to cope, what causes better adjustment in one family and worse adjustment in another? What child, parent, or family characteristics foster better

adaptation? To date, most work has examined such child characteristics as age and degree of impairment. Findings have been inconsistent for both variables. Some studies find it more stressful to parent older children with retardation; others suggest that families experience more stress when the child begins puberty (11 to 15 years) and again when the child reaches early adulthood (20 to 21 years). Still other studies find no relation between increased family stress and the child's age. Although most researchers feel that child maladaptive behaviour increases familial stress, increased familial stress may also elicit child behaviour problems (Sadock & Sadock, 2000).

Another important variable concerns the coping style of the parents. Across several studies, parents who more actively, constructively attempt to deal with their child did better than parents who adopted a palliative coping style, one that either dwells on or ignores parental emotions. This difference in personality style may help buffer parents and families from the increased stresses of parenting a child with mental retardation (Sadock & Sadock, 2000). An invaluable resource in evaluating and treating children with mental retardation is the **child's family**. Consequently, including the families of children with or at-risk for disabilities in every phase of intervention, from identification to planning to implementation through monitoring should be considered. However, including families in decisions about the treatment or management of their children's problems presents new challenges. Nevertheless, trying to understand and include families in the decision-making process can ultimately be rewarding and beneficial for all involved (Biasini et al, 1998).

### **\*Pharmacotherapy (by drugs):**

Medications are one part of overall treatment and management of children with mental retardation (Janicki et al, 1999). There is now significantly more evidence available to support the **rational prescribing of psychoactive agents** for several child and adolescent psychiatric disorders. It is important that we do not distance pharmacological studies from those evaluating the psychological therapies that continue to play a very important part in the treatment of many disorders. However there are still many gaps in our knowledge, and future studies are required before it will be possible to practice in a truly evidence-based manner (Coghill, 2002).

There are a few well controlled studies of drug treatments with children who have mental retardation (Batshaw & Perret, 1992). Drug prescription for those with disabilities should occur only when there is a specific indication. However, it

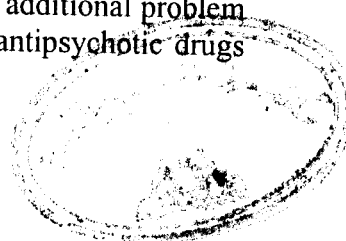
has been frequently reported that over-prescribing does occur, particularly in institutions (Puri et al, 1996). Fortunately, there is little reason to suspect that mechanisms of action of drugs change on the basis of retardation (Sadock & Sadock, 2000). It should also be noted that the use of medication as a form of *chemical restraint* should be avoided. In addition, when drug treatment is used, it should only be one component of an overall treatment approach (Batshaw & Perret, 1992).

Identification of the underlying cause of mental retardation has become increasingly important in considering biological treatments. Likewise, the diagnosis of mental disorders or syndromes in persons with mental retardation will guide and influence treatment strategies. However, because of **drug-drug interactions** that may affect the availability or effectiveness of concurrent medications, individuals with mental retardation may require **different dosing strategies**. Moreover, medical co-morbidity is the rule in individuals with developmental disabilities, and the importance of identifying and treating underlying medical problems (or refining that treatment) cannot be overstated. In institutionalized populations, for example, where 30 to 40 % of persons have **epilepsy** and as many as 70 % may have **some other significant medical condition**, drug interactions become an increasingly important consideration (Sadock & Sadock, 2000).

Hereafter, are the commonly used psychotropic and other drugs with mentally retarded persons along with their main positive and adverse effects:-

### **Antipsychotics:**

Antipsychotic medications have long been used, arguably indiscriminately, in persons with mental retardation. Antipsychotics continue to be the most widely prescribed class of psychotropic medication and are even more commonly prescribed than anticonvulsant drugs for persons with mental retardation. Given this ample experience with antipsychotics in persons with mental retardation, where in residential or institutional settings as many as 50 percent of individuals may be treated with this class of drug, the **adverse effects** are well-known. Individuals with mental retardation appear to be at greater risk of developing **tardive dyskinesia** than the general population; recorded rates range from 18 to over 30 %. On the other hand, **spontaneous abnormal involuntary movements** are not uncommon in this population, which may confound interpretation of rates of neuroleptic-induced tardive dyskinesia (Sadock & Sadock, 2000). In addition, the side effects of antipsychotic drugs, such as **akathisia**, may be confused with stereotypic behavioural patterns seen in people with learning disability. An additional problem for those patients is that the side effects of sedation of some antipsychotic drugs



may produce a reduced learning ability or a loss of skills. Moreover, those with brain damage may also show an increased sensitivity to the epileptogenic propensities of some antipsychotics (Puri et al, 1996).

Evidence supports the use of dopamine antagonists in self-injurious behaviour and aggression, both in theory and in practice. For **thioridazine (Mellaril)** in particular, ample experience indicates that both self-injurious behaviour and aggression may improve. Typical dosages average less than 300 mg a day, with dosages as low as 15mg a day reported for some children. Positive reports also exist for **most other neuroleptics** in this population. Although suggested by some, no convincing evidence suggests that these merely suppress behaviour generally through a non-specific sedating effect. Such an outcome is clearly undesirable in individuals with pre-existing cognitive impairment, and many of the earliest reports specifically note the absence of sedation. Nonetheless, enthusiasm for neuroleptic use in self-injurious behaviour should be dampened by significant adverse effect liability (Sadock & Sadock, 2000). Particularly, hyperactivity & aggression infrequently show good response to **conventional anti-psychotic drugs** (e.g. thioridazine or haloperidol), yet their resultant disabling adverse effects (eg. oversedation, overweight & extrapyramidal manifestations) put ahead limitations for their use both in adults or children (Gelder et al, 2000).

Interest in atypical neuroleptics for self-injurious behaviour and aggression has grown in recent years. The growing availability of serotonin-dopamine antagonists (**atypical antipsychotics**) and their use in persons with mental retardation suggest that these drugs may be very helpful considering their apparently reduced risk for tardive dyskinesia and extrapyramidal symptoms as well as cognitive toxicity. Studies reporting the effectiveness of clozapine (Clozaril), risperidone, and sulpiride through open trials now exist to support the use of these agents in individuals with mental retardation. (Sadock & Sadock, 2000) In the double-blind study done by Aman et al on 118 mentally retarded children with severe disruptive behaviour, risperidone produced both statistically and clinically significant improvements in these children. the positive effects of risperidone on behavior measures were shown to be independent of the sedative effects & occurred at a relatively low average dosage of 1.16 mg/ day (Aman et al, 2002) Scahill and his colleagues found that risperidone produced significant improvements in 69% of the studied autistic children. After 8 weeks of treatment, children treated with risperidone had a decrease in irritability scores of almost 57%, compared with 14% in the group given an inactive placebo. Tantrums and incidents of biting and scratching others dropped from a couple of times a day to a couple of times a week. Such improvement was a relief to parents (Scahill et al).

## Antidepressants:

The use of antidepressant medications in persons with mental retardation appears to remain relatively low (Sadock & Sadock, 2000). The treatment of depressive illness is based on the same principles whatever the age of the patient. Most cases respond to tricyclic antidepressants (TCAs). Depressed patients of any age are extremely demanding for any one to nurse, and a good deal of steadfastness is required to cope with their manipulative behaviour (Price, 1982).

Before initiating treatment with tricyclic antidepressants, the physician should obtain a careful history of cardiac symptoms, such as chest pain, dyspnea, actual or near syncope, palpitations, and tachycardia and should also do a complete physical examination including measurement of vital signs along with essentially baseline ECG that should be repeated at intervals as the dosage increases. If the history suggests head trauma or seizures, an EEG is indicated before starting treatment because TCAs lower the seizure threshold. Because of the potential toxicity of TCAs in overdose, clinicians must remind parents to supervise closely the administration of a TCA and to keep the medication in a safe place. TCAs used to treat depression in children include imipramine, nortriptyline and desipramine. Several open trials and retrospective case reports indicate that **fluoxetine** (20-40 mg/day) may improve mood, energy level, interest, and motivation in children suffering from depression. Clomipramine and fluoxetine have shown efficacy in the treatment of obsessive compulsive disorder (OCD) in children. Sertraline and fluvoxamine are promising. In many children with OCD, the response to medication is delayed for 8 or even 12 weeks after reaching the expected therapeutic dose. The efficacy of imipramine for school avoidance and separation anxiety is controversial. Sometimes it eases return to school and decreases subjective reports of separation anxiety. All TCAs are frequently effective in treatment of nocturnal enuresis. Tolerance may develop, requiring an increased dosage. For some children, TCAs lose their effect entirely. Maximum dosage is 2.5mg/kg/day (Dulcan & Martini, 1999).

Special considerations in the use of antidepressant medication include:

- The common medical co-morbidities.
- Risk of lowering seizure threshold. This risk in the general population is on the order of 1 in 1000, and in individuals with mental retardation it may increase to nearly 1 in 5.
- Cardiac anomalies are common in some mental retardation syndromes, and the anticholinergic adverse effects of some medications may be particularly significant in persons with Down syndrome.

- Disinhibition has been described with typical antidepressant doses of selective serotonin reuptake inhibitors (SSRIs). So, Individuals with mental retardation may require lower concentrations of antidepressant drug than their normally developing peers.
- Trials of SSRIs are increasingly common among **patients with self-injurious behavior**. Favourable results have been reported for fluoxetine, paroxetine, sertraline, trazodone, and clomipramine. However, of these agents, only clomipramine has been shown effective in well-controlled studies. Because it lowers seizure threshold, clomipramine is generally not a first-line treatment for compulsive self-injurious behavior in individuals frequently co-morbid for epilepsy.

(Sadock & Sadock, 2000).

### **Anxiolytics:**

Although **benzodiazepines** are commonly prescribed to treat anxiety in the general population, unique concerns arise in the context of developmental disorders, particularly regarding the possibility of increased confusion, cognitive impairment, unsteadiness, and paradoxical excitement. Jennifer Barron and Curt Sandman reviewed disinhibition associated with benzodiazepines, which occurred in 35 to 68 % of an institutionalized mentally retarded population compared with controls. Nevertheless, alprazolam, clonazepam, and lorazepam are widely used in the treatment of acute anxiety, particularly anxiety associated with medical or surgical procedures. **Buspirone (Buspar)** is another serotonergic agent that has been reported to benefit some persons with developmental disorders. John Ratey and colleagues reported the use of buspirone in an open trial in persons with diagnosed anxiety disorders manifested by aggressive and self-injurious behaviours. Its advantages include a relatively benign side-effect profile, specifically the absence of common motor or cognitive adverse effects at dosages used to treat anxiety (Sadock & Sadock, 2000).

### **Psycho-stimulants:**

A growing body of literature supports the use of stimulant drugs for the treatment of attention-deficit/hyperactivity disorder in the context of mental retardation. Additional study is needed concerning the likelihood of a response to stimulants in relation to the severity of cognitive disability. There are reports of paradoxical responses to stimulant medications in persons with mental retardation, with higher than expected rates of emergent motor tics and emotional lability. **Methylphenidate and dextroamphetamine** have been effective in the treatment of **ADHD** in mildly to moderately mentally retarded children (Sadock & Sadock,

2000). When conduct disorder or oppositional defiant disorder coexist with ADHD, stimulant medication can reduce defiance, negativism, and verbal and physical aggression. Whenever prescribed, stimulant medication should be initiated at a low dose and titrated every week or two according to response and side effects within the usual recommended range (Dulcan & Martini, 1999).

### **Opioid Antagonists:**

Some individuals who self-injure appear to have *altered pain sensitivity*, as Ireland suggested nearly a century ago. This observation, coupled with data indicating that opioid antagonists can attenuate **stereotypies and self-injury** in animal models and data indicating that opioids may modify the function of dopaminergic systems has fuelled interest in the opioids. **Naltrexone** (ReVia) is the opioid antagonist most widely used for self-injurious behaviour, but the literature is mixed. Typical dosages range from 0.5 to 2.0 mg/kg a day in children and up to 200 mg a day in adults. The relatively long half-life of naltrexone (72 hours in brain) must be kept in mind in designing titration strategies for this drug (Sadock & Sadock, 2000).

### **Anticonvulsants:**

- Data on the use of anticonvulsant medications **for indications other than epilepsy** are limited. However, considerable experience suggests that as in the population in general, some anticonvulsant drugs may improve cyclical mood disorders and impulsive aggression (Sadock & Sadock, 2000). **Antiepileptic drugs (AEDs)** have been used to reduce hyperactivity and aggression with promising results especially the extensively studied and widely used **Carbamazepine & Valproic acid** which have been proved effective in adults in controlling violent and aggressive behaviour and also in stabilization of mood for manic and hypomanic states (Rutecki & Gidal, 2002).
- Numerous studies have shown neuroleptics and mood stabilizers (anticonvulsants and lithium) to be effective in treating aggression in certain cases, even without evidence of a co-morbid psychotic, mood, or seizure disorder (Kaye et al, 2002).
- Self-injurious behaviour and aggression can also dramatically improve when **phenobarbital** administration is stopped. The potential behavioural toxicity of this or any drug should not be overlooked in designing treatment strategies for persons with mental retardation (Sadock,2000).
- Newer antiepileptics e.g. **lamotrigine & Gabapentin**, proved effective in treatment of epilepsy in developmentally disabled children, with less adverse effects esp. on cognition & behaviour (Rutecki & Gidal, 2002). Analysis of

**Tiagabine** (TGB) monotherapy showed that patients receiving TGB monotherapy did best, improving particularly in the areas of adjustment & mood (with low dose TGB) & in the area of abilities (with high dose TGB) (Dodrill et al, 1998).

- **Lamotrigine (Lamictal)** is an anticonvulsant drug that also appears to antagonize glutamate (Glutamate antagonist) by reducing its release. A recent case report suggests that lamotrigine may be useful in **reducing self-injury** in the context of a stereotyped movement disorder (Sadock & Sadock, 2000). Udall et al 1993, Stenbom et al 1998 and Genton 2000 concluded that 90% of Rett's patients with previously refractory seizures had improved seizure control and **45% of them had improved alertness, ability to concentrate, interaction and reduced autistic behavior and stereotyped movements** (Mikati, 2003). A larger study in which lamotrigine was added to the anticonvulsant regimen of children with **mental retardation** suggested that those with autism experienced **gains** that could not be readily attributable to better seizure control alone. However, a recent double-blind, placebo-controlled trial of lamotrigine in autism had **negative** results (Sadock & Sadock, 2000). **Lamotrigine**, a relatively new (1<sup>st</sup> marketed as antiepileptic in 1994), but more safe antiepileptic drug, has recently been approved also as a **mood stabilizer**. It also showed promising effects on modification of behaviour of persons with borderline personality disorder (Goldberg, 1997). Lamotrigine may be an effective treatment option for patients with refractory bipolar disorder (Calabrese et al, 1999). In the same study by Calabrese et al, LMG seemed to be equally effective as adjunctive therapy or monotherapy, and it was efficacious in reducing affective symptoms in patients presenting with treatment-refractory depressed, hypomanic, manic, and mixed phases of bipolar-I and bipolar-II disorder (Calabrese et al, 1999). In another study, LMG decreased ketamine-induced perceptual abnormalities and learning and memory impairment in healthy subjects. Open trials suggest an effect on posttraumatic stress disorder and borderline personality disorder without symptoms of mood disorder. Placebo-controlled trials suggest that LMG may have **antineuralgic properties**, prevent migraine aura (Malt, 2000).

Furthermore, Lamotrigine recently showed **favourable psychological effects** when used in treatment of some mentally retarded children suffering from epilepsy (Mikati, 2003). In another study, LTG, used as add-on therapy had significant positive or negative effects on behaviour in 7 patients with epilepsy and MR; in 4 of those pts (58%) lamotrigine may have induced very significant changes in behaviour. **Positive psychotropic effects** of lamotrigine included: reduction in irritability & hyperactivity, decreased



lethargy and diminished perseverative speech (producing a more appropriate speech), as well as improvement in co-operation & better social engagement. In all of the 4 pts, behavioural improvements were sustained at the time of latest follow up (6 months to one year) which is evidence of a genuine effect of the drug rather than a transient co-incidental change in the behaviour (Ettinger et al, 1998). In a different study, LMG may have **provoked aggressive behaviour** and violence (in about 47 % of pts) in intellectually handicapped pts with epilepsy, which may limit its use for such pts (Beran & Gibson, 1998).

### Other Drugs:

- **$\beta$ -Adrenergic receptor antagonists: (= Beta-blockers)** have also been reported to be of use in the population with developmental disorders, it is not clear whether the mechanism is central or peripheral (Sadock & Sadock, 2000). At dosages of propranolol (Inderal) above 1000 mg a day, the precise mechanism of action of this drug arguably becomes non-specific, but lower dosages of this and other b-adrenergic receptor antagonists are also reportedly beneficial. (Sadock & Sadock, 2000) Beta-blockers have also been useful, especially as adjunctive treatment of aggression in the developmentally disabled (Kaye et al, 2002).
- **Drugs acting at glutamate receptor subtypes:** A growing appreciation exists for the potential therapeutic utility of these drugs. Glutamatergic and dopaminergic interactions in the neostriatum are the focus of research on the pathogenesis of a host of neuropsychiatric illnesses including schizophrenia, obsessive-compulsive disorder, self-injurious behavior, and aggression (Sadock & Sadock, 2000).
- **Dextromethorphan:** (= Dimetane, Sudafed) an antitussive agent which also binds to the NMDA receptor, was reported by Leisa Welch and Robert Sovner to have markedly attenuated self-injurious behaviour in a 25-year-old individual with congenital rubella syndrome. The patient was maintained on dextromethorphan for 16 months with sustained benefit. Surprisingly (and perhaps ominously) no follow-up studies have been reported with this drug in self-injurious behaviour (Sadock & Sadock, 2000).
- **Lithium:** Lithium may be useful in mentally retarded youths with severe aggression directed towards themselves or others. **Cyclic mood disturbances** in mentally retarded patients have been treated successfully with **lithium**. However, side effects, including gastrointestinal distress, tremor, fatigue, and a worsening of eczema reduce compliance in almost two-thirds of the patients (Dulcan & Martini, 1999).

**In conclusion:**

**Mental retardation is a complex problem, with educational, medical, social, cultural and vocational aspects. Multi-modal treatment with a developmental orientation is the optimal treatment for mental retardation. This includes the coordination of medical and psychiatric evaluations, parent guidance and support, and education and skill development. Specialized professionals may provide educational and developmental training to enhance speech and language skills, motor, cognitive, social, and occupational functioning, and adaptive skills such as toilet training, dressing, grooming, and eating. Behavior management and pharmacotherapy are very useful for treating certain associated behaviors such as aggression or self-injurious behavior (Reiss and Aman, 1998). Excessive reliance on any particular approach to treatment can have adverse consequences (Puri et al,1996). There is no doubt that a supportive environment, especially at times of stress, may prevent psychopathology and preserve function (Shaner, 1997). The psychiatric component includes the coordination of medical and psychiatric evaluations, parental guidance (support, education, behaviour management, educational and environmental planning, long-term monitoring, and advocacy) and the standard psychiatric therapies for the concomitant psychiatric disorders. Ongoing follow-up is needed to monitor the overall speed of progress (Dulcan & Martini, 1999).**

# **Subject & Method**



# ***\*Subjects and Method:***

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## ❖ **Type of study:**

This is a **Case-control study** comparing 2 groups of non-epileptic mentally retarded children; one with EEG changes and the other without.

## ❖ **Subjects (Study Population):**

The study included 60 mentally retarded children attended (for the first time) the psychiatric outpatients of Bani-Sweif psychiatric hospital and reviewed the psychiatric outpatient once monthly during a 6-months period of follow up from January, 2004 till June, 2004.

### ▶ **Criteria of inclusion:**

1. Age range: 6-11½ years (Chronological age).
2. Both sexes were selected in the same common percentage of mental retardation in general population (1.5 males to 1 females) (APA, 1994)
3. Cases with mild or moderate MR only. (as per clinical assessment, IQ-testing and assessment of adaptive behaviour).
4. Cases living in Bani-Sweif city only (Urban).
5. Socio-economic status of all selected cases was the middle class (as assessed by Al-Shakhs Scale of Socio-economic Status of the Egyptian families).
6. Cases with 'intermediate or significant' level of maladaptive behaviour (as assessed by Vineland ABS, maladaptive domain, part-I).
7. Cases with normal EEG and cases with kindling changes in the EEG.

### ▶ **Criteria of exclusion:**

- 1- Other categories of mental retardation (Borderline , severe or profound MR).
- 2- Co-morbid significant neurological disorder (gross sensory-motor deficits) e.g. paralysis, muteness, deafness, blindness (most cerebral palsy cases).

- 3- Cases with gross medical disorder e.g.: cardiac, hepatic or renal failure, cases with endocrinal disturbances (e.g. cretinism), cases with inborn errors of metabolism and cases with brain tumour insult to avoid the effect of disease or of other drugs necessary for treating such medical problems.
  - 4- Cases with dual diagnosis; having known major co-morbid psychiatric illness e.g. schizophrenia, bipolar disorder, organic personality disorder, etc...
  - 5- Patients maintained on drugs for treatment of a chronic illness e.g. epilepsy, anemia, diabetes,... to avoid possible drug interaction with Lamotrigine (LMG) or possible adverse effects of other drugs.
- The whole study sample had been sub-classified into 2 groups:
    - ▶ **Group A:** = 30 children with EEG changes, referred to in this study as (**Study group**)
    - ▶ **Group B:** = 30 children with Normal EEG. referred to in this study as (**Control group**)

Both groups were given regular doses of Lamotrigine, ranging in the majority of cases between 25mg to 150mg (N.B: dose was escalated according to the response). The concerned patients were supervised by their caregivers & data were recorded for each child into a separate progress sheet and kept into separate files.

All parents or caregivers who agreed to enrol their children in the study had signed an informed “**consent**” to document their beforehand approval for the procedure of the study.

The study protocol was *approved* by **The Institutional Review Board, Institute of Postgraduate Studies, Ain Shams University** in May, 2003.

## ❖ Method:

### \*Medical, Psychiatric and Psychometric Diagnostic Approach:

The children selected for this study were chosen by simple randomization method “first seen first selected”, according to the criteria of inclusion (defined in the subject, page 57) including primarily to belong to average/middle socio-economic class (assessed by *Al-Shakhs Scale for socio-economic level of the family*, 2<sup>nd</sup> edition, 1995) and to have an IQ below 70 or above 35 (assessed by *Wechsler Intelligence Scale for Children*, Arabic version, by Melaika & Ismaeel, 1993) through individual interviewing with each child and his/her caregiver along with psychometric assessment of adaptive and maladaptive behaviour (using *Vineland Adaptive Behaviour Scale*, Arabic version, by Elwan Fadia, 1996). Recent IQ & EEG tracings were made sure ready for every child before start of treatment. Two doctors from the hospital psychiatrists were selected, trained and identified to other staff of the hospital as **(focal points)** to refer to them those cases suggested to be valid for our study seen in the psychiatric outpatients’ clinics by the other psychiatrists in the hospital so as to ensure proper filtering of such cases on every working day. All psychiatrists working in the outpatient department were given clear orientation about the research subject and procedure. They were asked to refer to the identified focal points every new case seen by them that fulfil the criteria of inclusion and exclusion.

The maladaptive behaviour of each child has been assessed (as a part of the psychiatric examination) across the 27 items of the *Vineland’s Adaptive Behaviour Scale, Maladaptive Behaviour Domain (part-I)* focusing on covering the following points: type of maladaptive behaviour, time of occurrence, duration each time it occurs, frequency and provoking and aborting stimuli. Maladaptive behaviour was assessed at the beginning and at the end of the study, then a comparison of both recorded scores (pre and post-treatment with Lamotrigine) was done to evaluate the different observable effects of Lamotrigine on behaviour. Reliability of the scale was tested by test-retest method on first 5 cases and proved reliable.

A responder (improved person) is defined as the subject with an endpoint rating in the range of “non-significant” on the Vineland ABS scale, Maladaptive Behaviour Domain, Part-I.

The caregivers of all children under study were asked to sign consents of their voluntary approval to include their children in the study and to meticulously document the changes in the maladaptive behaviour of their children on weekly basis in separate diaries, then, the collected data were discussed with the researcher once a month at least.

A comprehensive clinical evaluation of each patient was performed. In detail, all children of the study were subject to the following procedure:-

### **{1} General Information and History:**

A good history is always essential for proper diagnosis, and in many cases collateral informants (family members) are extremely useful to fill in important aspects of the developmental history of the child and also of the family pedigree. Detailed history was taken from caregivers of the patients especially about the following issues:

- Particulars of the patient.
- Complaint: (by parents or care providers)  
= Reason for referral to hospital (maladaptive behaviour, IQ assessment or other).
- Family history and consanguinity.
- Social background: Information necessary for assessment of patient's socio-economic status by Al-Shakhs Scale, (i.e. father's occupation, education and family's income were recorded here at this early step of assessment to select the patients from the middle 'average' social class only and to exclude from study those patients from other social classes. ).
- Developmental history = History of growth and development, both physically and psychologically stressing on '*developmental mile stones*'.
- Present history = **History of current abnormal (maladaptive) behaviour.**
- Present history of drug intake.
- Recent history of investigations and assessments done for the patient especially: EEG and IQ-testing.

N.B: The researcher designed a "**Patient's first interview form**" that summarizes history and examination data into one collective form to facilitate statistical dealing with data later. (Appendix, page xxx)



## **{2} Physical examination:**

### **(A) General medical examination:**

- ◆ Examination of head & neck, heart, chest, abdomen, limbs and genitalia.
- ◆ Assessment of minor physical anomalies e.g. heart defects.
- ◆ Description of skull & facial features suggestive of mental retardation (e.g.: microcephaly, micrognathia, hypertelorism, thin upper lip, protruded lower jaw, mongoloid features,..)

### **(B) Full neurological assessment:**

When neurological abnormalities are present, their incidence and severity generally rise in direct proportion to the degree of retardation.(Kaplan And Sadock, 1991) It was planned to exclude from our study any subjects suffering from gross neurological disorders though such disorders were not expected at this level of mental retardation. Nevertheless, all cases were subject to the following steps:

- ◆ Examination of motor and sensory functions, reflexes, co-ordination and gait.
- ◆ **EEG** was done routinely for all patients who had no recent EEG-records before starting treatment.

## **{3} Psychiatric examination:**

(using the classic structured psychiatric sheet )

Mental Status examination was applied to every child of the study population to **rule out dual diagnosis** and to specify the type of **the maladaptive behaviour** present. Items checked were:

- ◆ Intellect (attention and concentration, orientation, memory, grasp, judgement, thought, and insight)
- ◆ Perception
- ◆ Affect
- ◆ Conduct (Behaviour).

All selected cases matched the diagnostic criteria of mild or moderate mental retardation. Cases with dual diagnosis were excluded of our study. Psychiatric diagnosis of mental retardation necessitates psychometric assessment of intelligence and of adaptive behaviour to fit with the diagnostic criteria of **DSM-IV** (by APA, 1994).

#### **{4} Psychological assessment:**

##### **(A.) Assessment of Intelligence:**

All selected cases matched the diagnostic criteria of mild or moderate mental retardation (according to **DSM-IV** by APA, 1994) which necessitated fulfilling of 3 criteria; condition started before age 18 years, a below average I.Q, and a significant deficit in adaptive behaviour. So, the following two psychometric measures were applied:

##### **I. Assessment of IQ:**

By using **Wechsler Intelligence Scale for Children**, Arabic version, revised (**WISC-R**), by Melaika and Ismaeel, 1993. The scale is supposed to assess intelligence of children and adolescents aged 6-16½ years. The scale consists of 2 main parts;

1. The first part is the **Verbal Tests** which include 6 tests: Information, Comprehension, Similarities, Arithmetic, Vocabulary, and Digit Span.
2. The second part is the **Performance Tests** which include 5 tests: Block Design, Picture completion, Picture arrangement, Object assembly, and Coding.

The verbal and performance tests give rise to raw scores that are matched with standard scores from the manual of WISC to obtain the Mental age. Then, the IQ is calculated by the simple equation:

$$IQ = \frac{\text{Mental Age}}{\text{Chronological Age}} \times 100$$

Current mental age and recent IQ had been obtained by this test for each case of our study population individually as part of the first interview procedure.

## **II. Assessment of adaptive functioning:**

This was accomplished by use of **The Vineland Adaptive Behaviour Scale (VABS)**, the Survey Form, (by Sparrow et al, 1984), Arabic Version, by Elwan, Fadia, 1996, (unpublished). (Appendix, page ii)

The VABS measures **four domains** where each domain consists of 3 sub-domains, (except Motor Skills Domain consisting of 2 sub-domains). The domains and their corresponding sub-domains are:

1. **Communication Domain:** 3 sub-domains = Receptive, Expressive, and written.
2. **Daily Living Skills Domain:** 3 sub-domains = Personal, Domestic, and Community.
3. **Socialization Domain:** 3 sub-domains = Interpersonal Relationships, Play and Leisure time, and Coping Skills.
4. **Motor Skills Domain:** 2 sub-domains = Gross and Fine.

Each sub-domain consists of certain number of items (questions) to be answered by the caregiver of the mentally retarded child whose adaptive behaviour is planned to be assessed. The collected answers form “raw scores” of the sub-domains then, of their corresponding domains. They are converted into “standard scores” from the manual of the scale. The sum of such standard scores of the assessed domains (3 or 4 domains) is obtained then; it is to be matched in the manual to get the “Adaptive Behaviour Composite” for the three or four domains assessed. The “**Adaptive Level**” can be then obtained by matching the adaptive behaviour composites against their corresponding values in the manual of the Vineland’s scale. The resultant adaptive level may be high, moderate, adequate, moderately low or low. The low adaptive behaviour (corresponding to below 20 to 70 standard score of adaptive behaviour composite) indicates significant deficit in adaptive behaviour of the person. The following classification may be used for standard scores below 20 to 70:

1. Mild deficit: with standard score of 50-55 to 70
2. Moderate deficit: with standard score of 35- 50-55
3. Severe deficit: with standard score of 20-25 to 35-40
4. Profound deficit: with standard score below 20 or 25

Reliability of Vineland ABS by test-retest correlation proved reliable.

**(B.) Assessment of maladaptive behaviour:**

This was accomplished by use of **The Vineland Adaptive Behaviour Scale**, maladaptive domain, part-I, by Sparrow et al, 1984). **A Maladaptive Behaviour Domain** is also available (optional) along with the four main domains of VABS. It consists of two parts;

1. **Part-I:** measures **27 items** of common maladaptive symptoms. This is the domain used by the researcher in this study for assessment of maladaptive behaviour of the subjects studied. (page x, Appendix)
2. **Part-II:** measures **9 items** but it is to be applied for individuals who should be compared with a supplementary norm group which was not the case in our study.

The items of part-I were screened for every child studied by asking the caregiver of the child to answer the questions about the child and recording the equivalent scores for such answers. Thereafter, those scores were matched with their equivalent standard values at table-B12 in Vineland's manual of the VABS (Table-2) to obtain the level of maladaptive behaviour of the child.

**Scoring for Vineland's Maladaptive Behaviour Domain, Part-1:**

(Table-2) Vineland's Maladaptive Behaviour levels

Age (years)	Maladaptive levels (Ages: 06-00-00 through 11-11-30)		
	(Part-1 raw scores)		
	Non-significant	Intermediate	Significant
6	0-5	6-12	13-54
7	0-5	6-12	13-54
8	0-6	7-12	13-54
9	0-6	7-12	13-54
10	0-5	6-11	12-54
11	0-5	6-12	13-54

From: Vineland's Table B-12 (for ages: 05-00-00 through 18-11-30 and older)

The scores matching (Non-significant) mean no significant maladaptive behaviour. The scores matching (Intermediate or Significant) mean significant maladaptive behaviour at two levels different in severity; e.g. one is moderate and the other is severe).

Furthermore, the 27- items of part-I were classified by the researcher into **four clusters** (according to relevancy of symptoms) to see the effect of LMG on separate symptoms and on cluster symptoms (page xi, Appandix). The four clusters were:

01. **Neurotic symptoms cluster:** Thumb or fingers Sucking, Nail biting, Teeth grinding, Eating Disturbance, Sleep disturbance, Nocturnal Enuresis (NE), and Tics.
02. **Mood symptoms cluster:** Temper tantrums (TT), Over-Anxious mood, Depressed mood, Emotional lability, Indifference, and Lack of consideration.
03. **Attention Deficit Hyperactivity symptoms cluster (ADHD):** Poor attention, Hyperactivity, and Over-Impulsivity.
04. **Disruptive Behaviour symptoms cluster:** Potential aggression (bullying, intimidating or teasing), Physical aggressive (beating, biting or fighting) , Stubbornness or sullenness, Defiance or negativism, Swearing inappropriately, Lying, cheating or stealing, Over-dependency, Excessive withdrawal, Avoidance of going to school, Truancy from school, Run away from home, and Avoidance of direct eye contact.

### **(C.) Assessment of the socio-economic status:**

Assessment of socio-economic status of the families of our cases was done by using **Al-Shakhs Scale for socio-economic level of the family**, (Al- Shakhs, 1995) which included the following three parameters:

#### **1) The 1<sup>st</sup> Parameter: Occupation of Parents:**

This parameter described (9) levels according to the nature of occupation of parents. The researcher gave each level a label to facilitate recognition of different occupations at each level. (Table-2.a, Appendix)

#### **2) The 2<sup>nd</sup> Parameter: Educational Level of Parents:**

This parameter classified the level of education of parents into (8) levels from “illiterate” to “doctorate degree”. (Table-2.b, Appendix)

#### **3) The 3<sup>rd</sup> Parameter: Income per Capita per Month:**

This parameter distributed the income per capita per month (in Egyptian pounds) over 07-levels. The scale, issued in year 1995, defined the minimum income/capita/month to be below 20 Egyptian pounds and the maximum as

above 120 Egyptian pounds. The above-mentioned 07-levels were **re-standardized** by consulting a group of (10) qualified persons (specialized in: Education, Psychology, Psychiatry, Sociology, Medicine, Management, Engineering, Marketing and Statistics) to concord with current socio-economic structure of the Egyptian family. There was an average agreement ratio of 70% of the ten persons. (Table-2.c, Appendix) Accordingly, the modified third parameter ranged from below 40 Egyptian pounds to above 240 Egyptian pounds. (Table-2.d, Appendix)

Then, the score of the **level of the socioeconomic status of the family (X)** was calculated for each case individually according to the scale's equation. (Table-2.e, Appendix) Finally, the resultant score of (X) was matched with its corresponding level of socioeconomic status of the family (one of seven levels) (Table-2.f, Appendix) according to the score of the previous equation which range between 48 and 216 for the seven levels.

All children included in the study were first selected and assessed falling in the average level (**middle class**) of socioeconomic status; having scores of (X)s between 121 & 144. Children belonging to other socio-economic classes were banned from the study population.

## \* Statistical Work up:

In this study, the researcher applied the following statistical procedure:

### I. Defining all the variables to be dealt with:

According to the available data, The target variables considered were : **chronological Age, Gender, mental age, Age equivalent, I.Q (Intelligence Quotient), S.Q (Social Quotient), Socio-economic level, Adaptive behaviour level, Maladaptive behavioural clusters and items, mean Group Score of Maladaptive Behaviour** before and after treatment by Lamotrigine, **Dose of LMG, and Presence or absence of EEG changes.**

**II. Coding of different variables and entering them into computer system compatible with IBM system** according to SPSS, version-11 (Statistical Package of Social Sciences) at the purpose of processing the data and comparing groups statistically for significant differences.

### III. Statistical Analysis of data and getting conclusions:

Because of the limited size of the study population, the researcher used **Wilcoxon Signed Rank Test** mainly to compare variables and improvement inside each group separately and used **Mann-Whitney Test** mainly to compare between the two groups A & B (Study group and Control group). **Z-values** were obtained to highlight significant changes in maladaptive behaviour between both groups in relation to: Age, Sex, I.Q and Dose of the drug given and to compare Scores of maladaptive behaviour between both groups as well. **Pearson correlation** was used to find correlation between different variables. **t-test** was used to compare significance of the dose effect on improved maladaptive behaviour between both groups studied.

## Place of study:

**Bani-Sweif Psychiatric Hospital**, near Governorate building, Nile Korneesh Bani-Sweif.

Map (1): Map of Egypt

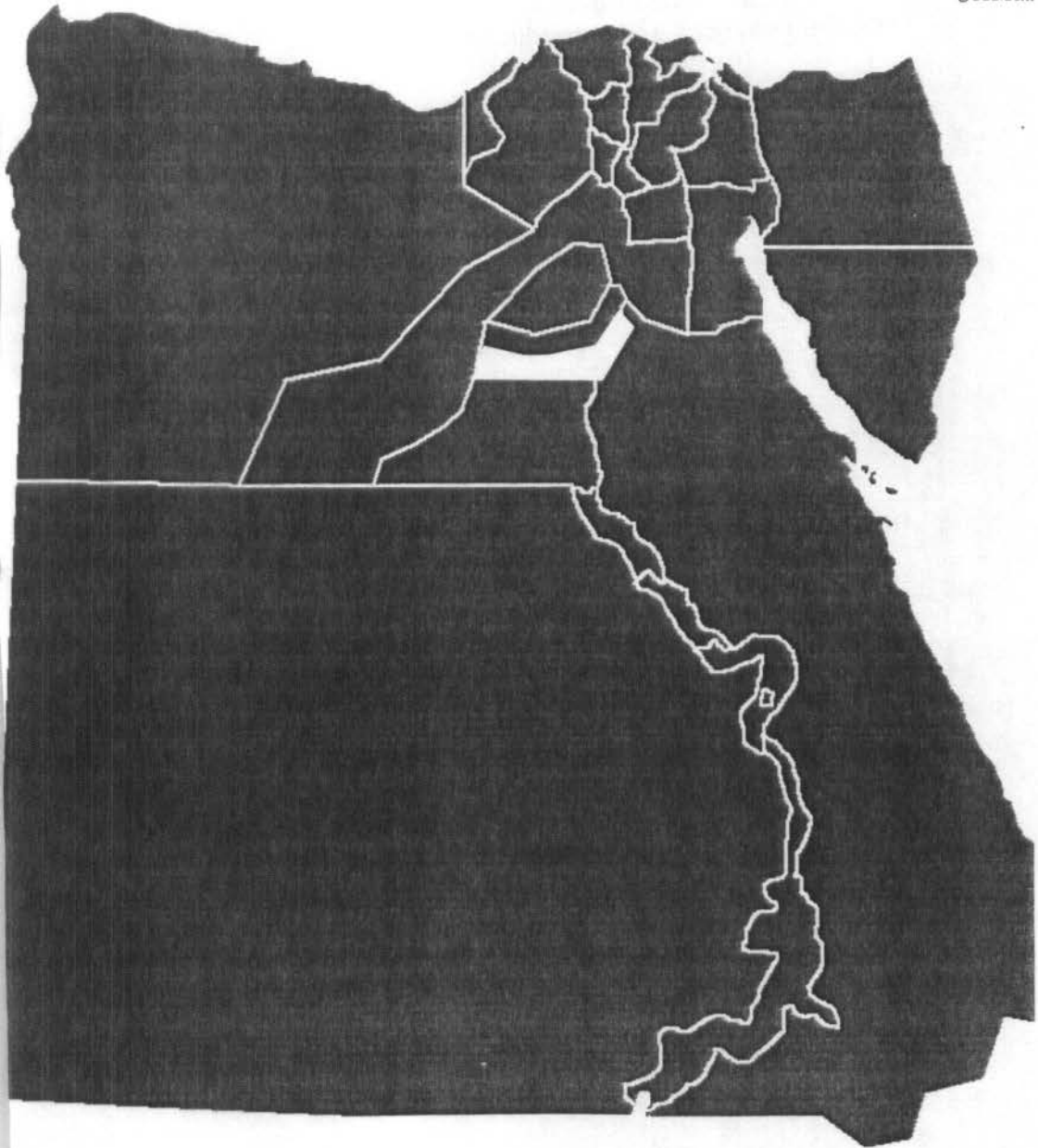


خريطة جمهورية مصر العربية



Map (2): Bani-Sweif Governorate

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محافظة بنى سويف



## The World Gazetteer

\* current population figures for cities, towns and places of all countries

\* largest cities of the world

\* current national flags

### Bani Suwayf - Egypt 2004

#### Bani-Suwayf Governorate (Bani-Suwayf city and subordinate towns) (Population in [1000])

(Table-3): Bani-Sweif Population

Place		Pop. 1996	Pop. 2004
Banī Suwaif	مدينة بنى سويف	172 000	203 600
Nāṣir	مركز ناصر	70 500	83 500
Al-Faṣn	مركز الفشن	52 600	62 300
Bibā	مركز ببا	49 400	58 500
Ihnāsiyā	مركز اهناسيا	31 500	37 300
Al-Waṣṭā	مركز الواسطى	30 900	36 500
Sumuṣṭā	مركز سمسطا	30 700	36 400
Al-Fant	مركز الفنت		22 500
Ṣawl	مركز صول		22 000
Total			562 600

( [www.world-gazetteer.com/d/d\\_eg\\_wj-htrm](http://www.world-gazetteer.com/d/d_eg_wj-htrm) )

# Time of study:

Total time = 15 months:

- ▶ Six months: (July, 2003 to December, 2003): Selection of patients.
- ▶ Six months: (January, 2004 to June, 2004): Examination, starting treatment and follow up of the cases along with collection of data.
- ▶ 03-months: (July, 2004 to September, 2004): Processing and revision of data, statistical analysis, conclusions and printing the work.

## Time table of the study:

(Table-4): Time table of the work

Number of months	Interval	Activity
06-months	July, 2003 – Dec., 2003	<ul style="list-style-type: none"> <li>▶ Selection of cases according to: criteria of inclusion + investigations (IQ + EEG + Vineland ABS assessment) + Assessment of maladaptive behaviour by Vineland ABS in the beginning of the study.</li> </ul>
06-months	Jan., 2004 – June, 2004	<ul style="list-style-type: none"> <li>▶ Starting treatment with Lamotrigine.</li> <li>▶ FU (every patient got the chance of regular follow up over six months period.</li> <li>▶ Re-assessment of maladaptive behaviour by Vineland ABS at the end of the study (end of treatment).</li> </ul>
03-months	July, 2004 – Sept., 2004	<ul style="list-style-type: none"> <li>▶ Processing of data:               <ul style="list-style-type: none"> <li>*Revision</li> <li>*Tabulation of data.</li> <li>*Statistical analysis</li> <li>*Discussion &amp; conclusions</li> </ul> </li> <li>▶ Language Revision and Printing the work</li> </ul>



# RESULTS



# \* RESULTS:

The results obtained by the researcher are summarized as follows:  
The following table (Table-5) summarizes the general results of the study:

## [1] General Clinical Results:

(Table-5): General clinical results

		Same	Worse	Adverse Effects	Improved	Total
<b>Study Group</b> (Abnormal EEG)	<b>Male</b>	<b>4</b>	<b>1</b>	<b>2</b>	<b>11</b>	<b>18</b>
	% to gp	13.3%	3.3%	6.6%	36.6%	
	<b>Female</b>	<b>3</b>	<b>0</b>	<b>1</b>	<b>8</b>	<b>12</b>
	% to gp	9.9%	0%	3.3%	26.6%	
	<b>Total</b>	<b>7</b>	<b>1</b>	<b>3</b>	<b>19</b>	<b>30</b>
	% to gp	23.3%	3.3%	9.9%	63.3%	
<b>Control Group</b> (Normal EEG)	<b>Male</b>	<b>5</b>	<b>2</b>	<b>1</b>	<b>10</b>	<b>18</b>
	% to gp	16.6%	6.6%	3.3%	33.3%	
	<b>Female</b>	<b>3</b>	<b>2</b>	<b>2</b>	<b>5</b>	<b>12</b>
	% to gp	9.9%	6.6%	6.6%	16.6%	
	<b>Total</b>	<b>8</b>	<b>4</b>	<b>3</b>	<b>15</b>	<b>30</b>
	% to gp	26.6%	13.3%	9.9%	50%	
<b>Grand Total</b>		<b>15/60</b>	<b>5/60</b>	<b>6/60</b>	<b>34/60</b>	<b>60</b>
% to all		25%	8.3%	10%	56.7%	

- In general, the mean percentage of responders (improved cases) was nearly 56.7% (63.3% in the Study group and 50% in the Control group).
- 25% of pts had to be classified as not improved (continued to have the same pre-treatment maladaptive behaviour).
- 8.3% of pts had to be classified as: worsened as developed increased aggression, disabling drowsiness, tiredness, or sleep disturbance.
- 10% discontinued treatment because of adverse effects; mainly vomiting, dizziness, tiredness and rash.

## [1] Description of the Sample:

### **{The psycho-demographic data of the sample}:**

The general features of the population sample of this study were as follows:

#### **2. Age:**

(Table-6.a): Age and Age Subgroups (Study and Control Groups)

	<b>Study Group</b>	<b>Control Group</b>
<b>Mean Group Age</b>	9.2	8.7
<b>S.D</b>	1.6955	1.6849
<b>Z value for Age</b> (Statistics Test: Mann-Whitney U)	-1.276 (Non-Significant) (Grouping Variable: Group Age)	

The cases were selected in the chronological age range of (6 to 11 ½) years with a mean chronological age of 9.2 in the Study Group and 8.7 in the Control Group, that gave a total mean chronological age of about 8 years. (Table-6.a) and (Figure 1).

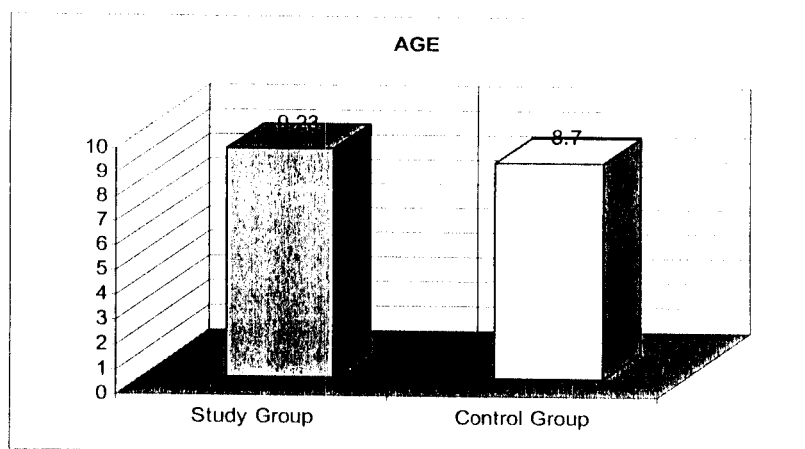


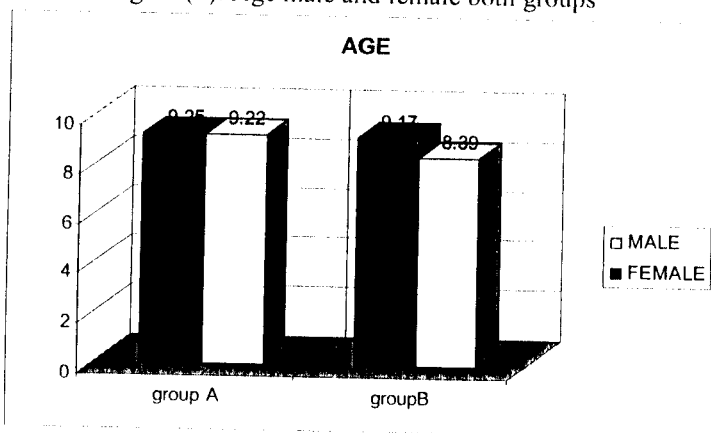
Figure (1): Age study and control groups

There was no significant difference between both groups as regards mean chronological age of each group before start of treatment. This can be concluded from the table above (Z value was **non-significant** for 'age variable' at the level of  $P>0.05$ ). Moreover, there were no remarkable age



differences between **males and females** in both groups together as shown in figure (2) below:

Figure (2): Age male and female both groups



### 3. Age Sub-groups:

(Table-6.b): Age and Age Subgroups (Study and Control Groups)

Age sub-groups (in years)	Study Group		Control Group	
	Number of cases	% to group	Number of cases	% to group
6-7	5	16.66%	6	20%
8-9	8	26.66%	14	46.66%
10-11½	17	56.66%	10	33.33%
<b>Total</b>	30	100%	30	100%

The age sub-groups inside the selected range were classified into 3 categories. (table-6.b) This shows that about half of the studied children belonged to the elder age group (10-11 ½) years, about one third belonged to the middle age group (8-9) years and about one fifth belonged to the younger age group of (6-7) years.

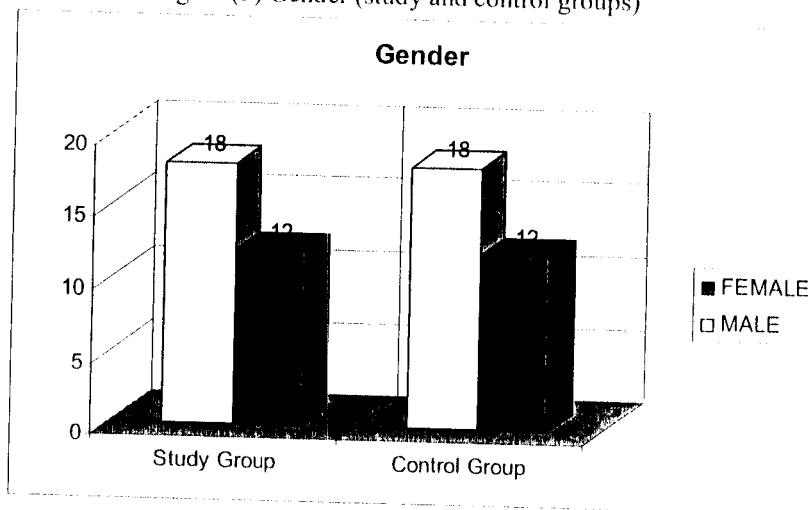
#### 4. Gender:

(Table-7): Gender: Study and control Groups

Gender	Study Group		Control Group	
	Number of cases	% to group	Number of cases	% to group
<b>Males</b>	18	60%	18	60%
<b>Females</b>	12	40%	12	40%
<b>Total</b>	30	100%	30	100%
<b>Z value for gender</b> (Statistics Test: Mann-Whitney U)	0.397 (Non-Significant) (Grouping Variable: Groups Gender in relation to age)			

Each sex was randomly selected separately inside each group to intentionally construct each group of exactly 30 children {in the actual prevalence rate of mentally retarded children in general societies (1½ male to 1 female)} giving our population sample the figures of 60% males and 40% females as shown above in (table-7) and below in figure (3):

Figure (3) Gender (study and control groups)



## 5. Socio-economic status:

(Table-8): Socio-economic level according to scores of Al-Shakhs Scale

	Study Group		Control Group	
	30	% to group	30	% to group
<b>Number of cases</b>				
<b>Mean score</b>	<b>131.43</b>	100%	<b>130.37</b>	100%
<b>S.D</b>	6.57416		6.03143	
<b>Socio-economic level</b>	<b>Average</b>		<b>Average</b>	
<b>Z value for Age</b> (Statistics Tests: Mann-Whitney U)	-0.587 (Non-Significant) (Grouping Variable: Groups socio-economic level)			

Every selected child was first evaluated for his/her socio-economic status (by using Al-Shakhs Scale for the socio-economic level of the family).

The scores on Al-Shakhs Scale and the level of the socio-economic status of the families of our studied children are summarized in tables (Appendix-VIII). All individuals of both groups belonged to the average (middle) socio-economic class. The mean socio-economic scores of the study group and Control group were respectively: 131 and 132 (denoting an average socio-economic class) as shown above, (Table-8):

## 6. Mental age and IQ :

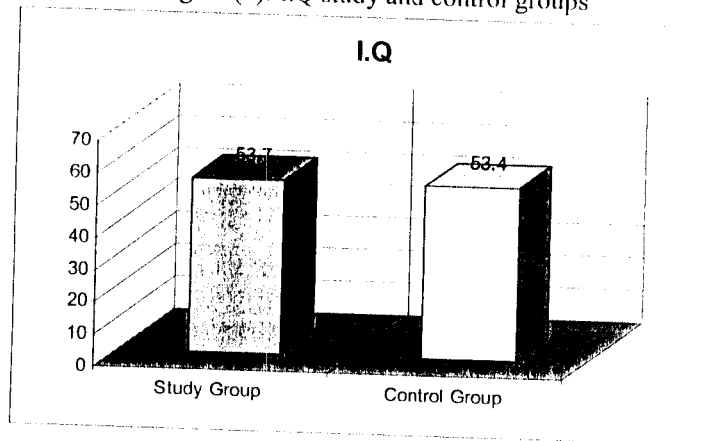
	Study Group	Control Group
<b>Number of patients</b>	30	30
<b>Mean Mental Age</b>	5year 1 month	4year 11month
<b>Mean IQ</b>	<b>54</b>	<b>53</b>
<b>Z value for IQ</b> (Statistics Test: Mann-Whitney U)	0.988 (Non-Significant)	

(Table-9): Mental Age and IQ: Study & Control Groups

In the beginning of the study, Mental Age was assessed as part of testing intelligence by Wechsler Intelligence Scale for Children (WISC) where the global IQ ratio (Intelligence Quotient) for patients in both groups ranged between 35 -70 with a mean total IQ of (54) and a mean Mental Age of (4 years and 11 months).

There was no significant difference between both groups as regards mean group IQ before start of treatment. This can be concluded from the table above (Table-9) where Z was non-significant for IQ means at the level of  $P > 0.05$ .

Figure (4): I.Q study and control groups



### 7. Degree of mental retardation according to IQ:

(Table-10): Degrees of M.R: Study & Control group

Degree of M.R	Study Group		Control Group	
	Number of cases	% to group	Number of cases	% to group
<b>Mild</b>	16	53.33%	13	43.33%
<b>Moderate</b>	14	46.66%	17	56.66%
<b>Total</b>	30	100%	30	100%

As obtained by psychometric assessment of IQ (Intelligence Quotient) using (WISC); the whole population study included two degrees of mental retardation (M.R) distributed in both groups (Table-10). It was observed that both groups consisted of nearly equal number of patients having mild or moderate M.R with mild increase in cases with mild M.R in group (A) than in group (B) and mild increase in moderate M.R cases in group (B) than in group (A). The total number of cases with mild M.R to that of moderate M.R in both groups was 29 to 31 cases (i.e. nearly each category formed about 50% of our population sample).

### 8. Age equivalent and Social Quotient (SQ):

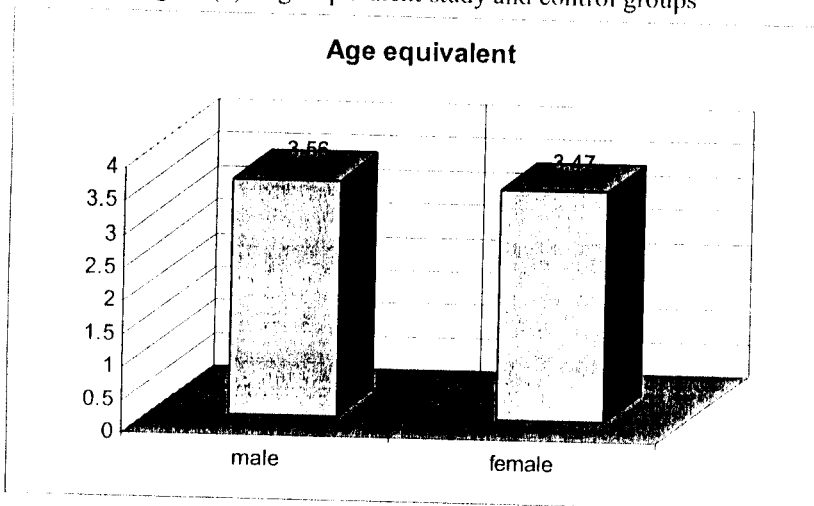
(Table-11): Age equivalent and S.Q

	Study Group	Control Group
Number of patients	30	30
<b>Mean Age Equivalent</b>	3year 7month	3year 5month
S.D	1.04426	0.99175
<b>Mean SQ</b>	<b>38</b>	<b>38</b>
S.D	10.34136	11.15435

Age equivalent and Social Quotient (SQ) were measured by Vineland ABS for all cases studied. **Age equivalent** is calculated from the sum of age equivalents of raw scores of Vineland's domains studied. The mean Age Equivalent for the population study was (3 years 6 months) The **SQ** is calculated by: multiplying Age equivalent by 100 & dividing the outcome by Chronological age. The mean SQs for both groups were (38) The values referred to here are illustrated in the above table (Table-11) (Detailed tables in Appendices III.B and IV)

(Age equivalent is illustrated between males and females (Figure 5) showed remarkable concordance between both groups.

Figure (5): Age equivalent study and control groups



**Matching both groups for consistency and differences considering Mental age, IQ, Age equivalent, and SQ:**

A correlation matrix was made (using **Pearson correlation**), included the above-mentioned four variables; [I.Q, S.Q, Mental age and Age equivalent]. showed *very* high correlation with significance at  $P < 0.001$  level in both groups separately except for correlation between mental age and social quotient in control group which also showed high correlation with significance at  $P < 0.01$  level. This means that all these items were highly significant in both groups at the beginning of the study confirming that **all the subjects were actually mentally retarded (mild or moderate M.R) with low I.Q, low S.Q, low mental age, and low adaptive behaviour level.** This can be inferred from the following tables (Table-12.a and 12.b):

**Correlation Matrix each group separately:**

**A. Correlation Matrix Study Group:**

(Table-12.a): Correlation matrix ages and quotients (Study group)

Correlation Matrix		Age Eq.	Mental Age	S.Q	I.Q
Age Equivalent	Pearson Correlation	1.000	.867	.716	.640
	Sig.	.	<b>0.000**</b>	<b>0.000**</b>	<b>0.000**</b>
	N	30	30	30	30
Mental Age	Pearson Correlation	.867	1.000	.567	.722
	Sig.	<b>0.000**</b>	.	<b>0.001**</b>	<b>0.000**</b>
	N	30	30	30	30
S.Q	Pearson Correlation	.716	.567	1.000	.876
	Sig.	<b>0.000**</b>	<b>0.001**</b>	.	<b>0.000**</b>
	N	30	30	30	30
I.Q	Pearson Correlation	.640	.722	.876	1.000
	Sig.	<b>0.000**</b>	<b>0.000**</b>	<b>0.000**</b>	.
	N	30	30	30	30

\*\* Correlation is significant at the 0.01 level (2-tailed).

## B. Correlation matrix Control Group:

(Table-12.b): Correlation matrix ages and quotients (Control group)

Correlation Matrix		Age Eq.	Mental Age	S.Q	I.Q
Age Equivalent	Pearson Correlation	1.000	.819	.745	.704
	Sig.	.	<b>0.000</b>	<b>0.000</b>	<b>0.000</b>
	N	30	30	30	30
Mental Age	Pearson Correlation	.819	1.000	.419	.763
	Sig.	<b>0.000**</b>	.	<b>0.021*</b>	<b>0.000**</b>
	N	30	30	30	30
SQ	Pearson Correlation	.745	.419	1.000	.739
	Sig.	<b>0.000**</b>	<b>0.021*</b>	.	<b>0.000**</b>
	N	30	30	30	30
IQ	Pearson Correlation	.704	.763	.739	1.000
	Sig.	<b>0.000**</b>	<b>0.000**</b>	<b>0.000**</b>	.
	N	30	30	30	30

\*\* Correlation is significant at the 0.01 level (2-tailed).

\* Correlation is significant at the 0.05 level (2-tailed).

## 9. Adaptive Behaviour level:

(Table-13.a): Adaptive Behaviour levels for (Study and Control Groups)

	Study Group			Control Group		
Number of cases	30			30		
<b>Domain</b>	<b>Communi- cation</b>	<b>Daily Living Skills</b>	<b>Sociali- zation</b>	<b>Communi- cation</b>	<b>Daily Living Skills</b>	<b>Sociali- zation</b>
Total Domain raw Sore	2155	2095	1724	2082	2083	1707
Mean of Domain raw score	71.83	69.38	57.46	69.40	69.43	56.9
Total 3 Domains standard score	3972			4099		
Mean 3 Domains Standard score	132.4			136.63		
Total Adaptive Behaviour Composite score	1221			1257		
Mean Adaptive Behaviour Composite score	40.7			41.9		
<b>Adaptive Behaviour level</b>	<b>Low</b> (=deficit in adaptive behaviour)			<b>Low</b> (=deficit in adaptive behaviour)		
<b>Z value</b> (Statistics Test: Mann-Whitney)	-0.577 (Non-Significant) (Grouping Variable: Group)					

Adaptive Behaviour for the selected cases was first assessed in raw scores by Vineland ABS for the three main Domains (Communication, Daily Living Skills and Socialization Domains) then the standard scores were obtained from the tables in the manual of the Vineland Scale to get the standard values of Adaptive behaviour Composite which showed that all cases had **low level of adaptive behaviour** with a mean Vineland Adaptive Behaviour Standard Score Composite of (41.3) as summarized in (Table-13.a). (Detailed tables in Appendix-V and VI).



(Table-13.b) Degree of low adaptive behaviour level of the study population

Degree of Low Adaptive Behaviour Level	Adaptive Behaviour Composite score (00-70)	Number of cases in Study Group	Number of cases in Control Group	Total
Mild	50-70	4	6	10
Moderate	35-50	26	24	50
<b>Total</b>		30	30	60

The above table shows that only few cases in either group had a mild degree of deficit in adaptive behaviour (4 and 6 patients) but the majority of cases had a moderate degree of such deficit, (Table-13.b)

**10. Maladaptive Behaviour Level before treatment by LMG: (Concern of the study):** Comparing both groups (**group to group**) for scores of Vineland Maladaptive Behaviour, part-I before starting treatment:

**(A). At the level of individual items of the Maladaptive Behaviour Domain:**

(Table-14.a): Vineland's Scores of individual items **group to group**

Scores of Maladaptive Behaviour Domain	Study Group		Control Group	
	Number of Group items	Scores of items	Number of Group items	Scores of items
Total score	231	377	241	375
Mean score	7.7	12.56	8.0	12.50
S.D	2.3		2.8	
Mean Level of Maladaptive Behaviour	Intermediate (≡Significant disturbance)		Intermediate (≡Significant disturbance)	
Z value (Statistics Test: Mann-Whitney)	-0.734 (Non-Significant) (Grouping Variable: Group)			

The scores of individual items of Vineland's Maladaptive Behaviour Domain for every patient, (27 items of Vineland ABS) and also of cluster symptoms were calculated. The obtained total scores of all individual items in each group showed a mean maladaptive behaviour score of (12.56) for

Study group and (12.50) for Control group before treatment by LMG as shown in table (Table-14.a).

**(B). At the level of item clusters:**

**1) Considering Age, I.Q and Dose: (Grouping Variable: GROUP)**

(Table-14.b): Cluster symptoms **group to group** for age, I.Q and dose (before LMG)

Statistics Test	Neurotic Before	Mood Before	ADHD Before	Disruptive Before
Mann-Whitney U	375.0	425.0	387.50	320.000
Wilcoxon W	840.0	890.0	852.50	785.000
Z	-1.142	-.397	-.945	-1.953
Sig.	0.254	0.691	0.345	0.051

As mentioned before, the maladaptive items of Vineland ABS were sub-grouped into **four clusters**. Mann-Whitney test was used to check any significant difference between both groups before treatment by LMG. The outcome in Z value was non-significant; i.e. there was no significant difference in maladaptive behaviour cluster symptoms between both groups (considering Age, I.Q and Dose) when compared together before treatment considering age, I.Q and dose of LMG given. (Table-14.b)

**2) Considering Gender: (Grouping variable: Gender)**

(Table-14.c): Cluster symptoms **group to group** considering **gender** (before LMG)

Statistics Test	Neurotic BF	Mood BF	ADHD BF	Disruptive BF
Mann-Whitney U	406.0	390.50	417.0	332.000
Wilcoxon W	1072.	1056.5	1083.0	632.000
Z	-.404	-.673	-.232	-1.533
Sig.	0.686	0.501	0.817	0.125

The above table (Table 14.c) shows that there was no significant difference between the study group and the control group in scores of Vineland's maladaptive behaviour when compared group to group before starting treatment with LMG considering gender variable.

## 11. Dose of Lamotrigine:

### (A) Considering total and improved cases:

	Study Group	Control Group
Total Number of cases	30	30
Mean Dose (for total)	125 mg	90.8 mg
Number of improved cases	19	15
<b>Mean Dose (for improved cases)</b>	<b>123.68 mg</b>	<b>88.33 mg</b>
S.D	65.34	35.19

(Table-15.a): Dose of Lamotrigine for Study & Control groups

The mean dose given to all group cases was 125 mg for Study group and 90.8 mg for Control group and the mean dose given to improved cases was 123.7 mg/day for Study group patients and 88.3 mg/day for Control group ones as shown in the table above (Table 15.a). Those patients who developed intolerable adverse symptoms or worsened by treatment had to discontinue LMG early and were dropped from follow up.

### (B) Dose associated with improvement in relation to group and gender:

(Table-15.b): Dose group to group and at gender level

Statistics Test	DOSE (Group to group)	DOSE (At gender level)
<b>Mann-Whitney U test</b>	319.5	372.5
<b>Z signifiacne</b>	0.359 (Non-significant)	
<b>Leven's test</b>	0.053 (Non-significant)	
<b>t-test (equality of means not assumed)</b>	0.068 (Non-significant)	

Comparing mean doses associated with improvement of symptoms in both groups showed no significant dose difference; whether when compared group to group or when compared at the level of gender (Z value at the level of  $P > 0.05$ ) measured through t-test (using Leven's test with equality of means not assumed) as shown in the above table (Table 15.b) and in Figures (6) and (7):

Figure (6): Dose Study and Control groups

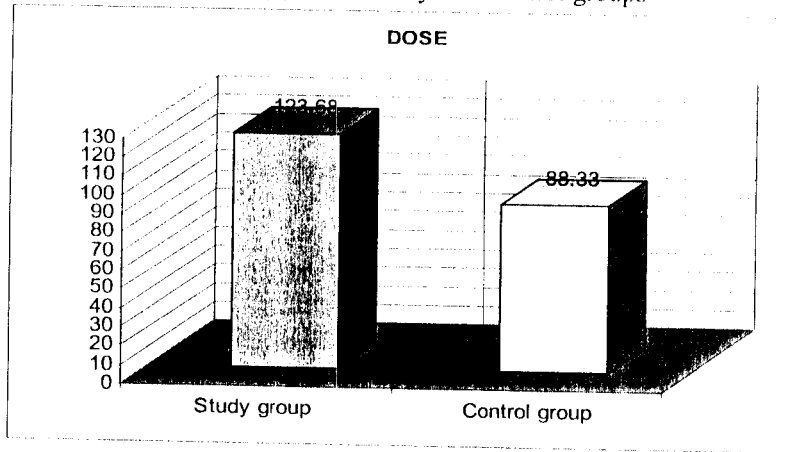
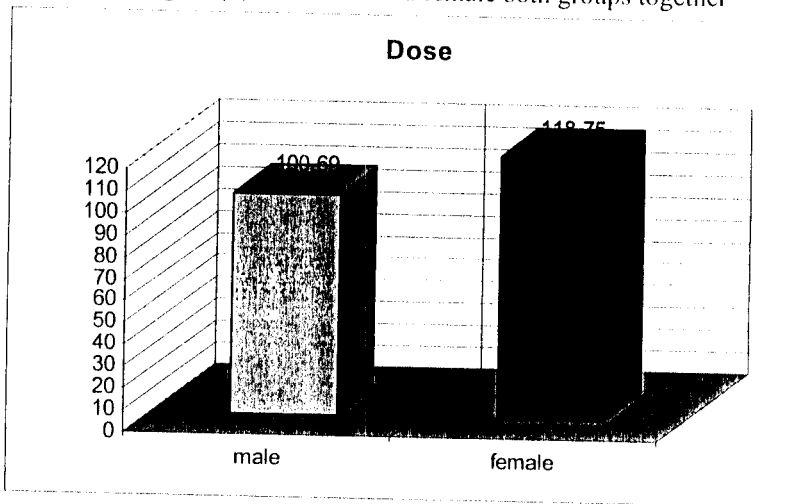


Figure (7) Dose male and female both groups together



## **[2] Statistical analysis of results Before and after LMG:**

{Comparing both groups together before and after LMG as regards improvement of maladaptive behaviour}:

**\*\*First: Comparing “group to group” before then “group to group” after treatment by LMG:**

**(A). At the level of total scores of individual symptoms of Maladaptive Behaviour:**

**1) considering Age, I.Q, and Dose:** (Grouping Variable: GROUP)

Statistics Test		Before LMG	After LMG
	Number of cases	60	49
	Mean score	12.5333	6.9796
	S.D	2.60030	3.55017
Mann-Whitney U		401.500	299.000
Wilcoxon W		866.500	575.000
Z		-0.734	0.000
<b>Sig.</b>		<b>0.463</b>	<b>1.000</b>

(Table-16.a) Total scores of individual items for age, IQ and dose before and after LMG

Considering Age, I.Q, and Dose, there was no significant difference in maladaptive behaviour cluster symptoms between both groups when compared with each other before or after treatment. (Table-16.a)

**2) Considering gender:** (Grouping Variable: Gender)

(Table-16.b) Total scores of individual items for gender before and after LMG

Statistics Test		Before LMG	After LMG
	Number of cases	60	49
	Mean score	12.5333	6.9796
	S.D	2.60030	3.55017
Mann-Whitney U		404.000	277.500
Wilcoxon W		704.000	742.500
Z		-0.432	-0.161
<b>Sig.</b>		<b>0.665</b>	<b>0.872</b>

Considering gender, there was no significant difference between the study group and the control group in scores of maladaptive behaviour when

compared group to group before treatment with LMG and also after LMG therapy. (Table-16.b)

**(B). At the level of clusters:**

**1) Considering Age, I.Q and Dose: (Group to Group):**

(Table-17.a): Cluster symptoms considering age, I.Q and dose (before and after LMG)

Statistics Test	Neurotic BF	Mood BF	ADHD BF	Disrupt. BF	Neurot AF	Mood AF	ADHD AF	Disrupt. AF
Mann-Whitney	375.0	425.0	387.50	320.0	338.0	422.0	413.0	327.0
Wilcoxon W	840.0	890.0	852.50	785.0	803.0	887.0	878.0	792.0
Z	-1.142	-.397	-.945	-1.953	-1.731	-.425	-.561	-1.887
Sig.	0.254	0.691	0.345	0.051	0.083	0.671	0.575	0.059

Grouping Variable: GROoup

In general, there was no significant difference before or after treatment by LMG between both groups when compared together (group to group) regarding Age, I.Q., Sex, or the Dose given or the score of maladaptive behaviour (Z at the level of > 0.05). This is clear in (Table-17.a):

**2) Considering Gender: (Group to Group): (as regards Gender)**

(Table-17.b): Cluster symptoms considering gender (before and after LMG)

Statistics Test	Neur. BF	Mood. BF	ADHD. BF	Disrupt. BF	Neurot. AF	Mood AF	ADHD. AF	Disrupt. AF
Mann-Whitney	406.0	390.50	417.0	332.0	405.0	337.5	386.0	395.5
Wilcoxon W	1072.	1056.5	1083.0	632.0	705.0	1003.5	1052.0	695.5
Z	-.404	-.673	-.232	-1.533	-.426	-1.463	-.712	-.571
Sig.	0.686	0.501	0.817	0.125	0.670	0.143	0.477	0.568

Grouping variable: Gender

In general, there was no significant difference before or after treatment by LMG between both groups when compared together (group to group) regarding gender (Z at the level of  $P > 0.05$ ) as evidenced above (Table-17.b)

The above tables show that there was no significant difference between the study group and the control group in scores of maladaptive behaviour when compared group to group before starting treatment with LMG (as regards age, I.Q, dose or gender or when compared group to group after treatment with LMG; there was also no significant difference regarding same variables. However, after treatment, both groups achieved significant improvement in all the clusters of maladaptive behaviour.

**\*\*SECOND: Comparing each group separately before and after treatment: (Cluster Symptoms Group (A) before and after LMG)**

**Study Group:** Cluster symptoms before and after LMG

(Table-18.a): Cluster symptoms study group before and after LMG

Statistics Test	Neurotic AF- Neurotic BF	Mood AF - Mood BF	ADHD AF ADHD BF	Disruptive AF- Disruptive BF
Z	-1.976	-3.680	-4.286	-4.154
<b>Sig.</b>	0.048*	0.000**	0.000**	0.000**

**Study Group:** (Total score of whole group before and after LMG)

(Table-18.b): Scores of maladaptive behaviour Study group before and after LMG

	N	Mean	S.D	Score BF-Score AF
<b>Score Before</b>	30	12.56	2.4	
<b>Score After</b>	26	6.88	3.6	
Z (Wilcoxon test)				4.124
<b>Sig.</b>				<b>0.000 **</b>

Tables-(18.a) and (18.b) show that there was significant improvement in maladaptive behaviour symptoms in Study group at high level of  $P < 0.001$

**Control Group:** Cluster symptoms before and after LMG

(Table-19.a): Cluster symptoms control group before and after LMG

Statistics Wilcoxon test	Neurotic AF- Neurotic BF	Mood AF - Mood BF	ADHD AF ADHD BF	Disruptive AF- Disruptive BF
Z	-2.640	-3.394	-2.695	-3.332
<b>Sig.</b>	0.008*	0.001**	0.007*	0.001**

**Control Group:** (Total score of whole group Before and After LMG):

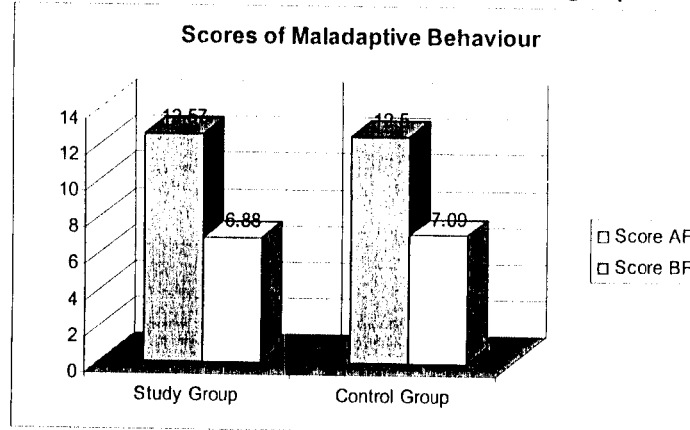
(Table-19.b): Scores of maladaptive behaviour control group

	N	Mean	S.D	Score BF-Score AF
<b>Score Before</b>	30	12.50	2.8	
<b>Score After</b>	23	7.08	3.5	
Z (Wilcoxon test)				3.526
<b>Sig.</b>				<b>0.001 **</b>

Tables-(19.a) and (19.b) show that there was significant improvement in maladaptive behaviour symptoms in Control group at high level of  $P < 0.001$

The outcome showed significant improvement in both groups after treatment with LMG with very high significance with Z value at  $P < 0.001$  level. The following figure illustrates this conclusion:

Figure (8) Scores of maladaptive behaviour both groups



There was a significant difference between all cluster-symptoms (of Neurotic symptoms, Mood symptoms, Attention deficit hyperactivity symptoms, and Disruptive behaviour symptoms) in the **Study group** before and after treatment with LMG in the following order: ( $P < 0.05$ ,  $P < 0.001$ ,  $P < 0.001$ , and  $P < 0.001$ ). There was also concomitant significant difference in the same cluster-symptoms in the **Control group** before and after treatment with LMG in the following order: ( $P < 0.01$ ,  $P < 0.001$ ,  $P < 0.01$ , and  $P < 0.01$ ) denoting that there was a universal improvement in all maladaptive behaviour symptoms in both groups, but the improvement was much more significant in the **Study group** subjects than in the **Control group** ones. This is evidenced by the following figures (9,10,11 and 12):



Figure (9): Neurotic symptoms both groups (before and after LMG)

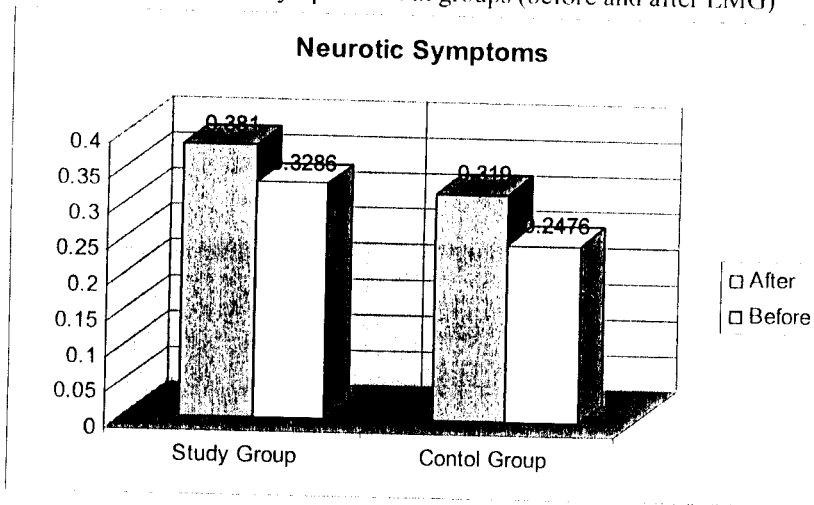


Figure (10): Mood symptoms both groups (before and after LMG)

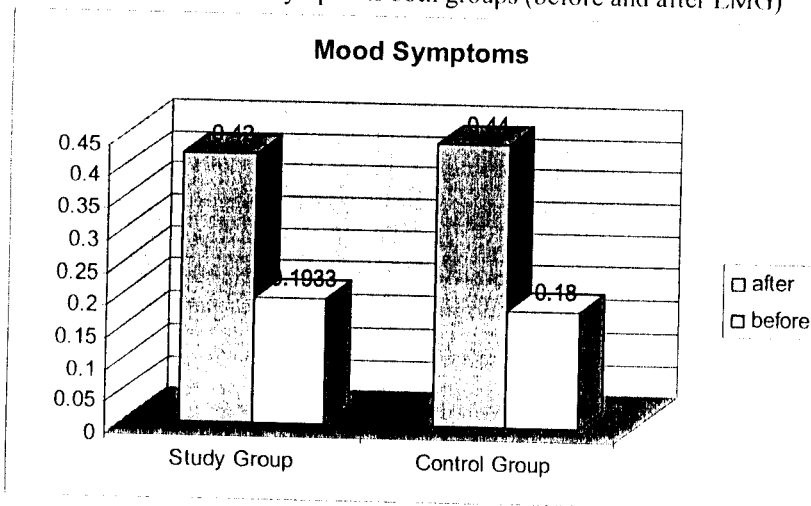


Figure (11): ADHD symptoms both groups (before and after LMG)

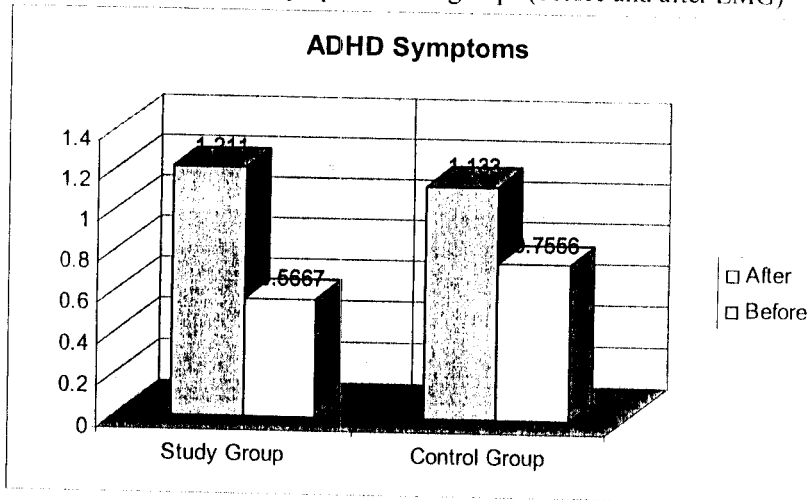
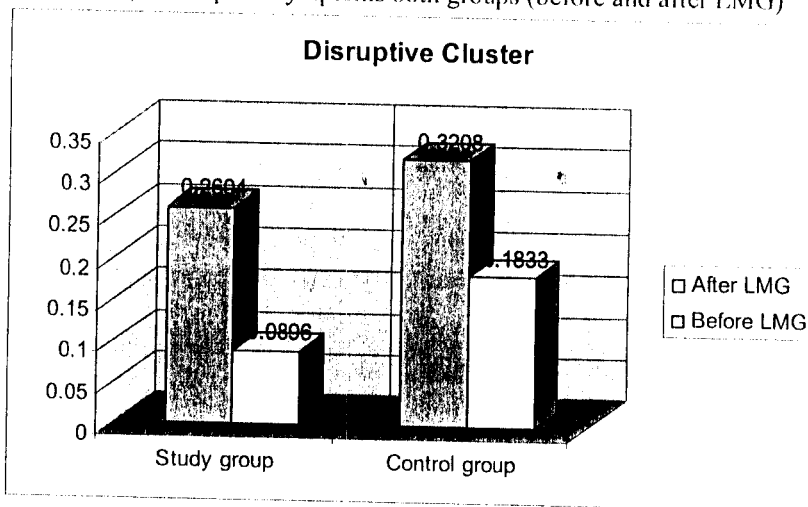


Figure (12): Disruptive symptoms both groups (before and after LMG)



**\*\*Third: Considering individual symptoms of maladaptive behaviour in each group separately before and after LMG:**

The following tables (Tables 18 & 19) illustrates the significant and non-significant improvement in different items of maladaptive behaviour in both groups separately:

**(A) Items (Statements) Study Group: (Table-20)**

(Table-20): Individual items before and after LMG (Study group)

Item's score (Before and After)	S1A - S1.B	S2A - S2.B	S3A - S3.B	S4A - S4.B	S5A - S5.B	S6A - S6.B	S7A - S7.B	S8A - S8.B	S9A - S9.B
Z (Wilcoxon)	-1.000	-1.732	.000	-1.342	-.828	-.368	-1.000	-2.530	-1.633
Sig.	.317	.083	1.000	.180	.408	.713	.317	.011	.102
								P<.05	

Item's score (Before and After)	S10A - S10.B	S11A - S11.B	S12A - S12.B	S13A - S13.B	S14A - S14.B	S15A - S15.B	S16A - S16.B	S17A - S17.B	S18A - S18.B
Z (Wilcoxon)	-2.428	-1.000	-2.165	-3.811	-2.739	-3.145	-1.000	-2.810	-3.624
Sig.	.015	.317	.030	.000	.006	.002	.317	.005	.000
	P<.05		P<.05	P<.001	P<.01	P<.01		P<.01	P<.01

Item's score (Before and After)	S19A - S19B	S20A - S20B	S21A - S21B	S22A - S22B	S23A - S23B	S24A - S24B	S25A - S25B	S26A - S26B	S27A - S27B
Z (Wilcoxon)	-2.251	-1.633	-1.732	-1.000	-1.732	-1.414	.000	-1.000	-1.342
Sig.	.024	.102	.083	.317	.083	.157	1.000	.317	.180
	P<.05								

**(B) Items (Statements) Control Group: (Table-21)**

(Table-21): Individual items before and after LMG (Control group)

Item's score (Before and After)	S1A - S1.B	S2A - S2.B	S3A - S3.B	S4A - S4.B	S5A - S5.B	S6A - S6.B	S7A - S7.B	S8A - S8.B	S9A - S9.B
Z (Wilcoxon)	.000	-1.000	-.447	-2.000	-1.414	-.791	-1.000	-3.000	-1.857
Sig.	1.000	.317	.655	.046	.157	.429	.317	.003	.063
				P<.05				P<.01	

Item's score (Before and After)	S10A - S10.B	S11A - S11.B	S12A - S12.B	S13A - S13.B	S14A - S14.B	S15A - S15.B	S16A - S16.B	S17A - S17.B	S18A - S18.B
Z (Wilcoxon)	-1.732	-2.070	-1.633	-2.038	-2.973	-.707	-.816	-1.623	-2.961
Sig.	.083	.038	.102	.042	.003	.480	.414	.105	.003
		P<.05		P<.05	P<.01				P<.01

Item's score (Before and After)	S19A - S19B	S20A - S20B	S21A - S21B	S22A - S22B	S23A - S23B	S24A - S24B	S25A - S25B	S26A - S26B	S27A - S27B
Z (Wilcoxon)	-.577	-1.414	-1.890	-.557	-1.890	-1.414	-1.000	-1.000	-2.333
Sig.	.564	.157	.059	.577	.059	.157	.317	.317	.020
			P<.05		P<.05				

The above results are illustrated here below: (Table-22)  
 (Table-22): Significant improvement of Vineland's items after LMG (both groups)

Vineland ABS		<b>Domain of: Maladaptive Behaviour, Part-I</b> (Individual symptoms before and after treatment by Lamotrigine)						
		Study Group			Control Group			
Z - Significance level		<.05	<.01	<.001	<.05	<.01	<.001	
Clusters	Items							
Neurotic symptoms	1. Sucks thumb or fingers							
	2. Bites fingernails							
	3. Grinds teeth day or night							
	4. Eating disturbance				*			
	5. Sleep disturbance							
	6. Wets bed (Nocturnal Enuresis)							
	7. Tics							
Mood symptoms	8. Temper tantrums	*				**		
	9. Exhibits extreme anxiety							
	10. Exhibits excess unhappiness	*						
	11. Cries or laughs too easily				*			
	12. Shows lack of consideration	*						
ADHD	13. Poor attention			***	*			
	14. Overly active		**			**		
	15. Too impulsive.		**					
Disruptive symptoms	16. Bullies or teases							
	17. Too physically aggressive		**					
	18. Stubborn or sullen			***		**		
	19. Negativistic or defiant	*						
	20. Swears inappropriately							
	21. Lies, cheats, or steals				*			
	22. Overly dependent							
	23. Withdraws				*			
	24. Avoids school or work							
	25. Truant from school or work							
	26. Runs away							
	27. Poor eye contact							
<b>Z - Significance level</b>		<b>&lt;.05</b>	<b>&lt;.01</b>	<b>&lt;.001</b>	<b>&lt;.05</b>	<b>&lt;.01</b>	<b>&lt;.001</b>	

The table above (Table-22) illustrates the Vineland's maladaptive Domain items and their corresponding significant improvement values.

**At the level of individual symptoms, the symptoms that showed significant improvement after treatment by Lamotrigine were as follows:**

- 1) Poor attention and concentration:** It showed significant improvement in both groups with very high significance in the study group (Z at the level of  $P < 0.001$ ) more than in the control group (Z at  $P < 0.05$ ).
- 2) Stubbornness or Sullenness:** It showed significant improvement in both groups with very high significance in the study group (Z at the level of  $< 0.001$ ) more than in the control group (Z at the level of  $P < 0.01$ ).
- 3) Hyperactivity:** It showed significant improvement in both groups with equally high significance in the both groups (Z at the level of  $P < 0.01$ ).
- 4) Temper Tantrums:** It showed significant improvement in both groups with high significance in the control group (Z at the level of  $P < 0.01$ ) more than in the study group (Z at the level of  $P < 0.05$ ).
- 5) Aggressive Behaviour:** It showed high significant improvement in the study group only (Z at the level of  $P < 0.01$ ) with no recordable significant improvement in the control group.
- 6) Impulsivity:** It showed significant improvement in the study group only (Z at the level of  $P < 0.01$ ) with
- 7) Depressed Mood:** It showed significant improvement in the study group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group.
- 8) Lack of Consideration:** It showed significant improvement in the study group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group.
- 9) Negativism and Defiant Behaviour:** It showed significant improvement in the study group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group.
- 10) Lying, Cheating or Stealing:** It showed significant improvement in the control group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the study group.
- 11) Withdrawal behaviour:** It showed high significant improvement in the control group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the study group.
- 12) Eating disturbance:** It also showed significant improvement in Control group only (Z at the level of  $P < 0.05$ ).



# Discussion





## **\*\* Discussion :**

Mental retardation is a universal problem found in every race, religion, culture and economic background. (Shapiro, 1996) It affects approximately **1-3% of the population** worldwide (Sadock, 2000 and Kolevzon & Simeon, 2002). Different studies have reported different rates depending on definitions used, methods of ascertainment, and population studied (APA, 1994). Higher values were recorded in different studies and populations.

Mentally retarded children frequently show one or more item of **mal-adaptive behaviour** that interrupt the process of their rehabilitation and learning and necessitate comprehensive psychiatric attention along with family & social support (Carr, 1999). There is a wide range of such maladaptive behaviour. It can be just simple & tolerable by other family members e.g. mild irritability or occasional stubbornness, or might be severe and very noisy e.g. poor frustration tolerance, persistent hyperactivity, impulsivity, destructive or aggressive behaviour or disinhibited & embarrassing talks & acts (Sadock & Sadock, 2000).

Early diagnosis of psychiatric disorders in children with mental retardation leads to early treatment. Medications are one part of overall treatment and management of children with mental retardation (Janicki et al, 1999).

Many drugs have been tried to treat associating maladaptive behaviour many cases of mental retardation especially antipsychotic drugs (e.g. thioridazine or haloperidol) and antiepileptic drugs e.g. Carbamazepine and Valproic acid. Newer antiepileptics e.g. **lamotrigine** & Gabapentin, proved effective in treatment of epilepsy in developmentally disabled children, with less adverse effects especially on cognition and behaviour (Rutecki & Gidal, 2002).

**Lamotrigine** has recently been approved also as a mood stabilizer. It also showed promising effects on modification of behaviour of persons with borderline personality disorder (Goldberg, 1997). Furthermore, Lamotrigine recently showed favourable psychological effects when used in treatment of some mentally retarded children suffering from epilepsy (Mikati, 2003). This aroused the hypothesis of this study; "Can lamotrigine ameliorate mood and attention and can it soothe the hyperactive, impulsive and aggressive behaviour of non-epileptic mentally retarded children who have such maladaptive symptoms with special concern to having normal or abnormal EEG.

To our knowledge, this study is only the first case-control trial to examine the use of **Lamotrigine** in treatment of mentally retarded patients with behavioural disorders and without having epilepsy. To our knowledge also, this is the first study in Egypt applying “**Maladaptive Domain, part-I**” of **Vineland Adaptive Behaviour Scale** on mentally retarded children with behavioural disorders, so, it may be considered as a country-wise standardization sample.

At the study entry, all the subjects were quite symptomatic (i.e. their maladaptive behaviour scores were “significant or intermediate” on Vineland ABS, Maladaptive Behaviour Domain, part-I). In the beginning of the study, a significant impairment in behaviour was confirmed by a mean baseline Vineland Maladaptive Behaviour score of (12.56) for Study group and (12.50) for Control group, denoting that there was a **significant universal disturbance in maladaptive behaviour in both groups**.

In the beginning of this study, each group was consistent and homogeneous in its structure. Also both groups were similar in their constitution as regards age, sex, mental age, Intelligence Quotient, Social Quotient, adaptive behaviour, age equivalent and maladaptive behaviour.

A correlation matrix (using **Pearson correlation**), included four variables; [I.Q, S.Q, Mental age and Age equivalent] showed a *very* high correlation with significance at  $P < 0.001$  level in both groups separately except for correlation between mental age and social quotient in control group which also showed high correlation with significance at  $P < 0.01$  level. This means that all these items were highly significant in both groups at the beginning of the study confirming that **all the subjects were actually mentally retarded (mild or moderate M.R) with low I.Q, low S.Q, low mental age, and low adaptive behaviour level**. The only different variable between both groups was the presence of abnormal EEG changes in the Study group and absence of similar changes in the control group.

The distribution of the **age-subgroups** of the included children into three categories showed that about half of the studied children belonged to the elder age group (10-11 ½) years, about one third belonged to the second age group (8-9) years and about one fifth belonged to the younger age group of (6-7) years. This may be explained by one or more of the following suggestions:

- a) Increased severity of maladaptive behaviour with increase in age; as the child becomes more physically developed and able to move around and cause problems. This suggestion agrees with the finding recorded by Holder and Kirkpatrick, (1991) that the mentally disabled interpret

emotions less accurately and spend more time identifying specific emotions, and the younger children require more time to interpret fear and anger than older ones.

- b) Negligence of caregivers to bring their children early for treating maladaptive behaviour considering it non-remediable and needs traditional healers not hospital management. This was highlighted by (Okasha and Maj, 2001) who explained that belief systems in the Arab world are derived from Islamic and non-Islamic roots e.g. beliefs related to the adversity of Zar demons to humans and beliefs that dead sheikhs of religion could bless or help those who invoke their support by visiting their shrines. In many cases the frustrated caregivers bring their children too late to the hospital for treatment after many years of suffering with them.
- c) The criteria of inclusion in this study excluded many factors that can affect age distribution in the population sample.
- d) The relatively small size of the sample might not be precisely expressive of the actual distribution of maladaptive behaviour in such age groups. Further studies may address larger samples.

Nevertheless, It is not expected to relate maladaptive behavioural changes to a particular age group as it is already known that prevalence of disruptive behaviour is related to many predisposing factors (detailed in page: 42) and it was only found more in urban than in rural areas (Spender et al, 2001) and more in people with lower IQ (Barker, 1988).

The population study included two **degrees of mental retardation**. It was observed that both groups were consisting of nearly equal number of patients having mild or moderate M.R. The total number of cases with mild M.R to total of moderate M.R in both groups was 29 to 31 cases (i.e. nearly each category formed about 50% of our population sample). There was no significant difference between both groups as regards mean group mental age and mean group IQ before start of treatment (IQ was 54 for Study group and 53 for Control group and Z value was non-significant for both means). Yet, both obtained IQs were at the lower limit of mild mental retardation (IQ = 50-70) and near to the upper limit of moderate mental retardation (IQ = 35 to 50). This implies that maladaptive behaviour was more in patients with lower degrees of mental retardation, which is concordant with the finding of (Barker, 1988) who concluded that there was a relationship between lower IQ and deviant behaviour, may be in part because greater intellectual capacity makes social adaptation occur easier.

The mean **Age Equivalents** for the population study were 3year 7month (study group) and 3year 5month (Control group) and the mean **SQ** was (38) for both groups which are clearly much lower values than their corresponding mean mental ages for both groups (5year 1 month for Study group) and 4year 11month (Control group) and mean IQs (54 and 53). Most patients in both groups (40) had moderate deficit in adaptive behaviour while only (10) patients had mild deficit. This observation is concordant with that of Sadock's, who highlighted that different studies found different correlations between cognitive and adaptive behaviour; strong correlations are especially observed in persons with moderate-to-profound mental retardation but weak correlations are found between cognitive and adaptive behaviour in persons with mild mental retardation. Thus, to resolves at least some of the controversy about the relative importance of these two constructs, I.Q. may be considered as an upper limit or ceiling to adaptive accomplishments (Sadock & Sadock, 2000). Also, the subjects in our population sample are selected having maladaptive behaviour, which no doubt interrupts the process of learning and rehabilitation of those children (Carr, 1999).

In our study **both sexes** were included in both of the studied groups with a **male-to-female ratio of 1.5:1** (18 males to 12 females in each group) to be more representative of the actual ratio in general population of mentally retarded children. Mental Retardation is more common among males, with a male to female ratio of approximately **1.5:1** as recorded by DSM-IV (APA, 1994). In **Egypt**, Okasha et al in 1983, reported a male to female ratio of **2:1** (Farrag, 1995). Male predominance may be the result of the culturally determined higher premium on male children in the society with parents being usually more inclined to report intellectual retardation in male children as compared to females. Additionally, relatively low emphasis on the education of girls (especially in rural areas) could also be responsible to some extent (Okasha et al, 1983). In our study, there was no significant difference between males and females as regards having disruptive behaviour or as regards improvement with Lamotrigine therapy. This agrees with the finding of Spector and Jackson on nursing home residents with cognitive and other disabilities, that being female did not affect the likelihood of being disruptive in general, but women were less likely to be verbally or physically abusive (Spector and Jackson, 1994).

Sadock, (2000) considered the prevalence rate of M.R. to be probably below 3% but above 1% and stated that this 3% of total population is not fixed in all societies; it increases with decreased economic and cultural levels

in society to reach up to 7% in areas crowded with **poor people**. Also, in a study by the Supreme Establishment for Care of the Handicapped in **Egypt** (1985), the prevalence of mentally handicapped in high and above average socioeconomic population was (3-3.3)% and reached **up to (7)%** in some districts with high-density and **poor socioeconomic population** (Ibrabim, 2000). The incidence of M.R. in the age group of 6-9 years was estimated in the primary school in Cairo city and it ranged between (7.6-12.7%). This ratio could be less than the true one because a large percentage of the mentally retarded do not go to school (Soltan et al, 1983).

The study done by the Research centre in Arizona University, (1982) found that more people with 'mild mental retardation' come from **minority groups and low socio-economic backgrounds** than would be expected from their percentages in the general population. Mental retardation was found in (6%) of **immigrants** from Mexico working in American cotton fields in Arizona. This over-representation of minority groups has been used to criticize I.Q. tests and to highlight the importance of both environmental-cultural and genetic influences on mental retardation (Sadock & Sadock, 2000).

In our study, all individuals in both groups selectively belonged to the average (**middle**) **socio-economic class** as people from higher socio-economic classes usually do not attend general hospitals, and people from lower socio-economic classes have more environmental stresses that may influence the adaptive and maladaptive behaviour of their mentally retarded children. The mean socio-economic score of the population study by Al-Shakhs Scale for socio-economic level of the family was 130.9 (average socio-economic class).

Mental ill-health during childhood and adolescence expresses itself, among other ways, as deviant behaviour, emotional problems, and delayed development. If the incidence and evolution of problems are to be assessed, it is necessary to delimit them. One way of doing this is to describe different problems individually; for example that a child who is often depressed is easily distracted by external stimuli and often has headaches. The advantage of such descriptions is that it is obvious what is being referred to. The disadvantage, however, is that it can be difficult to acquire a comprehensive overview of the great number of different problems that arise. In addition, it is often obvious that certain problems occur together. For example, a child who is distracted by external stimuli often has difficulties sitting still. Thus it serves the purpose better to try to group various problems together. Within psychological research so-called factor analysis is often used to group different characteristics. This is a statistical method, which takes into

consideration how often certain problems occur at the same time in the same children. Using this method it is possible to create classes of concepts, such as “attention deficit disorders” (APA, 1994). In this view, the individual items of Maladaptive Behaviour were grouped into four clusters; neurotic, mood, ADHD and disruptive clusters. The items were assessed as individual items and also as clusters before and after treatment with Lamotrigine.

All children of the study were given Lamotrigine, starting by a very small dose then escalating the dose gradually according to response. 10% of the patients (total 6 patients) developed early adverse effects so that they could not continue taking the drug. Other 8.3% of the patients (total 5 patients) developed worse symptoms when they started Lamotrigine therapy and had to stop the drug early also. More 25% of the patients (total 15 patients) did not show significant improvement till the end of the study. Improved cases constituted 56.7% of all studied cases (total 34 patients). This is in quite agreement with the percentage of improvement obtained by Ettinger who observed 58% improvement ratio in their studied epileptic and mentally retarded cases; (Ettinger et al, 1998) but it is nearly double the improvement ratio concluded by Huber et al, 1998 who observed that 26% of their epileptic and mentally retarded patients had positive psychotropic effects (Mikati, 2003). The mean dose given to our improved cases in both groups was 106 mg/day, while the mean dose given to all improved and non improved cases was 107 mg/day. The mean dose given to improved cases was 123mg/day for Study group patients and 88.33mg/day for Control group ones. As shown in (Table 15.a), it was observed that patients of the Study Group (with abnormal EEG changes) required higher doses than those of their Controls (with normal EEG). But comparing mean doses associated with improvement of symptoms in both groups showed no significant dose difference; whether when compared group to group at level of age and IQ or when compared at the level of gender (Z value at the level of  $P>0.05$ )

After statistical analysis of data before and after treatment with Lamotrigine drug, the following findings were concluded:

**At the level of cluster symptoms**, before treatment with LMG there was no significant difference between the Study group and the Control group in scores of maladaptive behaviour (as regards age, gender, IQ or dose), and also after treatment with LMG there was no significant difference regarding the same variables as Z was at the level of  $P>0.05$  (i.e., **both groups achieved significant improvement in maladaptive behaviour after treatment with**

LMG). There was remarkable improvement in both groups at significant levels in symptoms of the studied spectrum items of Vineland's Maladaptive Behaviour Domain especially in those symptoms belonging to Mood symptoms cluster and in Disruptive symptoms cluster.

Considering each group separately, in the *Study group* there was a significant difference between cluster-symptoms before and after treatment with LMG in the following order: **Neurotic symptoms** ( $P < 0.05$ ), **Mood symptoms** ( $P < 0.000$ ), **ADHD symptoms** ( $P < 0.000$ ), and **Disruptive behaviour symptoms** ( $P < 0.000$ ). There was also concomitant significant difference in the same cluster-symptoms in the *Control group* before and after treatment with LMG in the following order: ( $P < 0.01$ ,  $P < 0.001$ ,  $P < 0.01$ , and  $P < 0.001$ ) denoting that there was a **universal improvement in all maladaptive behaviour symptoms in both groups** but the improvement was much more significant in *Study group* subjects "who had abnormal EEG changes" than in *Control group* subjects "who had normal EEG", especially regarding improvement of mood symptoms, and ADHD symptoms.

**At the level of individual symptoms**, the symptoms that showed significant improvement after treatment by Lamotrigine were as follows:

1) **Poor attention and concentration and Lack of Consideration and Withdrawal behaviour:** Poor attention showed significant improvement in both groups with very high significance in the study group ( $Z$  at the level of  $< 0.001$ ) more than in the control group ( $Z$  at the level of  $P < 0.05$ ). This result agrees with the revised topic of Vajda Frank, (1999) that Lamotrigine does not appear to impair cognitive function and it appears to cause fewer side effects than other comparable medications, and may have a favourable effect on alertness. Also, in their separate studies on epileptic autistic patients, Udall et al 1993, Stenbom et al (1998), Genton (2000) concluded that 45% of their patients had improved alertness, ability to concentrate, interaction and reduced autistic behavior and stereotyped movements (Mikati, 2003).

In the same area, Lack of Consideration showed significant improvement but only in the study group ( $Z$  at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group, suggesting better effect of LMG on lack of consideration when there is EEG abnormality.

Withdrawal behaviour also showed high significant improvement in the control group only ( $Z$  at the level of  $P < 0.05$ ) with no recordable significant improvement in the study group may be on the same basis.

- 2) Stubbornness or Sullenness, Negativism and Defiant Behaviour:** Stubbornness showed significant improvement in both groups with very high significance in the study group (Z at the level of  $P < 0.001$ ) more than in the control group (Z at the level of  $P < 0.01$ ).

In a study by Ettinger et al, where LTG was used as add-on therapy had significant positive effects on behaviour in 7 patients with epilepsy and MR; in 4 of 7 patients (58% of total) lamotrigine may have induced very significant changes in behaviour. Positive psychotropic effects of lamotrigine included: reduction in irritability and hyperactivity, decreased lethargy and diminished perseverative speech (producing a more appropriate speech), as well as improvement in co-operation & better social engagement. In all the 4 patients, behavioural improvements were sustained at the time of latest follow up (6 months to one year) which is evidence of a genuine effect of the drug rather than a transient co-incidental change in the behaviour (Ettinger et al, 1998).

Negativism and Defiant Behaviour showed significant improvement in the study group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group.

- 3) Hyperactivity and Impulsivity:** Attention-deficit/hyperactivity disorder is one of the leading causes of academic underachievement and disruptive behaviours. Its causes are believed to be mostly genetic and/or perinatal in origin. The rates of attention-deficit/hyperactivity disorder in mental retardation are estimated to be between 9 and 18 %. ADHD is mainly characterised by the triad of poor attention, hyperactivity and impulsivity. For persons with mental retardation, the diagnosis of attention-deficit/hyperactivity disorder is qualified as being *excessive for an individual's mental age* (Sadock & Sadock, 2000).

In our results, hyperactivity showed significant improvement in both groups with equally high significance in both groups (Z at the level of  $P < 0.01$ ). This concurs with the above mentioned study of Ettinger et al, 1998 where LMG reduced hyperactivity in 58% of the studied patients.

Impulsivity showed significant improvement in the study group only (Z at the level of  $< 0.01$ ) suggesting better role for LMG in presence of EEG abnormality for impulsivity symptom.

- 4) Depressed Mood:** It showed significant improvement in the study group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the control group. But 'Mood symptoms cluster' showed significant improvement in both study and control groups. This fits well



with the recent approval of Lamotrigine as a **mood stabilizer** (Goldberg, 1997). Also in a study by Calabrese et al, (1999) LMG seemed to be equally effective as adjunctive therapy or monotherapy, and it was efficacious in reducing affective symptoms in patients presenting with treatment-refractory depressed, hypomanic, manic, and mixed phases of bipolar-I and bipolar-II disorder.

**5) Irritability, Aggressive Behaviour and Lying, Cheating or Stealing:**

Antiepileptic drugs (AEDs) have been used to reduce hyperactivity and aggression with promising results especially the extensively studied and widely used Carbamazepine & Valproic acid which have been proved effective in adults in controlling violent and aggressive behaviour (Rutecki & Gidal, 2002).

Aggressive behaviour showed high significant improvement in the study group only (Z at the level of  $<.01$ ) with no recordable significant improvement in the control group. In their study, Ettinger and co-workers observed positive psychotropic effects of lamotrigine in 58% of their studied seven epileptic patients with mental retardation. Amongst the improved symptoms were: reduction in irritability (Ettinger et al, 1998). This agrees well with our result as all Ettinger's patients were epileptic with abnormal EEG changes. Also our result is higher than that obtained by Huber et al., (1998) who observed 26% positive psychotropic effects in behaviour of 125 Rett's disorder children with mental retardation and epilepsy treated by Lamotrigine for epilepsy (Mikati, 2003). On the contrary, our result does not agree with the study of Beran and Gibson, who observed that LMG may have provoked aggressive behaviour and violence in about 47% of their intellectually handicapped patients with epilepsy, which may limit its use for such patients (Beran & Gibson, 1998).

Temper Tantrums showed significant improvement in both groups with high significance in the control group (Z at the level of  $P<0.01$ ) more than in the study group (Z at the level of  $P<0.05$ ).

Acute anxiety in children is shown by clinging and protesting behaviour, weeping, swearing and other manifestations of stress. Inevitably, acute anxiety is sometimes caused by unforeseen accidents, bereavements and other untoward events in a child's life. It is then necessary to recognize the child's own distress, which he may show in unusual ways; e.g. *soiling, wetting, stealing, ...behaviour* that is generally out of context with his usual pattern. The family needs help in helping him to come to terms with the traumatic experience of loss. Punishment

without sympathy here will reinforce the anxiety and make the behaviour worse without solving the child's problem (Price, 1982).

In our study, the item of Lying, Cheating or Stealing showed significant improvement in the control group only (Z at the level of  $P < 0.05$ ) with no recordable significant improvement in the study group.

**6) Eating disturbance:** Eating problems are much noticed in young mentally retarded children. They vary from excessive salivation, nausea, vomiting, anorexia, food fads colics, and indigestion to pica but little is known about eating disorders otherwise. Food refusal may reflect depression in a child with or without mental handicap (Holt et al, 1988). Obesity is much noticed in Down syndrome and non-hyperactive mentally retarded children (Chad et al, 1990).

In our work, eating disturbance symptoms showed significant improvement in the Control group only, (Z at the level of  $P < 0.05$ ).

**Although the total number of patients entering this study (60) was not particularly large, the magnitude of effect was quite pronounced, and the medication effect was striking. This study in 60 children with mild to moderate mental retardation (with sub-average IQ and deficit in adaptive behaviour) and with maladaptive behaviour demonstrated that:**

- LMG monotherapy was significantly effective in reducing problem behaviours in the two groups, (including mainly: Poor attention and Concentration, Stubbornness, Irritability, aggressive behaviour, Depressed mood, Hyperactivity, Impulsivity, Lying and Stealing, Negativism and social withdrawal.) in total of about 56.7% of the sum of patients in both groups. The study showed that the improvements associated with LMG in the studied group were of considerable to high clinical significance.
- LMG provided significantly greater symptom improvement in patients with EEG changes (63.3% improved in the Study Group) compared to less symptom improvement (of 50%) in the Control Group (i.e. patients with similar problem behaviours and with normal EEG) in the following symptoms: Poor attention, Stubbornness, Aggression, Impulsivity, Depressed Mood, Lack of consideration and in Negativistic Behaviour.
- LMG provided significantly greater symptom improvement in the Control Group patients (with problem behaviour and with Normal EEG) compared to less symptom improvement in the Study Group patients in the following symptoms: Irritability, Lying and Stealing, and social withdrawal.

- Both Groups (Study and Control Groups) showed equal symptom improvement in Hyperactivity symptom.
- Moreover LMG was well tolerated by the studied children throughout the 6-months of effective treatment. Earlier recommendations of the manufacturer, Glaxo-smithcline, were stressing not to give Lamotrigine for persons under age 12 years. Our conclusion about safety of Lamotrigine in children conforms to Messenheimer and co-workers who evaluated 13 studies that demonstrated efficacy of Lamotrigine in 1096 children with a variety of seizure types. Lamotrigine treatment in those clinical trials was generally given at higher initial doses and faster dose escalations than the currently recommended. The qualitative features of adverse events that occurred with lamotrigine treatment were similar for children and adults. Most adverse events associated with lamotrigine were mild to moderate in severity and did not result in discontinuation of treatment. The conclusion obtained was clear cut that Lamotrigine, an effective broad spectrum anticonvulsant, was well tolerated in children (Messenheimer et al, 2000).
- No unexpected adverse events occurred:
  - Although it has been suggested that the use of most conventional antiepileptic drugs for the treatment of conduct disorder may impair cognition, no evidence of added cognitive impairment was seen in this study. Moreover, attention and concentration showed significant improvement especially in patients with abnormal EEG.
  - Sedation was not expected in this study, the positive effects of LMG on behaviour measures were shown to be independent of sedative effects. This is important, since it has previously been postulated that the efficacy of antipsychotics for aggressive behaviour may be attributable to their sedative effect but, LMG appeared to exert other actions than sedation.
  - Weight gain may cause concern with the use of some antiepileptic drugs (e.g., Valproates) however; reports suggest that weight gain with LMG is minimal. In this study, as observed by the researcher and as reported by the caregivers of our patients, no significant weight gain was remarkable with LMG.
- Another important clinical finding was the rapid onset of therapeutic effect as reported by caregivers of 12 patients (20% of total). Significant improvements in behaviour occurred as early as 1 week after starting LMG. This onset of effect is faster than that of either carbamazepine or lithium used in treatment of conduct disorder, for which 2-6 weeks may be required for full therapeutic effect.
- The positive effects of LMG occurred at a mean average dosage of (106) mg/day at the end of the study (34 cases improved out of 60 total). The dose was

administered as 25-200 mg at a once-daily regimen for most subjects, which may help promote treatment adherence; (except for a few pts who tolerated as high doses as 250-300 mg/day).

- Furthermore, there is no need to monitor blood levels with LMG, unlike some other antiepileptic drugs (AEDs) (e.g. Carbamazepine and Valproate) or other drugs affecting mood and behaviour (e.g. lithium), which require regular monitoring of serum levels to ensure therapeutic level or to avoid life-threatening toxicity. Thus, LMG appears to have other clinical advantages over alternative drugs.
- The 6-months follow-up data, confirmed the durability of efficacy and the safety of long term administration of Lamotrigine in non-epileptic mentally retarded patients especially in case of presence of abnormal EEG changes.

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# **Summary & Conclusion**



## **Summary and Conclusion:**

Mental retardation is a universal problem accounting for at least 1% of people in different populations worldwide. Persons with mental retardation have increased risk of co-morbid psychiatric or behavioural dysfunction. Mentally retarded children frequently show one or more items of mal-adaptive behaviour that interrupt the process of their learning and rehabilitation and necessitate comprehensive medical and psychiatric attention along with family & social support. Improvement of such maladaptive behaviour can have its positive impact on mentally retarded children and also on their caregivers allowing for better achievement on either side.

There is a wide range of such maladaptive behaviour. It can be just simple and tolerable by other family members e.g. mild irritability or occasional stubbornness or might be severe and very noisy e.g. poor frustration tolerance, persistent hyperactivity, impulsivity, destructive or aggressive behaviour or disinhibited & embarrassing talks & acts.

Particularly, hyperactivity & aggression infrequently show good response to conventional anti-psychotic drugs (e.g. thioridazine or haloperidol), yet their resultant disabling adverse effects (e.g. oversedation, overweight & extrapyramidal manifestations) put beforehand limitations for their use both in adults or children.

Alternatively, Antiepileptic drugs have been used for the same purpose with promising results especially the extensively studied & widespreadly used Carbamazepine & Valproic acid which have been proved effective in adults in controlling violent & aggressive behaviour & also in stabilization of mood for manic & hypomanic states. Newer antiepileptics e.g. lamotrigine & Gabapentin, proved effective in treatment of epilepsy in developmentally disabled children, with less adverse effects esp. on cognition & behaviour. (Rutecki & Gidal, 2002).

Lamotrigine, a relatively new (1<sup>st</sup> marketed as antiepileptic in 1994), but more safe antiepileptic drug, has recently been approved also as a mood stabilizer. It also showed promising effects on modification of behaviour of persons with borderline personality disorder. (Goldberg, 1997) Furthermore, Lamotrigine recently showed favourable psychological effects when used in treatment of some mentally retarded children suffering from epilepsy. (Mikati, 2003)

This study **aimed at** evaluation of possible positive effects of lamotrigine in improving maladaptive behaviour of the mentally retarded with special concern to presence or absence of EEG changes.

This Case control study included 60 mentally retarded children with mild or moderate MR (Age range: 6-12 years and both sexes were included) who reviewed the psychiatric outpatients of Bani-Sweif Psychiatric Hospital during a 6-months period for each case. All Cases were living in Bani-Sweif city at the time of study and all were sure selected belonging to middle socio-economic class. The sample was sub-classified into 2 groups:

- ▶ **Study group:** 30 children with EEG changes.
- ▶ **Control group:** 30 children with Normal EEG.

Both groups were given regular doses of Lamotrigine, supervised by caregivers of the concerned children and checked monthly in the psychiatric outpatient.

All children of the study were thoroughly examined medically, neurologically and psychiatrically. Psychometric testing of Socio-economic status (by Al-Shakhs Scale), of intelligence (by WISC), of adaptive behaviour (by Vineland ABS), and of maladaptive behaviour (by Vineland ABS) was done for every child in both groups of our study.

**Results** showed dual improvement in both groups studied, with a general estimate of clinical improvement of 56.6% that was proved to be statistically significant. After treatment by Lamotrigine some maladaptive behavioural symptoms showed improvement in **(both groups)**. These symptoms were: poor attention and concentration, stubbornness or sullenness, hyperactivity, and temper tantrums. Other symptoms improved in **(the study group only)**, they were: aggressive behaviour, impulsivity, depressed mood, lack of consideration, and negativism and defiant behaviour. The symptoms that improved in **(the control group only)** were: Lying, Cheating or Stealing, Withdrawal behaviour and Eating difficulties.

In general, our study concluded that Lamotrigine had significantly positive psychotropic effects on maladaptive behaviour of the non-epileptic mentally retarded children and those effects were more prominent in cases having abnormal EEG.



# Recommendations



# Recommendations:

The following recommendations may be considered:

- ◆ Lamotrigine drug could be selected to modify some symptoms of maladaptive behaviour (particularly irritability, depressed mood, poor attention, stubbornness, aggression and social withdrawal) especially in presence of abnormal EEG tracing.
- ◆ In this study Lamotrigine was used as monotherapy. Future studies may address the use of Lamotrigine as add-on therapy with other drugs & also as combined therapy with behaviour therapy in patients with intellectual dys-functioning and behaviour disturbances.
- ◆ Like all anti-epileptics, Lamotrigine should be used cautiously, especially in children, whose brains are still developing. Starting with low dose for a few days or two weeks is the best regimen as recommended by the manufacturer particularly to avoid serious skin rash adverse effect.
- ◆ Because of the limited number of our population sample, further studies are needed to confirm our conclusions in a wider community of patients, including particularly rural inhabitants and other socio-economic classes. Future studies would expand our range of knowledge by addressing effects of Lamotrigine in more patients with intellectual dysfunctioning and behavioural disturbances.
- ◆ The results also suggested improvements in social competence with LMG. This observation is worthy of further research. If confirmed as a true drug effect, this finding may suggest that LMG actually enhanced prosocial adaptive behaviour and reduced maladaptive symptoms.
- ◆ Early discontinuation of Lamotrigine should be considered if serious or intolerable additional symptoms develop especially severe rash or aggressive behaviour.
- ◆ Lamotrigine, like all other drugs, should be kept out of reach of children.

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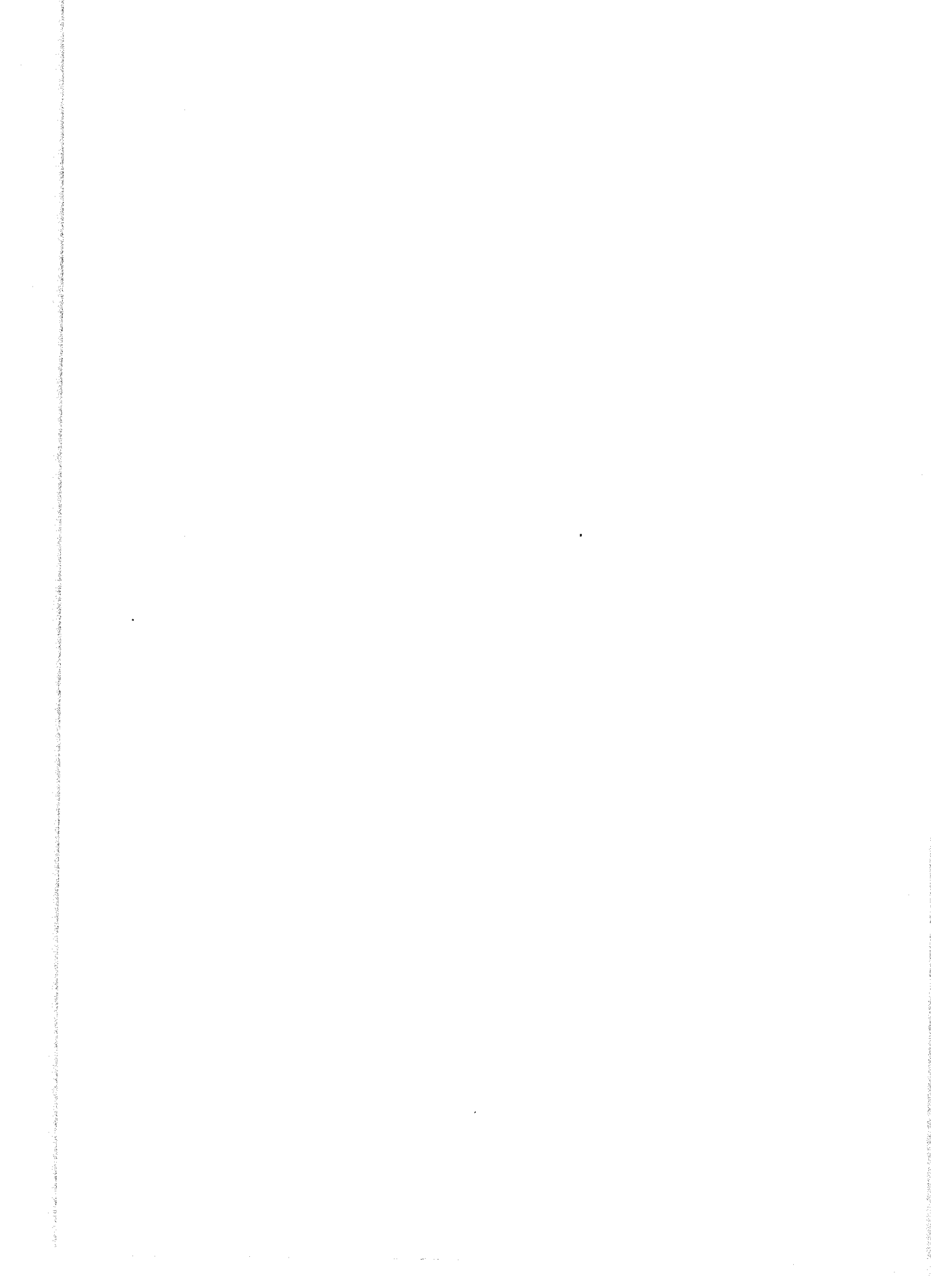
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\*\*\*\*\* تم بحمد الله وفضله \*\*\*\*\*



# Appendix



## Appendix –I: Vineland Adaptive Behaviour Scale (Vineland ABS)

### Description:

**Authors:** (Sara S. Sparrow, David A. Balla, and Dominick V. Cicchetti, 1984)

**Publisher:** American Guidance Service. (Manual + Booklets + Report to parents)  
The VABS is a revision of the Vineland Social Maturity Scale (Doll Edgar, 1953).  
(Biasini, 1998)

**Respondant:** V-ABS is an indirect assessment in that **the respondent** is not the individual in question but someone familiar with the individual's behavior.  
(Biasini, 1998)

**Purpose:** Designed to assess handicapped and non-handicapped persons in their personal and social functioning. (V-Manual, 1984)

**Population:** Birth to 18 years and low functioning adults. (V-Manual, 1984)

**Time:** (Interview Edition): (20-60) minutes (V-Manual, 1984)

**Measuring:** The VABS **measures four domains:** Communication, Daily Living Skills, Socialization, and Motor Skills. An **Adaptive Behavior Composite** is a combination of the scores from the four domains. A **Maladaptive Behavior domain** is also available with two of the three forms of administration. Each of the domains and the Composite has a mean of 100 and a standard deviation of 15. Three types of administration are available: the Survey Form (297 items), the Expanded Form (577 items, 297 of which are from the Survey Form), and the Classroom Edition (244 items for children age 3-13). (Biasini, 1998)

**Scoring:** Standard score -equivalents for domain raw scores and Adaptive Behavior Composite (V-Manual, 1984)

**Reliability:** Split-half and test-retest reliability coefficients for the Composite scores are good, ranging from median values of .83 for the Motor Skills domain to .94 for the Composite. Inter-rater coefficients are lower for the same measures: .62 to .78. When broken down by sub-domains, the coefficients fluctuate a great deal and some are quite low. (V-Manual, 1984)

**Validity:** Selected standardization subgroups were compared on the original Vineland, the ABIC, the K-ABC, the PPVT-R, and the VABS. These concurrent measures exhibited low to moderate correlations, with generally higher coefficients obtained when the comparisons were made on subjects with handicapping conditions. (V-Manual, 1984)

Appendix –II: Vineland Adaptive Behaviour Scale, Survey  
Form, 5 domains (including the Maladaptive one)

العمر زمنيا	مقياس فاينلاند للسلوك التوافقي (١) مجال التواصل		البنود
	السلوك الاستقبالي والفهم	السلوك التعبيري	
صفر			١ يرفع عينيه ويتجه براسه نحو الصوت
--			٢ يستمع ولو للحظات لمن يقوم برعايته
١			٣ يبتسم حينما يرى من يقوم برعايته
سنة			٤ يبتسم لشخص آخر مألوف لديه غير من يقوم برعايته [وليس الاغراب]
			٥ يرفع ذراعيه حينما يقول له من يقوم برعايته "إنهض" أو "تعال"
			٦ يظهر فهمه لكلمة "لا" [مثال (كف عن هذا النشاط)]
			٧ يقلد أصوات الأشخاص المحيطين به بعد الاستماع لهم
			٨ يظهر فهمه لبعض الكلمات (على الأقل ١٠ كلمات)
			٩ يستطيع أن يعبر بالإشارة أو الأيماء عن معنى كلمة "نعم" "لا" "أنا أريد"
< ١			١٠ ينصت بانتباه الى التعليمات [ إذا كان غير منصت يعطى صفر ]
-- ٢			١١ يظهر فهمه لمعنى كلمة "نعم" أو "وهو كذلك" [ إذا لم تقال له هذه الكلمات قبلا ]
			١٢ ينفذ تعليمات بسيطة تتضمن فعل
			١٣ يمكنه التعرف على جزء رئيسي من أجزاء جسمه
			١٤ ينطق باسم أخوته ، أصدقائه ، وأقرانه [لا يشترط أن يكون النطق سليما]
			١٥ ينطق بعض الجمل المكونة من (كلمتين) [لا يشترط أن يكون النطق سليما]
			١٦ يمكنه تسمية بعض الأشياء من حوله (٢٠ على الأقل) - لا تسجل (١)
			١٧ يمكنه الاستماع لقصة ما لمدة (٥) دقائق
			١٨ يعبر عن تفضيله لشيء ما إذا كان أمامه خيار
			١٩ ينطق عدد مناسب من الكلمات (على الأقل ٥٠) - لا تسجل (١)
+ ٢			٢٠ يمكنه أن يسرد بعض الأحداث الخاصة به بعبارة بسيطة
			٢١ يمكنه أن ينقل شفهيًا رسالة بسيطة
			٢٢ ينطق بجمل تتكون من (٤) كلمات أو أكثر
			٢٣ يشير بدقة إلى معظم أجزاء جسمه إذا سئل - لا تسجل (١)
			٢٤ ينطق عدد كبير من الكلمات (على الأقل ١٠٠ كلمة) - لا تسجل (١)
			٢٥ يتحدث في شكل (جمل تلمة)
			٢٦ يستخدم في الجمل التي ينطق بها أداة التعريف (ال)
			٢٧ يتبع التعليمات الشرطية (مثل: إذا..... ، سوف يحدث كذا...)
			٢٨ يذكر اسمه الأول و الأخير إذا سئل
			٢٩ يسأل أسئلة تبدأ ب: (متى ، ماذا ، كيف ، من ؟) - لا تسجل (١)
٣+ ٤			٣٠ يمكنه أن يقرر أن الشجرة < الوردية
			٣١ يذكر بعض الأحداث الشخصية بالتفصيل إذا سئل
			٣٢ يستخدم في الجمل التي ينطق بها كلمات: (وراء ، بين)

	٣٣	يستخدم في الجمل التي ينطق بها كلمة (حول)	
	٣٤	يستخدم في الجمل التي ينطق بها كلمات مثل: (لكن ، أو)	
	٣٥	ينطق بوضوح دون إبدال لبعض الأصوات	
	٣٦	يمكنه أن يحكى : قصة ، نكتة ، أو عرض شاهده في التلفزيون	
٥	٣٧	يمكنه استرجاع كل الحروف الأبجدية من الذاكرة [كتابة]	
	٣٨	يقرا على الأقل ٣ علامات عادية [؟ ، - ، + ، × ، ÷ ؟ علامات إعراب؟]	
	٣٩	يعرف يوم و شهر ميلاده إذا سئل	
	٤٠	يمكنه جمع الكلمات الشاذة لغويا	
	٤١	يكتب اسمه الأول و الأخير	٦
	٤٢	يعرف رقم تليفونه إذا سئل	
	٤٣	يعرف عنوان منزله بالكامل	
	٤٤	يقرا على الأقل ١٠ كلمات (بصوت عال أو منخفض)	
	٤٥	يكتب على الأقل ١٠ كلمات من الذاكرة	
	٤٦	يعبر عن أفكاره بأكثر من طريقة و بدون مساعدة	
	٤٧	يقرا قصص قصيرة بصوت مرتفع	
	٤٨	يكتب جملا بسيطة تتكون من ٢-٤ كلمات	٨+٧
	٤٩	ينتبه إلى الدرس لأكثر من ١٥ دقيقة	
	٥٠	يحب أن يقرأ من تلقاء نفسه	
	٥١	يمكنه قراءة كتب الصف الثاني الابتدائي	
	٥٢	يمكنه ترتيب بعض الكلمات حسب الأولوية الأبجدية	
	٥٣	يمكنه كتابة رسائل بسيطة	
	٥٤	يقول: يمين ، شمال بنفسه [يعطى الآخرين اتجاهات محددة]	+٩
	٥٥	يكتب البدايات الأولى للخطابات - لا تسجل (١)	
	٥٦	يمكنه قراءة كتب الصف الرابع الابتدائي أو في مستواها	
	٥٧	يكتب بخط رقعة معظم الوقت	
	٥٨	يستخدم القاموس - لا تسجل (١)	١٠-
	٥٩	يرجع إلى الفهرس حين يقرأ أى كتاب	١٨
	٦٠	يكتب تقارير أو موضوعات إنشائية - لا تسجل (١)	سنة
	٦١	يكتب العناوين الكاملة على الخطابات	
	٦٢	يستخدم الفهرس عند قراءة الكتب	
	٦٣	يقرا بعض الجرائد الخاصة بالراشدين	
	٦٤	لديه اهداف واقعية على المدى الطويل	
	٦٥	يكتب خطابات بشكل جيد	
	٦٦	يقرا اسبوعيا القصص التي تنشر في المجلات	
	٦٧	يكتب خطابات إدارية - لا تسجل (١)	
		مجموع درجات ١ و ٢ =	
		عدد الإجابات الحاصلة على ل، ع	
		عدد الإجابات الحاصلة على ل، ف	
		د.خ. للمجال النوعي = المجموع الكلي للدرجات الخام لكل مجال نوعي =	
		د.خ. للمجال العام = مجموع د.خ. للمجالات النوعية المرتبطة به =	

## مقياس فاينلاند للسلوك التوافقي (٢) مجال مهارات الحياة اليومية

المستوى الشخصي	المستوى الأسري	المستوى المجتمعي والجماعي	العمر	البنود
			١ >	١ يظهر توقعه للطعام عند رؤيته للطعام أو للبرازة أو لثدي الأم
				٢ يفتح فمه عندما تعرض عليه ملعقة بها طعام
				٣ يأخذ الطعام من الملعقة لفته [ يقرب فمه من الملعقة ويدخلها فيه ]
				٤ يمص البسكويت أو يمضغه
				٥ يأكل الطعام [ السائل- اللين ] بالملعقة
			١	٦ يشرب من الكوب أو الفئجان بدون مساعدة
				٧ يطعم نفسه بالملعقة
				٨ يظهر فهمه بأن الأشياء الساخنة خطيرة و محظورة
				٩ يهتمهم ببعض الأصوات أو يشير بيده إذا تبول على نفسه
				١٠ يستخدم الماصة عند الشرب
				١١ يسمح للقائم برعايته أن يسمح له أنفه
				١٢ يطعم نفسه بالشوكة
				١٣ يفتح الأزرار الأمامية للقميص أو الجاكيت أو البالطو بدون مساعدة
			٢	١٤ يطعم نفسه بالملعقة بدون دلق
				١٥ يظهر رغبته في تغيير ملابسه إذا كانت مبلولة أو متسخة
				١٦ يتبول في الحمام أو القصرية
				١٧ يستحم بنفسه بمساعدة الآخرين
				١٨ يتبرز في الحمام أو القصرية
				١٩ يطلب استخدام الحمام أو التواليت
				٢٠ يخلع و يلبس الملابس البسيطة (مثل بنطلون وسطه باسلك)
				٢١ يظهر فهمه لمعنى النقود
				٢٢ يضع حاجاته الخاصة جانبا عندما يطلب منه ذلك
			٣	٢٣ يدخل التواليت أثناء الليل
				٢٤ يشرب من الحنفية بدون مساعدة
				٢٥ ينظف أسنانه بدون مساعدة
				٢٦ يظهر فهمه لوظيفة الساعة (سواء تقليدية أو رقمية)
				٢٧ يساعد باقصى ما عنده (عندما يسأل)
				٢٨ يغسل و يجفف و جهه بدون مساعدة
				٢٩ يلبس الحذاء في القدم الصحيحة بدون مساعدة
				٣٠ يرد على التليفون بطريقة مناسبة
			٤	٣١ يلبس نفسه جيدا ماعدا ربط الحذاء
				٣٢ يخبر من على التليفون أن الشخص المطلوب غير موجود
				٣٣ يرتب المائدة بمساعدة
				٣٤ يعنى بكل احتياجاته للتواليت بمفرده أو بدون أن يذكره أحد بذلك - لا تسجل (١)
				٣٥ ينظر إلى اليمين أو الشمال قبل تعدية الشارع
				٣٦ يرتب ملابسه النظيفة بدون مساعدة (عندما يسأل)
				٣٧ ينظف أنفه بدون مساعدة
				٣٨ يرجع الأشياء من على المائدة بعد الأكل
				٣٩ يجفف نفسه بالفوطة بدون مساعدة
				٤٠ يربط حزامه

٥	٤١	يساعد في تحضير درجات الطعام التي تتطلب الطهي
	٤٢	يظهر فهمه إلى ان أخذ طعام أو نقود من الغرباء غير مأمون
	٤٣	يربط الحذاء بدون مساعدة
	٤٤	يستحم بدون مساعدة - لا تسجل (١)
	٤٥	ينظر إلى اليمين و اليسار و يمكنه ان يعدى الشارع
	٤٦	يغلق فمه و أنفه عند الكحة أو العطس
	٤٧	يستخدم الملعقة و الشوكة و السكين بكفاءة - لا تسجل (١)
	٤٨	ياخذ زمام المبادرة في الاتصال بالآخرين هاتفيا
	٤٩	يحترم إشارة المرور
	٥٠	يلبس نفسه تماما بما في ذلك ربط الحذاء أو أى شيء آخر - لا تسجل (١)
	٥١	يرتب سريره إذا طلب منه ذلك
	٥٢	يذكر اليوم الحالي من أيام الأسبوع (عندما يُسأل)
	٥٣	أثناء الطلوع و النزول من الباص ينط فيه أو منه و الباص ماشى
	٥٤	يدرك القيم المختلفة للنقود (٥ قروش ، ١٠ قروش ، ربع جنيه ، جنيه)
	٥٥	يستخدم أدوات أو عدد بسيطة
	٥٦	يحدد اليمين و الشمال بالنسبة له و للآخرين
	٥٧	يرتب المائدة بدون مساعدة ( عندما يُسأل)
	٥٨	يكنس و ينظف الأرض بكفاءة (عندما يُسأل)
	٥٩	يتصل بالإسعاف تليفونيا عند الضرورة
	٦٠	يطلب وجباته من الطعام كاملة إذا وجد في المطعم
	٦١	يذكر تاريخ اليوم ( عندما يُسأل)
	٦٢	يغير ملابسه مع تغيير الجو دون أن يذكره أحد
	٦٣	يبعد عن الأشخاص المعديين (المرض) دون أن يذكره أحد
	٦٤	يذكر الوقت بإضافة وحدة من الساعة (٥ق)
٩-	٦٥	يعتني بشعره بدون أن يذكره أحد [ودون مساعدة] - لا تسجل (١)
١٠	٦٦	يستخدم البوتجاز عند الطهي
	٦٧	يستخدم المنظفات عند الغسيل بطريقة سليمة
	٦٨	بعد النقود المتبقية بعد الشراء بطريقة صحيحة (جنيه فاكتر)
	٦٩	يستخدم التليفون بدون مساعدة [كافة أنواع الاتصالات الهاتفية]
	٧٠	يعتني بنظافة أظفاره دون أن يذكره أحد وبدون مساعدة - لا تسجل (١)
	٧١	بعد أصناف الطعام التي تتطلب الطهي بدون مساعدة
	٧٢	يستخدم التليفون المدفوع الأجر
	٧٣	يرتب حجرته دون أن يذكره أحد
	٧٤	يوفر و يشتري بند شيء واحد على الأقل يريد شراءه
	٧٥	يعتني بصحته ويمكنه قياس درجة حرارته و أخذ العلاج المناسب
	٧٦	يكسب نقوده و يصرفها على أساس سليم
	٧٧	يرتب سريره و يغير الملاء بصورة منتظمة - لا تسجل (١)
	٧٨	ينظف أماكن أخرى في المنزل غير حجرته دون أن يُسأل
	٧٩	يصلح أو يصون أدوات المنزل دون أن يُسأل
	٨٠	يخيط/تخيط الزراير والكبسونات و الكيشات في الملابس حينما يُسأل
١٧-	٨١	يضع ميزانية خاصة بمصروف الأسبوع
١٨	٨٢	ينظم و يدير نقوده دون مساعدة
	٨٣	يخطط و يجهز لوجبات اليوم دون مساعدة
	٨٤	يصل إلى عمله في الوقت المحدد
	٨٥	يعتني بكل ما يخص ملابسه دون أن يذكره أحد

	يخير مديره في العمل إذا تعذر أن يصل في الموعد المقرر	٨٦
	يخير مديره في العمل إذا تغيب بسبب المرض	٨٧
	ينظم ميزانية الشهر	٨٨
	يخطط/تخطيط ذيل البنطلون أو القستان دون أن يُسال- دون مساعدة	٨٩
	يحترم مواعيد العمل و الراحة	٩٠
	يتحمل مسئولية العمل	٩١
	لديه حساب في البنك و يستخدمه بكفاءة	٩٢
	مجموع درجات ١ و ٢ =	
	عدد الإجابات الحاصلة على ل، ع	
	عدد الإجابات الحاصلة على ل، ف	
	د.خ. للمجال النوعي = المجموع الكلي للدرجات الخام لكل مجال نوعي =	
	د.خ. للمجال العام = (مجموع د.خ. للمجالات النوعية المرتبطة به) =	



## مقياس فاينلانند للسلوك التوافقي (٣) مجال التنشئة الاجتماعية

مهارات التوافق	أوقات اللعب و الفراغ	العلاقات الشخصية والاجتماعية	العمر	
			البنود	
١			١	ينظر إلى وجه من يقوم برعايته
			٢	يستجيب إلى صوت من يقوم برعايته أو إلى أى شخص
			٣	يميز بين من يقوم برعايته و بين الآخرين
			٤	يظهر اهتمامه بالأشخاص الجدد أو بالأشياء الجديدة
			٥	يظهر بعض الانفعالات الواضحة (مثل الفرح ، الخوف ، الحزن)
			٦	يرفع ذراعيه عندما يطلب منه الشخص القائم برعايته ذلك
			٧	يستجيب وجدانيا للأشخاص المألوفين لديه
			٨	يظهر اهتمامه بالأطفال الآخرين غير أخوته
			٩	يزحف حتى يصل إلى شخص مألوف لديه
			١٠	يلعب بمفرده أو مع الآخرين بلعبه أو بأى شيء
			١١	يلعب ألعاب جماعية بسيطة جدا
			١٢	يستخدم أدوات المطبخ فى اللعب
			١٣	يظهر اهتمامه بما يفعله الآخرون من نشاط أو لعب
			١٤	يقلد حركات الآخرين مثل التصفيق أو يلوح بيده حينما يخرج
٢-١			١٥	يبتسم و يضحك عندما يثاب على شيء
			١٦	يذكر أسماء أشخاص مألوفين لديه ( اسمين على الأقل)
			١٧	يظهر رغبته فى إسعاد من يقوم برعايته
			١٨	يشارك الآخرين فى نشاط ما أو فى لعبة ما
			١٩	يقلد بعض الأعمال التى يشاهدها بعد فترة
			٢٠	يقلد بعض العبارات التى يسمعاها من الكبار فى مواقف معينة
٣			٢١	يشارك فى نشاطات أو ألعاب من وحي خياله بمفرده أو مع آخرين
			٢٢	يفضل بعض أصدقاء عن أصدقاء آخرين
			٢٣	يقول من فضلك حينما يطلب شيئا ما
			٢٤	يعبر عن نفسه بعبارات: السعادة ، الخوف ، الحزن ، الغضب
٤			٢٥	يُعرف الناس بخصائصهم و ليس بأسمائهم حينما يسأل
			٢٦	يشارك الآخرين فى لعبه و وممتلكاته دون أن يخبره أحد
			٢٧	يسمى و يتذكر عرضا شاهده فى تى.فى. و يحدد يوم المشاهدة و القناة
			٢٨	يتبع التعليمات الخاصة بالألعاب التى يلعب بها دون أن يذكره أحد
٥			٢٩	له صديق من أحد الجنسين
			٣٠	لا يخالف أوامر و تعليمات المدرسة
			٣١	يستجيب بصورة إيجابية و لفظية للأحداث السعيدة للآخرين
			٣٢	يعتذر عن أخطائه غير المقصودة
			٣٣	لديه مجموعة من الأصدقاء
			٣٤	لا يخالف أوامر الجماعة التى يعيش فيها
			٣٥	يلعب أكثر من لعبة تتطلب مهارات خاصة (مثل اتخاذ قرار: مثلما فى الكوتشينة)
			٣٦	لا يتحدث و الطعام فى فمه
٦			٣٧	لديه صديق مفضل من نفس الجنس
			٣٨	يعامل الغرباء بصورة لائقة

٣٩	٨-٧	يبادر بشراء هدية أو كارت لأى عضو فى الأسرة ، وذلك فى أى مناسبة
٤٠		يحافظ على الأسرار لأكثر من يوم
٤١		يعيد الكتب المستعارة للمكتبة ، و يعيد أى لعب أو نقود استلفها إلى صاحبها
٤٢		يختتم محادثاته بصورة لبقة
٤٣	٩	يحترم الوقت المسموح به للعب أو للأكل [أو لزيارة الآخرين]
٤٤		يتحاشى قول أى عبارات أو أسئلة تجرح شعور الآخرين
٤٥		يتحكم فى مشاعر الغضب أو الأذى و يحاول إنكارها أو إخفاءها بطريقة الخاصة
٤٦		يحافظ على الأسرار أطول فترة ممكنة
٤٧	-١٠	يحافظ على آداب المائدة دون أن يذكره أحد ... لا تسجل (١)
٤٨	١١	يشاهد ال تى فى. أو يستمع للراديو بغرض سماع خبر أو برنامج يريده أو يرغبه
٤٩		يذهب إلى مدرسة ليلية أو إلى حفلة مع الأصدقاء بمصاحبة أحد من الراشدين
٥٠		يزن بنفسه النتائج المترتبة على أفعال معينة ، وذلك قبل أن يقوم بها
٥١		يعتذر عن أخطائه فى الحكم على شىء ما أو شخص ما
٥٢	-١٢	يتذكر أعياد ميلاد أو أعياد زواج أصدقائه القريبين أو أقربائه فى الأسرة
٥٣	١٥	يبادر بالحديث فى موضوعات خاصة تهم الآخرين
٥٤		لديه هواية
٥٥		يعيد النقود التى استلفها ممن يقوم برعايته
٥٦	-١٦	أثناء الحديث يلمح المعانى وراء الكلمات و يستجيب لها
٥٧	١٨	يشترك فى الألعاب و الأنشطة الرياضية خارج المدرسة
٥٨		يتابع نشرات الأخبار اليومية فى الإذاعة و التلفزيون
٥٩		يحافظ على المواعيد (مثل تمرين رياضى أو درس موسيقى)
٦٠		يعتمد على نفسه فى متابعة برامج الراديو أو التلفزيون
٦١		يذهب إلى حفلات الأصدقاء دون مصاحبة شخص كبير معه
٦٢		يذهب إلى السينما أو الأحداث الرياضية دون مصاحبة شخص كبير معه
٦٣		يشترك فى نادى اجتماعى
٦٤		يرافق أصدقاءه من الجنس الآخر إلى مكان عام أو لحدث هام فى حضور جمهور
٦٥		يمكنه عمل أكثر من لقاء ناجح مع أصدقائه
٦٦		يحرص على مراعاة التقاليد و القيم فى تعامله مع الجنس الآخر
		مجموع درجات ١ و ٢ =
		عدد الإجابات الحاصلة على ل،ع
		عدد الإجابات الحاصلة على ل، ف
		د.خ. للمجال النوعى = المجموع الكلى للدرجات الخام لكل مجال نوعى =
		د.خ. للمجال العام = (مجموع د.خ. للمجالات النوعية المرتبطة به) =

		مقياس فاينلاند للسلوك التوافقي (٤) مجال المهارات الحركية	
دقيقة	كبيرة	البنود	العمر
		١ يحفظ رأسه منتصباً (١٥ ثانية) عندما يحمله الشخص الراعى فى وضع عامودى	١ >
		٢ يجلس بمساعدة لمدة (١ ق) على الأقل	
		٣ يلتقط أشياء صغيرة بيديه بأى طريقة	
		٤ ينقل الأشياء من يد لأخرى	
		٥ يلتقط الشيء الصغير بالإبهام والأصبع	
		٦ يرفع نفسه لوضع الجلوس و يحافظ على وضعه لمدة (١ق) على الأقل بمفرده	
		٧ يزحف على الأوض بيديه و ركبته دون أن تلمس بطنه الأرض	
		٨ يفتح الأبواب التى يتطلب فتحها الدفع أو الجذب	
		٩ يدحرج الكرة أثناء الجلوس	١
		١٠ يسير بصورة مبدئية كأنه يدور حول نفسه	
		١١ يمكنه تسلق (أو الهبوط من) سرير أو مقعد من مقاعد الراشدين	
		١٢ يتسلق لعبة ارتفاعها بسيط (حصان)	
		١٣ يرسم أى خط بالقلم الرصاص أو الطباشير	
		١٤ يصعد السلالم واضعاً كلتا قدميه على كل سلمة	٢
		١٥ ينزل السلالم واضعاً كلتا قدميه على كل سلمة	
		١٦ يجرى فى جميع الاتجاهات أحياناً بسرعة و أحياناً ببطء	
		١٧ يفتح الأبواب بشد الأكرة أو بتدويرها	
		١٨ يقز على شيء صغير	
		١٩ يمكنه فتح غطاء علبة بتدوير غطائها	
		٢٠ يقود عجلة بثلاث عجلات لمدة قصيرة	
		٢١ ينط على رجل واحدة ممسكاً بشخص آخر أو بشيء ثابت دون أن يقع	
		٢٢ يبنى نماذج ذات ثلاثة أبعاد باستخدام (٥) الأقل مكعبات عل	
		٢٣ يفتح و يقفل المقص باستخدام يد واحدة	
		٢٤ ينزل السلم واضعاً رجلاً بعد أخرى بدون مساعدة	٦-٣
		٢٥ يصعد على أى لعبة ارتفاعها عالى (مرجحة)	
		٢٦ يستطيع قص ورق بالمقص	
		٢٧ ينط على رجل واحدة ٣ مرات على الأقل بدون مساعدة - لا تسجل (١)	
		٢٨ يمكنه تجميع شكل بسيط يتكون من ٦ أجزاء منفصلة	
		٢٩ يرسم أكثر من شكل بسيط باستخدام القلم الرصاص أو ألوان الشمع	
		٣٠ يستطيع قص ورق على شكل خط واحد مستقيم	
		٣١ يستخدم המחاة بدون أن يمزق الورق	
		٣٢ ينط على قدم واحدة بسهولة - لا تسجل (١)	
		٣٣ يفتح القفل بمفتاح	
		٣٤ يمكنه قص أشكال أكثر صعوبة بالمقص	
		٣٥ يمكنه الإمساك بكرة القيت من على بعد ٣م حتى لو اضطر أن يجرى ليمسكها	
		٣٦ يركب دراجة بعجلتين دون أن يقع	
		مجموع درجات ١ و ٢ =	
		عدد الإجابات الحاصلة على ل، ع	
		عدد الإجابات الحاصلة على ل، ف	
		د.خ. للمجال =	
		د.خ. للمجال النوعى = المجموع الكلى للدرجات الخام لكل مجال نوعى =	

٦	أحياتا	٧	مقياس فاينلاندا للسلوك التوافقي (٥) مجال السلوك غير التوافقي (الجزء الأول)	
			البند	
			١	يمص إبهامه أو أصابعه.
			٢	اعتمادى جدا (يعتمد تماما على الآخرين).
			٣	انسحابى (يميل إلى العزلة).
			٤	يبال فرشه أثناء النوم.
			٥	لديه اضطراب فى تناول الطعام.
			٦	لديه اضطراب فى النوم.
			٧	يقضم أظافره.
			٨	يتحاشى الذهاب إلى المدرسة (أو العمل).
			٩	يلاحظ عليه قلق شديد.
			١٠	يظهر لزمات (خلجات).
			١١	يبكى أو يضحك بسهولة جدا.
			١٢	يتحاشى النظر للآخرين (لديه قصور فى التقاء العينين المباشر).
			١٣	تبدو عليه تعاسة زائدة (يفرط فى الحزن).
			١٤	يطحن أسنانه نهارا أو ليلا.
			١٥	نزوى جدا (يميل للاندفاعية الشديدة).
			١٦	ضعيف الانتباه و التركيز.
			١٧	لديه نشاط حركى زائد (كثير الحركة).
			١٨	لديه نوبات صراخ (مقلب المزاج ، حاد الطبع ، نوبات غضب).
			١٩	خلفى أو متحدى (يزدرى الآخرين و يحقر منهم).
			٢٠	يشاكس ، يشاغب (يعاكس الآخرين) أو يضارب ، مستبد.
			٢١	يظهر عدم اعتبار للآخرين (منعدم الشعور بالمسئولية) ، سطحى.
			٢٢	يكذب ، يخدع أو يسرق.
			٢٣	عدوانى جسديا جدا.
			٢٤	يحلف كثيرا بدون داع (باحقيقته فيما يملك) وفى مواقف غير ملائمة
			٢٥	يهرب من المسكن ، يجرى بعيدا بغير هدى.
			٢٦	عنيد و مشاكس أو متجهم (مقطب الجبين) .
			٢٧	يزوغ (يتهرب) من المدرسة أو العمل.
			مجموع درجات ١ و ٢ =	
			مجموع الدرجات الخام (الجزء الأول)	
			(Non-significant – Intermediate – Significant)	
			النتيجة:	

الدرجة الكيفية	مقياس فاينلاند للسلوك التوافقي مجال السلوك غير التوافقي (الجزء الثاني)	
	البنود	
شديد	متوسط	
		يصدر سلوكيات جنسية شاذة وغير ملائمة. ٢٨
		يصدر سلوكيات غريبة (أو زائدة) عند مسكه للأشياء أو أدائه للأنشطة. ٢٩
		يعبر عن أفكار غير مقبولة. ٣٠
		يقوم بعادات أو نمطيات غريبة و مميزة . ٣١
		يصدر سلوكيات مؤذية للذات. ٣٢
		يقوم عمدا بتخريب ممتلكاته أو ممتلكات الغير. ٣٣
		يستخدم حديثا محيرا (كلامه شاذ و غريب). ٣٤
		غير واع بما يجرى فى بيئته المباشرة. ٣٥
		يهز نفسه للأمام و الخلف عندما يكون جالسا أو واقفا. ٣٦
		مجموع درجات ١ و ٢ =
		مجموع الدرجات الخام (الجزء الثاني)
		مجموع الدرجات الخام (الجزء الأول و الثاني)
		Non-significant – Intermediate – Significant النتيجة:

Vineland ABS		Scores		
Domain of: <b>Maladaptive Behaviour,</b> Part-I (Modified into clusters)				
Clusters	Items	Yes = 2	Sometimes = 1	No = 0
Neurotic symptoms	1. Sucks thumb or fingers			
	2. Bites fingernails			
	3. Grinds teeth day or night			
	4. Eating disturbance			
	5. Sleep disturbance			
	6. Wets bed (Nocturnal Enuresis)			
	7. Tics			
Mood symptoms	8. Temper tantrums			
	9. Exhibits extreme anxiety			
	10. Exhibits excess unhappiness			
	11. Cries or laughs too easily			
	12. Shows lack of consideration			
ADHD cluster	13. Has poor attention			
	14. Overly active			
	15. Too impulsive			
Disruptive symptoms	16. Bullies or teases			
	17. Too physically aggressive			
	18. Stubborn or sullen			
	19. Negativistic or defiant			
	20. Swears inappropriately			
	21. Lies, cheats, or steals			
	22. Overly dependent			
	23. Withdraws			
	24. Avoids school or work			
	25. Truant from school or work			
	26. Runs away			
	27. Has poor eye contact			
	<b>Sum of answers with 1 &amp; 2 scores</b>			
	<b>Total raw score of the domain</b>			

## Appendix –III.A: Vineland ABS: Results Scoring Summary

مقياس فاينلاند للسلوك التوافقي Vineland Adaptive Behaviour Scale						
تاريخ الاختبار:						
اسم الطفل:						
تاريخ الميلاد:						
السنة الدراسية:						
محول من:						
الفاحص:						
	العمر المكافئ	مستوى التوافق	الدرجة المعيارية	الدرجة الخام	المجالات النوعية	المجالات العامة
	B-10,11	B -6,8	B -1,2			Vin. Table No
Receptive					السلوك الاستقبالي	التواصل
Expressive					السلوك التعبيري	
Written					الكتابة و القراءة	
					الدرجة الكلية للمجال	مهارات الحياة اليومية
Personal					السلوك الشخصي	
Domestic					السلوك الأسري	
Community					الجماعة والمجتمع	التنشئة الاجتماعية
					الدرجة الكلية للمجال	
Inter-personal Relationship					العلاقات الشخصية والاجتماعية	
Play & Leisure time					أوقات اللعب والفراغ	Socialization Domain
Coping skills					مهارات التوافق	
					الدرجة الكلية للمجال	
Gross					كبيرة	المهارات الحركية
Fine					دقيقة	
					الدرجة الكلية للمجال	
						مجموع الدرجات الكلية للمجالات العامة الدرجة الكلية للسلوك التوافقي
المجموعة الطبيعية المساندة	المستوى غير التوافقي	الدرجات الخام Raw scores (Table-B-12)				مجال السلوك غير التوافقي (اختياري)
				الجزء الأول		
				الجزء الثاني		

**Appendix –III.B:**

**Vineland ABS – Scoring (Age Equivalent & SQ)**

**Name:**

**Date:**

**DOB:**

**Chronological Age (CA):**    y    m

**(1) Age Equivalent:**

	Domains (Main)	Raw Score	Domain Age-Equivalent (Table B-6)		Sum of all (Age-Equivalents) for all domains (3or4)	
			Year	Month	in years / Number of domains	in months (= Q for example)
1	<b>Communication</b>					
2	<b>Daily living skills</b>					
3	<b>Socialization</b>					
4	<b>Motor skills</b>					
<b>Result =</b>						
Age-Equivalent = (in years)		= (Q) ÷ (12) (month of the year) =    y    m				

**(2) Scoring Social Quotient (SQ):**

$$SQ = \text{Age Equivalent} \times 100 \div \text{Chronological Age} =$$



## Appendix IV: Age Equivalent scores (Vineland's)

### Age Equivalent scores: (Study group)

S N	Raw Scores (Domains)			Age Equivalent (for Domains)				Age Equivalent		Mental Age		SQ	IQ
	Com	DLS	Socz	Com	DLS	Socz	Emons	Y	m	Y	m		
1.	75	80	48	4.2	4.2	2.3	127 / 3	3	6	4	11	31	44
2.	69	52	66	3.8	2.8	4.0	124	3	5	4	5	31	40
3.	81	95	74	4.9	5.2	4.10	182	5	0	6	10	50	68
4.	65	52	72	3.3	2.8	4.7	126	3	6	4	2	30	38
5.	59	44	54	2.10	2.3	2.9	99	2	9	4	9	49	66
6.	59	69	62	2.10	3.6	3.6	118	3	3	4	9	30	44
7.	69	78	61	3.8	4.1	3.0	129	3	7	4	4	58	70
8.	80	73	56	4.8	3.9	2.11	127	3	6	5	3	39	58
9.	82	86	69	4.10	4.8	4.4	166	4	7	6	6	46	65
10.	91	95	63	5.10	5.2	3.8	176	4	10	6	8	42	58
11.	68	64	59	3.7	3.3	3.2	120	3	4	3	6	38	40
12.	71	61	57	3.10	3.1	3.0	126	3	6	4	7	38	50
13.	87	79	53	5.5	4.1	2.8	146	4	0	6	1	46	70
14.	81	73	42	4.9	3.9	1.9	123	3	5	5	8	30	49
15.	65	56	47	3.3	2.10	2.2	99	2	9	4	5	24	39
16.	38	36	34	1.8	1.11	1.3	58	1	7	2	9	25	46
17.	78	80	60	4.6	4.2	3.3	143	3	11	6	0	44	67
18.	94	102	68	6.2	5.8	4.2	192	5	4	7	0	52	69
19.	62	43	64	3.1	2.3	3.9	109	3	0	3	9	31	39
20.	77	96	67	4.4	5.3	4.1	164	4	6	5	8	41	55
21.	70	39	60	3.9	2.0	3.3	108	3	0	4	5	44	65
22.	88	110	69	5.6	6.2	4.4	192	5	4	6	6	42	60
23.	72	68	48	3.11	3.5	2.3	115	3	2	5	3	28	47
24.	58	37	50	2.9	1.11	2.5	76	2	1	3	6	21	35
25.	42	40	47	1.10	2.1	2.2	78	2	2	2	8	36	44
26.	101	112	72	6.11	6.4	4.7	214	5	11	7	5	56	70
27.	71	64	56	3.10	3.3	2.11	120	3	4	4	8	33	46
28.	70	74	37	3.9	3.10	1.6	109	3	0	6	6	27	59
29.	86	78	66	5.3	4.1	4.0	160	4	5	6	3	50	70
30.	46	59	43	2.1	3.0	1.10	73	2	0	3	10	21	40

### Age Equivalent scores: (Control group)

S N	Raw Scores (Domains)			Age Equivalent (for domains)				Age Equivalent		Mental Age		SQ	IQ
	Com	DLS	Socz	Com	DLS	Socz	Emons	Y	m	Y	m		
1.	74	70	48	4.1	3.7	2.9	119 / 3	3	3	4	5	51	70
2.	90	89	67	5.8	4.10	4.1	165	4	7	6	2	50	68
3.	39	28	54	1.8	1.7	2.9	72	2	0	2	5	33	40
4.	66	43	62	3.4	2.3	3.6	109	3	0	3	7	38	45
5.	55	54	41	2.7	2.9	1.9	85	2	4	3	5	24	35
6.	68	66	59	3.7	3.4	3.2	121	3	4	4	0	50	60
7.	77	110	74	4.4	6.2	4.10	174	4	4	5	9	38	50
8.	81	114	56	4.9	6.7	2.11	161	4	10	5	11	43	52
9.	63	58	49	3.2	2.11	2.4	91	2	5	4	0	24	40
10.	93	96	64	6.0	5.3	3.9	180	5	6	6	2	62	70
11.	88	78	57	5.6	4.1	3.0	151	4	2	6	6	35	55
12.	86	106	71	5.3	5.11	4.6	188	5	2	6	2	59	70
13.	53	50	62	2.5	2.7	3.6	102	2	10	3	9	35	47
14.	48	78	53	2.2	4.1	2.8	107	2	0	5	10	22	65
15.	45	63	54	2.0	3.2	2.9	95	2	7	3	5	42	55
16.	53	33	62	2.5	1.6	3.6	89	2	5	3	10	25	40
17.	83	96	59	5.0	5.3	3.2	161	4	5	6	8	44	66
18.	72	62	56	3.11	3.2	2.11	120	3	4	4	0	56	48
19.	72	44	50	3.11	2.3	2.5	103	2	10	4	4	26	39
20.	76	92	70	4.3	5.0	4.5	164	4	6	6	11	45	70
21.	41	45	53	1.9	2.4	2.8	81	2	3	2	11	27	35
22.	90	77	62	5.8	4.0	3.6	158	4	4	7	3	38	63
23.	73	71	58	4.0	3.8	3.1	129	3	7	5	4	41	60
24.	81	75	50	4.9	4.11	2.5	145	4	0	5	7	50	70
25.	62	78	49	3.1	4.1	2.4	114	3	2	4	0	29	36
26.	48	54	54	2.2	2.9	2.9	92	2	6	3	1	28	35
27.	90	62	69	5.8	3.2	4.4	158	4	4	6	1	49	68
28.	69	53	52	3.8	2.8	2.7	107	2	11	4	11	27	45
29.	64	65	46	3.2	3.3	2.1	102	2	10	3	8	36	46
30.	86	69	64	5.3	3.6	3.9	150	4	2	6	6	38	59

## Appendix –V: Adaptive Behaviour (Raw scores)

### Adaptive Behaviour: Vineland's Raw Scores: (Study Group)

	Communication Skills				Daily Living Skills				Socialization Skills			
	Receptive	Expressive	Written	Sum	Personal	Domestic	Community	Sum	Inter- Personal	Play & leisure time	Coping	Sum
31.	24	48	3	75	60	9	11	80	36	9	3	48
32.	22	45	2	69	30	11	11	52	35	21	10	66
33.	24	54	3	81	69	14	12	95	38	25	11	74
34.	24	39	2	65	36	8	8	52	40	23	9	72
35.	23	32	4	59	32	3	9	44	33	18	3	54
36.	24	34	1	59	58	3	8	69	34	20	8	62
37.	24	45	2	69	57	7	14	78	36	20	5	61
38.	24	46	10	80	58	6	9	73	36	18	2	56
39.	26	50	6	82	59	12	15	86	40	24	5	69
40.	24	59	8	91	70	10	15	95	37	25	1	63
41.	24	42	2	68	52	3	9	64	34	22	3	59
42.	24	44	3	71	46	5	10	61	37	18	2	57
43.	24	52	11	87	64	1	14	79	36	17	0	53
44.	24	47	10	81	62	2	9	73	26	16	0	42
45.	24	40	1	65	47	1	8	56	31	16	0	47
46.	18	20	0	38	31	0	5	36	30	4	0	34
47.	22	50	6	78	64	0	16	80	39	20	1	60
48.	24	58	12	94	71	11	20	102	40	22	6	68
49.	22	38	2	62	33	3	7	43	41	18	5	64
50.	24	50	3	77	70	6	20	96	39	26	2	67
51.	24	44	2	70	31	1	7	39	38	21	1	60
52.	26	52	10	88	75	10	25	110	40	25	4	69
53.	26	44	2	72	43	7	18	68	32	14	2	48
54.	22	36	0	58	28	2	7	37	27	22	1	50
55.	21	21	0	42	32	1	7	40	28	18	1	47
56.	26	57	18	101	74	9	29	112	42	27	3	72
57.	24	45	2	71	51	2	11	64	34	20	2	56
58.	24	40	6	70	58	3	10	74	27	9	1	37
59.	24	53	9	86	62	3	13	78	40	23	3	66
60.	20	24	2	46	51	0	8	59	26	13	4	43

### Adaptive Behaviour: Vineland's Raw Scores: (Control Group)

	Communication Skills Domain				Daily Living Skills Domain				Socialization Skills Domain			
	Receptive	Expressive	Written	Sum	Personal	Domestic	Community	Sum	Inter-Personal	Play & leisure time	Coping	Sum
1.	23	46	2	70	60	3	11	74	30	16	2	48
2.	24	56	10	90	68	2	19	89	40	24	3	67
3.	16	23	0	39	20	1	7	28	36	18	0	54
4.	24	42	0	66	36	1	6	43	38	19	5	62
5.	22	33	0	55	44	2	8	54	24	14	3	41
6.	23	44	1	68	53	3	10	66	38	17	4	59
7.	24	50	3	77	70	20	20	110	42	20	12	74
8.	24	54	3	81	72	20	22	114	34	18	4	56
9.	21	40	2	63	46	1	11	58	30	19	0	49
10.	26	54	13	93	73	2	21	96	40	24	0	64
11.	26	55	7	88	65	1	12	78	36	20	1	57
12.	24	47	15	86	71	20	15	106	42	20	9	71
13.	18	34	1	53	40	3	7	50	40	19	3	62
14.	24	22	2	48	64	4	10	78	34	18	1	53
15.	21	24	0	45	44	9	15	63	34	16	4	54
16.	21	32	0	53	23	1	9	33	40	20	2	62
17.	21	53	9	83	71	4	21	96	33	21	5	59
18.	24	48	0	72	47	8	7	62	34	20	2	56
19.	24	45	3	72	30	4	10	44	24	20	6	50
20.	22	52	2	76	63	12	17	92	40	22	8	70
21.	20	21	0	41	39	2	4	45	36	16	1	53
22.	24	54	12	90	59	6	12	77	39	19	4	62
23.	22	48	3	73	60	1	10	71	37	20	1	58
24.	24	45	12	81	54	4	17	75	32	17	1	50
25.	24	34	4	62	62	8	8	78	32	16	1	49
26.	20	28	0	48	47	1	6	54	36	16	2	54
27.	26	55	9	90	50	2	10	62	40	26	3	69
28.	22	44	3	69	46	1	6	53	32	20	0	52
29.	22	42	0	64	48	9	8	65	30	16	0	46
30.	24	50	12	86	52	7	10	69	36	21	7	46

## Appendix –VI: Adaptive Behaviour Level (Standard)

**Adaptive Behaviour Level: Vineland’s Standard Scores: (Study Group)**

S N	Raw Scores (Domains)			STANDARD SCORES				Adaptive Behaviour Composite (st.scores)	Adaptive Behaviour Level
	Communication	Daily Living Skills	Socialization	Communication	Daily Living Skills	Socialization	SUM		
61.	75	80	48	40	22	43	105	32	LOW
62.	69	52	66	38	<20	52	110	34	LOW
63.	81	95	74	48	46	60	154	47	LOW
64.	65	52	72	34	<20	54	108	33	LOW
65.	59	44	54	50	31	58	139	43	LOW
66.	59	69	62	34	<20	51	105	32	LOW
67.	69	78	61	64	64	61	189	58	LOW
68.	80	73	56	51	33	52	136	42	LOW
69.	82	86	69	48	37	56	141	43	LOW
70.	91	95	63	49	37	50	136	42	LOW
71.	68	64	59	46	27	55	128	39	LOW
72.	71	61	57	46	20	53	119	37	LOW
73.	87	79	53	60	45	53	158	49	LOW
74.	81	73	42	43	<20	38	101	31	LOW
75.	65	56	47	34	<20	41	95	29	LOW
76.	38	36	34	45	34	51	130	40	LOW
77.	78	80	60	51	43	105	199	61	LOW
78.	94	102	68	57	53	55	165	51	LOW
79.	62	43	64	39	<20	54	113	35	LOW
80.	77	96	67	41	40	52	133	41	LOW
81.	70	39	60	60	30	65	155	48	LOW
82.	88	110	69	49	56	54	159	49	LOW
83.	72	68	48	38	<20	43	101	31	LOW
84.	58	37	50	38	<20	49	107	33	LOW
85.	42	40	47	48	41	58	147	46	LOW
86.	101	112	72	63	59	56	178	54	LOW
87.	71	64	56	42	<20	50	112	34	LOW
88.	70	74	37	37	<20	33	90	27	LOW
89.	86	78	66	57	41	60	158	49	LOW
90.	46	59	43	34	<20	47	101	31	LOW

### Adaptive Behaviour Level: Vineland's Standard Scores: (Control Group)

S N	Raw Scores (Domains)			STANDARD SCORES (Domains)				Adaptive Behaviour Composite (st.scores)	Adaptive Level
	Communication	Daily Living Skills	Socialization	Communication	Daily Living Skills	Socialization	SUM		
31.	74	70	48	65	62	57	184	56	LOW
32.	90	89	67	59	49	59	167	51	LOW
33.	39	28	54	47	30	64	141	43	LOW
34.	66	43	62	48	<20	60	128	39	LOW
35.	55	54	41	36	<20	45	101	31	LOW
36.	68	66	59	61	55	65	181	55	LOW
37.	77	110	74	41	53	54	148	46	LOW
38.	81	114	56	43	58	48	149	46	LOW
39.	63	58	49	38	<20	47	105	32	LOW
40.	93	96	64	65	57	58	180	55	LOW
41.	88	78	57	48	<20	46	114	35	LOW
42.	86	106	71	57	64	64	185	57	LOW
43.	53	50	62	42	22	60	124	38	LOW
44.	48	78	53	37	41	52	130	40	LOW
45.	45	63	54	49	57	64	170	52	LOW
46.	53	33	62	36	<20	54	110	34	LOW
47.	83	96	59	49	47	51	147	46	LOW
48.	72	62	56	53	37	56	146	45	LOW
49.	72	44	50	38	<20	44	102	31	LOW
50.	76	92	70	46	46	58	150	46	LOW
51.	41	45	53	36	<20	53	109	33	LOW
52.	90	77	62	49	<20	49	118	36	LOW
53.	73	71	58	48	34	54	136	42	LOW
54.	81	75	50	58	46	53	157	48	LOW
55.	62	78	49	33	20	44	97	30	LOW
56.	48	54	54	37	<20	52	109	33	LOW
57.	90	62	69	61	25	62	148	46	LOW
58.	69	53	52	38	<20	47	105	32	LOW
59.	64	65	46	49	40	52	141	43	LOW
60.	86	69	64	46	<20	51	117	36	LOW

## Appendix –VII: Maladaptive behaviour: Vineland’s Scores

**Maladaptive behaviour Vineland’s Scores: (Study Group)**

S.N	Before LMG		After LMG	
	Items	Score	Items	Score
1.	7	11	D/C	
2.	7	12	4	5
3.	9	13	4	5
4.	8	12	3	5
5.	7	10	WORSE	
6.	8	12	4	5
7.	7	12	3	4
8.	7	11	4	6
9.	8	13	5	5
10.	6	10	*SAME	10
11.	7	12	4	4
12.	7	12	*SAME	12
13.	11	20	D/C	
14.	9	14	4	5
15.	11	19	*SAME	19
16.	8	14	4	5
17.	7	12	4	5
18.	9	13	*SAME	13
19.	6	11	4	5
20.	8	13	*SAME	13
21.	6	10	3	4
22.	7	10	4	5
23.	7	11	4	5
24.	7	11	*SAME	11
25.	7	12	4	5
26.	7	13	3	5
27.	8	11	4	4
28.	10	17	4	5
29.	8	13	*SAME	13
30.	7	13	D/C	
Total				

**Maladaptive behaviour Vineland's Scores: (Control Group)**

S.N	Before LMG		After LMG	
	Items	Score	Items	Score
1.	7	10	2	4
2.	8	12	*SAME	12
3.	7	11	WORSE	
4.	7	12	2	3
5.	7	11	*SAME	11
6.	7	12	3	5
7.	8	12	D/C	
8.	8	12	4	4
9.	9	11	WORSE	
10.	8	11	4	6
11.	6	10	*SAME	10
12.	7	11	4	4
13.	9	15	5	6
14.	10	17	4	5
15.	9	15	4	5
16.	9	13	*SAME	13
17.	8	12	*SAME	12
18.	6	11	3	4
19.	7	11	WORSE	
20.	7	10	*SAME	10
21.	8	13	4	5
22.	7	12	*SAME	12
23.	7	10	D/C	
24.	8	11	4	5
25.	10	19	3	4
26.	9	13	D/C	
27.	7	11	4	5
28.	13	23	*SAME	23
29.	10	12	WORSE	
30.	8	12	4	5
<b>Total</b>				



**Appendix-VIII: Al-Shakhs Scale, Socioeconomic level**

**(1) Al-Shakhs Scale: 1<sup>st</sup> Parameter, Occupation of Parents:**

<b>Level</b>	<b>Group Label</b>	<b>Occupation of Fathers (examples)</b>	<b>House wives</b>
<b>First</b>	Unskilled workers	1.Un-skilled labourers in agriculture or industry. 2.Manual workers 3.Door keepers, Porters 4.Sellers 5.Policemen (caporal)	Non-educated house wives
<b>Second</b>	Skilled workers	1.Barbers 2.Tailors 3.Drivers 4.Medical orderlies 5.Policemen (sergent)	House wives with middle qualification (like technical school graduate or its level)
<b>Third</b>	Simple Jobs	1.Primary school teachers 2.Nurses 3.Cashiers 4.Train drivers 5.Small employees (secretaries) 6.Small merchants e.g. (grocers, fruitiers, Café or restaurant owner 7.Storekeepers	House wives with level of education above middle and below university.
<b>Forth</b>	Technical Jobs	1.assistant engineers 2.mechanics 3.electricians 4.Painters 5.teachers of prep. Stage 6.old govt. employees with old middle qualification 7.Headmasters of primary schools	House wives with university grade
<b>Fifth</b>	University-graduate jobs	1. Assistant lecturers at university 2. Teachers of secondary schools 3. University graduated govt. employees 4. medium-rank officers (leftnant or captain) 5. High nurses	

<b>Sixth</b>	Smart Jobs	<ol style="list-style-type: none"> <li>1. Doctors, Engineers, Lawyers, Directors at govt. units ,Senior teachers at secondary stage</li> <li>2. Big Officers (major, colonel)</li> <li>3. Big merchants (furniture, carpets, electronics) - Jewellers - Contractors -Brokers</li> </ol>
<b>Seventh</b>	High jobs	<ol style="list-style-type: none"> <li>1. Lecturers at university</li> <li>2. General directors at govt. units or companies, Managers at banks</li> <li>3. Consultants in companies or corporations</li> </ol>
<b>Eighth level</b>	Higher Jobs	<ol style="list-style-type: none"> <li>1. Assistant professors at university</li> <li>2. Diplomats</li> <li>3. Chief justice</li> <li>4. High-rank officers at army or police.</li> <li>5. Consultants in governmental administrations</li> </ol>
<b>Ninth level</b>	Uppermost Jobs	<ol style="list-style-type: none"> <li>1. Heads of universities, Dean of faculties, Head of faculty section, Professors at university.</li> <li>2. Heads of councils of companies</li> <li>3. Ministers and their proxies</li> <li>4. Chief senior attorneys, Court consultants,</li> <li>5. Ambassadors</li> <li>6. Governors</li> </ol>

The scale classified the level of occupation of parents into (9) levels

**(2) Al-Shakhs Scale: 2<sup>nd</sup> Parameter, Educational Level of Parents:**

	<b>Level of Education of Parents</b>
1.	Illiterate or not qualified.
2.	Primary school.
3.	Preparatory school.
4.	Secondary school and its levels
5.	Certificate above secondary school and below university.
6.	University graduate.
7.	Post graduate studies (Master degree)
8.	Doctorate degree

The scale classified the level of education of parents into (8) levels

### Al-Shakhs Scale: Suggested distribution of income

الدخول المقترحة						كما جاء في المقياس		
و	هـ	د	ج	ب	ا	دخول الفرد	الدرجة	الفئة
٤٩-٠٠	٤٤-٠٠	٣٩-٠٠	٣٤-٠٠	٢٩-٠٠	٢٤-٠٠	١٩-٠٠	١	منخفض جدا
٩٩-٥٠	٨٩-٤٥	٧٩-٤٠	٦٩-٣٥	٥٩-٣٠	٤٩-٢٥	٣٩-٢٠	٢	منخفض
-١٠٠	-٩٠	-٨٠	-٧٠	٨٩-٦٠	٧٤-٥٠	٥٩-٤٠	٣	دون متوسط
١٤٩	١٣٤	١١٩	١٠٤					
-١٥٠	-١٣٥	-١٢٠	-١٠٥	-٩٠	٩٩-٧٥	٧٩-٦٠	٤	متوسط
١٩٩	١٧٩	١٥٩	١٣٩	١١٩				
-٢٠٠	-١٨٠	-١٦٠	-١٤٠	-١٢٠	-١٠٠	٩٩-٨٠	٥	فوق متوسط
٢٤٩	٢٢٤	١٩٩	١٧٤	١٤٩	١٢٤			
-٢٥٠	-٢٢٥	-٢٠٠	-١٧٥	-١٥٠	-١٢٥	-١٠٠	٦	مرتفع
٢٩٩	٢٥٩	٢٣٩	٢٠٩	١٧٩	١٤٩	١١٩		
٣٠٠	٢٦٠	٢٤٠	٢١٠	١٨٠	١٥٠	١٢٠	٧	مرتفع جدا
	فاكثر	فاكثر	فاكثر	فاكثر	فاكثر	فاكثر		
١	...	٧	١	١	...			عدد الأفراد المستشارين

#### Suggested distribution of income

ملحوظة:

- \* جاء في المقياس أن متوسط دخل الفرد في الشهر يمتد من ١٠ جنيهات إلى ٢٠٠٠ جنيه.
- \* اتفقت آراء معظم الأشخاص المستشارين على أن متوسط دخل الفرد في الشهر حاليا (وقت بدء الدراسة في يوليو ٢٠٠٣) يمتد من ٣٠ جنيه إلى ٣٠٠٠ جنيه.
- \* متوسط دخل الفرد في الشهر بالنسبة لحضر الوجه القبلي (بالجنيه) حسب آخر دراسة لمركز التعبئة والإحصاء سنة ٢٠٠٠م = ٢١١٧٥ جنيه مصرى

### (3) Al-Shakhs Scale: 3<sup>rd</sup> Parameter, Income per Capita per Month:

Level	Income/capita/month (Egyptian pounds)
1.	Below 40
2.	40-79
3.	80-119
4.	120-159
5.	160-199
6.	200-239
7.	Above 240

The scale classified the income/capita/month into (7) levels

#### (4) Al-Shakhs Scale:

#### Calculation of the level of Socio-economic Status:

The level of the socioeconomic status (X) is calculated by the following equation:

$$X = F \{(A) + (B_1 S_1) + (B_2 S_2) + (B_3 S_3) + (B_4 S_4)\}$$

Where:

X = the socioeconomic level to be calculated

A = fixed number

B = variables

F = Constant Factor = 10

The fixed numbers "A" and "B" variables are calculated by using the **Dummy variable method** where:-

$$A = 2.259$$

$$B_1 = 1.016$$

$$B_2 = 0.886$$

$$B_3 = 0.622$$

$$B_4 = 0.013$$

And:

(S1) = score of **income per capita per month**

(S2) = score of **work of father**

(S3) = score of **education of father**

(S4) = score of **work of mother**

So, the equation becomes:

$$X = 10 X \{2.259 + 1.016 (S_1) + 0.886 (S_2) + 0.622 (S_3) + 0.013 (S_4)\}$$

N.B: It was noticed that the level of education of the mother (S4) was neglected by the author of the scale as it gave negligent or zero values during analysis of data; hence, it was excluded from the equation.

So, the final equation became as follows:

**Level of socioeconomic status =**

$$X = 10 X \{2.259 + 1.016 (S_1) + 0.886 (S_2) + 0.622 (S_3)\}$$

**Al-Skakhs Scale: Level of socio-economic status**

<b>Level of socio-economic status</b>	<b>Score</b>
Very low	48-72
Low	73-96
Below average	97 -120
<b>Average (= middle class)</b>	<b>121-144</b>
Above average	145-168
High	169-192
Very high	193-216

## Al-Shakhs Scale: matching socio-economic level

### Study Group: Socio-economic level

Pt SN	1 <sup>st</sup> Parameter	2 <sup>nd</sup> Parameter	3 <sup>rd</sup> Parameter	X = { A + B1S1 + B2S2 + B3S3 } X 10 =	Socio- economic Level
	Score of Father's Occupation	Score of Father's Education	Score of Income / Capita / Month.		
1.	5	4	4		
2.	5	3	4	134	Average
3.	3	6	6	124	Average
4.	7	3	3	144	Average
5.	5	3	4	140	Average
6.	5	3	4	125	Average
7.	5	5	4	125	Average
8.	5	3	6	143	Average
9.	5	3	4	137	Average
10.	5	3	4	125	Average
10.	5	4	3	127	Average
11.	7	2	3	130	Average
12.	4	3	6	127	Average
13.	4	3	5	121	Average
14.	5	3	4	125	Average
15.	5	4	3	137	Average
16.	4	4	4	124	Average
17.	5	3	4	125	Average
18.	7	3	2	133	Average
19.	5	4	4	134	Average
20.	7	3	3	139	Average
21.	4	3	5	130	Average
22.	5	4	5	140	Average
23.	6	2	6	139	Average
24.	6	2	4	126	Average
25.	5	6	2	139	Average
26.	5	4	4	129	Average
27.	3	5	3	135	Average
28.	6	3	3	129	Average
29.	4	5	4	132	Average
30.	5	3	4	125	Average

### Control Group: Socio-economic level

Pt SN	1 <sup>st</sup> Parameter	2 <sup>nd</sup> Parameter	3 <sup>rd</sup> Parameter	$X = \{ A + B1S1 + B2S2 + B3S3 \}$ $X 10 =$	Socio-economic Level
	Score of Father's Occupation	Score of Father's Education	Score of Income / Capita / Month.		
1.	6	4	3	130	Average
2.	6	2	4	126	Average
3.	5	3	4	125	Average
4.	6	2	4	126	Average
5.	4	4	6	136	Average
6.	6	3	4	135	Average
7.	6	3	4	135	Average
8.	5	4	4	134	Average
9.	5	3	5	131	Average
10.	5	2	6	128	Average
11.	5	3	4	125	Average
12.	4	4	4	123	Average
13.	4	3	6	127	Average
14.	4	5	5	138	Average
15.	5	6	2	139	Average
16.	3	5	6	135	Average
17.	5	3	4	125	Average
18.	4	4	4	124	Average
19.	4	3	6	127	Average
20.	3	4	6	121	Average
21.	5	4	3	128	Average
22.	5	3	4	125	Average
23.	5	6	2	139	Average
24.	5	3	4	125	Average
25.	6	6	1	143	Average
26.	4	4	4	124	Average
27.	6	2	4	127	Average
28.	5	6	2	139	Average
29.	3	5	6	135	Average
30.	4	4	6	136	Average

## Appendix –IX: Patient’s First Interview Form

### Patient’s First Interview Form

**Patient #:**

**Name:**

**DOB:** / /

**Address & Tel:**

**Father’s Job:**

**Father’s Educational level:**

**Date:** / /

**Sex:** M / F

**Age:** y m

#### (1) HISTORY:

##### (1) Perinatal H:

- |   |        |          |                    |
|---|--------|----------|--------------------|
| <input type="checkbox"/> <b>Labour Process:</b>   | Normal | Assisted | Operated by CS     |
| <input type="checkbox"/> <b>Age at labour:</b>    | FT     | Preterm  | Post-term          |
| <input type="checkbox"/> <b>Status at labour:</b> | Fair   | LBW      | Incubated for: ( ) |

##### (2) Post-natal H:

- **Early Feeding:** Breast-fed      Bottle-fed      Passive feeding
- **Physical Milestones:** Teething: ( )      Sitting: ( )      Walking: ( )
- **Psychological m/s:** Social smile ( )      Mother recognition ( )  
Talking: ( )      Toilet control: ( )

##### (3) Current Difficulties (C/O) in the following areas:-

- ▶ **Feeding:** Refusal of food      Food-fads      Hyperphagia      PICA
- ▶ **Sleep:** Insomnia      Night mares/terrors      Sleep-walking/talking
- ▶ **Speech:** Lispings      Slurring      Stuttering/Stammering      Echolalia
- ▶ **Oral Behaviour:** Thumb/fingers      Sucking      Teeth grinding      Nail biting
- ▶ **Sphincteric control:** NE / DE -      Encoporesis
- ▶ **Emotional Behaviour:** Anxious/Fearful/phobic      Depressed/Tearful  
Irritability/T.Tantrums      Emotional Lability
- ▶ **Attention & Memory:** Poor attention/Distractible/Forgetful
- ▶ **Motor Activity:** Hyperactive      Inert      Impulsive
- ▶ **Social Behaviour:** (Disruptive Behaviour)
  - Aggressive: Abusive/Bullies/Intimidates      Bites/Beats/Fights
  - Destructive/Vandalistic / Sets fires
  - Stubborn / Sullen
  - Defiant/Negativistic
  - Indifferent/Irresponsible/Lack of consideration
  - Swearing excessively (swears in inappropriate situations)
  - Lying/Cheating/Stealing
  - Dependent overly



- Withdrawn
  - Tics / Stereotypy
  - Self Injurious Behaviour
  - Other: (Disinhibited / Sexually inappropriate behaviour, runs away,..)
- **Family H/O MR:**      Yes              No
- **Consanguinity:**      P+ve              N-ve

**[2] School History:**

- Present Educational Status: Nil    Normal (Class: )    Special (Class: )
- Failure in Classes:
- Avoids going to school:    Yes    No
- Truancy from school:      Yes    No

**[3] Examination:**

❑ **General Appearance:**

- Built:            Average    Stunted    Obese
- Retarded facies or features: Nil    Yes ( )

❑ **General Demeanour:**

- **Ability for Direct Eye Contact:** Yes    Avoids DEC
- **Attention:**            Poor attention    Fair
- **Physical activity:**    Hyperactive    Average    Inert    Impulsive
- **Co-operability:**      Co-operative    No

❑ **Recognition of own age & sex :** Yes    No

❑ **Recognition of familiar food:**    Yes    No

❑ **Recognition of familiar animals:** Domestic (Y / N)    Wild (Y / N)

❑ **Recognition of colours:** Nil    1    2    3    4    5

❑ **Calculation till 10:**      Nil    (1&2)    (1-5)    (1-10)

❑ **Writing own name:**      Nil    First Name    Full Name

❑ **Writing numbers:**      Nil    (1&2)    (1-5)    (1-10)

❑ **Reading ability:**            Nil    Yes

❑ **Copying geometric figures:**

- Nil or makes lines Only
- Simple figures:    Yes    No
- Complex ones:    Yes    No

**IQ:**

**EEG:**

**Diagnosis: MR:            Mild            Moderat**

## Appendix -X: Consent Form

### إقرار

اسم الطفل: \_\_\_\_\_

تاريخ الميلاد: / /

العنوان:

أقر أنا: \_\_\_\_\_

ولّى أمر الطالب المذكور أعلاه ، بأننى موافق على إعطائه يوميا دواء لاموتريجين ( المعروف تجاريا باسم : لامكتال ) فى حدود الجرعات العلاجية المناسبة و لمدة ستة أشهر بانتظام وذلك حسب تعليمات الطبيب المعالج / د. محسن على عبد العال أخصائى الطب النفسى ، و الذى أوضح لى الفوائد المتوقعة و كذلك الأعراض الجانبية المحتملة للعلاج المذكور ، وهذا إقرار منى بذلك.

المقر بما فيه:

بطاقة:

التوقيع:

م / / تحريرافى:

## Appendix -XI:

# LAMOTRIGINE PRESCRIBING INFORMATION

### LAMICTAL (Lamotrigine)

Useful pharmaceutical information about Lamotrigine  
(Glaxo-Smithline Pamphlet)

**Prescribing Information** (Please refer to the full data sheet before prescribing)

**Presentation:** Pale yellow tablets containing 25 mg, 50 mg, 100 mg and 200 mg lamotrigine and white dispersible/chewable tablets containing 5 mg, 25 mg and 100 mg lamotrigine.

#### Uses:

**Monotherapy:** *Monotherapy in children 12 years and younger is not recommended.* Adults and children over 12 years for partial epilepsy with or without secondarily generalized tonic-clonic seizures and in primary generalised tonic clonic seizures. **Add-on therapy:** Adults and children over 2 years: for partial epilepsy with or without secondarily generalized tonic-clonic seizures and in primary generalized tonic-clonic seizures. Lamictal is also indicated for the treatment of seizures associated with the Lennox-Gastaut syndrome.

**Dosage and administration:** The initial dose and subsequent dose escalator are a maximum and should not be exceeded to minimize the risk of rash.

**Monotherapy:** Adults and Children over 12 years: The initial dose in monotherapy is 25 mg daily for two weeks followed by 50 mg daily for two weeks. Thereafter the dose should be increased by a maximum of 50- 100 mg every 1 -2 weeks until optimal response is achieved. **The usual maintenance dose is 100-200 mg/day** given once a day or in two divided doses.

**Add-on therapy:** Adults and Children over 12 years: In patients taking sodium valproate with or without ANY other antiepileptic drug (AED) the initial lamictal dose is 25 mg every alternate day for two weeks followed by 25 mg/day for two weeks. Thereafter the dose should be increased by a maximum of 25-50 mg every 1-2 weeks until optimal response is achieved. **The usual maintenance dose is 100 to 200 mg/day** given once a day or in two divided doses.

For patients taking enzyme-inducing AEDs with or without other AEDs (but NOT valproate) the initial lamictal dose is 50 mg daily for two weeks followed by 100 mg/day in two divided doses for two weeks. Thereafter the dose should be increased by a maximum of 100 mg every 1-2 weeks until optimal response is achieved. **The usual maintenance dose is 200 to 400 mg/day** given in two divided doses.

**Children aged 2- 12 years:** *Children should be dosed on a "mg/kg" basis until the adult recommended titration dose is reached.* For patients taking sodium valproate with or without ANY other AED, the initial dose 0.2 mg/kg bodyweight/day given once a day for two weeks, followed by 0.5 mg/kg/day given once a day for two weeks. Thereafter the dose should be increased by 0.5-1 mg/kg every 1-2 weeks until optimal response s

achieved. **The usual maintenance dose is 1-5 mg/kg/ day** given once a day or in two divided doses. If the calculated dose is 2.5-5 mg/day, then 5 mg may be taken on alternate days for the first two weeks. *If less than 2.5 mg/day, lamictal should not be administered.* For patients taking enzyme-inducing AEDs with or without other AEDs (but NOT valproate), the initial dose is 2 mg/kg bodyweight/day given in two divided doses for two weeks, followed by 5 mg/kg/day for two weeks given in two divided doses. Thereafter, **the dose should be increased by a maximum of 2-3 mg/kg every 1-2 weeks** until optimal response is achieved. **The usual maintenance dose is 5-15 mg/kg/day** given in two divided doses. The weight of the child should be monitored and the dose adjusted as appropriate during maintenance therapy.

**Use in the elderly:** While there is no evidence to suggest that the elderly respond differently to the young, elderly patients should be treated cautiously.

**Contra-indications:** Hypersensitivity to lamotrigine and significant hepatic impairment.

**Precautions:** Adverse Skin reactions have been reported and generally occur during the first 8 weeks of treatment. The majority are mild and self limiting. However rarely serious, potentially life-threatening rashes including Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) have been reported. All patients who develop rash should be promptly evaluated and lamotrigine withdrawn unless the rash is clearly not drug related. High initial dose, exceeding the initial recommended dose, and concomitant use of sodium valproate have been associated with increased risk of rash. Patients who acutely develop symptoms suggestive of hypersensitivity such as rash, fever, lymphadenopathy, facial oedema, blood and liver abnormalities, flu-like symptoms, drowsiness or worsening seizure control, should be evaluated immediately and lamictal discontinued if an alternative etiology can not be established.

**Concomitant AED therapy:** Avoid abrupt withdrawal except for safety reasons.

**Pregnancy and lactation:** lamictal was not carcinogenic, mutagenic or shown to impair fertility in animal studies. While volunteer studies with lamictal have shown no effect on co-ordinations or reaction time, the individual response to AEDs should be considered with respect to driving.

**Interactions:** AEDs which alter drug metabolising enzymes in the liver ( eg Phenytoin, carbamazepine, phenobarbitone, primidone, sodium valproate) alter the metabolism and pharmacokinetics of lamictal (see Dosage and administration). This is also important during AED withdrawal.

**Side and Adverse Effects:**

With monotherapy: headache, tiredness, rash, nausea, dizziness, drowsiness, and insomnia. In addition with add-on-therapy: Diplopia, blurred vision, conjunctivitis, unsteadiness, GIT-disturbances (Including vomiting), irritability/aggression, agitation, tremors, , confusion and haematological abnormalities. Severe skin reactions including angioedema, Stevens-Johnson syndrome and toxic epidermal necrolysis have occurred

(see precautions). Rarely hepatic dysfunction, lymphadeopathy, leucopenia, thrombocytopenia have been reported in conjunction with skin rash (see precautions)  
Lamictal is a trade mark

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For further information:

GlaxoSmithline

Near East-Regional Office,

P.O.Box: 26756, NICOSIA 1647, Cyprus

NE/LAM/08/NOV/01



# ***Arabic Summary***





# ملخص عربي

## تقويم الآثار الإيجابية لعقار لاموترجين على السلوك التوافقي المضطرب للأطفال المتخلفين عقليا غير الصرعيين

هناك نسبة معتبرة من الأشخاص الذين يعانون من التخلف العقلي تصدر عنهم سلوكيات غير متسقة (بنسبة عامة تصل إلى الثلث تقريبا)، تؤثر سلبا على أدايمهم و على عمليات إعادة تأهيلهم ، لذا تحتاج إلى رعاية طبية نفسية خاصة بالإضافة طبعا إلى الدعم الأسرى و الاجتماعي.

هناك تشكيلة واسعة من أعراض هذا السلوك غير التوافقي ، و الذي يمكن أن يكون بسيطا و ممكن تحمله من جانب باقى أفراد الأسرة (مثل نوبات عصبية بسيطة و فترات قليلة من العنادية) كما يمكن أن يكون شديدا و مزعجا جدا (مثل فرط النشاط الحركى، سلوك عدواني أو تخريبى ، أو أحاديث وأفعال فاضحة و محرجة).

أظهرت بعض أعراض السلوك غير التوافقي (مثل فرط النشاط الحركى و السلوك العدوانى) استجابة جيدة للأدوية المضادة للذهان (مثل هالوبيريديول و ثيوريدازين) إلا أن الآثار الجانبية الناتجة عنهم (مثل زيادة الوزن و اضطرابات الجهاز العصبى الجار هرمى) وضعت قيودا مسبقة لاستعمالهم ، خصوصا فى الأطفال.

عوضا عن ذلك ، استعملت الأدوية المضادة للصرع لنفس الغرض و أعطت نتائج واعدة ، و أثبت بعضها فعالية فى ضبط السلوك العنيف و العدوانى و أيضا فى استقرار الاضطرابات الهوسية فى البالغين. هذا وقد أثبتت بعض الأدوية الحديثة المضادة للصرع (مثل عقار لاموترجين وجابانتين) فعالية مماثلة فى علاج الصرع فى الأطفال المتخلفين عقليا، مع ملاحظة حدوث آثار جانبية أقل خصوصا على الإدراك و السلوك.

إن عقار لاموترجين المضاد للصرع هو دواء حديث نسبيا و أكثر أمنا ، وقد سوق أولا كمضاد للصرع عام ١٩٩٤ ، ثم تم اعتماده مؤخرا كمتبب للمزاج ، كما أنه أظهر تأثيرات واعدة أيضا فى تعديل سلوك الأشخاص ذوى اضطراب الشخصية البينى. علاوة على ذلك ، أظهر لاموترجين آثارا نفسية مستحبة مؤخرا عندما استعمل فى معالجة بعض الأطفال المتخلفين عقليا و الذين يعانون من الصرع فى الدراسة التى عرضها ميقاى سنة ٢٠٠٣م.

### فرضية الدراسة (الهدف من الدراسة):

"هل يُمكن لعقار لاموترجين أن يعدل من أعراض السلوك التوافقي المضطرب ( كان يُحسن المزاج و يطف من شدة السلوك العدوانى أو النزوى و يحد من فرط النشاط الحركى) فى

الأطفال المتخلفين عقلياً غير المصابين بالصرعَ وهل يرتبط هذا التحسن في السلوك بوجود تغيرات في رسم المخ الكهربى.

### الموضوع والطريقة:

- \* نوع الدراسة: دراسة حالة منضبطة
- \* عينة الدراسة: الدراسة شملت ٦٠ طفلاً متخلفاً عقلياً راجعوا العيادات الخارجية النفسية بمستشفى الطب النفسى ببنى سويف خلال ١٢ شهراً ،هى فترة الدراسة الميدانية.
- \* معايير الاحتواء: الأطفال المختارين للدراسة تم انتقاؤهم عشوائياً بطريقة (اختيار الأول مشاهدة و تنطبق عليه الشروط) ، و الشروط هى:
  - المدى العُمري :٦- ١١ ½ سنّوات.
  - المنزلة الإجتماعية الإقتصادية: الطبقة المتوسطة (باستخدام مقياس الشخص لقياس المستوى الإقتصادى و الاجتماعى للأسرة).
  - النوع: كلا الجنسين متضمنً بنسبة تواجدهم فى المجتمعات و هى: ذكور : إناث ( ١ ½ : ١ ) .
  - درجة التخلف: حالات التخلف العقلى البسيط و المتوسط (بموجب التقويم العيادى واختبار معامل وكسلر للذكاء للأطفال و اختبار مقياس فاينلاند للسلوك التوافقى)
  - الحالات التى لديها سلوك غير توافقى (بحسب مقياس فاينلاند أيضاً).
  - الحالات المقيمة فى مدينة بنى سويف.

### الطريقة:

- عينة الدراسة تم تقسيمها إلى مجموعتين:
  - مجموعة تجريبية: (مجموعة أ): = ٣٠ طفلاً لديهم تغيرات فى رسم المخ الكهربى .
  - مجموعة ضابطة: (مجموعة ب): = ٣٠ طفلاً ليس لديهم تغيرات فى رسم المخ الكهربى .
- كلتا المجموعتين كانتا متجانستين عند بدء الدراسة من حيث العمر الزمنى والعقلى و العمر المكافىء ومعامل الذكاء و المعامل الاجتماعى و القصور فى السلوك التوافقى وكذلك فى اضطراب السلوك غير التوافقى. هذا و قد أعطى أطفال المجموعتين جرّعات منتظمة من دواء لاموتريجين لمدة ستة أشهر تحت إشراف أولياء أمور الأطفال المعنيين و الذين قاموا بتوقيع "إقرارات" تُؤكد موافقتهم المسبقة على إدراج أطفالهم فى الدراسة.
- تم فحص كل طفل إكلينيكيًا و عصبياً حيث طبقت كل الحالات معايير تشخيص التأخر العقلى البسيط أو المتوسط (طبقاً للإصدار الرابع من تشخيص و تصنيف الأمراض النفسية لعام ١٩٩٤ من الجمعية النفسية الأمريكية).
- تم قياس السلوك غير التوافقى لأطفال الدراسة قبل بدء العلاج و عند نهاية فترة العلاج المفترضة (٦ شهور) على مقياس فاينلاند للسلوك التوافقى ، مجال السلوك غير التوافقى ، الجزء الأول.

مكان الدراسة: العيادات الخارجية بمستشفى الطب النفسى ببنى سويف.

وقت الدراسة: الوقت الإجمالي = ١٥ شهراً

- ▶ ٦ شهور: لجمع الحالات
- ▶ ٦ شهور: لإعطاء العلاج و متابعة المرضى و جمع البيانات.
- ▶ ٣ شهور: لمعالجة البيانات، عمل التحليل الإحصائي بواسطة الحزمة الإحصائية للدراسات الاجتماعية SPSS ، واستخراج الاستنتاجات ثم المراجعة الفنية و اللغوية و طباعة البحث.

### النتائج:

أثبتت هذه الدراسة أن لعقار لاموتريجين (لامكتال) آثاراً إيجابية ذات دلالة إحصائية على تحسن السلوك غير التوافقي لأطفال الدراسة ، و كان التحسن العام بدرجة أكبر في أطفال المجموعة التجريبية الذين لديهم تغيرات في رسم المخ الكهربى (بنسبة ٦٣ر٣%) مقارنة بتحسن عام في ٥٠% من أطفال المجموعة الضابطة. ولم يرتبط التحسن بالعمر الزمنى ولا بمعامل الذكاء (الدالين على درجة التخلف العقلى) ولا بنوع الطفل ذكراً أم أنثى. علماً بأن ٢٥% من حالات الدراسة لم يحدث لهم تحسن ذو دلالة ، و ٨ر٣% ساءت حالاتهم بالإضافة إلى ١٠% آخرين اضطروا لقطع العلاج بسبب آثار عكسية من الدواء أهمها القيء و الطفح الجلدى.

أما على مستوى التحسن النوعى فى الأعراض المختلفة للسلوك غير التوافقى، فقد كان التحسن بدرجة متوسطة متساوياً فى فرط النشاط الحركى فى المجموعتين ، ولكن كان التحسن أكبر فى المجموعة التجريبية عن المجموعة الضابطة فيما يخص نقص الانتباه و التركيز، العنادية ، السلوك العدوانى ، السلوك النزوى ، المزاج الاكتئابى ، نقص المنطقية ، و السلوك الخلفى و المتحدى ، بينما كان التحسن أكبر فى المجموعة الضابطة عن المجموعة التجريبية فيما يخص العصبية ، الكذب و السرقة ، و السلوك الانسحابى.

### التوصيات:

تمت التوصية بأنه يمكن انتقاء دواء لاموتريجين لتعديل السلوك غير التوافقى للأطفال المتخلفين عقلياً غير الصرعيين خصوصاً أعراض العصبية و العنادية و اضطراب المزاج و السلوك العدوانى و السلوك الانسحابى خاصة فى وجود تغيرات فى رسم المخ الكهربى. نظراً للصغر النسبى فى حجم عينة البحث ، فإنه قد يلزم إجراء أبحاث مستقبلية على مجموعات أكبر لتأكيد نتائج لدراسة.

\*\*\*\*\*



## المستخلص:

افترض الباحث أن لدواء لاموتريجين (لامكتال) المضاد للصرع تأثيرات إيجابية على السلوك غير التوافقى فى الأطفال المتخلفين عقلياً غير الصرعيين بناءً على الدراسات السابقة لإتنجر (١٩٩٨) و ميقاتى (٢٠٠٣) و غيرهم ، وقد عرض الباحث موجزاً تاريخياً عن معاملة المجتمعات المختلفة للمتخلفين عقلياً من الامتهان و التعذيب و القتل إلى الاهتمام و الرعاية الموجهة و الدفاع عن حقوقهم ، كما قام بعرض التعريفات المختلفة للتخلف العقلى ، و أسبابه و العوامل المساعدة لتكوينه ، و كيفية تشخيصه ، وأشهر الأمراض الجسمية و النفسية المصاحبة له ، و طرق الوقاية و العلاج الممكنة.

و قد بيّن الباحث أن التطبيق العملى للبحث تم عن طريق اختيار مجموعتين من الأطفال المتخلفين عقلياً و غير الصرعيين المراجعين للعيادات النفسية بمستشفى الطب النفسى بنى سويف ، أحدهما مجموعة تجريبية قوامها ٣٠ طفلاً لديهم تغيرات فى رسم المخ الكهربى ، و الثانية مجموعة ضابطة من ٣٠ طفلاً مماثلين و لكن رسم المخ الكهربى لديهم طبيعى . و قد تم إعطاء الجميع دواء لاموتريجين و متابعتهم بالعيادة النفسية لمدة ٦ أشهر لمن استكمل مدة العلاج.

أظهرت النتائج وجود تحسن ذى دلالة فى المجموعتين بنسبة ٦٣,٣% فى المجموعة التجريبية و ٥٠% فى المجموعة الضابطة مما يدل على ان لدواء لاموتريجين تأثيرات إيجابية على السلوك غير التوافقى فى الأطفال المتخلفين عقلياً غير الصرعيين ، خصوصاً الذين لديهم تغيرات فى رسم المخ الكهربى.

أوصى الباحث بضرورة إجراء مزيد من الأبحاث فى نفس الاتجاه على عينات أكبر و أشمل لتدقيق نتائج الدراسة الحالية.

**الكلمات الكاشفة:** السلوك التوافقى - السلوك غير التوافقى - أدوية مضادة للصرع - آثار إيجابية نفسية - لامو تريجين - بنى سويف - التخلف العقلى - المتخلفين عقلياً - غير صرعي - الطبقة الاجتماعية المتوسطة - مقياس الشخص لقياس المستوى الإقتصادى و الاجتماعى للأسرة - مقياس فاينلاند للسلوك التوافقى - مقياس وكسلر لذكاء الأطفال



جامعة عين شمس  
معهد الدراسات العليا للطفولة  
قسم الدراسات الطبية

شكر

أشكر السادة الأساتذة الذين قاموا بالإشراف

وهم:

- ١- أ.د. / علوية محمد عبد الباقي - أستاذ ورئيس القسم الطبى بالمعهد.
- ٢- أ.م.د. / رحاب عبد القادر محمود - أ.م. الدراسات الطبية بالمعهد.
- ٣- أ.م.د. / هويدا حسنى الجبالى - أ.م. الدراسات الطبية بالمعهد.

ثم الأشخاص الذين تعاونوا معى فى البحث

وهم:

- ١- أ.د. / عبد العزيز الشخص - أستاذ علم النفس بكلية تربية عين شمس.
- ٢- د. / حسن إبراهيم الضبع - مدير مستشفى الطب النفسى بنى سويف.
- ٣- الأستاذ / محمد حافظ - أخصائى نفسى بمستشفى الطب النفسى بنى سويف.
- ٤- د. عادل سلطان - رئيس قسم الإحصاء بمعهد البحوث الجنائية و الاجتماعية.

و كذلك الهيئات الآتية:-

- ١- مستشفى الطب النفسى بنى سويف.
- ٢- مدرسة التربية الفكرية بنى سويف.





جامعة عين شمس  
معهد الدراسات العليا للطفولة  
قسم الدراسات الطبية

صفحة العنوان

اسم الطالب: محسن على عبد العال  
الدرجة العلمية: ماجستير  
القسم التابع له: الدراسات الطبية  
اسم المعهد: معهد الدراسات العليا للطفولة  
الجامعة: عين شمس  
سنة التخرج: ١٩٧٩ م  
سنة المنح: ٢٠٠٥ م

شروط عامة:  
يوضع شعار الجامعة على الغلاف الخارجي



رسالة : ماجستير

اسم الطالب : محسن على عبد العال

عنوان الرسالة : ( تقويم الآثار الإيجابية لعقار لاموترجين على السلوك التوافقي  
المضطرب للأطفال المتخلفين عقليا غير الصرعيين )

أسم الدرجة : الماجستير

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تاريخ البحث : ٢٠٠٢ / ٥ / ٢٠

الدراسات العليا

ختم الإجازة :

أجيزت الرسالة بتاريخ :

٢٠٠٥ / ٥ / ١٢

١٩٩ / /

موافقة مجلس الجامعة

موافقة مجلس الكلية

١٩٩ / /

٢٠٠٦ / ٥ / ٢٢





١٥٢  
٥

١٤٤٦



قسم الدراسات الطبية

## تقويم الآثار الإيجابية لعقار لاموترجين على السلوك التوافقي المضطرب للأطفال المتخلفين عقليا غير الصرعيين

مسودة بحث توطئة لنيل درجة الماجستير في دراسات الطفولة  
قسم الدراسات الطبية (صحة و تغذية الطفل)

مقدمة من الطبيب

محسن على عبد العال

بكالوريوس الطب والجراحة جامعة القاهرة  
دبلوم نفسية وعصبية جامعة القاهرة

تحت إشراف

أ.د. علوية محمد عبد الباقي

أستاذ الطب النفسى للأطفال

رئيس قسم الدراسات الطبية ( صحة و تغذية الطفل)

معهد الدراسات العليا للطفولة

جامعة عين شمس

أ.م. د. / هويدا حسنى الجبالي

أستاذ مساعد بقسم الدراسات الطبية

معهد الدراسات العليا للطفولة

جامعة عين شمس

أ.م. د. / رحاب عبد القادر محمود

أستاذ مساعد بقسم الدراسات الطبية

معهد الدراسات العليا للطفولة

جامعة عين شمس

القاهرة ٢٠٠٥ م