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Psychological Aspects of Chronic Adenotonsillitis in Late Childhood

Thesis
Submitted For Fulfillment Of PhD Degree In
Childhood Studies

Presented by
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M.B., B.CH. -M.Sc.

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بسم الله الرحمن الرحيم

وَفِي أَنفُسِكُمْ أَفَلَا تُبْصِرُونَ

صدق الله العظيم

سورة الذاريات الآيه ٢١

Abstract

This study was conducted on 83 children (age 7-12 years) suffering from chronic adenotonsillitis of duration more than one year going for adenotonsillectomy; besides 30 apparently normal control children.

Full general, otorhinolaryngologic and psychiatric evaluation, according to DSM- IV criteria, were done for all children; besides the psychometric studies using Revised Behavior Problem Checklist (RBPC), Children Depression Inventory (CDI) and Children Anxiety Scale (CAS), preoperatively and 5-6 months after surgery. Psychiatric morbidity in the study group was 39.75 % in contrast to 16.66 % in the control group.

Attention deficit disorder, oppositional defiant disorder and nocturnal enuresis were significant among the cases than the controls and showed gradual increase with the severity and duration of upper airway obstruction.

Both attention deficit disorder and nocturnal enuresis showed significant decrease after surgery. Although there was no significant improvement in clinically diagnosed oppositional defiant disorder. There was significant improvement on attention problem immaturity, motor tension excess and conduct disorder subscales of RBPC.

There was no significant changes in conduct disorder, depression and anxiety between the cases and the controls preoperatively. However, there was significant reduction in anxiety postoperatively in cases with huge adenoids and tonsils. Anxiety disorder was decreased with the duration of obstruction while depression was increased with the duration of disease.

Key words: psychological – childhood – chronic adenotonsillitis – upper airway obstruction .

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INTRODUCTION

Introduction

Although tonsils and adenoids serve as a defense against infection, yet they may become a site of acute or chronic infection.

The principal disturbances of the tonsils and adenoids are infection and hypertrophy (Potsic, 1989).

Local enlargement of the lymphoid tissue secondary to chronic infection can be a problem (Zalzal and Cotton, 1993) which causes upper airway obstruction and respiratory distress in children that may be severe enough to cause pulmonary hypertension and possible death.

In 1994, Ley, assumed that if cognition is a product of the brain's activity, and if the brain's activity is dependent on adequate supply of oxygen, then the connection between disordered breathing and psychiatric disorders should be clear, suggesting thereby the closest correspondence between disease and behavior.

Deutch, 1996, reported that most upper airway obstruction in children is caused by adenotonsillar hypertrophy in which prolonged partial airway obstruction during sleep may result in significant hypercarbia and hypoxaemia.

Baily and Croft, 1997, reported that chronic adenotonsillitis most often causes upper airway obstruction; frequent waking and disturbed sleep are the consequences. A second but perhaps major consequence of disturbed sleep is daytime hypersomnolence which clearly impairs cognitive functions, increasing inattentiveness and

difficulty in concentration, in addition, many experience personality changes (Moran et al., 1984).

Richardson et al., 1980, reported that sleep deprivation in those children may result in retarded growth and development.

During the school years illnesses that interfere with the participation of children in normal family and school events are conceptualized to be particularly potent stressors (Marzek, 1994).

Aim of the Work

The aim is to study the psychological effects of chronic adenotonsillitis with its possible upper airway obstruction in late childhood and to determine the effect of severity and the duration of upper airway obstruction in chronic adenotonsillitis on the psychology of the affected children.

REVIEW OF LITERATURE

Tonsils and Adenoids

Anatomy

Waldeyer's ring of lymphoid tissue consists of the palatine (faucial) tonsils, lingual tonsils, pharyngeal bands and adenoids. The palatine tonsil is a lymph node with prominent germinal centers that is separated from the lateral pharyngeal wall by a capsule of connective tissue. The tonsil lies in the tonsillar fossa, which has three muscles: the palatoglossus, which is the anterior pillar; the palatopharyngeus, which is the posterior pillar; and the superior constrictor, which forms the bed of the tonsil. Medially there are crypts lined with stratified squamous epithelium (Zalzal and Cotton, 1993); the tonsillar crypts may penetrate nearly the whole thickness of the tonsil and distinguish it histologically from other lymphoid organs.

The medial surface of the tonsil is free and projects to avariable extent into the oropharynx, depending partly on its size but, probably more importantly, on the degree to which it is embeded into the tonsillar fossa (Beasley, 1997).

The adenoids (Nasopharyngeal tonsils) is a collection of lymphoid tissue found in the mucous membrane overlying the basisphenoid.

The nasopharyngeal tonsil has a rectangular shape similar to a truncated pyramid, dependent from the roof of the nasopharynx. Its surface is deeply grooved in a longtudinal way in line with the airflow across it. The anterior edge of the block of tissue is vertical and in the same plane as the posterior nasal aperture. The posterior

edge gradually merges into the posterior pharyngeal wall: the lateral edges incline toward the midline (Beasley, 1997).

The arterial blood supply for the tonsil is from the descending and ascending palatine arteries and the lingual tonsillar and descending pharyngeal arteries. The venous blood drains through the peritonsillar plexus into the lingual veins and pharyngeal veins, which in turn drain into the internal jugular vein (Zalzal and Cotton, 1993).

The nerve supply of the tonsils is through the lesser palatine nerves to the pterygopalatine ganglion and through the glossopharyngeal nerve.

The arterial supply of the nasopharyngeal tonsils is provided by the pharyngeal branches of the external carotid artery. These vessels include the ascending pharyngeal artery and the minor palatine branches of the maxillary artery, as well as, the minor palatine branches of the facial artery.

Venous drainage passes through the pharyngeal plexuses to the internal jugular vein.

Sensory innervation is provided by nasopharyngeal branches of the glossopharyngeal and vagus nerves (Beasley, 1997).

The efferent lymph drainage of palatine tonsils is through the upper deep cervical lymph nodes, especially to the jugulodiagastric group of lymph nodes behind the angle of the mandible. While adenoids lymph drainage is to the retropharyngeal

and pharyngomaxillary space lymph nodes (Zalzal and Cotton, 1993).

The relation between the anatomic location and pathophysiology of the tonsil and adenoids is extremely important in understanding the etiology of the diseases most commonly encountered in the tonsils and adenoids.

The pathogenesis of disease in the tonsils and adenoids is probably multifactorial and depends on the interactions among the anatomy, microbiology and immunology of these structures.

In the broadest terms, it is likely that alterations in the ecology of the nasopharynx and oropharynx are primarily responsible for both infectious and hyperplastic disease (Brodsky et al., 1988).

The microbiology and immunology of the tonsils and adenoids are considered to be similar.

Group A B haemolytic streptococci have classically been considered the only important bacteria in the development of acute tonsillitis, yet evidence is increasing that other bacteria, which are usually pathogenic elsewhere in the upper aerodigestive tract, can cause acute, recurrent and chronic tonsillitis. These include Streptococcus pneumoniae, Staphylococcus aureus and Hemophilus influenzae (Brodsky, 1989).Their role in acute adenoiditis has also been well established.

These bacteria, which are often pathogenic else where, but only considered colonizers of the tonsils, are now believed to

become pathogenic in the presence of inflammation (often viral in origin) or recurrent bacterial infection (Brook, 1987).

Viral infections of the oropharynx and nasopharynx are prevalent. Sprinkle and Veltri, 1974, proved that adenoviruses can cause tonsillitis, Epstein Barr virus and herpes simplex as well may be the sole agent responsible for acute tonsillitis in children.

The existence of polymicrobial infections, the emergence of B lactamase – producing organisms and encapsulated anaerobes (Reilly et al., 1981), in addition to the recognition of the role of bacterial antigenic load deep in the tonsil and adenoid cores (Surrow et al., 1989) are all important concepts in understanding the development of chronic disease in the tonsils and adenoid.

The immunology of the tonsils and adenoids has been the subject of many reviews, with two major immunological functions have been proposed for the tonsils and adenoids: Local immunity and immune surveillance (Brodsky et al,1989)

Lal et al., 1984, demonstrated local antibody production by tonsil and adenoid B cells in response to specific antigens. Persistent antigenic stimulation of tonsil's lymphocytes by H. influenzae, S. aureus and Bacteroids melaninogenicus, may underlie the emergence of tonsillar hyperplasia as the commonest reason for tonsillectomy even in the absence of clinical history of infection (Brodsky et al 1989).

Clinical classification of diseases in the tonsils and adenoids can be confusing, Brodsky, 1989, proposed clinically useful classification of diseases of tonsils and adenoids.

- **Infection**
  - Acute tonsillitis or adenoiditis.
  - Recurrent acute tonsillitis or adenoiditis.
  - Chronic tonsillitis or adenoiditis.

- **Obstruction**
  - Airway
    Nasal
    Oropharyngeal
  - Eustachian tube (adenoids).
  - Swallowing (tonsils).

- **Neoplasia**
  - Benign.
  - Malignant.

- **Trauma**

Tonsillitis

This is a common disorder in children, with a peak incidence at around 5 to 6 years of age.

It is unusual for a child not to have at least one or two episodes of tonsillitis. These attacks are liable to occur when the child is exposed to large numbers of other children for the first time, that is on entering nursery school or primary school (Cowan and Hibbert, 1997).

Patients can be predisposed to tonsillitis by having low resistance, by having tonsils that are at a disadvantage secondary to previous recurrent tonsillitis, as a part of generalized pharyngitis, or secondary to a virus infection (usually adenovirus, which renders the tonsils susceptible to bacterial invasion).

Overcrowding, poor nutrition and inadequate ventilation help propagate epidemics of tonsillitis. Fatigue and exposure to cold may lower the body's resistance to infection (Zalzal and Cotton, 1993).

The onset of sore throat, fever, dysphagia, and tender cervical adenitis in the presence of erythematous or exudative tonsils is consistent with acute tonsillitis (Stjernquist – Desatnik et al., 1987).

The definition of recurrent acute tonsillitis varies from four to seven episodes of acute tonsillitis in one season to five episodes per year for 2 years (Paradise et al., 1984).

The presence of chronic sore throat; tonsilloliths; malodorous breath; and persistent tender cervical lymphadenopathy are all consistent with a diagnosis of chronic tonsillitis when not attributable to other causes (Brodsky, 1989).

Obstructive tonsillar hyperplasia is problematic when functional obstruction of the air or food passages occur.

The diagnosis of acute adenoiditis is somewhat controversial because the signs and symptoms resemble those of acute rhinosinusitis,These include nasal obstruction, fever and often

otitis. As a result acute adenoiditis is probably underdiagnosed or misdiagnosed (Brodsky, 1989).

More than four infections in one season can be considered recurrent acute adenoiditis.

In obstructive adenoid hyperplasia the size of adenoids in relation to that of the nasopharynx is important in considering its harmful effects. It is certainly possible that recurrent acute infections are the sole cause of abnormally large adenoids, although it has been suggested that allergic episodes also result in adenoidal enlargement (Cowan and Hibbert, 1997).

The effect of such enlargement produces impairment of nasal respiration as large adenoids can partially or totally obstruct nasal respiration causing snoring, hyponasal speech and forcing the child to breathe through the mouth leading to high incidence of crossbite; narrow upper arch; retroclined upper and lower incisors due to lip tention and small sagittal depth of the nasopharynx (Linder – Aronson. 1972).

The child may have classical adenoid facies with an open-mouthed, dull appearance, elongated face, dark circles around the eyes. Although Cowan and Hibbret, 1997, argued that open lip posture with prominent teeth and short upper lip are likely to be result of thumb sucking.

Nearly all children with pharyngeal obstruction snore at night when they are sleeping, and many have periods of interrupted breathing (Potsic et al., 1986).

Even minimal hyperplasia of the adenotonsillar tissue may contribute to severe obstruction in these children during sleeping. In addition, aspiration of pharyngeal secretions during snoring and interrupted breathing may aggrevate the lower respiratory disease (Potsic, 1989).

Modern assessment of the tonsils and adenoids is based on an appreciation of new concepts pertaining to the pathogenesis of tonsil and adenoid disease.

The performance of a precise history in which the triad of chronic mouth breathing, snoring and hyponasal speech are most often seen (Hibbert, 1981). While sleep disturbance can occur, it is often due to a combination of nasopharyngeal and oropharyngeal obstruction (Albert, 1997) , as obstructive hyperplasia often occurs in tonsils and adenoids simultaneously (Brodsky, 1989).

Brodsky, 1989, developed a standardized system for evaluation of tonsils:

0: Tonsils are situated in the tonsillar fossa, with no impingement on the oropharynx.

+1: Tonsils sit just outside of the tonsillar fossa with obstruction less than 25 percent of the airway.

+2: Tonsils are readily seen in the airway 25 - 50 percent of the airway is obstructed.

+3: Tonsils denote a 50 to 75 percent obstruction of the airway.

+4: Tonsils involve a greater than 75 percent obstruction of the airway.

However, clinical examination is unlikely to be a decisive factor in the assessment of a child for tonsillectomy and / or adenoidectomy (Weir, 1972).

The use of the lateral neck film can occasionally be helpful in identifying the inferior extension of the tonsils; however, the relative size of the tonsils in relation to the airway cannot be accurately determined by this modality. The use of a small amount of barium to coat the tonsils, which then can be further visualized in a posteroanterior view, is sometimes worthwhile when used with lateral neck film (Brodsky, 1989). Yet lateral radiograph is considered the most reliable way of assessing the size of the adenoids. This will give a measure of the absolute size of adenoids and also an assessment of the relation to the size of the airway through the use of adenoid – nasopharyngeal ratio (Cowan and Hibbert, 1997) .The presence of adenoidal tissue in a small post-nasal space rather the adenoidal hypertrophy itself is the main cause of symptoms of obstructive sleep apnea.

Bacteriological examination of throat swabs is most unlikely to be helpful in assessing children with recurrent tonsillitis, Veltri et al., 1972, explored serological tests as a possible indicator of recurrent or chronic tonsillitis, IgM, IgA or IgG are almost non-specific (Cowan and Hibbert, 1997).

Tonsillectomy and Adenoidectomy

Every pediatrician has wrestled with the question of appropriate criteria for recommending tonsillectomy, adenoidectomy or both for an individual child; and each develops a personal algorithm for decision making. Rigidly structured criteria are tempered by the anecdotal experiences of the physician and the family and a lack of universally accepted criteria (Deutsch 1996). The pendulum of public and professional openion concerning tonsillectomy and adenoidectomy continues to swing between enthusiasm and condemation.

Although the total number of tonsillectomies has decreased, the percentage performed for upper airway obstruction has increased (Rosenfeld and Green, 1990).

Obstructive sleep apnea (OSA) caused by adenotonsillar hypertrophy constitutes a definite indication for surgery (Paradise, 1996). In 1988, Sofer et al., reported several children with OSA related respiratory distress, pulmonary edema and corpulmonale with severe right ventricular and right atrial dilatation and reduced right ventricular ejection fraction. All children improved following removal of hypertrophied tonsils or adenoids or both. Adenotonsillectomy is considered the most frequent treatment of children with OSAS (Joosten and Vanden Berg, 1998).

Adenotonsillar hypertrophy with upper airway obstruction, even without OSA, is another definite indication for adenotonsillectomy (Deutsch, 1996).

Mozata and Carrascosa, 1999, postulated a new indication for tonsillectomy & adenoidectomy which is pharyngeal breathing

due to obstruction by hypertrophied lymphoid formations in the Waldeyers ring,

Paradise, 1996, proposed dysphagia resulting from adenotonsillar hypertrophy as another definite indication for surgery. Hyponasal speech as a result of blockage of nasal airflow due to adenoid hypertrophy may be alleviated by adenoidectomy where as hypernasal speech which results from an inability to prevent inappropriate airflow through the nasopharynx because of inability to seal the soft palate against the posterior pharyngeal wall may result from tonsillar hypertrophy and constitute a definite indication for tonsillectomy (Brodsky, 1989).

Moreover adenotonsillectomy is indicated in Orofacial growth abnormalities and dental malocclusion due to upper airway obstruction which is particularly effective in normalizing malocclusion when performed before the child is 6 years of age (Hultcrantz et al., 1991). Yet recurrent throat infections are generally accepted as a relative indication for surgery.

In recent study conducted by Matsuda et al., 1999, serum and urinary concentration of macrophage colony stimulating factor M-CSF in patients with IgA nephropathy associated with chronic tonsillitis was measured; the results showed that tonsil stimulation contributes to the progression of IgA nephropathy via enhancement of glomerular production of M-CSF; suggesting thereby tonsillectomy as an idication for patients with IgA nephropathy.

Cowan and Hibbert, 1997, stated that no doctor has any right to promise more from the removal of tonsils other than that the child will have no further attacks of acute tonsillitis.

Obstructive Sleep Apnea Syndrome

Obstructive sleep apnea is a common disorder. However, it is a recently described disorder for which most primary care physicians do not have formal training.

Childhood obstructive sleep apnea syndrome (OSAS) is characterized by episodes of partial or complete upper airway obstruction that occur during sleep, usually associated with a reduction in oxyhemoglobin saturation and / or hypercarbia (Carroll and loughlin, 1995).

Children with OSAS may be seen by various specialists: pediatricians for failure to thrive, neurologists for sleepiness, otolaryngologists for adenotonsillar hypertrophy, psychiatrists for behavioral problems and pulmonigists for snoring and difficult breathing.

The cardinal symptom of obstructive sleep apnea is loud and persistent snoring, which is a sign of partial airway obstruction, and the progression of events leading to this obstruction will eventually lead to shut down of the airway as increasing velocity of inspiratory airflow through the narrowed pharynx creates an irrisistible collapsing force on the complaint pharyngeal walls (Venturi effect). This produces the classical obstructive apnoeic episodes which is the second hallmark symptom of OSAS (Baily and Croft, 1997).

During the periods of apnea the high negative intrathoracic pressures and decrease in oxygen saturation interact to produce arousal. The airway is restored as the patient wakes. A major consequence of multiple arousals and disturbed sleep pattern is

daytime hypersomnolence (Baily and Croft, 1997); however, the observation that EDS "Excessive Daytime Sleepiness" is present only in a minority of children with OSAS suggests two possibilities: most children with OSAS get sufficient adequate quality sleep or the daytime manifestations of sleep disruption are different in children than adults (Leach et al., 1992).

Many investigators have described restless sleep in children with OSAS, which is defined as persistent or recurrent body movements, arousal and brief awakening in the course of sleep.

Stradling and associates, 1990, found that about 75% of children with OSAS were often perceived as restless sleepers, compared to 10% of controls; suggesting that restless sleep is associated with upper airway obstruction.

Enuresis is said to be a common feature of childhood type OSAS, although, it is an unusual symptom of OSA in adults. Frank et al., 1983, reported enuresis in 33% of children with OSAS over 4 years old. Weider and Hauri reported in 1985 that 26 of 35 children with nocturnal airway obstruction have enuresis. The role of OSAS per se is not clear.

Pathophysiology

The mechanisms of OSA in children are not known. Clearly, there is not a single mechanism in all children with OSAS.

Three variables are important in the development of the collapse and obstruction of the upper airway: the decreased activity of the muscle dilators of the pharyngeal airway, the relative vacuum generated in the upper airway during inspiration, and the surgical

anatomy of the upper airway (Remmers, 1978). The inspiratory obstruction usually begins in the pharynx and extends downwards during the apnea.

In children with craniofacial abnormalities, the site of obstruction is variable, such children may show pharyngeal or hypopharyngeal obstruction during inspiration. Most of otherwise normal children with OSAS have enlarged tonsils and / or adenoids and no clinically apparent craniofacial or upper airway structural abnormalities (Swift, 1988).

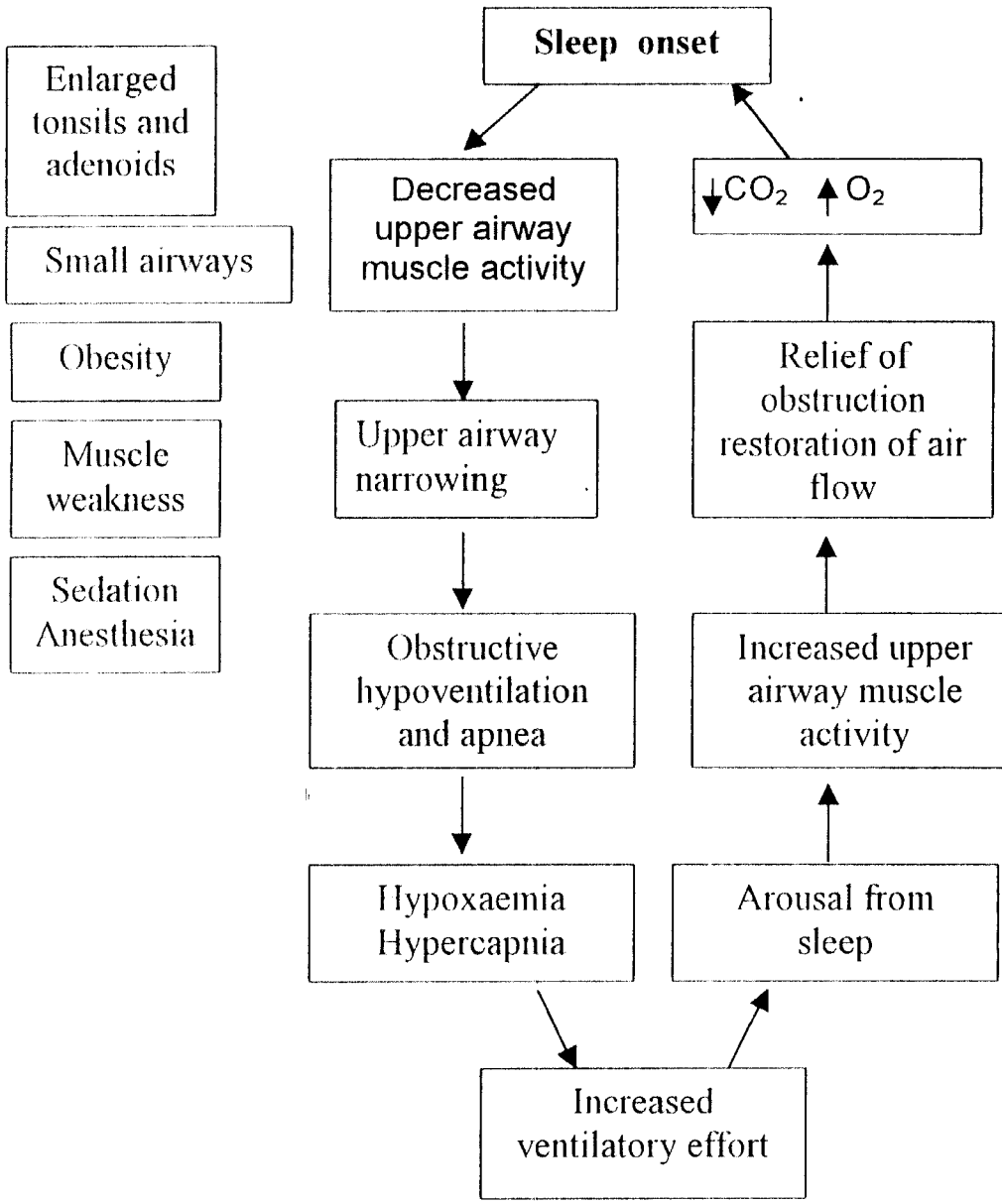
Carroll and Loughlin, 1995, proposed that when the airway is preloaded with large tonsils and adenoids, the increased resistance leads to activation of upper airway dilator muscles which then play a significant role in the maintenance of upper airway patency; still it appears likely that other etiologic factors such as subtle abnormalities of airway configuration, abnormal upper airway dilator muscle control or abnormal arousal mechanisms are necessary to fully explain childhood OSAS.

Although studies suggest that tonsillar enlargement alone cannot account for OSAS in children, there is little information about the role of adenoidal hypertrophy.

The observation that most children improve after adenotonsillectomy leads many to assume that adenotonsillar hypertrophy causes OSAS.

Moreover, it would appear that enlargement of the tonsils and adenoids leads to major changes in airway size and anatomic relationships between mucosal surfaces.

It is also possible that the properties of the mucosal surfaces or the mucous itself may be different in children with OSAS from that in normal children (Caroll and Loughlin, 1995).



Pathophysiology of Obstructive Sleep Apnea and Hypoventilation in Children . (Rosen and Haddad , 1996)

OSAS is a clinical diagnosis based on information obtained by clinical history, physical examination, and laboratory studies of which polysomnography [PSG] is the only one – Carroll and Loughlin, 1995, proposed that it can generally be said that if there is no snoring and no other symptoms, then the child probably does not have OSAS. Attal et al., 1994, went further as he considered snoring without apneas as a form of sleep apnea syndrome.

The 1990 International Classification of Sleep Disorders (ICSD) diagnostic criteria for OSAS do not apply to children, as it require a complaint of excessive daytime sleepiness (EDS) and more than five obstructive events per hour lasting longer than ten seconds.

Suggested diagnostic criteria for childhood OSAS were modeled after International Classification of Sleep Disorders: Diagnostic and Coding Manual criteria for adult OSAS, 1990,by Marcus et al,1992.

Diagnostic Criteria for OSAS in Children

A. Caregivers complain of child having noisy or disturbed breathing during sleep and / or inappropriate daytime sleepiness or behavioral problems.

B. Episodes of complete or partial airway obstruction during sleep.

C. Associated features include:
   1. Snoring
   2. Paradoxial chest / abdomen motion and / or retractions.
   3. Apnea or difficulty breathing observed by caregivers.

4. Excessive daytime sleepiness.
5. Behavior problems.
6. Adenotonsillar hypertrophy.
7. Daytime mouth breathing.
8. Failure to thrive or obesity.

D. Polysomnographic monitoring demonstrates:
1. Obstructive hypoventilation and / or.
2. One or more obstructive apnea / hour, usually with one or more of the following:
    a. Arterial oxygen desaturation below 90-92%.
    b. Arousals from sleep associated with upper airway obstruction.
    c. An MSLT demonstrating an abnormal sleep latency for age.

E. Usually associated with other medical disorders. e.g. adenotonsillar enlargement.

F. Other sleep disorders can be present. e.g. narcolepsy.

The great difficulty in diagnosing sleep apnea on general examination is that there may be few, if any, daytime physical signs However, in the child, a poor nasal airway with constant mouth – breathing is usual. The upper lip is often coated with nasal mucous and daytime respiration may be noisy with variable stretor present.

Examination of the throat may show "midline tonsils" which appear to occupy the oropharynx completely and nasal airflow may

be minimal on objective testing. Examination of the nasal airway should be carefully undertaken, usually with the help of fibreoptic endoscopic equipment (Baily and Croft, 1997).

Polysomnography

The "gold standard" for the diagnosis of abnormal breathing during sleep is nocturnal, full night PSG.

It should be remembered that PSG can detect and quantify sleep – associated upper airway obstruction, but the diagnosis of OSAS can only be made by clinician, based on the total clinical picture.

As knowledge about childhood OSAS increases, pediatric sleep laboratory with pediatric – oriented technicians is crucial for obtaining successful PSG in children.

A significant benefit of PSG is determination of severity, as patients with severe OSA on PSG are at higher risk of postoperative complications (Joostan and Vandenberg,1998). However, in a recent study conducted by Loube and Andara, 1999, nocturnal PSG showed the same abnormal nocturnal respiratory measurements of body mass index and gender- matched cohorts of upper airway resistance syndrome (UARS) and OSAS patients with the exception of Apnea Hypoapnea Index (AHI).

Polysomnographic features of OSAS in children varies, as some children exhibit a predominant pattern of repetitive obstructive apnea, while others may have continuous obstructive hypoventilation lasting hours and many children exhibit both

obstructive hypoventilation and apnea during a single night (Carroll and Loughlin, 1995).

Hypoxemia is usually a feature of OSAS in children; although the prevalence of hypoxemia during sleep in children with OSAS is not known since the definitions vary from study to study.

Children have been documented to have OSA without hypoxemia, as well as significant obstructive hypoventilation with no hypoxemia, this could be explained by brief obstructions (that would not be scored as significant by adult criteria)which can lead to hypoxemia in children (Carroll and Loughlin, 1995). Much less is known about hypercarbia than hypoxemia in children with OSAS.

Marcus et al., 1992, reported peak PET CO_2 values in normals should not exceed 53 mmHg, greater PET CO_2 values may indicate sleep - related upper airway obstruction.

Paradoxical inward rib cage motion of inspiration (PIRCM) occurs during upper airway obstruction, due to the large subatmospheric intrathoracic pressures, is a PSG feature of OSAS in children occurring in NREM sleep, and in REM sleep in children over 3 years of age (Gaultier, 1990).

Although very little has been published on the quantification of disturbed sleep by PSG in children. It seems reasonable to consider a sleep efficiency of 85% or less abnormal (Carroll and Loughlin, 1995).

Before a PSG can be used to diagnose OSAS, it must first be scored. This requires tremendous data reduction, since a PSG consists of nearly 1000 pages of continuous recording of 14 to 22 channels. Pertinent information must be identified and data is extracted in a usable form.

At present, there are no standards for scoring of cardiorespiratory sleep studies in children.

Sleep Sonography has been proposed by Potsic,1989, as a reliable method for evaluating children with upper airway obstruction.

It has been shown to agree with PSG findings in children with OSAS. Although, it has the disadvantage of inability to distinguish central from obstructive apnea.

Radiology

A plain lateral X-ray of the postnasal space and upper airway is extremely useful in documenting adenoidal obstruction of the nasopharynx. It also shows the position of the lower jaw and tongue base in addition to outlining the airway.Radiography also has a place when obstrucion has been diagnosed but the etiology is obscure as it might well demonstrate an anomaly of the upper airway (Baily and Croft,1997).

Evaluation of Obstructed Sleep Apnea by Polysomnography Prior to Pediatric Adenotonsillectomy

Although there are probably a considerable number of patients in the pediatric age group who undergo Tonsillectomy and Adenoidectomy based on infection criteria alone. Yet, Adenotonsillar hypertrophy even with no history of infectious criteria is the most common cause of sleep related breathing disorder in an otherwise healthy child, in which Tonsillectomy and Adenoidectomy is the most common treatment.

Sleep related breathing disorders range in disease severity from primary or simple snoring through upper airway resistance syndrome (UARS)and at the severe end of the spectrum obstructive sleep apnea syndrome (OSAS), in which polysomnography is recognized as the most useful laboratory test to assess the presence and severity of the condition. Whether polysomnogroghy documenting OSAS should be obtained prior to tonsillectomy and adenoidectomy is challenging to many physicians.

Goldstein et al., 1997, prospectively evaluted 30 children of whom 18 were predicted to have OSAS. They conculded that the clinical assessment was sensitive (92.3%) but not specific (29.4%) for OSAS.

Using different PSG parameters, 82 children were evaluated by Wang et al., 1998, they reported that there was no significant association between clinical parameters and the presence of OSAS as defined by PSG; other four trials were conducted by Suen et al., 1995, Leach et al., 1992, Carroll et al., 1995; and Nieminen et al.,

1997. The authors of each trial concluded that clinical diagnosis was not accurate in predicting OSAS, when PSG was used as a criterion standard test. Suggesting that PSG should be used for all children prior to Tonsillectomy and Adenoidectomy(T & A) for OSAS to prevent unnecessary surgery.

However, there are 2 factors that must be considered when reviewing these studies. First, in several of these studies the definitions of apnea and / or OSAS that are used are appropriate for adults, but not for children, thus underdiagnosing OSAS. Second, in all the studies, children were not evaluated for UARS, a form of sleep related breathing disorder, which although milder than OSAS, can result in similar daytime symptoms. Children with UARS may benefit from T & A and may not be diagnosed as having UARS from results of PSG.

Childhood sleep – related breathing disorder currently is a clinical diagnosis based on the history, physical examination, and sometimes laboratory studies. Messner, 1999, proposed that it may be possible to predict a significant sleep – related breathing disorder by obtaining the medical history and a physical examination. Yet, the accuracy of the typical clinician's diagnosis of prediatric sleep – related breathing disorders (including both OSAS and UARS) in not known.

A serious problem with recommending that all children undergo PSG prior to T & A for obstructed night time breathing is the expense and availability of PSG, moreover insisting that all children with obstructive symptoms should undergo a PSG prior to T & A, would likely delay treatment for some affected children and possibly deny treatment to those with no access to PSG (Messner, 1999).

Nocturnal Enuresis

Enuresis is the repeated voiding of urine into the patient's clothes or bed; the voiding may be involuntary or intentional (Kaplan et al., 1994)

At all ages the enuretic population comprises a mixture of children who have always been wet and children who started to wet after a period of continence (primary and secondary enuresis). As many as 25% of preschoolars who achieve continence for at least 6 months will start to wet again (Oppel et al, 1968), and over half of 7-12 year olds enuretics have previously been dry for at least 6 months will start to wet again (Oppel et al, 1968); Secondary enuresis has its onset most often between the ages of 5-7 years and is uncommon after age 11 (Shaffer, 1994). The likelihood that a child will start to wet if he or she has stayed dry until age 7 is only about 1% (Oppel et al , 1968) .

Stehr et al, 1998, noted that enuresis must be diffrentiated from urinary incontinence in children, which is any kind of loss of urine without normal emptying of the bladder. Wetting in those cases is a symptom of a disease (structural, neurogenic, psychogenic or functional) while they considered enuresis as delayed development of bladder function.

According to DSM- IV the child must exhibit a developmental or chronological age of at least 5 years, and that behavior must occur twice weekly for a period of at least three months. DSM- IV breaks down the disorder into three types: nocturnal only; diurnal only and noctournal and diurnal (American Psychiatric Association

1994). The DSM- III- R types primary and secondary, depending on whether the disturbance was preceded by a year long period of urinary continence, have been removed from DSM- IV diagnostic criteria (Kaplan et al , 1994) .

Daytime wetting is more common in girls than boys . At age 5 about 1 in 6 boy and 1 in 3 girl nocturnal enuretics also wet during the day. These proportions fall by a half by age 7 (Järvelin et al, 1988). Daytime wetting alone or in combination with night wetting is associated with higher rates of psychiatric disturbance (Rutter et al , 1973) and genitourinary tract infection than nocturnal enuresis (Shaffer, 1994) .

Normal bladder control is acquired gradually and is influenced by neuromuscular and cognitive development, socioemotional factors, toilet training and possibly genetic factors. A new concept is put forward by El Hemaly, 1998, to explain the act of micturation and urinary continence. This depends mainly on, the presence of an intact, sound and strong internal sphincter, and an acquired behavior, gained by learning in early childhood, how to maintain a high alpha- sympathetic tone, thus keeping the internal sphincter closed all the time .

On desire and at the appropriate time and place, this acquired high alpha- sympathetic tone is inhibited, thereby opening the internal sphincter and allowing voiding to occur.

Fergusson et . al, 1986, found that a family history of enuresis was the most important predictor of delayed bladder control; Hublin et al, 1998, reported childhood enuresis probandwise concordance rate was 0.43 for monozygotic and 0.19 for dizygotic pairs. The proportion of total phenotypic variance attributed to

genetic influences (due to dominance), which confirm the central role of genetic liability in enuresis. The molecular genetic aspects of enuresis are attracting increased attention. Studies have substantiated the involvement of numerous loci on certain chromosomes,in excess of 10 chromosomes are thought to be involved (Djurhuns, 1999).

Linkage of nocturnal enuresis to loci on chromosomes 8, 12, 13 and recently 22 were reported (Von- Gontard et al ,1999) and provide support for genetic hypothesis.

Pondar et al, 1999, reported hypoexcitability of sphincter nuclei along with hyperexcitability of bladder motor nuclei, using electrophysiological techniques; minor dysfunction in the neurocontrol of lower urinary tract in at least a subgroup of enuretic children was postulated.

A number of studies (Esperanca and Gerrard , 1969 , Järvelin et al, 1990, 1991) have found lower maximum urinary voided volumes in enuretics than controls, although there was considerable overlap between both groups.

A cystometric study conducted by NØgaard , 1991, measured spontaneous bladder activity of enuretics during sleep, and concluded that bladder function is essentially normal , but that urine production was abnormally large.

Urine output is normally reduced at night to approximately one third of daytime rate. During the first year of life this rhythm is abscent and infants excrete urine at a constant rate (Shaffer, 1994).

Rittig et al, 1989, have studied diurnal variation of circulating vassopressin, urinary excretion rate and lower urine osmolality; enuretic children had a significantly less marked change in diurnal rhythm with higher nocturnal urinary excretion rate and lower urine osmolality; although Eggert et al, 1999, results contradict that enuretic children have an AVP (Arginine Vasopressin) deficiency, rather the results point to a defect at the AVP receptor level or of the signal transduction pathway.

Airway obstruction- induced bedwetting

Enuresis is described as a classic symptom of childhood upper airway obstruction. The mechanism of enuresis in patients with OSA is probably multifactorial.

Typically, patients with sleep apnea complain of frequent awakenings to urinate (Pressman et al, 1996) . The fact that a small subset of patients do not fully awaken to urinate, but rather have enuresis, suggests that there may be something abnormal with their arousal response. Perhaps the inordinately high number of arousals that sleep apnea patients experience may blunt their ability to fully wake up (Kramer et al 1998). Data from Berry et al , 1996, suggest that OSA itself (perhaps due to sleep fragmentation) decreases the arousal response to airway occlusion.

Krieger et al, 1998, demonstrated higher fractional urinary flows, higher fractional sodium and chloride excretion, and a lower percentage of filtered sodium reabsorption in patients with sleep apnea compared with those values in normal subjects. Treatment with nasal continuous positive airway pressure (CPAP) tended to normalize the renal function in patients with OSA.

A role for atrial naturetic peptide (ANP) in these changes in renal function is suggested (Lin et al, 1993). Negative intrathoracic pressure swings associated with respiratory effort against a closed airway are associated with increased venous return. In addition, the hypoxia associated with an apneic event may induce pulmonary vasoconstriction, which can cause right ventricular overload and right atrial distension. The subsequent atrial dilatation may lead to ANP release, and this increase in ANP levels enhances urinary excretion (Kramer et al, 1998).

In support of this, the work by krieger and his colleagues, 1991, which suggested that the ANP levels correlated with the degree of hypoxaemia as well as the degree of negative intrathoracic pressure associated with apneas.

Historically, a distinction has been made between primary and secondary enuresis and between nocturnal and diurnal enuresis. These distinctions are based on the presence (secondary) or absence (primary) of a significant period of bladder control, and whether it occurs with or without (nocturnal) daytime wetting. One of the implicit notions underlying the concept of secondary enuresis is that it is secondary to some psychosocial events and hence less likely to have a biological etiology (Mc Gee et al, 1984).

Bed-wetting is a distressing, and in some cases, stigmatizing condition. There is evidence of a dynamic link between psychiatric disturbance and enuresis, although psychiatric disorders are present in only a minority of enuretics, it is two to six times more common than in non enuretics (Shaffer, 1994) .

The association is strongest in girls, in children who wet during the day and in secondary enuretics (Mc Gee et al , 1984).

Systematic studies have failed to show a consistent or specific pattern for associated psychiatric symptoms (Rutter et al, 1973), yet number of symptoms have been thought to be specifically related to enuresis which include: tics, temper tantrums , nail- bitting, firesetting and cruelty to animals (Jacobson , 1985) . Diaz - Atienza et al, 1999, reported a 19.5 percent of the patients with enuresis presented a comorbid psychiatric disorder, which was significantly associated to both male gender in subjects younger than 9 and enuresis of the type secondary or mixed.Von Gontard, 1998, reported significantly higher rate of behavior disorders among bedwetting children, especially among day wetter and secondary nocturnal enuretics.

Enuresis has been linked to a variety of emotional and behavioral problems in pediatric, psychiatric and community samples.

The consistency of the reported prevalence of enuresis among children with ADHD (about 30%) found in various samples of both children (Bhatia et al, 1991; and Ornitz et al, 1992) and adults (Biederman et al , 1993) is striking. This consistency suggests that enuresis may yet turn out to be a developmental maker in children with ADHD.

In a longitudinal study of 161 Israeli kibbutz children followed from birth to age four, Kauffman and Elizur, 1977, found that children who had not attained bladder control by age four had more behavioral symptoms, and scored higher on tests of motor activity, aggression and dependency, and lower on achievement motivation.

Couchells et al, 1981, found that children seeking treatment for enuresis had more conduct problems and immature behaviors;

Feehan et al, 1990, extended these findings into early adolescent years showing a significant association between enuresis and measures of psychopathology by the age of 13 years.

Other interesting study conducted by Tapia et al, 1960, surveyed 830 families with children in the third grade, and found no association between enuresis and emotional disturbance, as rated by teachers, although parent ratings indicated much higher levels of behavioral disturbance among the children with enuresis. In the Isle of Wight study, Rutter et al, 1973, reported that children with enuresis showed behavioral deviance prior to their becoming enuretic, suggesting that children with enuresis have a prior emotional or behavioral disturbance .

However Rutter and associates also found that children with primary enuresis showed levels of behavioral deviance similar to children with secondary enuresis .

Biederman et al , 1995, showed that enuresis is not associated with psychosocial adversity or developmental immaturity, and that enuresis does not increase the risk for psychopathology in children; these findings were obtained after stratifying the children with enuresis by primary versus secondary and by nocturnal versus diurnal subtypes.

Yet Biederman et al, 1995, reported that enuresis was associated with an increased risk for learning disability, impaired intellectual functioning, and impaired school achievement.

Such findings would be compatible with enuresis being either a cause or a consequence of psychiatric disorder.

Several factors are common to both enuresis and psychiatric disorder.

Enuresis is more common in children living in broken or single-parent homes, in families in which there are extremes of poverty, repeated disruptions of maternal care, a reliance on welfare support, inadequate nutrition and clothing, and parental delinquency, in large crowded families and in children who have received institutional care (Rutter et al , 1973).

Umphress et al, 1970, found that parents of bed- wetters were less concerned over homemaking, wage earning or the mental and physical health of their children. Oppel et al., 1968, found that poor marital adjustment was significantly more common amongst the parents of girl enuretics. Most of these factors are known to be associated with psychiatric disorder in childhood. Shaffer, 1994 proposed that it could be that families with these characteristics have child rearing practices that are conductive to the persistence of wetting, but that other aspects of their lives predispose to psychiatric disturbance.

It is also possible that there is shared relationship with biological mechanisms.

Investigating Enuresis

No single laboratory finding is pathognomonic of enuresis. A detailed anamnesis is the most important diagnostic tool in enuresis, whereas in the case of urinary incontinence a lot of diagnostics from non- invasive to invasive have to be performed (Stehr et al , 1998).

Frequently associated conditions such as developmental delay, learning difficulties, urinary tract infection, constipation and concurrent psychiatric disturbance should be ruled out. (Shaffer 1994).

Shaffer, 1994, recommended that the routine investigation of enuretic children should comprise four components, structured interview, physical examination urine analysis and ultrasound investigation.

Attention Deficit Disorder

Children who display extreme levels of attention deficits and hyperactivity have been noticed, bemoanded and stigmatized for centuries. Yet only since the advent of compulsory education, which mandates self-controlled behavior in large group settings, have children with such difficulties emerged in sufficient numbers to receive systematic inquiry (Hinshaw, 1994).

Definition

Attention deficit can refer to behavioral trait, a pattern of test performance or a hypothesized deficit in a psychological process (Taylor, 1994).

Clinical Features

The predominant perspective in today psychiatric community is that categories of disordered behavior exist.

Inattention is a central characteristic of this disorder (Weiss and Hechtman, 1993), but it does not completely define attention deficit disorders; hyperactivity is an important characteristic that can refer to either a dimension or a category of psychopathology (Goodman and Stevenson, 1989) that is characterized by an increased tempo of normal activities, an increase in purposeless, minor movements that are irrelevant to the task in hand, or an amount of movement of the whole body that is excessive for the situation (Taylor, 1994).

Impulsivity is another hallmark of the condition which is characterized by acting before weighing alternative responses (Kendall and Braswell, 1985).

Diagnosis

DSM-IV criteria for attention deficit disorder reflect a considerable body of past research, as well as specific investigation performed in field trials held specifically to develop the nosology (Hinshaw, 1994), as it has reintroduced the distinction between those with symptoms of motor overactivity and those without, that is similar to DSM-III major scheme (American Psychiatric Association, 1980).

An additional criterion in DSM-IV that was not present in the revised third edition of DSM (DSM-III-R) is the presence of symptoms in two or more situations. However, changes from DSM-III-R and DSM-IV resulted in minimal changes in case identification, providing support for diagnostic continuity between the two classification systems (Beiderman, 1997).

ICD-10 and DSM-IV are very similar in the behaviors that are considered to be the basis for the diagnosis. They vary, however, in the rules for weighting them.

Impulsiveness and hyperactivity are pooled in DSM-IV. In ICD-10, impulsiveness and hyperactivity are considered separately.

Accordingly, DSM-IV lists three subtypes of attention – deficit / hyperactivity disorder: predominantly inattentive type, predominantly hyperactive impulsive type and combined type.

Recently, Jensen et al., 1997, suggested the delineation of two new subclassifications of ADHD (1) ADHD aggressive subtype and (2) ADHD anxious subtype; another study conducted in Japan by Inoue et al., 1998, classified ADHD into eight subtypes by combining scores of Actigraph and CPT: hyperactive – impulsive; hyperactive – inattentive; impulsive – inattentive, hyperactive, impulsive, inattentive, mixed and unspecified.

Epidemiology

ADHD comprises a major public health problem with respect to both physical and psychological well-being.

Epidemiological studies have identified populations with striking differences in prevalence (Hinshaw, 1994); reports in the United States have varied from 2 to 20 percent of grade - school children (Kaplan et al., 1994); yet the most widely repeated figure is the 3.5%, suggested by the DSM manual (American Psychiatric Association, 1980, 1987); In Great Britain, the incidence is reported to be lower than in the United States, less than 1 percent (Kaplan et al., 1994); this reflects chiefly differences in diagnostic practices.

Etiology

Intensive research has been in progress for decades to clarify the etiology of ADHD, and recently there has been growing interest in the genetic basis of ADHD.

Family studies suggest that ADHD is a highly familial condition (Mc Guffin et al., 1994); However, this may be explained

by shared environmental factors such as social disadvantage, as well as by genes.

The methodological design of twin studies consistently report higher correlations in hyperactivity / inattentive scores for MZ twins rather than DZ twins (Thapar et al., 1999), which indicates that genetic factors account for a substantial amount of variance.

There is thus a compelling argument for now searching for susceptibility genes at a molecular level.

An empirical rationale for the current interest in candidate genes involved in dopaminergic pathways is largely due to the rapid symptomatic improvement of ADHD children with methyl phenidate, which increases extracellular dopamine levels (Thapar et al., 1999).

To date, genes encoding the dopamine transporter (DAT 1) and dopamine DRD-4 receptor have both been implicated, findings emerging from molecular genetic studies of ADHD look promising (Thapar et al., 1999).

Pennington et al., 1997, suggested one plausible theory of brain mechanisms in ADHD as follows: The symptoms of ADHD are caused by functional hypofrontality, which is caused by either structural and / or biochemical changes in the prefrontal lobes, or closely related structures such as basal ganglia, and is detectable as reduced frontal blood flow.

Biochemically, the cause would be low dopamine levels; yet Oades and Muller, 1997, argued that changes in the balance

between dopamine and serotonin systems may contribute to abnormal cognitive development in ADHD.

Oades, 1987, previously argued the combined action of dopaminergic and nonadrenergic systems in the biology of ADHD. It seems no single neurotransmitter is exclusively involved in pathogenesis of ADHD.

Obviously researchers have given less attention to the psychological environment than they have to the working of the brain.

Poverty, bad housing and low socioeconomic status have been investigated several times but their role is still uncertain (Taylor, 1994). While adversity in close personal relationships has a robust association with hyperactive behavior ; impulsivity and hyperactive behavior characterized youngesters for whom familial interactions are instigating (Bauermeister et al., 1992). Yet implicating such psychological factors as primary causes is fraught with difficulty.

Assessment

Assessment of children with attention deficits and hyperactivity can not take place in the absence of some overarching conceptions about the nature of the disorder and of child psychopathology in general – or without consideration of key psychometric, developmental and theoretical issues (Hinshaw, 1994).

Rating instruments are perhaps the most widely utilized assessment tools for the evaluation of youngesters with attention

problems and hyperactivity, as they constitute an ideal "first wave" of assessment. Broad scales related to wide conceptions of child psychopathology provide a comprehensive picture of behavioral and emotional functioning. Child Behavior Checklist (Achenbach, 1991) is the paradigmatic rating scale for the field; other broad scales in wide use are the Revised Behavior Problem Checklist of Quay and Peterson, 1983; the Conners Parent Rating Scale and the Conners Teacher Rating Scale (Goyette, Conner and Ulrich, 1978).

Narrower scales related solely to externalizing behavior: Conners Abbreviated Symptom Questionnaire, which is a 10 item scale; Du Paul ADHD Rating Scale (Du Paul, 1990) for assessment of ADHD children ages 2-11 years which is composed of 14 items, they are well suited for repeated assessments.

Interviewing is the corner stone of child clinical assessment. Unstructured clinical interviews; despite their flexibility, are quite unreliable with respect to obtaining a diagnosis; on the other hand, structured interviews are particularly helpful for ascertaining a definitive diagnosis of ADHD,as well as the full range of possible comorbid diagnosis that may accompany ADHD (Hinshaw, 1994).

Semistructured interviews regarding parental impressions of child's developmental history are of critical importance for ascertaining key information about motoric, cognitive and language performance earlier in the child's history (Barkley, 1990).

Behavior observation is the most direct form of assessment. Although quite costly and difficult to coordinate logistically, systematic behavioral observations in the natural environment yield the potential advantage of precise and objective evaluation (Gelfand et al., 1997).Cognitive testing that is helpful in confirming

the child's inattention and impulsivity, includes the continuous performance task (CPT) (Kaplan et al,1994). Although impaired overall level of performance in ADHD children porbably has multiple causes, among which motivation, non compliance,IQ and age may be major ones, and attention may play a relatively minor role(Koelega,1995).

Comorbidity

Since the introduction of DSM III / III- R; clinicians and investigators have shown increasing interest in the study of conditions comorbid with attention deficit disorders (Jensen et al., 1997)

The largest available body of literature concerned the comorbidity with ADHD and conduct disorder / aggression was found in Jensen et al., 1997, review of ADHD literature from the past 15 years, in which rates of comorbidity range from 30% to over 50% in both clinic – referred and community samples (Hinshaw, 1994).

Still learning disabilities are comorbid with ADHD at rates well above chance with recent figures ranging from less than 10% to approximately 25% (Semrud – Clickeman et al., 1992).

Disruptive Behavior Disorders

With the establishment of clinics attached to juvenile courts and the ascendancy of the mental hygiene movement, tension emerged between those who regarded delinquent youths as budding psychopath and those who believed their behavior to be reactive to adverse circumstances.

As the profession moved beyond court clinics into community and medical settings, as younger children were seen in these contexts, and a more bengin and developmental perspective began to replace notions of constitutional inferiority.

It was in these contexts that new diagnostic concepts evolved. Firstly, conduct disorder and later oppositional disorder were introduced into the lexicon of child psychiatrists.

The term "disruptive behavior disorder" is used to cover any disorder of antisocial behavior as oppositional defiant disorder and hostile sibling interactions are part of the spectrum of distruptive behavior disorder (Spender and Scott, 1996).

A group in Sydney, Australia, found that disruptive behavior disorder in adolescence predict antisocial and other personality disorders in early adulthood (Rey et al., 1995). Moreover, in contrast to previous finding that life event lead to psychiatric disorder, a group in London , UK, showed that behavior problem in 10 year old children could predict life stressors and difficulties in early adult life, suggesting that disorder can also trigger events (Champion et al., 1995).

Oppositional defiant disorder

DSM-III (American Psychiatric Association, 1980) offered the first definition of oppositional disorder as part of an official nomenclature. In the revision of this manual, DSM-III-R (American Psychiatric Association, 1987) the list of symptoms was expanded from 3 to 10 symptoms.

Although ICD-9 (WHO, 1978) did not include oppositional disorder among its categories, ICD-10 has done so ,in keeping with the original plan to make ICD-10 and DSM-IV identical, the same list of symptoms and diagnostic criteria for oppositional defiant and conduct disorder were adopted (Earls, 1994).

The core of this disorder is negativistic, defiant, disobedient and hostile behaviors toward authority figures (Gelfand et al., 1997) in the absence of serious violations of societal norms; the child's temper outbursts, active refusal to comply with rules, and annoying behaviors exceed the expectations for these behaviors compared with others of the same age. (Kaplan and Sadock, 1998).

Etiology

A person's asserting his or her own will and opposing others' will is crucial for normal development. Normal oppositional behavior peaks between 18 and 24 months; pathology begins when this developmental phase persists (Kaplan and Sadock, 1998).

Oppositional defiant disorder probably has several different causes, there is probably no single element, but instead is caused by an interaction between many factors.

Gelfand, et al., 1997, suggested inhereted tempermental characteristics such as lowered levels of anxiety, inhibition and fear to be problematic.

School and family difficulties rise to prominence in cases of oppositional defiant disorder, hence the authority imposed by home and school may help to set the tone for the genesis of the disorder. (Adams and Fras, 1988).

Behaviorists have assumed coercive behaviors as the origin of opposionalism, coercive behaviors such as tantrumming, teasing or aggression are learned through modeling and reinforcement of the behavior (Gefland et al., 1997).

In late childhood, environmental traumata, illness or chronic incapacity may trigger oppositionalism. as a defense against helplessness, anxiety and loss of self-esteem (Kaplan and Sadock, 1998).

Epidemiology

Although oppositional defiant disorder can begin as early as 3 years of age, it typically begins by 8 years of age (Kaplan and Sadock,1998)

Age and gender patterns for oppositional defiant disorder suggest developmental stage–associated risks,as Cohen et al., 1993, showed in their population based study low levels among the 10 to 11 year olds with prevalence rates for boys and girls 14.2% and 10.4 % respectively, rising to high levels among 13 to 16 year olds with prevalence rates 15.4% for boys and 15.6% for girls, and a

sharp fall there after, suggesting that the disorder is more prevalent in boys than in girls before puberty (Kaplan and Sadock, 1998).

An overall estimate of negativistic traits in non clinical population of school age children ranges from 16-22 percent, with no distinct family pattern (Kaplan and Sadock, 1998).

Oppositional defiant disorder and conduct disorder are two closely related syndromes.

Both ICD-10 and DSM-IV view oppositional disorder as a milder and developmentally related form of conduct disorder (Earls, 1994) or a precursor to the more serious forms of conduct disorders, especially when the effects of negativistic, defiant behaviors are considered (Gelfand et al., 1997).

Biederman et al., 1996, in their study to evaluate the overlap between oppositional defiant disorder and conduct disorder, found that both disorders are part of the same disease process and suggested two subtypes of oppositional defiant disorder: one that is prodromal to conduct disorder and another that is subsyndromal to conduct disorder but not likely to progress to conduct disorder in later years.

Conduct Disorder

Over the past 2 decades, there has been a proliferation of research into the etiology and treatment of conduct disorder. This has been fuelled by increasing awareness of the social and financial costs of delinquency.

The essential feature of conduct disorder is a repetitive and persistent pattern of behavior in which either the basic rights of others or major age appropriate societal norms or rules are violated (Kaplan et al., 1994).

The criteria in the fourth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) are similar to those in the revised third edition (DSM-III-R).

Although DSM-IV has enlarged the list of potential symptoms from 13 to 15, DSM-III-R divided conduct disorder into three subtypes: group type, solitary aggressive type and undifferentiated type.

Yet DSM-IV divides conduct disorder into two types with respect to age of onset; childhood and adolescent onset types. Tolan, 1987, separated delinquent youths into early – under 12 and later – onset groups, and found that the earlier-onset group reported 3 ½ times the rate of self-reported delinquency as the later-onset group. Other studies tracing onset back as far as the pre school period have also found a higher rate of antisocial behavior (Loeber et al., 1990) indicating that early activation is related to a more aggravated course than later onset, also DSM-IV labels severity ranging from mild, moderate to severe (Kaplan et al., 1994).

On the other hand, both ICD-9 and ICD-10 include socialized and unsocialized types of conduct disorder (Earls, 1994).

Both ICD-10 (World Health Organization, 1992) and DSM-IV (American Psychiatric Association, 1994) require the presence of at least three of 15 symptoms.

The integration of a common list of symptoms which has become a feature of both DSM-IV and ICD-10 permits an improved description of children in a transitional state between conduct disorder and oppositional disorder.

Assuming the developmental link between the two disorders.

Etiology

Although no single factor can account for children's antisocial behavior or conduct disorder. There is increasing evidence of genetic influences on antisocial behavior. Ratings of aggressive behavior in early and middle childhood showed a consistently high monozygotic and dizygotic discordance, with evidence that the same genes are operating at both time points (Schmitz et al., 1995).

Assuming a heritable component exists, functions of central nervous system especially that of its physiological processes may be under a degree of genetic control; Raine et al., 1990, observed the association between uninhibited temperment and sluggishness of the autonomic nervous system, characterized by low heart rate, decrease amplitude and slow recovery of skin conductance and excessive slow wave activity in the electroencephalogram.

Other plausible indicators of a physiological basis for antisocial behavior relate to central neurotransmitter system, low levels of platelet monoamine oxidase and plasma dopamine beta-hydroxylase characterized the profiles of boys with both attentional and conduct disorders (Earls, 1994). Pliska et al., 1988, reported that some conduct disordered juvenile offenders have increased blood serotonin (5 – hydroxytryptamine (5-HT)) levels.

Yet Moffitt and Henry, 1991, have suggested that impaired frontal lobe functions produce many of the deficits seen in chronic offenders, such as the inability to plan, to redirect potentially harmful behavior and to learn from negative consequences of their actions.

Aside from genetic factors, a host of environmental insults could contribute to the cognitive deficits detected on neuropsychological measures. These include, but are not limited to, prenatal exposure to alcohol and drugs, absorption of heavy metals such as lead, injury to and infections of the developing brain (Earls, 1994); yet this is not the complete picture as sociological studies showed that poverty and low socioeconomic status have important links with conduct disorder (Smith, 1995).

Beyond the effects of social stratifacion, there is profound effects of parental neglect and poor quality of parenting. Spender and Scott,1996, reported parental psychiatric disorder, marietal breakdown, criminality, large family size, inaddition to high rates of deviancy in the community to be associated with conduct problems.

Scerbo and Kolko, 1995, found that physical abuse in childhood is a predictor of aggressive behavior. However, they suggested that aggression follows from physical abuse only if the abuse has caused emotional distress.

Children brought up in a chaotic negligent conditions generally become angry and demanding and as their role models are poor and often frequently changing, the basis for developing both ego - ideal and a conscience is lacking (Kaplan et al., 1994).

McArdle et al., 1995, suggested that there are different aetiological paths to conduct disorder at different ages, Schmitz et al., 1995, in their study to estimate importance of genetic and environmental influences on problem behavior in children showed that shared environmental influences may be more important in early childhood than in middle childhood, while the reverse holds for genetic influences; indicating the importance and utility of a developmental perspective in research into child psychopathology.

Epidemiology

Conduct disorder was found to be by far the most common disorder in males in the Isle of Wight study, which was one of the first investigations to estimate the prevalence of conduct disorder in general population (Rutter et al., 1970).

More recently, the Ontario Health Survey has provided a comprehensive picture of conduct disorder in the general population (Offord et al., 1991) the overall rate of conduct disorder 5.5% was similar to the rate reported 25 years earlier in the Isle of Wight study. The male to female ratio was also similar, 8.2% for males and 2.8% for females, with urban areas slightly higher than the rural rate: 5.6% compared to 5.2%.

Cohen et al.,1993,reported conduct disorder to be about as twice as prevalent in boys as in girls. However the shapes of the prevalence curves were significantely different.For boys the prevalence was highest in the younger age group .For girls, in marked contrast, an increase in prevalence to a peak age 16 was followed by a sharp decline.

McArdle et al., 1995, in their population - based study of two different age groups showed 12.8% prevalence of conduct disorder in juniors7-8years old and 5.3% in seniors 11-12 years old.

Assessment

Clinicians faced with the evaluation of a child with oppositional or conduct disorder should view the diagnosis as only one component of an overall process. As a first step in assessment, the various manifestations of antisocial behavior must be recorded along with other symptom areas (Earls, 1994).

The theoretical assumptions about the types of information gathering differ widely. Observations, checklists and curriculum based measurement generally sample behavior directly. Personality inventories, projective tests and some intelligence tests are assumed to measure underlying traits or construct which control behavior across different situations and time. The direct sample techniques value observable behavior, and the trait techniques value underlying traits and consider observable behavior as only a reflection of these underlying traits (Gelfand, 1997).

In behavior checklists child's behavior is scored along several predetermined dimensions, factor analysis techniques are commonly used to identify behavioral dimensions included in checklists (Gelfand, 1997).

General problem behavior checklists such as the Child Behavior Checklist (CBCL); (Achenbach et al. 1989) and the Revised Behavior Problem Checklist (RBPC; Quay, 1983).

The two methods include nearly the same number of items (38 and 39) and considered to be two of the most widely used in current research (Earls, 1994).

Recently two scales were developed to be completed by clinicians: the Paddington Complexity Scale (PCS) (Yates et al., 1999) and Health of the Nation Outcome Scale for Children and Adolescents (HoNOSCA) (Gowers et al., 1999) in which both scales are useful to describe clinical intake to the child and adolescent mental health service. Although PCS requires staff to be trained in making psychiatric diagnosis and using a multiaxial perspective, HoNOSCA was designed to assess outcome, and it has the advantage of measuring changes (Gowers et al., 1999).

Specific checklists such as Child Depression Inventory (Kovacs, 1981), Social Skills Checklists (Demaray et al., 1995) are used to identify distinct behavior problems in children.

The advantage to most behavior checklists is that they have good validity especially if they are derived using multivariate statistics. In addition, most behavior checklists have good reliability characteristics (Gelfand et al., 1997).

Interviewing has been called the clinician's basic technique and is considered to be the corner- stone of child clinical assessment.

It is preferable to interview the child separately since the nature of conduct disturbances are such that they are more likely to be of concern to others than do to the patient (Earls, 1994). The evaluation should include a search for diagnosable and possibly

treatable diseases of the CNS, in addition, to a neurological examination (Earls, 1994).

A neuropsychological assessment is critically important to the overall evaluation including measures of intellectual capacity, language development, reading and arithmetic skills(Gelfand et al,1997).

Depressive Disorders

Until relatively recently, it was widely believed that depressive disorders resembling adult depression were extremely uncommon in young people, due to the immaturity of their psyche. However, in the past 15 years there has been an increasing recognition that conditions meeting the full criteria for major depression in adults occur in children (Harrington et al., 1993).

Despite these developments, there is still much controversy surrounding the idea that depression in children represents the same psychiatric condition as depression in adults, as young children differ from adults in their abilities both to experience and to report certain features of depression (Rutter, 1986).

Toolan, 1981, showed that the manifestations of depression in children differ depending on their age and developmental stage, as younger children with age ranging from 5 – 8 years verbalize feelings of sadness more directly, whereas older children (8 – 12) years old are more likely to show increasingly poor self esteem. As children pass through adolescence, guilt themes become more prominent and the depressive syndromes resemble those of adults; this variation in the expression of disturbed mood in childhood is probably due to three developmental issues: the substantial age differences in the occurrence of most forms of affective phenomena (Rutter, 1986). Secondly, children differ from adults in their ability to experience some of cognitive features said to characterize adult depression such as guilt (Rutter, 1986). Thirdly, the valid application of adult criteria to children requires not only that they are capable of experiencing depression, but also that they can report it accurately (Rutter, 1986).

Several investigators suggest that it might be better to identify age-appropriate symptoms of depression that take into account the child's level of functioning in the various cognitive and affective domains (Harrington, 1991).

While the cardinal symptoms of mood disorders persist across all ages, manifestations of mood disorders may vary with age (Alyward, 1985). Depressed infants, as may be seen in neglectful or abusive situations, often have eating and sleeping difficulties, failure to thrive, more crying and self-injurious behavior, such as head-banging. Depressed preschoolers exhibit more behavioral problems, irritability, withdrawal, apathy and regression. School-aged children display sad, irritable or depressed mood, sad facies, crying spells and lack of pleasure. Adolescents often describe mood symptoms similar to adults or "atypical" depressive symptoms such as hyperphagia, hypersomnia, and excessive fatigability (Kaminiski et al.., 1995).

Diagnostic Criteria

Diagnostic criteria for mood disorders now appear stable across the lifespan, and are described in the Diagnostic and Statistical Manual, 4th Edition (DSM-IV) (American Psychiatric Association, 1994), with a primary care, pediatrician – friendly version now avaliable (Bostic et al., 1997).

DSM-IV differentiates between mood disorders in which there may or may not have been episodes of mania (bipolar disorders and depressive disorders respectively). The two major mood disorders are major depressive disorder and bipolar I disorder.

Two additional mood disorders which are chronic in course and with less severe symptoms are dysthymic disorder and cyclothymic disorder (Kaplan et al., 1994). DSM-IV has codified additional mood disorders related to depression namely: Minor depressive disorder; recurrent brief depressive disorder and premenstrual dysphoric disorder, in which there is functional impairment, although symptoms are less severe or less in duration.

The diagnostic criteria in the fourth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) for major depressive disorder, dysthymic disorder, and bipolar I disorder are the same for children and adolescents as they are for adults, with some minor modifications (Kaplan and Sadock,1998). The modifications in the criteria for childhood and adolescence major depressive disorder include irritable mood and failure to gain expected weight. In dysthymic disorder; also irritable mood can replace depressed mood, and the duration criterion has been modified to one year instead of two years in adults. The criteria for bipolar I disorder are the same for children and adolescents as for adults.

ICD-10 (World Health Organization, 1992) makes an explicit distinction between unipolar and bipolar disorders, and also ICD-10 distinguishs between milder forms of affective disorders and severe affective disorder which are mostly included under mood disorders (Harrington, 1994).

There is however, a separate childhood category for mixed disorders characterized by both conduct disorder and depression (depressive conduct disorder). ICD continues to be different from DSM in its use of such combination categories.

Epidemiology

Mood disorders in children and adolescents have received increasing recognition and attention over the past few decades, however uncertainties surrounding the concept of depression in young people and understandardized methods of assessment led to huge variability in the rates of depressive disorders that were found in early studies (Harrington, 1994). Although recent epidemiological surveys have also had methodological problems, yet the use of similar diagnostic criteria together with more comparable methods of data collection has lead to greater consistency in estimates of the prevalence of current rates in the range from 0.5 – 2.5 % in preadolescents (Velez et al.., 1989).

Lewinsohn et al., 1994, reported prevalence of major depressive disorder of approximately 2% of children, and up to 5% of adolescents, dysthymia in 2% of children and 8% of adolescents; this goes with a recent study conducted by Abd El-Ghany, 1997, who showed prevalence of mild depressive symptoms in 53.66%, moderate depressive symptoms 43.31% and prevalence of severe depressive symptoms in 3.03%.

There are also strong age trends for depression – related conditions such as suicide which shows a huge rise over the adolescent years (McClure, 1988).

On the other hand, Carlson, 1979, reported a 16% occurrence rate of severe depression in pediatric population. Another study conducted by Earls, 1984, confirmed an increase in the rates of depression from middle childhood through life to adulthood, adolescent's rates of depression remain constant being 8.6%.

Epidemiological studies of adolescents have generally reported higher prevalence with current rates of major depression ranging from 2.0 – 8.0% (McGee et al., 1992), indicating that mood disorders increases with increasing age.(Kaplan and Sadock, 1998).

Some studies showed that there is equal sex distribution of depression in children (Velez et al., 1989); other studies confirmed male preponderance (Anderson et al., 1987). Cohen et al., 1993, reported different prevalence patterns for boys and girls. In late childhood , rates were low and comparable for the two genders, and this prevalence rate remained approximately at the same levels throughout the adolescent years for boys; however in girls a very sharp increase in prevalence of major depressive disorder was present in the immediate post-puberty years, reaching up to 14% . In the older adolescent years the rates of major depressive disorder were effectively the same for boys and girls. In contrast to Angold and Rutter, 1992, who suggested that the switch begins at round the age of 10 years, with rates of depression increasing steadily in both boys and girls, but with the increase being more marked in girls.

Etiology

Interest in the genetics of depressive disorders arising in childhood has been stimulated by data from different studies, the most important was the cross-sectional and longitudinal studies of children of depressed parents which have found that they have greater than expected rates of depression (Weissman, 1992). However, this risk seems non specific as non depressive symptoms are increased as well. Other studies reported high rates of affective disorders among relatives of depressed children probands, A recent study was conducted by Harrington et al., 1993, reported that the lifetime prevalence of depression in first degree relatives of

depressed children proband was significantly higher than depression in relatives of control children, and that the higher rates of depression were found to be among the female relatives of both control and depressed probands.

Goodyer et al., 1993, proposed that the increase in the life time prevalence of depression in relatives of depressed children may be due to that these families become life event-prone as a result of parental psychopathology. Yet family studies cannot discriminate effectively between genetic and environmental mediation; the best discriminator is the twin study. However, the validity of the twin method depends on the equal environment assumption – that monozygotic (MZ) and dizygotic (DZ) twins are equally correlated in their exposure to environmental factors of aetiological importance for the disorder. Kendler and Gardner, 1998, found that differential environmental experience of MZ and DZ twins in childhood are unlikely to represent a substantial bias in twin studies of major depression. However, the finding that identical twins do not have a 100 percent concordance rate suggests a role for non-genetic factors (Kaplan and Sadock, 1998).

Research on the psychosocial correlates of childhood depression is at an early stage (Harrington, 1994). Some researches showed the impact of parental depression on the child and its influences on the child's environment (Rutter, 1990). Yet this path may be bi-directional as child characteristics can elicit negative parental reactions, which in turn increases the risk for psychopathology in the child.

Wickramartne and Weissman, 1998, showed that parental major depressive disorder is associated with eight fold increased risk in offspring of childhood – onset MDD, but not adolescent

onset MDD, especially when the parent had an early –onset depression (Before age of 30 years).

Brown and Harris, 1978, identified three kinds of risk factors: acute life events, chronic adversities and vulnerability factors.

It was found that in depressed young people acute life events often occurred in the context of long-standing problems. Although, the relationship between stress and the type of emotional disorder that occurs in the child appears to be non-specific as in the presence of acute adversity children are equally likely to become anxious as they are to become depressed (Goodyer et al., 1988).

Depressive symptoms have been found in association with many types of adverse life experiences such as divorce (Aro and Palosaari, 1992), disasters (Yule et al., 1990), beareveement (Kranzler, 1990) and physical and sexual abuse (Goldston et al., 1989). Although multivariate analysis suggest that depression is unlikely to be a direct consequence of childhood sexual abuse (Goldstone et al., 1999).

Vulnerability factors is still conceived as only raising the risk of depression in the presence of a provoking agent (Brown and Harris, 1978).

Many psychological models have been devised to explain the links by which external stresses lead to the internal mood state of depression. At the root of the psychodynamic formulation of depression was Freud's, 1957, idea of the actual or perceived loss

of the love object. This loss was followed by self-rejection and self criticism, the anger toward the parent is turned inward.

Recently Kohut, 1971, redefined depression in term of self psychology, when self object needs for mirroring, twin-ship or idealization are not forthcoming from significant people; the depressed person feels a sense of incompleteness and despair as not receiving the longed for response. Seligman, 1975, introduced the concept of "Learned helplessness" positing that when people cannot influence events in their lives that very experience or expectation leads to depression.

The reformulated learned helplessness theory, now called the hopelessness theory (Abramson et al., 1989), and has many similarities with the so-called cognitive theories of depression.

The occurrence of such cognition has been documented in several cross-sectional studies of depressed children, whose distorted style of processing self-evaluative information distinguished them from children with other psychiatric disorders (Kendall, 1992).

A recent psychological construct is the competence theory, which provides evidence of an association between perceived incompetence and depressive symptoms (Adams and Adams, 1991).

Studies of prepubertal major depressive disorder and adolescent mood disorders have revealed biological abnormalities (Kaplan et al., 1994). Deakin and Crow, 1986, proposed that depression results from hypoactivity of monoamine reward systems.

Several studies of young people with depressive disorders have reported abnormalities of the biological markers that are thought to reflect the activity of these systems (Yaylayan et al., 1992).

A recent study conducted by Arborelius and Coworkers in 1999, showed that Corticotrophin-Releasing Factor (CRF) is hypersecreted from hypothalamic, as well as, from extra hypothalamic neurons in depression, resulting in hyperactivity of the Hypothalamic-Pituitary-Adrenal (HPA) axis and elevations of cerebrospinal fluid concentrations of CRF. This increase in CRF neuronal activity is also believed to mediate certain behavioral symptoms of depression, involving sleep and appetite disturbances and psychomotor changes. Other variety of neuroendocrine dysregulations have been reported in young people with depressive disorders such as secretion of growth hormone after hypoglycaemia (Puig-Antich et al., 1981), and thyroid releasing hormone test (Kutcher et al., 1991).

The major implicated transmitters are monoamines, indoleamines and catecholamines, with indications that deficits in these agents would produce depression, whereas excesses would produce mania. It should also be borne in mind that early-onset depressive conditions are likely to be biologically heterogeneous (Goodyer et al., 1991). Noradrenaline, acetylcholine and serotonin in certain levels have all been indicated as present in those suffering from depression. These agents regulate neuroendocrine levels controlling hormone production and in some cases pituitary functioning.

Assessment

Depressed children usually have multiple problems, such as educational failure (Forness, 1988), impaired psychosocial functioning (Puig-Antich et al., 1985), and comorbid psychiatric disorders. Moreover, they tend to come from families with high rates of psychopathology and may have experienced adverse life events (Goodyer et al.,1988) including maltreatment (Kaufman, 1991). All these problems need to be identified and assessed.

Assessment of depression in young people should start with a thorough evaluation of depressive symptomatology. Efforts to improve the reliability and validity of the diagnosis have been focused on two areas: the development of standardized assessment instruments and biological tests.

Accounts from children and parents can be supplemented by information from other sources such as teachers and peers and / or direct observations (Harrington, 1994). It is not enough to rely on accounts obtained from parents alone, as children usually give a better account of symptoms related to internal experience.

Several standardized interviews have been devised for use with children and many of them will generate depressive diagnoses, as a result, structured psychiatric interviews are being used more as a diagnostic tools in clinical settings (Harrington, 1994).

The most frequently identified instruments in the literature are the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Chambers, 1985) which include both parent and child versions. This semi-structured interview presents multiple items that are keyed to DSM-III.

A more structured interview is the Diagnostic Interview Schedule for Children-Revised (DISC-R) which is applicable to children between 8 and 17 years of age, with parent and child version. Nevertheless, there are several unresolved difficulties: The first one is the test-retest reliability as affective symptoms are particularly unstable in the younger age group. The second is the low agreement between parent and child on depressive symptoms (Barret et al., 1991).

Although the advantage of using a structured interview in that information that might otherwise to be overlooked or minimized is collected in a comprehensive way. Structured interviews cannot take the place of clinical interviews as they do not adequately address the chronology of symptoms, nor the interplay between environmental stressors and emotional response (Kaplan et al., 1994).

Self report questionnaires provide a convenient way of screening for symptoms, that are not part of the presenting complaint, and may be helpful in monitoring subjective feelings (Harrington, 1994).

In research settings, questionnaires have been used both as primary source of data and as a screening instrument to select subjects for further in-depth interviews (Harrington, 1994). The most currently used appear to be the Children's Depression Inventory (CDI; Kovacs, 1981) which is designed to assess cognitive, behavioral and affective signs of depression.

A number of factors need to be considered in selecting an instrument to assess depression among the young, as some of these scales may be used as discriminator between depressed and non

depressed children, or as a measure for change in the course of treatment of depression (Costello and Angold, 1988).

As measurement of mood state by the Mood Adjective Checklist which measures 8 mood constellations namely: calm, anxious, sad, confused, resentful, angry, expectant and happy (Plutchick, 1989).

The Suicide Risk Scale (SRS) is a self report instrument that include items on past history of suicide attempts, present strength of suicidal impulses, feelings of depression and hopelessness (Plutchik et al., 1989).

Another development has been the use of psychobiological measures as markers of depressive disorder (Harrington, 1994).

Although no single laboratory test is useful in making a diagnosis of a mood disorder (Kaplan et al., 1994), Dexamethasone-suppression test (DST), was initially thought to be of diagnostic usefulness, however, several recent studies have shown that in both children and adolescents the DST is a poor discriminator between depressed and non depressed cases (Tyrer et al., 1991).

Indeed, one study of depressed adolescents found abnormalities of nocturnal growth hormone secretion (Kutcher et al., 1991).

Yet screening test for thyroid function can rule out the possibility of an endocrinological contribution to a mood disorder (Kaplan et al., 1994).

Comorbidity

DSM-IV and ICD-10 take different approaches to the overlap between depression and other child psychiatric problems. In DSM-IV there is no separate category for conditions characterized by two problems. Rather, it is assumed that comorbidity between depression and other psychiatric conditions represents the co-occurrence of separate disorders. By contrast, in the ICD-10 the expectation is that mixed clinical picture is more likely to mean a single disorder with varied manifestations (Harrington, 1994).

It seems that most children who meet research criteria for depressive disorders are given a primary diagnosis of other disorders this has been consistent finding from research population.

In Cohen et al., 1993, epidemiological study diagnostic overlap was greater between major depressive disorder and disruptive disorders (62% of cases with depression).

Harrington et al., 1991, reported an association with conditions as diverse as conduct disorder, while major depressive disorder and the anxiety disorders reported 43% diagnostic overlap in depressed children in the study conducted by Cohen et al., 1993. In contrast to Anderson et al., 1987, who reported in their study that anxiety disorders were the major comorbid disorder; followed by attention deficit disorder and conduct disorder. McGee et al., 1990, reported that adolescents with depressive disorders seem to be more likely to have an additional psychiatric conditions than depressed adults.

Finally, it should be borne in mind that depressive disorders may present not only with non depressive psychiatric symptoms but also with somatic complaints. Conversely physical conditions or endocrine disorders may be associated with significant depression (Harrington, 1994).

Anxiety Disorders

The Origins of Anxiety:

Prior to the early 20th century, childhood anxiety was largely ignored; when noted, its sole purpose was to elaborate on adult psychopathology (Klein and Last,1989).

The psychological views, psychoanalytic (Freudian and non-Freudian), learning, and cognitive theories differ sharply in their identification of the processes that activate anxiety, but they share the notion that anxiety is a unitary emotion with similar psychological mechanisms underlying normal and pathological states. (Klein, 1994).

The ICD-10 and DSM-IV have very similar approaches to the classification of anxiety disorders, but differ in several ways.

DSM-IV Anxiety Disorders

DSM-IV lists the following anxiety disorders

1. Panic disorder with and without agoraphobia.
2. Agoraphobia without a history of panic disorder.
3. Specific and social phobias.
4. Obsessive- compulsive disorder.
5. Post traumatic stress disorder.
6. Acute stress disorder.
7. Generalized anxiety disorder.

8. Anxiety disorder due to general medical condition.
9. Substance induced anxiety disorder.
10. Anxiety disorder not otherwise specified, including mixed anxiety-depressive disorder (American psychiatric association, 1994).

Separation anxiety disorder is the only anxiety disorder currently contained in the child and adolescent section of DSM-IV. In contrast, the child and adolescent section of revised 3rd edition of DSM (DSM-III-R) included overanxious disorder and avoidant disorder of childhood or adolescence in addition to separation anxiety disorder.

In DSM-III-R, overanxious disorder was marked by excessive anxiety unrelated to separation issues. Children with symptoms consistent with overanxious disorder are currently covered by the DSM-IV adult category of generalized anxiety disorder. In the DSM-III-R category of avoidant disorder of childhood or adolescence, a child exhibits warm and satisfying relationships with family members but avoides contact with unfamiliar people, children with symptoms of avoidant disorder meet the DSM-IV diagnostic criteria for social phobia which is also used for adults. Children may also present with anxiety disorders described by DSM-IV, including specific phobia, panic disorder, obsessive compulsive disorder and post traumatic stress disorder (Kaplan et al, 1994).

Separation Anxiety Disorder

Developmentally inappropriate and excessive anxiety emerges concerning separation from the major attachment figure (Kaplan et al., 1994).

As cognitive development proceeds during the first year of life and the infant acquires object constancy, fear of strangers and distress upon caretakers departure emerge, and separation anxiety appears (Lewis and Brooks, 1974). In most of anxiety disorders, there is congruence between the developmental timing of normal and abnormal forms of these affective states, exceptionally, the onset of maladaptive separation anxiety does not fit this pattern, as the peak age in community and clinical samples is reported in late childhood rather than infancy (Last et al, 1992), this goes well with the DSM-IV criteria for diagnosis, yet it does not fit the ICD-10 classification which requires onset before age of six (Klein, 1994) .

According to DSM-IV, the patient exhibits peripheral manifestations of anxiety, plus concerns about death, dying and the integrity of the family or the major attachment figure.

Typically the child will refuse to visit friends or go to school, this latter situation pertaining to school has been referred to as school refusal, which represent school phobia, however often it is a part of the larger picture of separation anxiety. DSM-III-R retain the term school phobia for those cases in which anxiety and fear reactions occur on account of school perse, and when school refusal is due to fear of being separated; separation anxiety is the more appropriate term. School refusal prevalence is 1-2% of school aged children, and appears to peak during periods of major traumas and transitions, as first beginning school. Generally,separation anxiety disorder is the most common anxiety disorder in childhood (Cantwell and Baker, 1988) and it is more common in young children than in adolescents, and has been reported to occur equally in boys and girls with onset most commonly seen in 7-8 year olds (Kaplan et al.,1994) . Cantwell and Baker, 1988, estimated

prevalence of separation anxiety disorder to be 3-4% in all school age children.

Moreover separation anxiety disorder is considered a common predisposing factor for a large number of psychiatric disorders, Segui et al, 1998, reported previous occurrence of separation anxiety disorder in 13.3% of patients with major depression, 16.7% of patients with dysthymia, 13.3% in generalized anxiety disorder patients, 33.3% of patients with social phobia and 15.3% of patients with panic disorder.

Generalized anxiety disorder:

With the advent of DSM-III in the USA a new disorder termed generalized anxiety disorder was established separately from panic disorder. Because generalized anxiety disorder was reglected to a residual category, it soon became a confusing diagnosis. Although revisions in DSM-III-R removed generalized anxiety disorder as a residual category, they also complicated the clinical examination necessary to arrive at generalized anxiety disorder diagnosis.

With the publication of DSM-IV, generalized anxiety disorder has further refined in an attempt to improve the reliability and discriminality. The DSM-IV has eliminated overanxious disorder, which was the childhood variant of generalized anxiety disorder. Instead, children may receive a diagnosis of generalized anxiety disorder as in the case in ICD-10 (Klein, 1994). Wervy, 1991, was the first to show overanxious disorders' poor validity, and scrutiny of the diagnostic criteria for overanxious disorder reveals its ambiguities. Overanxious disorder in DSM-III-R characterized by chronic and excessive worry and fearful behavior.

Now overanxious disorder in DSM-IV has combined with generalized anxiety disorder, focuses on over concerns, not reflected in other anxiety disorders, such as worries about untoward events.

Generalized anxiety disorder is defined as an uncontrollable disposition to worry about one's welfare and that of one's immediate kin. Associated manifestations include arousal, vigilance, tension, irritability, unrestful sleep and gastrointestinal distress.

There is growing evidence for the life-long nature of this condition among many of its sufferers. Akiskal, 1998, considered generalized anxiety disorder as an exaggeration of a normal personality disposition that can be named: Generalized Anxious Temperament (GAT).

Generalized Anxious Temperament seems to have distinct profile with altruistic overtones.

Panic Disorder

Panic disorder is characterized by spontaneous, unexpected occurrence of panic attacks (Kaplan et al., 1994). Panic attacks are relatively short- lived, usually less than one hour, periods of intense anxiety or fear which are accompanied by somatic symptoms such as palpitation and tachypnea (Kaplan et al., 1994). DSM-IV does make panic disorder the predominant disorder in a dyad as it contains diagnoses for panic disorder with or without agroraphobia. Although panic attacks and panic disorders are less common in children, they are nonetheless present, yet their expression in childhood may vary from the clinical features seen in

adolescence and adulthood, as it is suggested that most panic attacks in childhood are associated with particular events and are not unexpected. Moreover, non catastrophic interpretation of the symptoms of panic prevail (Ollendick, 1998).

Only 0.5% of all adolescents meet the DSM-IV criteria for panic disorder, while panic attacks, occur more frequently, 18% of adolescents reported having had at least one panic attack (Essau. et al.,1999). The occurrence of panic attack and panic disorder is greatest among the 14-15 year olds with slightly more prevalence rates among girls than boys.

Obviously whether panic disorder occurs at all in preadolescents, it is still an unresolved issue, but it seems clear that if it does, it is very rare (Klein, 1994). The most common previous psychiatric disturbance is separation anxiety disorder, which suggests that separation anxiety disorder may be a precursor to panic disorder, or perhaps an early form of it in some children (Ollendick et al., 1994).

Obsessive Compulsive Disorder:

Obsessive-compulsive disorder (OCD) is a common, childhood- onset, psychiatric condition, the most recent and scientifically rigorous data regarding age of onset indicates that 80% of adults with OCD identify their onset of symptoms before age 18 (Pauls et al., 1995).

Obsession is a recurrent and intrusive thought, feeling, idea, or sensation; while compulsion is a conscious standardized recurrent thought or behavior (Kaplan et al., 1994).

Obsessive-compulsive disorder is classified as a separate disorder under the heading of Neurotic stress-related and somatoform disorders in ICD-10.

The rationale for its inclusion as an anxiety disorder in the DSM-III (American Psychiatric Association, 1980) relates to the organizational principles of the DSM-III; the anxiety disorders provided the least incongruous locus for it (Klein, 1994).

Whereas DSM-III-R defined obsessions as thoughts and compulsions as actions, DSM-IV introduces the clinical observation that thoughts (that is, mental acts) can be either obsessions or compulsions, depending on whether they increase anxiety (obsessions), or reduce anxiety (compulsions).

Prevalence estimates for adolescents are similar to adults 1-3% (Flament et al., 1998), unfortunately, prevalence data for obsessive-compulsive disorder in preteenagers are non-existent.

Given the limits of current knowledge, there are probably only subtyping dichotomies that are of clinical value at this time: tic related versus non-tic-related obsessive compulsive disorder and familial versus non familial obsessive compulsive disorder (Riddle, 1998).

Although cognitive-behavioral approaches are important in the treatment of obsessive compulsive disorder, behavioral explanations are generally not accepted as having primary etiological significance (Rachman and Hodgson, 1980). Likewise, conflicts of psychodynamic origin are no longer thought to play an etiological role in obsessive compulsive disorder; yet genetic etiology has not been established for the disorder, although family

genetic studies published over the past 60 years have repeatedly shown that OCD is familial (Black, 1996). At least two types of biological, non- genetic, etiologies of obsessive compulsive disorder have been documented: focal brain lesions with basal ganglia structures involvement, which is rare (Berthier et al, 1996), and immune mediated sequelae of group A beta haemolytic streptococcal (GABHS) infections .

Clinical researchers at the National Institute of Mental Health and Brown University who were interested in tic disorders and obsessive compulsive disorder made the theoretical connection between Sydenham's chorea, tics and obsessive compulsive disorder and showed that OCD and tics occur in some children with Sydenham's chorea (Swedo et al., 1993). An abnormal immune response to group A β-heamolytic strepotcocci might play an etiological role in the development of OCD and / or tic disorders in some children who did not have Sydenham's chorea (Riddle, 1998).

Phobias :

The term (phobia) is derived from the Greek word (phobos) meaning fear or panic (Knoff, 1979). A phobia is an irrational fear, resulting in a conscious avoidance of the feared object, activity or situation; either the presence or the anticipation of the phobic entity elicits severe distress in the affected person, who recognizes that the reaction is excessive, this phobic reaction results in a disruption of the persons ability to function in life (Kaplan et al, 1994).

Phobias are increasingly recognized as being highly prevalent (Magee et al., 1996); as frequently having onsets early in life and as risk factors for the later development of disorders such as major depression (Kessler et al., 1996) and alcoholism (Kessler et al.,

1997). Hence appropriate classification could assist in understanding disorders that develop subsequently, as well as in understanding the phobias themselves (Curtis et al, 1998) .

Phobia is classified under the heading of neurotic stress–related and somatoform disorders in the ICD-10 classification, furthermore, ICD-10 includes phobic disorders of childhood. However, in DSM-IV phobia is included under anxiety disorders. The Fourth Edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) lists 3 types of phobia, namely: agoraphobia, specific phobia and social phobia; which is more or less similar to ICD-10 subdivisions; the divisions in DSM-IV match the nomenclature in the ICD-10.

Subsequently, reports of heterogeneity among the simple / specific phobia suggested possible re-splitting of the category. The DSM-IV (American Psychiatric Association, 1994) work group commissioned literature reviews and re-analysed existing data to evaluate these questions. The results judged insufficient to prove heterogeneity, but sufficiently suggestive to define five subtypes: animal, natural environment, blood-injection injury, situational and other. (Craske et al, 1996) .

The category was also renamed specific phobia, partly for congruence with ICD-10 (World Health Organization, 1992).

Estimates of prevalence obviously depend on where the boundary between phobia and other irrational fears is set. Those set by the ICD and DSM systems are of course, arbitrary.

Using impairment as the boundary, as the DSM systems do, has the practical logic of estimating the illness burden in the population and the number of cases possibly warranting treatment.

Curtis et al, 1998, study of specific fears and phobias showed that most simple phobias involve multiple fears. This contrasts with the notion of these phobias as isolated and circumscribed. Another robust finding was that increasing numbers of specific fears, independently of content, are associated with more likelihood of full criteria simple / specific phobia diagnoses, more disability and more comorbidity with other anxiety disorders. This suggests that numbers of fears may mark some general predisposition to anxiety disorders.

Social phobia, also called social anxiety disorder, only recently garnered recognition as a unique anxiety disorder.

ICD-10 includes social phobia that is equivalent to dysfunction DSM-III-R avoidant disorder; however in the DSM-IV this disorder is combined with adult diagnosis of social phobia (Klein, 1994). It involves persistent withdrawal from contact with strangers, to the extent that it impairs social functioning and peers relationship (Kleinknecht, 1991).

Although this fear is considered normal during the first years of life, having an average onset of about 8 months of age, a diagnosis of social phobia is only considered after the intense fear of strangers has persisted well past the normal developmental phase, roughly up to 2 and half years of age (Francis et al, 1992). However these children report comfort and satisfying involvement with significant others, such children often present as embarrassed and socially phobic.

Epidemiological studies have identified two subtypes of social phobia: speaking only social phobia, which is characterized by the fear of public speaking situation and complex social phobia, which is characterized by fear of multiple social sittings. Speaking only social phobia corresponds to the DSM-IV's non-generalized social phobia, while complex social phobia resembles generalized social phobia; the complex form is usually more disabling, familial and longer lasting (Stein and Chivara, 1998).

Epidemiology

Unexpectedly, epidemiological studies have observed high rates of anxiety disorders in children and adolescents as well as in adults (Christi et al., 1988). Interest in childhood anxiety has been heightened by reports that early onset characterizes adult anxiety disorders (Christi et al., 1988).

Benjamin, et al, 1990, conducted psychiatric interviews with 789 child and their parents, with ages ranging from 7-11 years, using Diagnostic Interview Schedule for Children (DISC), and showed a 1 year- prevalence of anxiety disorders 6.6% based on parent interview and 10.5% based on child interview, based on DSM-III diagnostic criteria .

Separation Anxiety Disorder is reported to be the most common of the anxiety disorders occurring in childhood; according to Anderson et al., 1987, the one year prevalence in prepubertal children has been 3.5 to 4.1 percent, with girls to boys ratio 3:1. However, Kaplan et al., 1994, reported equal distribution among both sexes, and age distribution between 7-8 years, which goes with Cohen's et al, study conducted in 1993, that showed rates of

separation anxiety to be comparable for boys and girls and comparable age trends in the two groups.

The overanxious disorder among prepubertal children has been estimated by Anderson et al., 1987, and found it to be ranging from 2.9 to 4.1 percent, in the same previous study conducted in Newzealand, they also reported that most anxiety disorders had an over- representation of females, except for overanxious disorder which showed male predominance 1.7 :1 this prevalence figure was exaggerated by Kleinknecht, 1991, who reported a prevalence ratio 2-9 % of overanxious disorder in preadolescent children .

There are notably different age trends. For boys a strong linear decline in overanxious disorder is apparent from age 10 to age20. For girls the prevalence rates show much less decline (Cohen et al, 1993).

Children's phobia have been studied by numerous researchers for many decades .Body et al., 1990, reported that prevalence of phobia is 6.2 % in community sample, yet phobia is considered relatively uncommon in child psychiatric practices as it is seen in less than 5% of all pediatric patients (Dupont, 1983).

Specific phobia is more common than social phobia, prevalence of social phobia was reported to range from 0.9 to 2.2 percent (Myers et al., 1984), while specific phobia was recorded to be 7.7 percent (Agars et al.,1969), fears of hights and darkness made 2% for each in their sample. Dalton, 1996, reported school phobia to be 1-2 percent.

Although epidemiological studies shows female pre-dominance, in clinical samples the reverse is often true. The reason

of these varying observations is still unknown (Kaplan and Sadock, 1998).

Kashani and Ovrasche, 1990, of university of Missouri examined a randomly selected group of 210 children aged 8-17 years and administered to them Anxiety- Disorders Interview, they found that 21% of their sample had one or more anxiety disorder. Of those with an anxiety disorder 36% had more than one diagnosis, with the most frequent diagnosis being separation anxiety and overanxious disorder.

Assessment of Anxiety Disorders

Despite the presence of multiple anxiety disorders, anxiety is a unitary emotion with similar psychological mechanisms underlying normal and pathological states, in all anxiety disorders anxiety varies in severity but not in its fundamental nature. Assessment of anxiety is a complex problem as anxiety follows an identifiable evolution that parallels other aspects of growth (Klein, 1994). Rutter, 1986, viewed developmental patterns as critical to the definition of psychopathology in children. On the other hand, current reports of comorbidity among the anxiety disorders also support the notion of the complexity of children's fears and anxieties (Last et al., 1987).

Anxiety is expressed through three modalities: behavior, subjective experience and physiological response.

Direct observation of anxious behavior can be made in a laboratory when the controlling stimulus is available for presentation, but this procedure has faded due to ethical consideration (Bandura et al.,1967); instead, behavior can be

evaluated in the field by direct observers e.g. parents or participants for daytime activities and bed time behavior, and teachers for observation of behavior pattern in response to different scholastic tasks e.g. examinations, peer relationship, and speaking in front of the class, those observers, whether parents, teachers or participants, require training to ensure collection of good data.

The current methodology for the assessment of childhood anxiety disorders includes rating scales and direct interviews.

1. Rating anxiety scales.

These scales are all designed to quantify level, or type of anxiety, they are either self rating anxiety scales, or parent and teacher- rated anxiety scales.

(a) Self- rating anxiety scales :

These are paper and pencil measures derived from adult scales, designed to obtain self-perception of anxiety in children.

Children Manifest Anxiety Scale (a modified version of the Manifest Anxiety Scale, Castaneda et al., 1956) , can be applied to 6-19 years old, self administered, or can be read by the examiner, it has the unique feature of providing a lie scale.

Spielberger and Colleagues, 1970, devised a scale for adults derived from a two-part model of anxiety, one tapping stable consistent tendencies (traits), and the other reflecting situational temporary reactions (states): [the state – trait anxiety inventory: Spielberger et al, 1970]. A child version was adopted subsequently, the State-Trait Anxiety Inventory for Children [STAIC], this is

used along with parent version; yet there is a question as to the instrument's ability to accurately measure "state" anxiety in children.

The Wolpe-Lang Fear Survey (Wolpe and Lang, 1964) was modified for use in children and became the Fear Survey Schedule for Children (Scherer and Nakamura, 1968), revised to be applied to children below age 9 (FSSC-R) (Ollendick, 1983). FSSC-R had no significant differences across diagnostic subgroups, however it does discriminate separation anxiety from overanxious disorder and school phobia, using a total intense fear score.

Another scale designed to assess anxiety related to negative social evaluation in adults, modified for use in children (Social Anxiety Scale for Children; La Greca et al, 1988) which is a semistructured 10-item self report measure, it pin points two factors of social anxiety, Fear of Negative Evaluation (FNE) and Social Avoidance and Distress (SAD) . The short term reliability (1-2 weeks) of these scales found to be satisfactory (Ollendick, 1983; La Greca et al, 1988). However evidence for the diagnostic validity of self-rating scales is limited and not encouraging, although higher scale ratings have been obtained in children with anxiety disorders compared to normal subjects (Ollendick 1983), this finding has not been consistent.

Although some claim that self rating can distinguish the affects of anxiety and depression in children; reviews of the adult literature document the poor discriminative ability of such scales. (Dobson and Cheung, 1990).

(b) Parent and teacher- rated anxiety scales

Anxiety factor scores are included in several parent scales such as Louisville Behavior Chicklist, which was elaborated into Louisville Fear Survey (Miller et al, 1972); the Personality Inventory for Children (PIC: Lachar, 1982); and the Child Behavior Chick List (CBCL, Achenbach 1989). The most widely used parent scale is the CBCL, although it has two limitations: the first it has no anxiety factor at all ages, the second is that the content of the anxiety factor varies between boys and girls; these discrepancies in the CBCL suggest that item content may fail to capture relevant aspects of children's anxious symptomatology (Klein, 1994).

Personality assessment is not a generally recommended method (Gittleman, 1988), due to length, necessary reading skills and breadth of information covered. However the PIC has been found to distinguish between anxious and depressed cases.

Obviously: parents are poor, invalid informants for assessing children's anxiety (Gittleman, 1988). The picture seems even worse with regard to teacher scales. Although the test –retest reliability of the anxiety scores of the teacher CBCL appear to be satisfactory (Achenbach et al, 1989) , teachers ability to identify anxious children is questionable .

II. Clinical Interviews

The inclusion of diagnostic criteria DSM-III (American Psychiatric Association, 1980), gave impetus to the development of clinical interviews designed to elicit diagnostic information (Klein, 1994). Now it is considered the most widely used method for

obtaining information. Clinical interviews include history taking, mental status examination, and direct behavioral observation.

The clinical interviews are helpful in providing systematic, comprehensive coverage of symptomatic status and, as such may be a resource to clinicians. (Klein, 1994).

Structured and semistructured interviews have gained popularity, and several of these interview schedules are in general use.

Omnibus interviews that include DSM-III-R childhood anxiety disorders are Schedule for the Affective Disorders and Schizophrenia for School- Age Children (Kiddie- SAD or K-SADS; Chambers et al, 1985).

This semistructured interview presents multiple items with some space for future clarification of symptoms; it is applicable for children between ages 6-17 years. It comes into 2 forms, one for the use for the parents about the child, and the other to be used with the child directly.

The Diagnostic Interviews for Children and Adolescents (DICA, Reich et al, 1992). The Diagnostic Interview Schedule for Children (DISC; Costello et al, 1985). Both are structured interviews which assess symptoms of a multitude of diagnoses keyed to the revised third edition of DSM (DSM- III- R).

The Child Assessment Schedule (CAS; Hodges, 1989) and the Interview Schedule for Children (ISC; Kovacs, 1985).The coverage of adult anxiety disorders is partial in these omnibus interviews. This omission is important since the onset of adult anxiety

disorders is frequently in childhood (Christie et al, 1988), therefore, the results obtained reflect only a partial picture of anxiety disorder in youth.

The World Health Organization, 1992, introduced another structured clinical interview based on ICD-10 diagnostic criteria, the Schedules for Clinical Assessment in Neuropsychaitry (SCAN).

Interviews specific to anxiety disorders are another type of interviews:

The Anxiety Disorders Interview Schedule for Children (ADIS; Sliverman, 1991) provides inquiry for all anxiety disorders. This semistructured interview has 2 versions: ADIS-C-Child version and ADIS-P parent version, it is symptom oriented, independent of informant, the ADIS-C and ADIS- P are reported to discriminate between simple phobias, overanxious disorders and school phobias, two other interviews are undergoing testing. The first is a new version of the DISC for use in epidemiological studies (NIMH, 1991) includes detailed inquiry about all anxiety disorders. The Child and adolescent psychiatric assessment (CAPA; A. Angold; A. Cox; M. Prendergast; M. Rutter and E. Sinonoff, unpublished observation) designed for clinical, and elicits symptoms of all anxiety disorders over a 3 month period.

MATERIAL AND METHODS

Materials and Methods

This study included 83 patients with varying degrees of upper airway obstruction due to chronic adenotonsillitis of duration more than one year.

The patients were chosen from the Otorhinolaryngology Clinic of Ain Shams University Hospital and Nasr City Health Insurance Hospital.

The patients were in late childhood, with age ranging from 7 – 12 years with a mean of 9.38 ; 37 were females and 46 were males.

Control Group Character

Thirty apparently normal children of nearly the same age 7 – 12 years (mean 9.57) and sex distribution (18 males and 12 females).

They were relatives of patients visiting the otorhinolaryngologic clinic of nearly the same social level and family construction.

Procedures

Full general and otorhinolaryngologic examination were done for all children of both groups to exclude any other associated general medical condition or otorhinolaryngologic disease.

All of the patients were school children of low to medium socioeconomic class of non-specified family construction.

Cold mirror test was used in diagnosis of nasal obstruction.

Tympanometry was done to exclude middle ear effusions in suspected cases.

Lateral soft tissue film of the nasopharynx.

Full psychiatric evaluation was done for all children of both groups to detect depression, anxiety, attention deficit disorder and disruptive behavior disorders, before and 5 to 6 months after their surgical operation for only 39 patients who were available in the follow up.

Using semi-structured psychiatric interview with each child and his (or her) parent – this psychiatric interview is the one applied in Ain Shams Psychiatric Department – The psychiatric diagnosis was done according to DSM-IV (1994). In addition to specific psychological tests used in Ain Shams Psychiatric Center which were also applied to all children before and in the follow up of all groups namely:

1) Children's Depression Inventory.

2) Children's Anxiety Scale.

3) Revised Behavior Problem Checklist.

Revised Behavior Problem Checklist (RBPC)

This scale is one of the most widely used in current research, designed by Quay and Peterson, 1983. The original Behavior Problem Checklist (BPC) first made available in 1967, A revision of the BPC was undertaken in 1980 to strengthen its psychometric characteristics. The RBPC was completed by parents. It consists of four major subscales Conduct Disorder, Socialized Aggression, Attention Problems – Immaturity and Anxiety-Withdrawal and two additional subscales: Psychotic Behavior and Motor Tension – Excess.

Four subscales were chosen for evaluation of children namely: Conduct Disorder and Socialized Aggression subscales for evaluation of disruptive behavior disorders.

Attention Problem Immaturity for evaluation of attention deficit disorder, and Motor Tension – Excess for evaluation of hyperactivity.(Appendix)

Children's Depression Inventory (CDI)

This scale is an Arabic version derived from the Children's Depression Inventory (CDI) developed by Kovas M., 1981 – It was designed by Ghareeb, 1988.

The CDI is a downward extension of the Beck Depression Inventory (Beck and Beames Derfer, 1974), CDI is a self – report instrument which is widely used in epidemiological studies to evaluate the prevalence of depressive symptoms in children as regards cognitive, affective and behavioral aspects. (Appendix)

Children's Anxiety Scale (CAS)

This is the Arabic version derived from Children's Manifest Anxiety Scale (CMAS) by Castaneda et al., 1956; It is designed by Abdel Hamid and .El- Nial, 1991. The Children's Anxiety Scale was prepared to be applied on children at the primary and preparatory schools. The scale consists of 36 statements, which measures all aspects of anxiety: somatic features, motor features, emotional features, physiological features and social and mental features.

It is designed to obtain self – perception of anxiety, both validity and reliability were assessed by the authors (Appendix).

STATISTICAL ANALYSIS

Statistical Analysis

The data collected was introduced to IBM computer. Statistical analysis of the whole work was done at the department of community medicine, Ain Shams University.

The following statistical parameters have been used in this field research:

1. Prevalence Rate

Definition:- The prevalence of disease in population is the proportion of that population having disorder at a given point of time.

$$\text{Prevalence} = \frac{\text{Number of individuals with attribute or event.}}{\text{Total number of individuals in a group}}$$

* The numerator encompasses both new and on going cases of the disorder.

2. The mean = the arithmetic average

$$\textbf{The mean } M = \frac{\sum X}{N}$$

Where $\sum$ = the sum of the individual values .

X = individual values.

N = The number of cases.

3.Standard Deviation (SD)

It is the square root of the variance. It gives an estimate of the average deviation around the mean.

$$S.D = \sqrt{\frac{\sum X^2 - \frac{(\sum X)^2}{n}}{n-1}}$$

Where $\sum X^2$ = The sum of squares of the individual values.

$(\sum X)^2$ = the square of the sum of the individual values .

4. T- Test:

$$t = \frac{\overline{X_1} - \overline{X_2}}{\sqrt{\frac{SD_1^2 x n_1 + SD_2^2 x n_2}{(n_1 + n_2) - 2}}}$$

For the value of t, consult tables at the degree of freedom n_1+n_2 -2

5- Chi- Square: X^2

$$X^2 = \sum \frac{(O-E)^2}{E}$$

Where O = the observed value .

E = The expected value.

6. Correlation Matrix

Was examined between the various parameters and statistical tests of significance.

7. MC Nemar Test

Non parametric test of the differences between group measures

8. Kruskal Wallis One- Way Anova

A non parametric test (distribution- free) used to compare three or more independent groups of sampled data.

The One Way Anova is an extention of the independent group t-test used to compare the means of more than two independent groups.

9. Wilcoxon Matched – Pairs Signed Ranks Test .

It is non- parametric test that compares two groups. It calculates the difference between each set of Pairs.(Hill, 1991).

RESULTS

Results

This study was conducted on 83 children suffering from chronic adenotonsillitis of more than one year duration, besides 30 apparently normal control subjects of nearly the same age and sex distribution as shown in table I and II.

These children were divided into the following groups :

Group A : 17 patients suffering from recurrent tonsillitis with no marked enlargement of adenoids .

Group B : 23 patients presented with moderately enlarged tonsils and adenoids.

Group C : 19 patients presented mainly with huge adenoids .

Group D : 24 patients presented with huge tonsils and adenoids.

The results of psychiatric and psychologic evaluation of all children of the study and control groups and that of the available children of postoperative group were analysed in the Public Health Department of Ain Shams University using IBM Computer.

Table I : Age Distribution

Cases	Mean	S.D.±	Minimum	Maximum
Group A	10.00	1.77	8	12
Group B	9.57	1.56	7.5	12
Group C	9.47	2.01	7	12
Group D	9.17	1.58	7	12
Total	9.38	1.73	7	12
Controls	9.57	1.87	7	12

The age range in patients with chronic adenotonsillitis and controls varied from 7-12 years with a mean value of 9.38 $\pm$ 1.73 in study group and 9.57 $\pm$ 1.87 in controls, there is no significant difference in age distribution , using Kruskal Wallis 1- Way Anova test, as shown in table I .

Table II : sex distribution

Cases	Male	Female
Group A	9 52.9%	8 47%
Group B	11 47.8%	12 52.1%
Group C	11 57.8%	8 42.1%
Group D	15 62.5%	9 37.5%
Total	46 55.4%	37 44.5%
Controls	18 60 %	12 40 %

Both cases and controls have nearly the same sex distribution as shown in table II.

Table III: Nocturnal Enuresis

Cases	Preoperative (P = 0.014)	Postoperative (P = 0.455)
Group A	1 5.88 %	1 14.3 %
Group B	4 17.4 %	1 11.1 %
Group C	6 31.6 %	- 0 %
Group D	8 33.30 %	1 7.69 %
Total	19 22.8%	3 7.69 %
Controls	1 3.33 %	

Using Chi- square test, nocturnal enuresis was more among the cases than the controls, and there was significant increase with the severity of upper airway obstruction. There was also significant reduction in nocturnal enuresis postoperatively especially in cases with huge adenoids and tonsils.

Table IV : Clinically diagnosed attention deficit disorders

Cases	Preoperative (P = 0.044)	Postoperative (P = 0.528)
Group A	1 5.9 %	- 0 %
Group B	3 13.0 %	1 11.1 %
Group C	5 26.3 %	1 10 %
Group D	6 25 %	1 7.69 %
Total	15 18 %	3 7.69 %
Controls	1 3.33 %	

Using Chi-square test, clinically diagnosed attention deficit disorder was more significant among cases than the controls, and showed marked increase in cases with huge adenoids.

The number of cases with attention deficit disorder was significantly reduced post- operatively.

Table V: Attention Problem – Immaturity (AP) subscale of RBPC

Cases	Preoperative	Postoperative	
Group A	5.94 ± 4.84	5.14 ± 1.86	0.760 Non significant
Group B	7.43 ± 4.48	6.22 ± 3.27	0.027 significant
Group C	10.42 ± 6.48	5.90 ± 4.93	0.007 Highly significant
Group D	10.33 ± 7.63	5.62 ± 4.03	0.0051 Highly significant
Controls	4.77 ± 4.00		

Using Kruskal – Wallis 1-Way Anova and Wilcoxon Matched – Pairs tests, the psychometric scores on the AP subscale of the cases were significantly higher than the controls, and the levels were significantly reduced postoperatively .

The scores increased significantly with the severity of upper airway obstruction .

Table VI : Motor Excess (ME) subscale of RBPC .

Cases	Preoperative	Postoperative	
Group A	1.47 ± 2.15	1.71 ± 1.50	0.760 Non significant
Group B	3.35 ± 2.64	2.00 ± 2.18	0.043 significant
Group C	3.32 ± 2.43	1.80 ± 1.69	0.005 Highly Significant
Group D	3.79 ± 2.21	2.15 ± 2.38	0.036 significant
Controls	1.87 ± 2.08		

Using Kruskal- Wallis 1 – Way Anova and Wilcoxon Matched – Pairs test, Motor Excess (ME) subscale scores were significant among cases than the controls, and the postoperative levels were significantly reduced especially in cases with huge adenoids .

Table VII: Clinically diagnosed oppositional defiant disorder.

Cases	Preoperative (P = 0.049)	Postoperative (P = 0.068)
Group A	2 11.8%	1 14.3%
Group B	4 17.4%	1 11.1 %
Group C	5 26.3%	2 20.0%
Group D	8 33.3 %	4 30.7 %
Total	19 22.8 %	8 20.5 %
Controls	2 6.7 %	

Using Chi- square test , clinically diagnosed oppositional defiant disorder was significantly higher among the cases than the controls , and increased with severity of upper airway obstruction. However, there was no much improvement after surgical management.

Table VIII: Clinically diagnosed conduct disorder

Cases	Preoperative (P = 0.248)	Postoperative (P = 0.669)
Group A	- 0 %	- 0 %
Group B	- 0 %	- 0 %
Group C	1 5.3 %	- 0 %
Group D	2 8.3 %	1 7.69 %
Total	3 3.6 %	1 2.56 %
Controls	- 0 %	

Using Chi – square test, there was no significant difference in conduct disorder prevalence between the cases and the controls.

Table IX : Conduct Disorder (CD) Subscale of RBPC

Cases	Preoperative	Postoperative	
Group A	7.29 ± 5.8	6.00 ± 5.45	0.715 Non significant
Group B	9.61 ± 7.02	8.33 ± 8.15	0.442 Non significant
Group C	13.16 ± 8.05	9.3 ± 5.06	0.008 Highly Significant
Group D	13.96 ± 7.02	10.92 ± 6.97	0.004 Highly significant
Controls	6.23 ± 5.3		

Using Kruskal- Wallis 1- Way Anova test and Wilcoxon Matched – Pairs tests, Conduct Disorder (CD) subscale scores were higher among the cases than the controls and increased with the severity of upper airway obstruction.

The preoperative levels were significantly higher than the postoperative levels.

Table X : Socialized Aggression (SA) subscale of RBPC.

Cases	Preoperative	Postoperative	
Group A	0.65 ± 0.49	1.29 ± 2.14	0.654 Non significant
Group B	0.78 ± 1.00	0.67 ± 0.87	0.317 Non significant
Group C	1.68 ± 2.91	0.90 ± 1.37	0.179 Non significant
Group D	2.00 ± 3.36	2.15 ± 3.46	0.916 Non significant
Controls	0.43 ± 0.77		

There was no significant difference in Socialized Aggression (SA) subscale between the cases and controls by using the Kruskal – Wallis 1- Way Anova Test.

There was also no significant difference between the preoperative and postoperative levels using the Wilcoxon Matched – Pairs test.

Table XI : Clinically diagnosed depression.

Cases	Preoperative (P = 0.948)	Postoperative (P = 0.658)
Group A	1 5.9 %	-
Group B	1 4.3 %	-
Group C	1 5.3 %	-
Group D	2 8.3 %	1 7.6 %
Total	5 6.02 %	1 2.56 %
Controls	1 3.3 %	

Using Chi- square test, clinically diagnosed depression showed no significant difference between the cases and the controls, also there was no significant difference between preoperative groups and postoperative groups.

Table XII : Children Depression Inventory (CDI) Psychometric scores .

Cases	Preoperative	Postoperative	
Group A	5.82 ± 4.33	4.71 ± 2.56	0.345 Non significant
Group B	5.87 ± 5.27	5.78 ± 1.72	0.456 Non significant
Group C	7.63 ± 3.29	7.30 ± 2.88	0.179 Non Significant
Group D	8.46 ± 4.05	7.69 ± 4.68	0.916 Non significant
Controls	4.57 ± 4.88		

Using Kruskal – Wallis I- Way Anova and Wilcoxon Matched – Pairs test, there was significant difference in psychometric scores between the cases and the controls, but there was no significant difference between the preoperative and postoperative levels.

Table XIII : Clinically diagnosed anxiety disorder.

Cases	Preoperative (P = 0.252)	Postoperative (P = 0.738)
Group A	-	1 14.3 %
Group B	1 4.3 %	1 11.1 %
Group C	3 15.8 %	1 10%
Group D	4 16.7 %	1 7.69 %
Total	8 9.63%	4 10.2 %
Controls	2 6.7 %	

Using Chi- square test clinically diagnosed anxiety disorder showed no significant difference between the cases and the controls.

There was significant reduction in anxiety postoperatively in cases with huge tonsils and adenoids (Group C and D) .

Table XIV : Children Anxiety Scale (CAS) Psychometric scores .

Cases	Preoperative	Postoperative	
Group A	6 ± 2.94	7.43 ± 3.69	0.529 Non significant
Group B	8.83 ± 3.47	8.11 ± 3.22	0.465 Non significant
Group C	8.63 ± 3.89	8.30 ± 4.79	0.918 Non significant
Group D	10.08 ± 4.26	8 ± 3.72	0.100 Non significant
Controls	6.17 ± 5.39		

Using Kruskal – Wallis I- Way Anova test, psychometric scores for anxiety were significant in preoperative study group than the controls. However, there was no significant difference on CAS levels post- operatively using the Wilcoxon Matched – Pairs test.

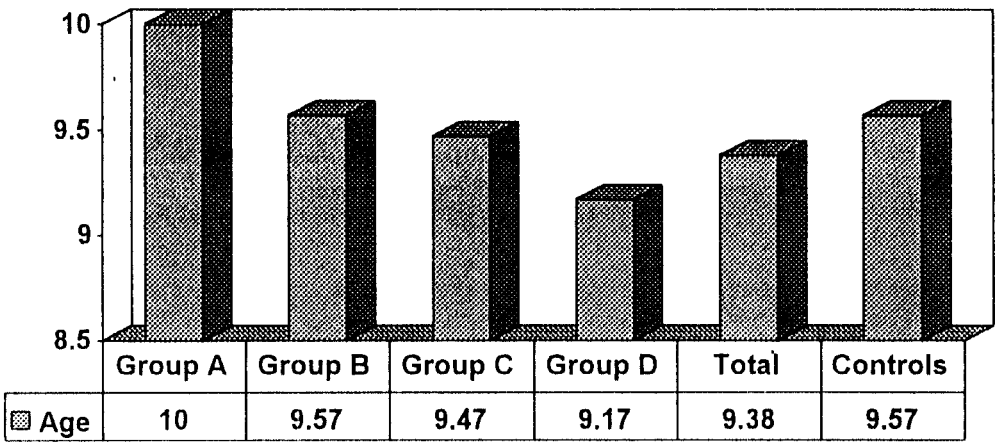


Figure 1: The distribution of mean age

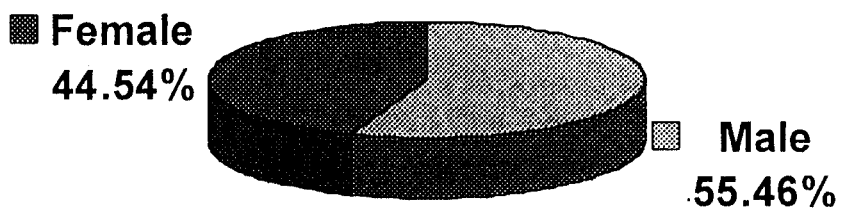


Figure 2: Percentage of total sex distribution

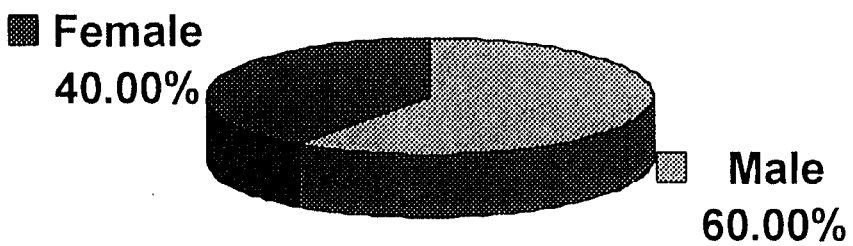
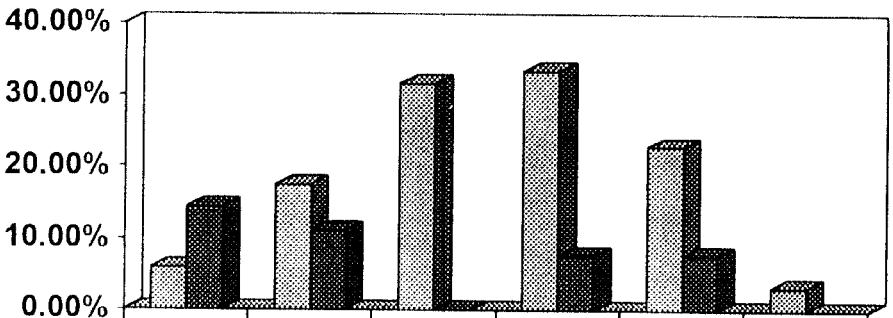
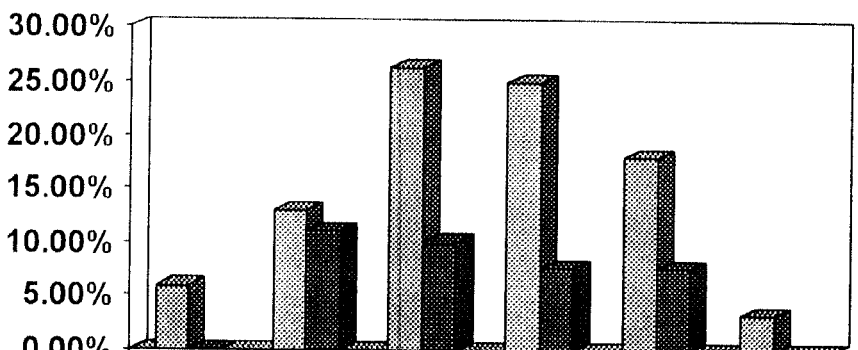


Figure 3: Percentage of controls sex distribution



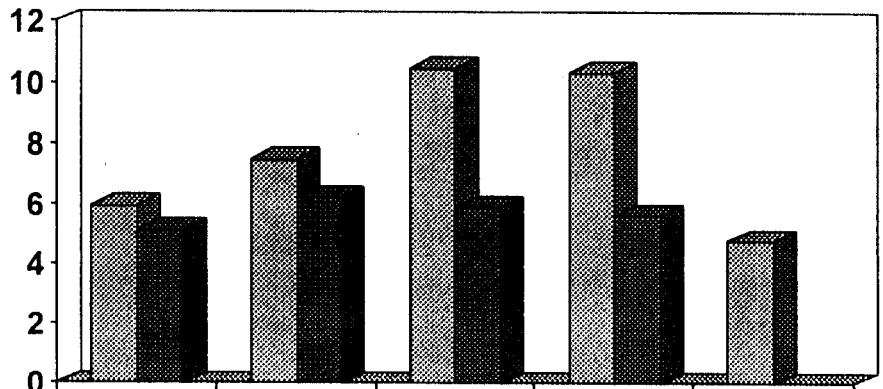
	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	5.88%	17.40%	31.60%	33.30%	22.80%	3.33%
Postoperative	14.30%	11.10%	0.00%	7.69%	7.69%	

Figure 4: The nocturnal enuresis of preoperative (P=0.014) and postoperative (P=0.455) cases.



	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	5.90%	13.00	26.30	25.00	18.00	3.33%
Postoperative	0.00%	11.10	10.00	7.69%	7.69%	

Figure 5: Clinically diagnosed attention deficit disorder of preoperative (P=0.044) and postoperative (P=0.528) cases.

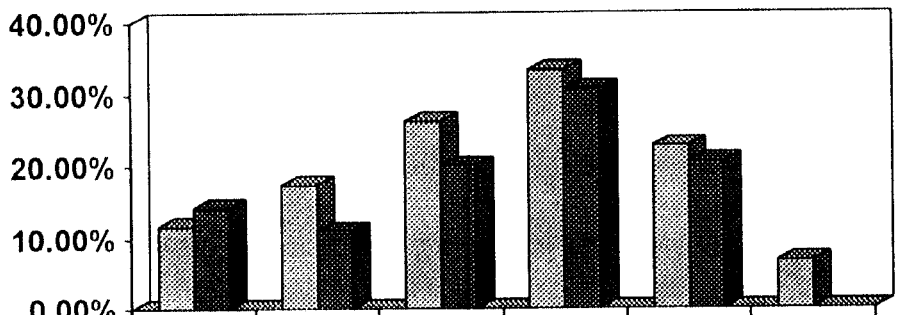


	Group A	Group B	Group C	Group D	Controls
Preoperative	5.94	7.43	10.42	10.33	4.77
Postoperative	5.14	6.22	5.9	5.62	

Figure 6: Attention problem – Immaturity (AP) subscale of RBPC for preoperative and postoperative cases.

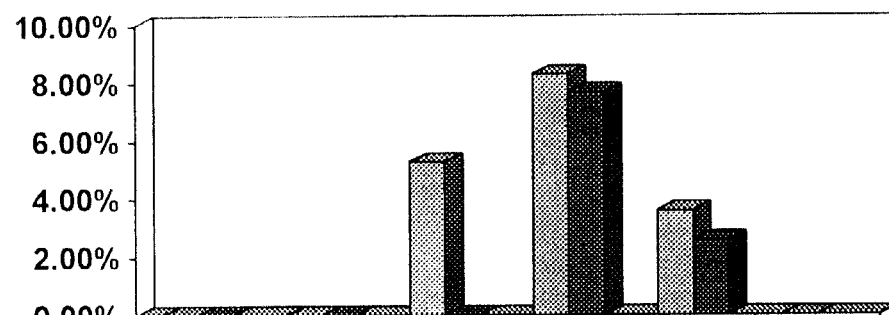
	Group A	Group B	Group C	Group D	Controls
Preoperative	1.47	3.35	3.32	3.79	1.87
Postoperative	1.71	2	1.8	2.15	

Figure 7: Motor tension-excess (ME) subscale of RBPC for preoperative and postoperative cases.



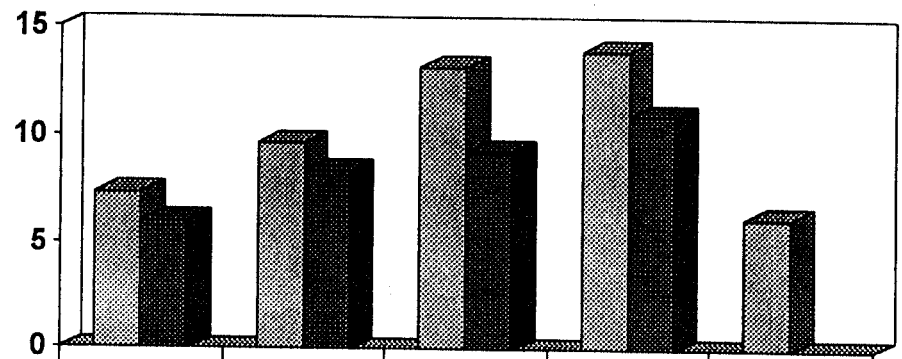
	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	11.80%	17.40%	26.30%	33.30%	22.80%	6.70%
Postoperative	14.30%	11.10%	20.00%	30.70%	20.50%	

Figurer 8: Clinically diagnosed oppositional defiant disorder of preoperative (p=0.049) and postoperative (p=0.068) cases.



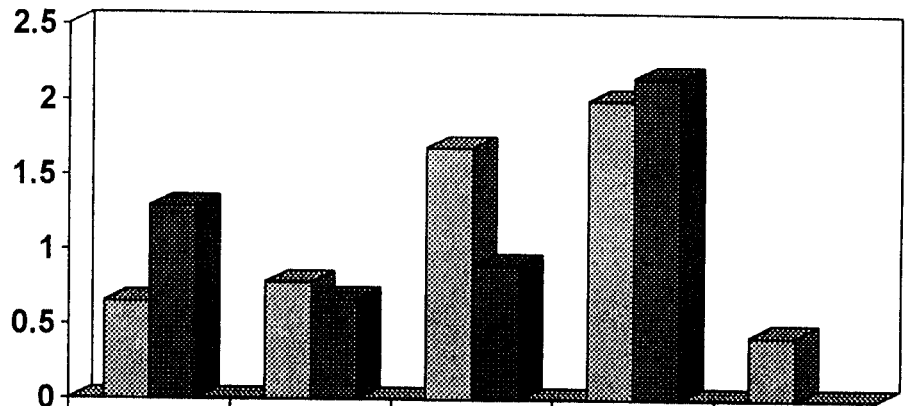
	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	0.00%	0.00%	5.30%	8.30%	3.60%	0.00%
Postoperative	0.00%	0.00%	0.00%	7.69%	2.56%	

Figure 9: Clinically diagnosed conduct disorder of preoperative (p=0.248) and postoperative (p=0.669) cases.



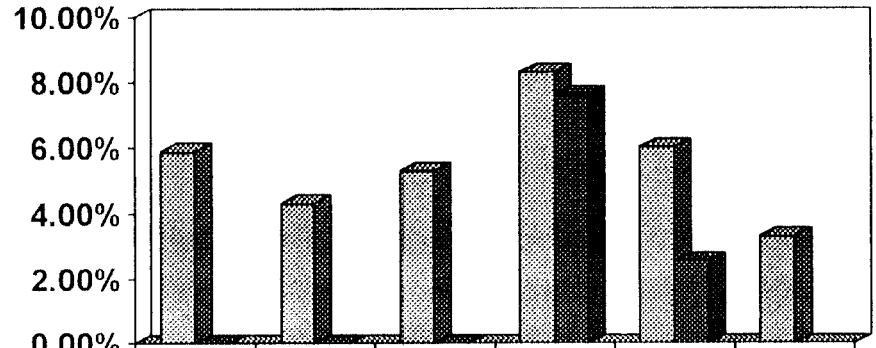
	Group A	Group B	Group C	Group D	Controls
Preoperative	7.29	9.61	13.16	13.96	6.23
Postoperative	6	8.33	9.3	10.92	

Figure 10: Conduct disorder (CD) subscale of RBPC of preoperative and postoperative cases.



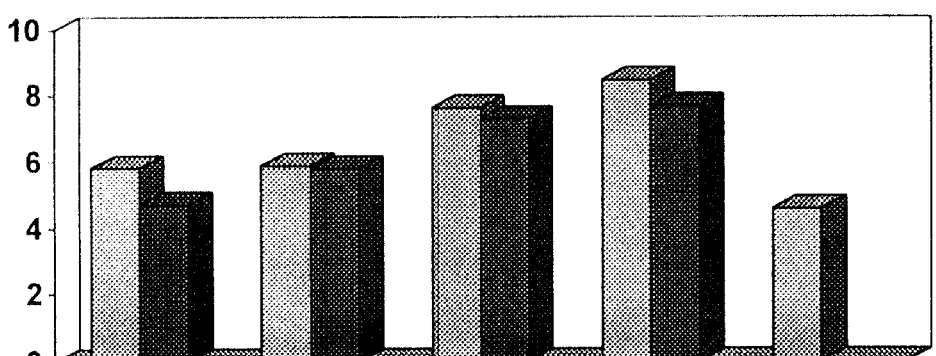
	Group A	Group B	Group C	Group D	Controls
Preoperative	0.65	0.78	1.68	2	0.43
Postoperative	1.29	0.67	0.9	2.15	

Figure 11: Socialized aggression (SA) subscale of RBPC of preoperative and postoperative cases.



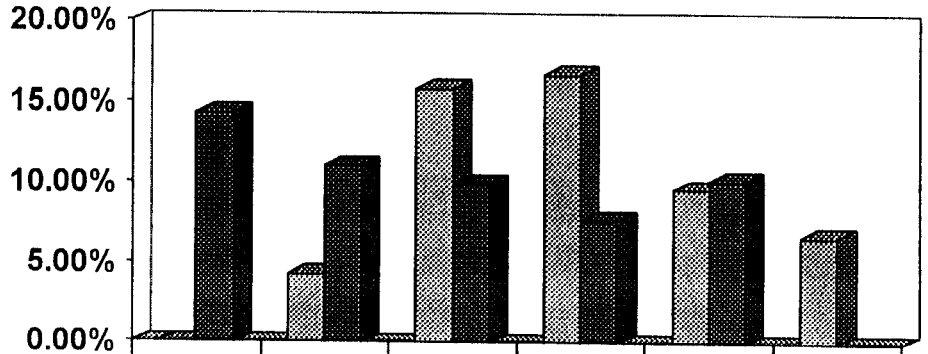
	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	5.90%	4.30%	5.30%	8.30%	6.02%	3.30%
Postoperative	0%	0%	0%	7.60%	2.56%	

Figure 12: Clinically diagnosed depression of preoperative (p=0.948) and postoperative (p=0.658) cases.



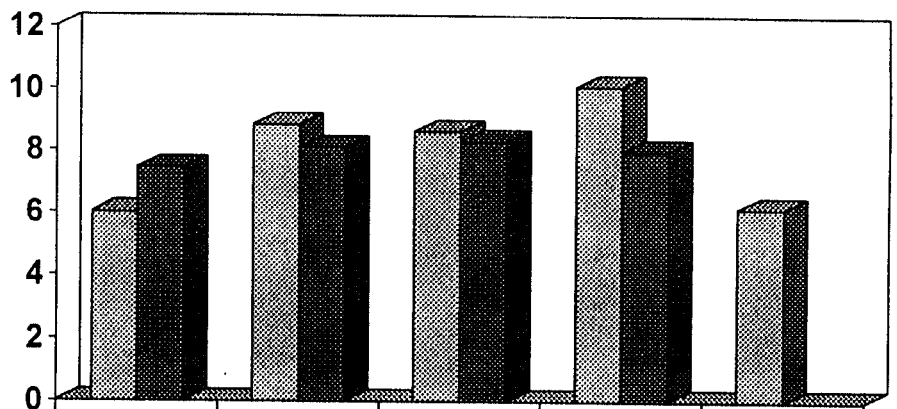
	Group A	Group B	Group C	Group D	Controls
Preoperative	5.82	5.87	7.63	8.46	4.57
Postoperative	4.71	5.78	7.3	7.69	

Figure 13: Children depression inventory (CDI) psychometric scores of preoperative and postoperative cases.



	Group A	Group B	Group C	Group D	Total	Controls
Preoperative	0.00%	4.30%	15.80%	16.70%	9.63%	6.70%
Postoperative	14.30%	11.10%	10.00%	7.69%	10.20%	

Figure 14: Clinically diagnosed anxiety disorder of preoperative (p=0.252) and postoperative (p=0.738) cases.



	Group A	Group B	Group C	Group D	Controls
Preoperative	6	8.83	8.63	10.08	6.17
Postoperative	7.43	8.11	8.3	8	

Figure 15 Children anxiety scale (CAS) psychometric scores of preoperative and postoperative cases.

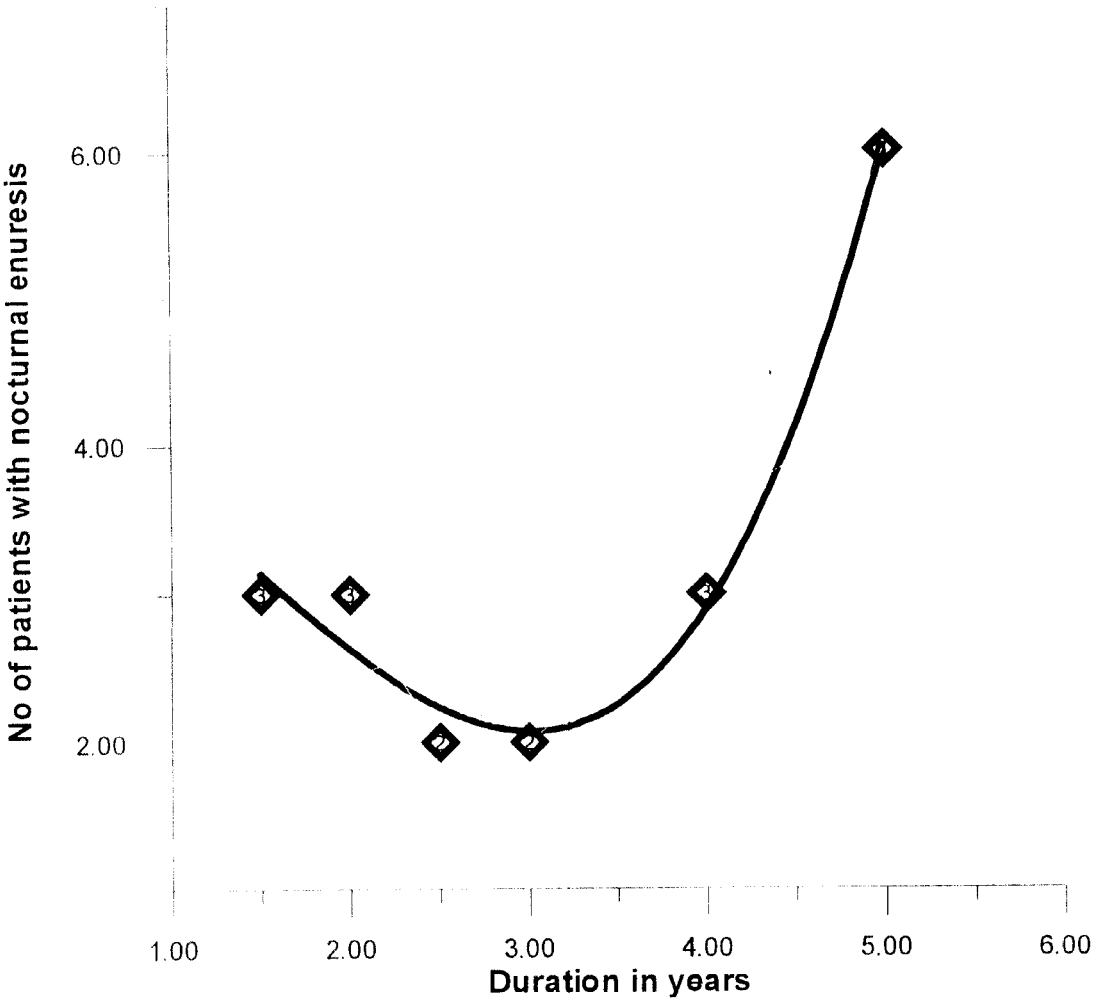


Figure 16: Relationship between prevalence of nocturnal enuresis and the duration of upper airway obstruction.

A fitting technique has been used to predict the relationship between the number of patients with nocturnal enuresis Y with the duration in years X which can be expressed in a polynomial function of the third degrees following:

$$Y = 2.6 - 0.51\,X + 0.24X^2 + 0.129X^3$$

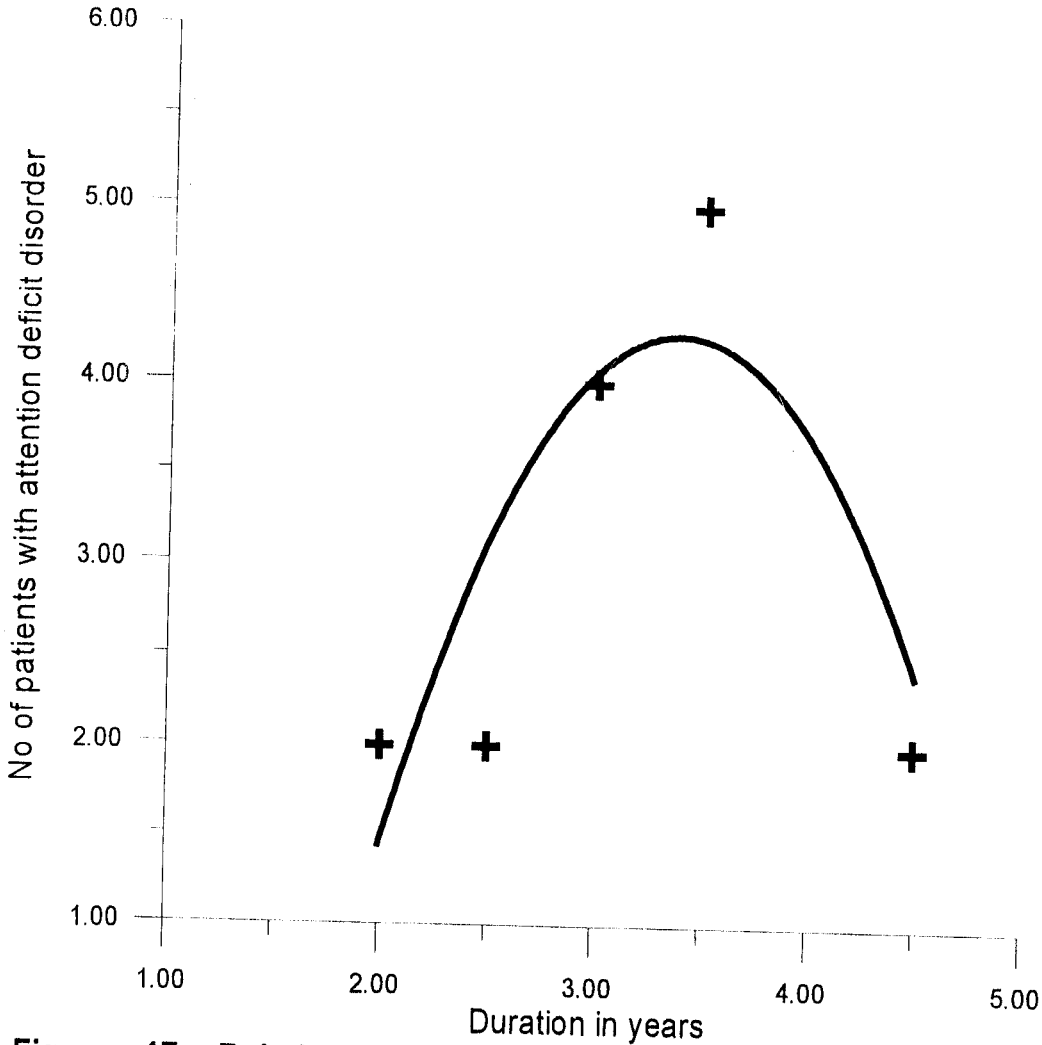


Figure 17: Relationship between the prevalence of attention deficit disorder and the duration of upper airway obstruction.

The fitting technique was applied to predict the relationship between the number of patients with attention deficit disorder Z with the duration in years X which is expressed in the following polynomial function:

$$Z = 3.06 + 0.206\,X - 0.581\,X^2$$

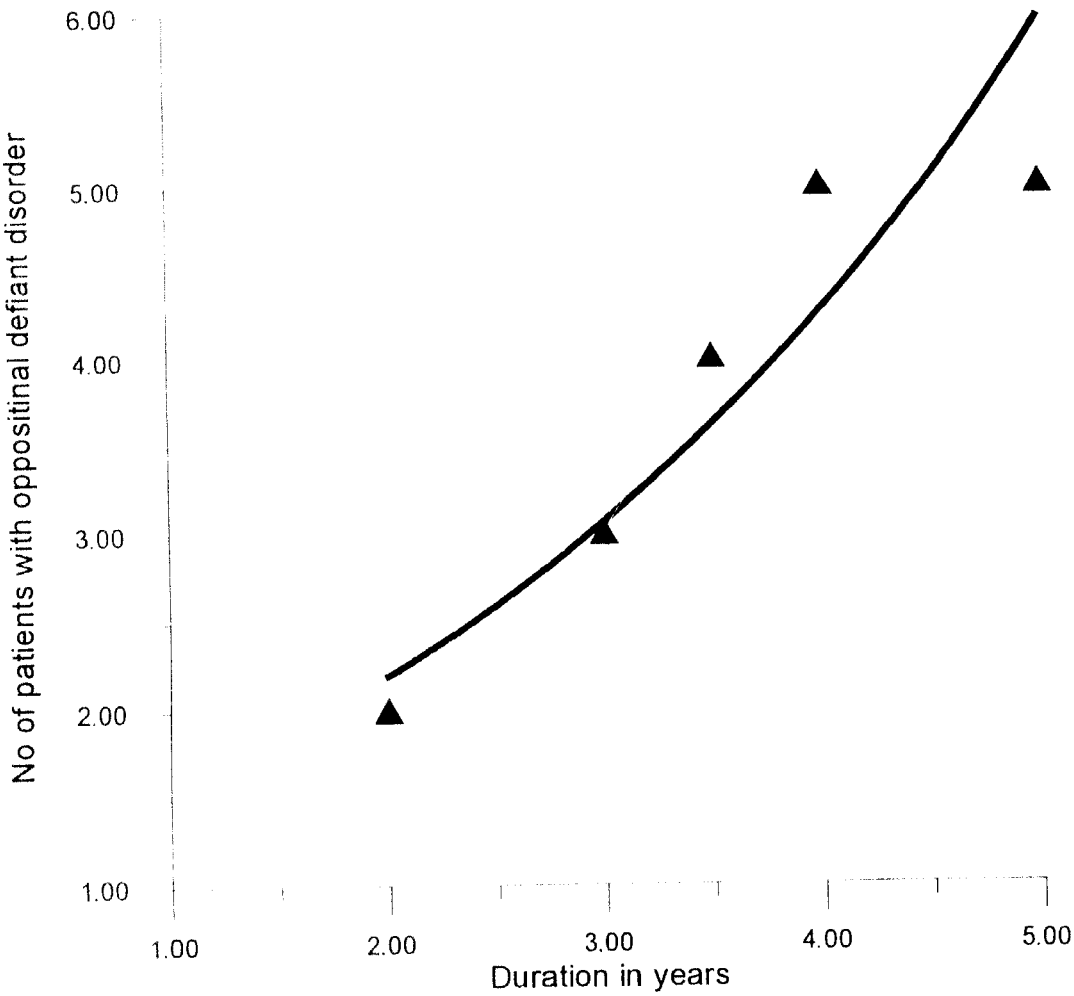


Figure 18: Relationship between the prevalence of oppositional defiant disorder and the duration of upper airway obstruction.

The fitting technique was used to predict the relationship between the number of patients with oppositional defiant disorder ***N*** with the duration in years ***X*** which is expressed in a logarithmic function as following:

$$Log\ [N] = 0.332\ X\ + 0.123$$

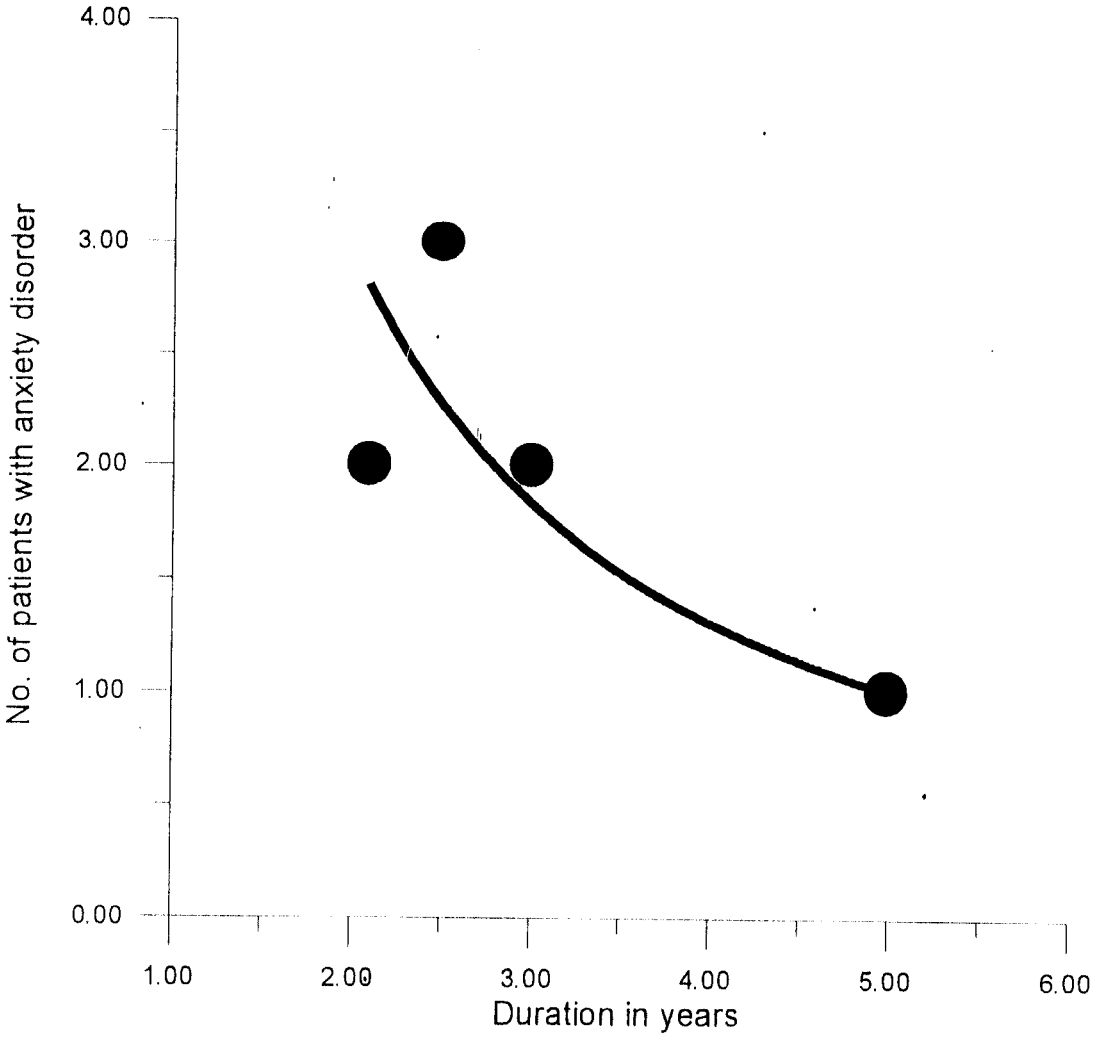


Figure 19: Relationship between the prevalence of anxiety disorder and the duration of upper airway obstruction.

The fitting technique was used to predict the relationship between the number of patients with anxiety disorder ***K*** with the duration in years ***X*** which is expressed in a logarithmic power function as following:

$$Log\ [K] = -\ 1.0988\ Log\ [X] + 1.835$$

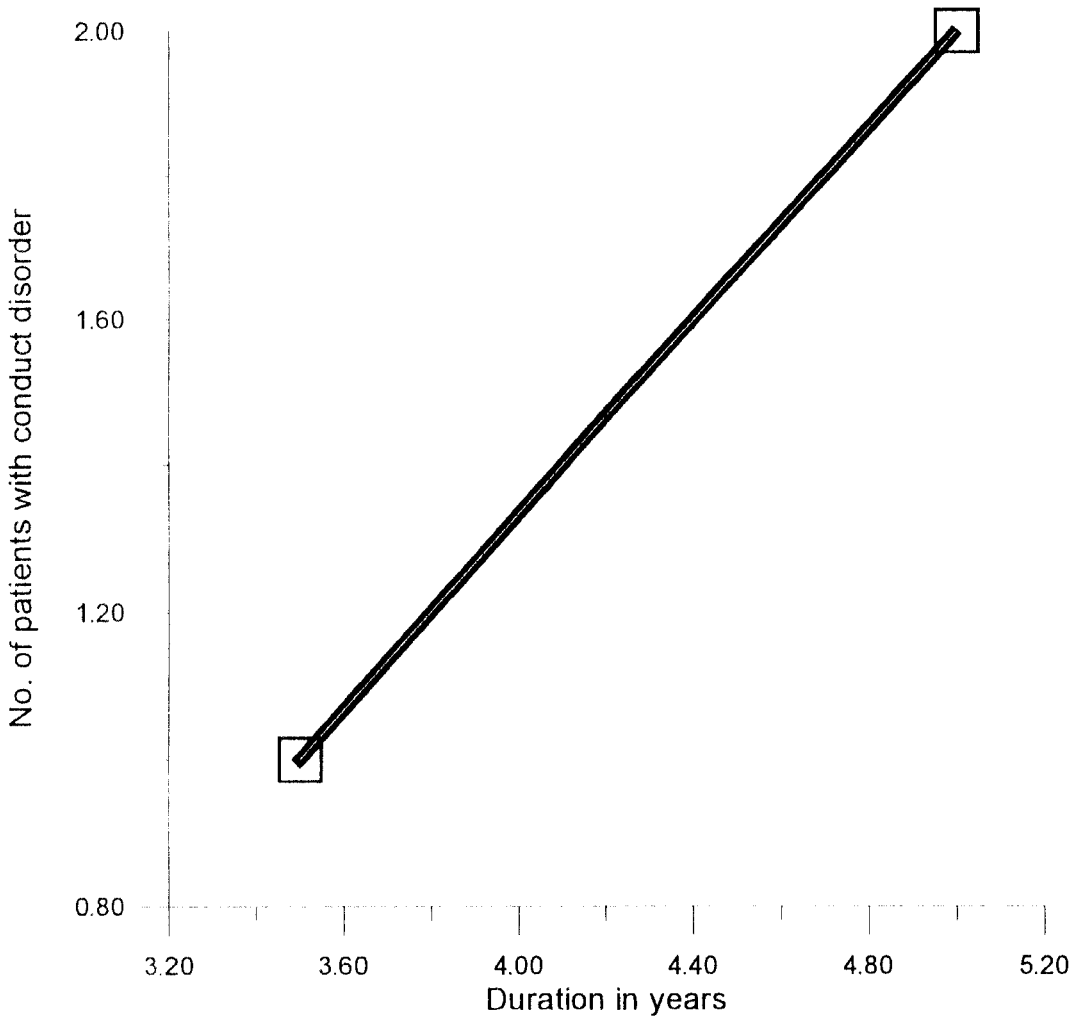


Figure 20: Relationship between the prevalence of conduct disorder and the duration of upper airway obstruction.

The fitting technique was used to predict the relationship between the number of patients with conduct disorder ***H*** with the duration in years ***X*** which is expressed in a linear function as following:

$$H = 0.667 X - 1.333$$

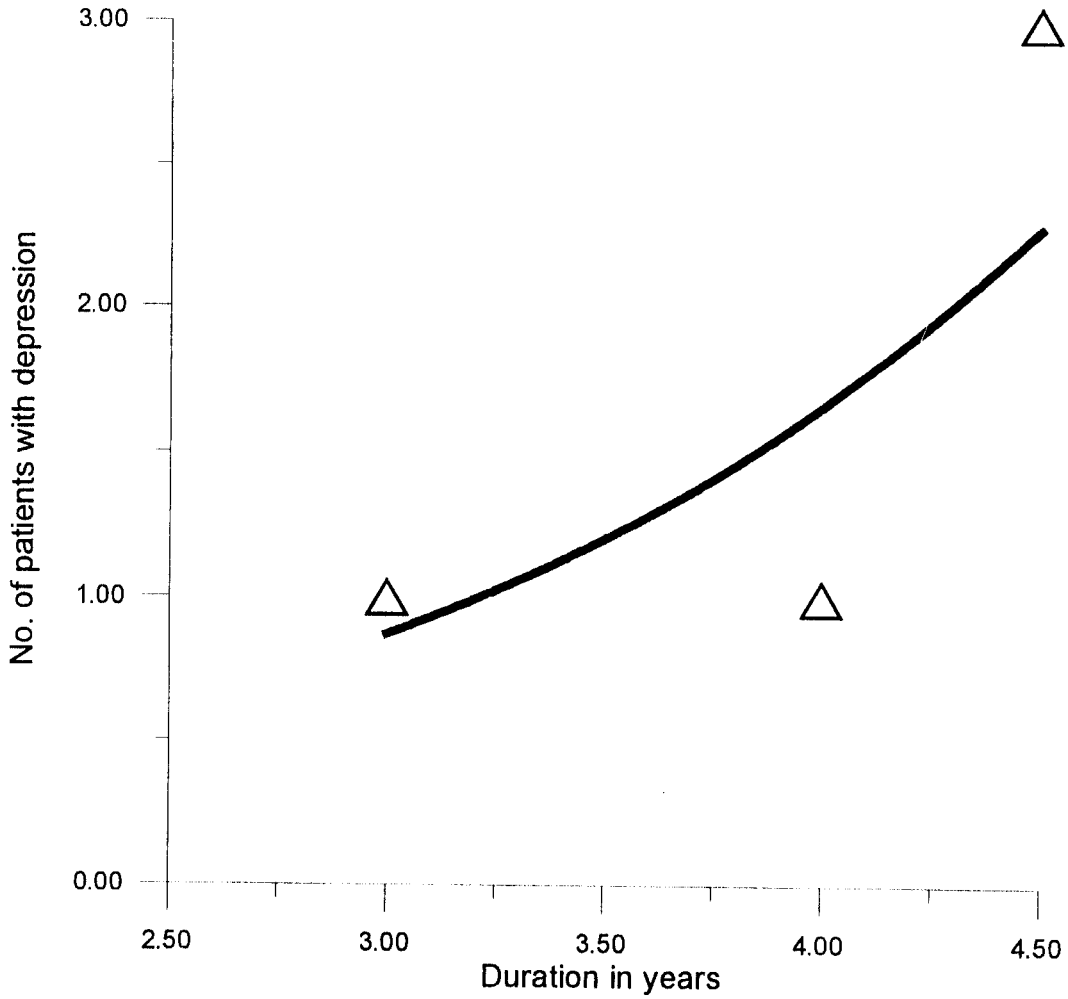


Figure 21: Relationship between the prevalence of depression and the duration of upper airway obstruction.

The fitting technique was applied to predict the relationship between the number of patients with depression ***G*** with the duration in years ***X*** which is expressed in **a log.** function as following:

$$Log\,[G] = 0.641\,X - 2.061$$

DISCUSSION

Discussion

In 1889 Dr. William Hill presented a paper to the autumn meeting of the Royal Society of Medicine in Leeds entitled "On some cases of backwardenss and stupidity in children relieved by adenoidal scarification ". In this paper Hill associated symptoms of snoring and restless sleep with mental dullness and lethargy which he presumed were due to the disturbed sleep pattern.

The children Hill described, benefited from adenoidectomy and the credit for first recognizing the association between partial airways obstruction and significant respiratory disturbances during sleep must go to him.

Sixty- seven years passed before Spector and Bautista alerted the medical community (again) to the possibility that adenotonsillitis could cause upper airway obstruction and respiratory distress in children (Spector and Butista, 1956) .

Potsic, 1989, reported that any child has the potential for developing airway obstruction because the most common cause is hyperplasia of the adenoid and tonsils. Adenotonsillitis is a common disorder and it is unusual for a child to have at least one or two episodes (Cowan and Hibbert, 1997); yet airway obstruction is rarely precipitated by an acute infection (Potsic, 1989) and rather caused by local enlargement of the lymphoid tissue secondary to chronic infection (Zalzal and Cotton, 1993).

At birth the tonsils and adenoids are very small because of immunoglobulin transfer from the mother, then they enlarge in size

until the age 4 or 5 years because of immune activity and subjection to infection (Brodsky et al, 1988) .

The tonsils and adenoids decrease in size as puberty is reached and often atrophy in the adult. Zalzal and Cotton, 1993, reported that chronic adenoid infection is expected between 3 and 6 years of age with concomitant hypertrophy.

Obstructive sleep apnea in children is caused by either physical or dysfunctional obstruction of the upper airway during sleep. Physical obstruction of upper airway is especially common in children, the most common cause of obstructive sleep apnea in children is undoubtly non- inflammatory enlargement of the adenoids and tonsils (Baily and Croft, 1997). OSAS probably represents the most severe syndrome related to obstruction of upper airway; less severe forms include upper airway resistance syndrome (UARS) and primary snoring (Chervin and Guilleminault, 1996) .

Prolonged partial airway obstruction during sleep may result in significant hypercarbia and hypoxaemia (Brouillette et al, 1982).

Many authors have studied the role of adenotonsillar enlargement on pulmonary ventilation and blood gases (Deutch, 1996); very few have considered the role of enlarged adenoids alone. El Assy et al, 1994, gave additional support to the adenoidal etiology of the nasal obstruction. Arterial blood gases and oxygen saturation were measured before and after adenoidectomy, results showed that arterial PO_2 and O_2 saturation increased significantly and arterial PCO_2 decreased; suggesting that ventilatory impairment evident before surgery was reversible.

The relationship between disordered breathing and psychiatric disorders is not broadly understood. Ley, 1994, proposed that if some psychiatric disorders are a manifestation of faulty cognition, if cognition is a product of the brain's activity, and if the brain's activity is dependent on an adequate supply of oxygen, then the connection between disordered breathing and psychiatric disorders should be clear.

Prevalence estimates of psychopathology in children and adolescents range from approximately 1 to nearly 51% with a mean prevalence of 13.2 % in preadolescent sample (Roberts et al, 1998).

The current study examines the behavioral and psychological effects of chronic adenotonsillitis, as a cause of upper airway obstruction, in 83 children going for adenotonsillectomy. Psychiatric morbidity in the study group was 39.75% in contrast to 16.66 % in the control group. Guilleminault and associates, 1976, reported high incidence of impaired school performance, hyperactivity, decreased intellectual performance and emotional problems that 50% were receiving psychiatric counseling in children with upper airway obstruction.

In our study psychiatric morbidity was prevalent among patients suffering huge adenoids with and without tonsillar enlargement leading to considerable degree of upper airway obstruction. This goes with Taylor and Eminson, 1994, who proposed that the degree of psychological impact generally parallel the severity of physical illness.

Stewart et al, 2000, showed that overall health status and quality of life (QOL) of children with tonsil and adenoid disease is significantly worse than those of healthy normal children as

demonstrated by lower mean scores on general health, physical functioning, behavior, bodily pain and parental impact subscales.

Moreover several aspects of health status were significantly worse than children with asthma and juvenile rheumatoid artheritis.

The assumption made by Marzek, 1994, that if two diseases share similar illness-specific risk factors, it is logical to expect that their impact on emotional development should be similar. This discussion looks at studies performed on psychological effects of airway obstruction due to different causes both in children and adults, since little published data are available on the psychological effects of chronic adenotonsillitis in children.

Several authors have studied psychiatric morbidity in relation to upper airway obstruction both in adults and in children.

The International Classification of Sleep Disorders Manual describes depression, irritability, anxiety, and profound despair as commonly associated with OSAS in adults, as well as blackouts, disorientation, and periods of automatic behavior with amnesia (Diagnostic Classification Steering Committee, 1990).

In Children, Carroll and Loughlin, 1995, suggested that subtle, currently unrecognized developmental impairment may occur due to OSAS; while other studies have shown correlations between nocturnal hypoxaemia and daytime cognitive impairment in patients with continuous partial airway obstruction (Greenberg et al, 1987 and Findley et al, 1986). However Noel and Guarisco, 1994; and Suen et al, 1995, suggested chronic sleep disruption in upper airway obstruction to cause behavioral abnormalities,

including poor school performance, daytime hypersomnolence and learning disabilities.

Mubarak et al, 1996, demonstrated that chronic obstructive pulmonary disease can produce major changes in the psychological adaptation, promoted by exertional limitations, as the disease has effect on the higher brain functions of short term visual memory, attention and concentration which was correlated with both Pa O_2 and pulmonary functions.

Similar results was reported by Lishman, 1987. Depression was reported in 76.2% of patients with COPD (Mubarak et al, 1996) and anxiety in 42.9% in the same study group. Barak et al, 1992, reported depression in half of COPD patients; Porzelius et al , 1992 , reported panic disorder in 37 % of their COPD sample.

Sacre-Hazouri, 1999, assessed in a prospective fashion a cohort of pediatric patients, 12 years old and under, with clinical complaints of recurrent upper airway obstruction, two thirds of these patients had a suggestive pattern of nasal allergy and infection at the same time.

Case studies have been used to describe a pediatric behavioral syndrome called the " allergic irritability syndrome ", the hallmarks of which are irritability, anger, and poor concentration. (Klien et al, 1985). Investigators noted that these behaviors disappeared with appropriate treatment, but reappeared when treatment was discontinued.

Unfortunately, the children described in these cases were sometime incorrectly labeled as hyperactive, where in fact their

behavioral problem were secondary to the symptoms of chronic rhinitis. (Bender, 1997).

In children, the most common cause of lower respiratory tract obstruction is bronchial asthma. Asthma is defined as a reversible reactive airway disease triggered by a variety of immunological, infectious, physiological and emotional triggers, Severely asthmatic children have been shown to be at increased risk for emotional disturbance (Marzek, 1994), in which depression is the most common.

Hamlett et al, 1992, compared the behavioral correlates of asthmatic children, using Child Behavior Checklist, and showed that mean externalizing behavior scores for asthmatics were not significantly higher than those of controls, and that the mean internalizing scores showed significant increase among asthmatic children.

Stores et al, 1998, studied subjective and objective sleep disturbance in children with nocturnal asthma. Relations between such disturbance and daytime psychological functions were explored, children with asthma had significantly more disturbed sleep, tended to have more psychological problems and performed less well on some tests of memory and concentration.

Nocturnal enuresis (NE) is considered a bengin condition and common in healthy children with a known rate of spontaneous resolution. El- Shamy, 1992, declared 7.1% prevalence of enuresis among primary school children, while Salama, 1992, proposed 44.5 % prevalence of enuresis in the same age group in east region of Cairo. An overall prevalence of nocturnal enuresis was suggested by Gumus et al, 1999, to be 13.7% with male preponderance.

Enuresis may result from either a disturbance of awakening or a disturbance of bladder function (Wantabe et at , 1994).

Weider et al, 1991, suggested a relation between nocturnal enuresis and disturbed sleep patterns in children with upper airway obstruction, this is confirmed by El Asfour et al, 2000, study of enuretic children with manifestations of upper airway obstruction due to adenotonsillar hypertrophy, in which sleep studies of Apnea Hypoapnea Index (AHI) and SaO_2 revealed the presence of pathological events ranging from mild to severe obstructive sleep apnea .

In the present study nocturnal enuresis was significantly correlated with upper airway obstruction; children presented with huge adenoids with or without tonsillar enlargement were more susciptable to enuresis; suggesting thereby the closest correspondence between severity of upper airway obstruction and nocturnal enuresis.

In our series of patients nocturnal enuresis also increased with the duration of upper airway obstruction.

Poor school performance was reported by many authors as a result of prolonged partial airway obstruction during sleep caused by adenotonsillar hypertrophy (Deutch, 1996). Children with school problems pose a challenge for many physicians, in which complex learning problems are often the cause of poor school performance.

Attention deficit hyperactivity disorder (ADHD) is one of the most common disorders in childhood and adolescence, with a prevalence of approximately 5 %, causing school failure and social problems. It has been reported that during the past decade the

prevalence of ADHD has increased dramatically (Robison et al, 1999). Wolraide et al, 1998, showed prevalence rates of ADHD 16.1% (all types); 8.8 % inattentive; 2.6 % hyperactive- impulsive; 4.6% combined types; rates were 6.8 %; 3.2 %; 0.6 % and 2.9% respectively when impairment was taken into consideration. In our study ADHD was significantly higher among cases with upper airway obstruction than controls (18% versus 3.3%).

ADHD was prominent in cases with huge adenoids, and appears directly proportional with the severity of obstruction of upper airway. Brouillette et al, 1982, reported hyperactivity in 48 % and learning problems in 40% in children with continuous partial airway obstruction.

Effects of OSAS on daytime cognitive function are virtually unexplored in children; yet adults with OSAS have been shown to have impaired attention span, concentration, memory, vigilance and motor skills. Findley et al, 1986, have shown a correlation between daytime cognitive impairment and nocturnal hypoxaemia.

Since many children with OSAS suffer moderate-to-severe hypoxaemia during sleep, it is likely that their daytime cognitive function is similarly impaired (Carroll and Loughlin, 1995). Marcotte et al, 1998, took a different approach as they revealed that ADHD children have more sleep breathing problems.

Psychometric scores, have confirmed clinical results, and showed highly significant scores in moderate and severe upper airway obstruction including Attention Problem-Immaturity (AP) and Motor Tension-Excess (ME) subscales of RBPC among our study group. This goes with Ali et al, 1993, who reported more

hyperactive, inattentive behavior in children at high risk of sleep and breathing disorders.

In the present study adenoidal hypertrophy plays an important role in the development of Attention deficit in our series of patients with chronic adenotonsillitis, with a peak incidence of ADHD in those with duration of 3-4 years of chronic adenotonsillitis with upper airway obstruction.

Owens et al, 1997, reported daytime disruptive behaviors more likely to be associated with the milder sleep disturbances due to upper airway obstruction in children in a primary care unit.

Bizzare behavior, discipline problems, hyperactivity, and aggressive behavior are reported to varying degrees in 9 to 31% of children with OSAS. Similar figure 38% prevalence of disruptive behavior disorders was reported in 100 rheumatic children aged 8-15 years, 63.6% of those who suffered from nocturnal enuresis had also disruptive behavior (Aiad, 1997).

Ali et al, 1993, reported that parents of children with sleep breathing disorders thought them more aggressive.

Aggressive and rebellious behavior was recorded in 40% of children with continuous partial airway obstruction (Guilleminault et al, 1982).

In the present study, clinically diagnosed oppositional defiant disorder (ODD) was significantly higher among the study group 22.8% in contrast to prevalence rate of 6.7% in the controls. ODD was more prevalent in cases with severer upper airway obstruction.

On the other hand clinically diagnosed conduct disorder did not differ between study group and controls.

Psychometric scores using RBPC showed highly significant scores on Conduct Disorder (CD) subscale but no significant difference on the Socialized Aggression (SA) subscale . It is clear that our study group did not have severe disruptive behavior problems.

Oppositional defiant disorder was directly proportional to the degree of upper airway obstruction. Both oppositional defiant disorder and the more severe conduct disorder were more prevalent as the duration of upper airway obstruction increased in patients with chronic adenotonsillitis.

Several studies all over the world in varying populations have been carried out to estimate the prevalence of depression; yet they are still far away from being truly representative because of nosological and diagnostic controversies (Kielholz et al, 1982). In Egypt, Okasha et al, 1988, reported overall estimate of depressive disorders 15.3 %; Abdel- Baky et al, 1988, reported 22.5% prevalence of affective disorders among psychiatric morbidity in primary school children.

Ample evidence indicates that depression is likely to be associated with airway obstruction in cases of bronchial asthma in children. Apter et al, 1997, demonstrated that mood states are associated with perception of airway obstruction in asthmatic patients.

In the present study there was no significant difference in prevalence of depression between study group and control being

6.03 % and 3.3 % respectively. However, psychometric scores of Child Depression Inventory (CDI) was higher among cases than controls.

Kaplan, 1995, argued that sleep fragmentation was most related to feelings of depression in patients with nasal obstruction; Katz and Mcttorneyt, 1998, reported that patients with chronic insomnia including patients with airway obstruction are more likely to develop affective disorders. Yet children with OSAS have normal amount of delta sleep (Carroll et al, 1992) and children with continuous partial obstruction during sleep do not show sleep fragmentation (Carroll and Loughlin, 1995); however our results show that depression is very likely to be associated with other behavioral problems in some cases of chronic adenotonsillitis.

In the present study depression was found to be directly proportional to the duration of chronic adenotonsillitis. Perrin et al, 1987, suggested a variety of reasons to expect increased psychosocial morbidity among those children: chronic or recurrent episodes of pain and diminished or altered physiologic function may promote anxiety and depression.

The presence of a chronic condition may also limit or alter social interaction and distinguish children from their peers, which in turn increase the risk of problems with normal psychological adjustment.

A large number of studies was conducted to assess the prevalence of anxiety disorders in normal children population.

Sayed et al, 1994, has studied the prevalence of anxiety symptoms among primary school children; it was 7.9%; Abdel-

Baky et al, 1988, reported prevalence of 3.9% of anxiety disorder among primary school children; Mousa et al, 1990, reported 2.24% prevalence rate of anxiety.

In the current study anxiety disorders were insignificantly higher among the study group 10.2% opposite to 6.6% recorded in the control group. Anxiety disorder were more prevalent among children with huge adenoids with and without tonsillar hypertrophy. Psychometric scores, however, using the Child Anxiety Scale (CAS) were higher among cases than the controls.

A predominance of generalized anxiety disorder, separation anxiety disorder in our study was noted. In contrast to a study conducted by Pollack et al, 1996, which revealed 41% and 17% occurrence of panic attacks and disorders respectively in airway obstruction due to different respiratory diseases; yet their study was conducted on adult population and most of patients were suffering COPD.

Swedo et al, 1993, described a group of children with obsessive compulsive disorder (OCD) and / or tic disorder with onset from age 3 to puberty, presumably due to a re–infection with GABHS, they were called PANDAS (Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections).

In this work no OCD cases were found, this may be due to two reasons : first, only small proportion of children exposed to GABHS develop PANDAS while the vast majority do not; second, the fact that GABHS is found in only 17% of patients with acute tonsillitis (Cowan and Hibbert, 1997).

Anxiety disorder in the current study decreased in prevalence as the duration of illness increased, this may be due to the adaptational nature of anxiety disorders.

In the present study an excess of externalizing behavior, inattention, aggression and hyperactivity was noticed among our study group more than internalizing behavior problems such as anxiety and depression. Our results go partly with Ali et al, 1996, who reported excess of externalizing behavior on the Conners scale, although insignificant from controls; this could be explained by insensitivity of Conners scale; relatively mild upper airway obstruction, in addition to small number of children enrolled in their study .

In our study the externalizing behaviors (attention problems and oppositional behaviors) not only were associated with the degree of upper airway obstruction but also with the duration of chronic adenotonsillitis.

Ali et al, 1994, wondered if many cases of this condition will improve spontaneously as their tonsils involute. Yet Lynham, 1996, reported that children who manifest symptoms of hyperactivity-impulsivity- attention problems (HIA) and conduct problems (CP) are at the greatest risk for chronic offending. As HIA increases risk in those already at risk and HIA leads to symptoms of CP. In addition children with symptoms of HIA and CD are afflicted with a virulent strain of conduct disorder, best described as fledging psychopath; high lightening the need for early intervention for chronic offending.

In children it is difficult to justify surgery as a therapeutic trial, particularly as the long – term effects of sleep breathing

disorders due to adenotonsillar enlargement on children's development are still not known.

The approach to tonsillectomy and adenoidectomy has become progressively more conservative. As this conservative approach becomes a part of collective medical thought, and as fewer children undergo tonsillectomy and adenoidectomy, there may be an increasing tendency for children to develop the sequelae of adenotonsillar hypertrophy.

The pediatric literature contains mostly anecdotal reports of improved behavior after surgical relief of chronic upper airway obstruction.

Lind and Lundell, 1982, described 14children with OSAS, growth impairment, and CO_2 retention and stated that a great improvement in school results was noted in several children after tonsillectomy.

Guilleminault et al, 1982, reported on children with continuous partial airway obstruction during sleep who showed abnormal sleep latencies and impaired cognitive function preoperatively and all returned to normal after tonsillectomy and / or adenoidectomy. Karvath et al, 1977, described also children with apparent mental retardation who appeared more intelligent after surgery.

Nocturnal enuresis in children can at times be alleviated or resolved completely with surgery on the upper airway. Nowak and Weider, 1998, presented a report of a child of whom nocturnal enuresis began immediately after reconstructive surgery of the pharynx that caused upper airway obstruction and diminished

immediately following adenoidectomy. In the current study there was significant reduction in nocturnal enuresis postoperatively from 22.8% to 7.69 %. The improvement was most marked in cases presented with severe degrees of upper airway obstruction. Swift, 1988, reported that 16% of children with OSAS had enuresis preoperatively but only one third of them became dry after surgery. Weider and Hauri reported in 1985, that children with nocturnal airway obstruction and enuresis, was cured or significantly improved after surgical removal of adenoids or the tonsils. Weider et al, 1991, reported that upper airway surgery dramatically diminished enuresis in 76% of their patients; leading them to conclude that enuresis should be an indication for adenotonsillectomy or other airway surgery; and speculated that improvement of enuresis after surgery was due to elimination of sleep disruption and the subsequent normalization of arousal mechanisms. El Asfour et al, 2000, declared that removal of the upper airway obstruction by adenotonsillectomy lead to complete cure of monosymptomatic nocturnal enuresis but inadequate or no response in polysymptomatic enuretic children.

Biederman et al, 1994, reported that enuresis was associated with increased risk for learning disability, and impaired school achievement in children.

Developmental delay in a child with severe OSAS has been reported to improve within days of tracheostomy and, overtime, to resolve completely (Brouillette et al, 1982).

In our study significant improvement was noted in attention deficit disorders postoperatively from 18% to 7.69 % especially in patients suffering from huge tonsils and adenoids. Improvement on the Attention Problems immaturity (AP) and Motor Tension-Excess

(ME) subscales were marked even in the less severe groups of upper airway obstruction. This goes with Ali et al, 1996, who showed improvement of children on measures of inattention and hyperactivity on the Conners rating scale. They suggested that the improvement was due to relief of upper airway obstruction.

On the other hand in the current study clinically diagnosed disruptive behavior disorders both oppositional defiant and conduct disorders showed no significant improvement after adenotonsillectomy.

In contrast to our results kotiniemi et al, 1996, reported behavioral improvement in 33% of children after adenotonsillectomy. This may be due to different age group of children and that middle ear diseased children were included in their study.

The Socialized Aggression (SA) subscale did not show any significant improvement along all groups of the study. Yet the Conduct Disorder subscale (CD) showed a highly significant improvement in patients presenting with huge adenoids with and without tonsillar enlargement after surgical intervention.

This goes with Ali et al, 1996, who recorded improvement on aggression subscale in cases with sleep breathing disorders after adenotonsillectomy.

Our series of children with moderate degree of upper airway obstruction improved in terms of attention and hyperactivity but not as much as children with severe degree of upper airway obstruction who showed improvement in attention, hyperactivity and conduct problems. However the magnitude of improvement was related to

the degree of quality of life impairment prior to treatment rather than to the severity of disease in OSAS patients treated with CPAP. (Ambrosio et al, 1999) .

Our results indicate that even mild degree of upper airway obstruction has measurable effects on children's daytime behavior, something that was suggested by the results of Ali et al, 1993.

During the past four decades, the distress that can accompany hospitalization and surgery of children has been recognized. In the 1950s, it was first noted that separation from parents and family and exposure to medical procedures could produce emotional distress in children. It was also recognized that behavioral problem could persist in children after hospitalization.(Jessner et al , 1952).

In 1995, Klausner et al, reported three children who underwent tonsillectomy and adenoidectomy and post operatively suffered disruptive behavior problems that were later recognized as forms of depression.

Although the actual incidence of postoperative depression in children undergoing tonsillectomy is not known. It is conceivable that the emotional trauma surrounding the surgery could push a child predisposed to depression over the edge.

In the present study there was no significant difference between preoperative and postoperative prevalence of clinically diagnosed depression. Also psychometric scores of Children Depression Inventory (CDI) showed the same results.

Clinically diagnosed anxiety disorders were slightly higher in the postoperative group, yet non significant, in the present study.

However significant reduction in anxiety postoperatively in cases with severe degrees of upper airway obstruction was noted.

Psychometric scores of Children Anxiety Scale (CAS) showed non significant difference between preoperative and postoperative groups.

Klausner et al, 1995, proposed that children may interpret tonsillectomy, or similarly any hospitalization or surgical procedure, as failure, punishment, or retribution for bad behavior.

Our results could be explained by the fact that one factor may contribute to vulnerability to postoperative depression is the age of the child at time of surgery, as our study group children were 7-12 years old, while adverse reactions to surgery have been noted to occur more frequently in preschool-aged children than in older children (Wolfer and Visintainer, 1975).

Our results go with Engleman et al, 1997, study on patients with moderate and severe apnea / hypoapnea syndrome (SAHS), who were on continuous positive airway pressure (CPAP) and showed significant reduction of self rated depression and anxiety, on the Hospital Anxiety and Depression Scale (HADS), in addition to cognitive improvement skills.

Ambrosio et al, 1999, reported that all aspects of the quality of life from physical, and emotional health to social functioning, are markedly impaired by OSAS, and nasal CPAP therapy improved those aspects related to vitality, social functioning, and mental health, although they reported either worsening or no changes in some of psychological symptoms such as depression, this could be explained by older age of patients enrolled in that study, that

majority of patients had severe OSAS, and short time of the trial, (8weeks).

Löth et al, 1999, showed that snorer adults, due to partial or total nasal obstruction, experienced a better quality of life when nasal breathing was improved at night, using nostril dilator, more pronounced in the energy dimension, suggesting there by the importance of nasal breathing during sleep.

Because snoring reflects a mild degree of partial upper airway obstruction, Carroll and Loughlin, 1995, reported that parents noted remarkable improvement in their snoring child's behavior only in retrospect, after adenotonsillectomy, even if OSAS could not be documented preoperatively.

It is conceivable that adenotonsillectomy could result in behavioral changes; the mechanism of postoperative improvement in such children is still unknown.

Data indicate that what happens during sleep in chronic adenotonsillitis suffering children, with its possible upper airway obstruction, can not be ignored, especially since sleep period is likely to be considerably longer in the child. Furthermore, it cannot be assumed that observations on adults with lung disease or even upper airway obstructive diseases are applicable to children. In fact the experience in children with OSAS strongly suggests that breathing disorders associated with sleep are significantly different in children (Carroll and Loughlin 1992).

In children, the clinician must account for the influences of age, growth, development and the effect of disease on the psychology of child.

It seems that upper airway obstruction in children whether due to OSAS (Carroll and Loughlin , 1995) or allergic rhinitis (Klein et al, 1985) or chronic adenotonsillitis, as in the current study, is exceptionally associated with externalizing behaviors in terms of inattention, hyperactivity and oppositionalism or aggression; in contrast to documented effects of upper airway obstruction in adults (Diagnostic Classification Steering Committee, 1990) and lower airway obstruction both in adults with COPD (Mubarak et al , 1996) and children with bronchial asthma (Hamlett et al, 1992) in which internalizing problems in term of anxiety and depression are significantly recorded.

SUMMARY AND CONCLUSIONS

Summary and Conclusions

Waldeyer's ring is most prominent during childhood, when the size of the oro-naso-pharyngeal space is not yet fully developed, but decreases spontaneously with age. In the child, enlarged tonsils and/ or adenoids may cause eustachian tube dysfunction / otitis media, rhinosinusitis, upper airway obstruction, obstructive sleep opnea, voice changes , change in facial growth, swallowing problems and can affect overall quality of life.

This study was conducted on 83 children aged 7-12 years with varying degrees of upper airway obstruction due to chronic adenotonsillitis of duration more than one year, going for adenotonsillectomy.

Our patients were selected from the Otorhinolaryngology Clinic of Ain Shams University Hospital and Nasr City Health Insurance Hospital, besides 30 apparently normal control children.

Full general and otorhinolarygologic examination were done for all children of both groups in addition to lateral soft tissue film of the nasopharynx.

The psychiatric diagnosis for all of them was done according to DSM-IV (1994) as well as specific psychological tests used in Ain Shams psychiatric Center were applied to all the diseased children and controls.

Namely: Child Behavior Problem Checklist
Children's Depression Inventory.
Children's Anxiety Scale

Both before and 5-6 months after adenotonsillectomy operation.

Enuresis was more significant among cases with chronic adenotonsillitis than the controls; and increases with the severity and duration of upper airway obstruction.

Clinically diagnosed attention deficit disorder was more prevalent among cases than the controls; and increases also with the severity of obstruction of upper airway, with peak incidence at 3-4 years duration. Psychometric scores using RBPC were highly significant in patients presenting with adenoidal enlargement in the inattention and hyperactivity subscales, confirming the clinical results.

Clinically diagnosed oppositional defiant disorder was more significant among cases than the controls; most prevalent in cases with huge tonsils and adenoids, whereas clinically diagnosed conduct disorder showed no significant difference among both groups. Using RBPC, the Socialized Aggression subscale showed no significant difference between cases and controls, yet the Conduct Disorder subscale was higher among cases than the controls and increased with the severity of upper airway obstruction. Both oppositional defiant disorder and conduct disorder increased with the duration of chronic adenotonsillitis .

Depression was not significantly higher among cases than controls, psychometric scores, however, showed significant difference. Depression was recorded in patients with longer duration of upper airway obstruction.

Clinically diagnosed anxiety showed no significant difference between the cases and controls, however psychometric scores were higher among cases than the controls. Anxiety disorder was inversely proportional to the duration of chronic adenotonsillitis.

Postoperatively there was a significant reduction in nocturnal enuresis most marked in cases presented with huge tonsils and adenoids. Also number of cases of attention deficit disorder was significantly reduced postoperatively and psychometric scores improved significantly on measures of attention and hyperactivity.

No significant improvement in clinically diagnosed disruptive behavior disorders postoperatively, yet there was significant improvement on Conduct Disorder subscale of RBPC.

Depression and anxiety disorders showed no significant change postoperatively also psychometric scores of their specific psychological tests showed no significant difference after surgery.

From this work we may conclude that upper airway obstruction in children is exceptionally associated with externalizing behaviors in term of inattention, hyperactivity and oppositionalism, in contrast to documented effects of upper airway obstruction in adults and lower airway obstruction in cases of chronic obstructive pulmonary diseases and bronchial asthma both in children and adults in which internalizing problems in term of anxiety and depression are significantly recorded .

APPENDIX

I Revised Behavior Problem Chicklist

القائمة المعدلة لمشاكل السلوك

	صفر	١	٢
١. غير مستقر لايستطيع الجلوس ساكناً .			
٢. يستعرض يسعى لجذب الانتباه .			
٣. يتأخر بالخارج ليلاً .			
٤. واع لذاته يخاف من ردود أفعال الاخرين تجاهه " يعمل حساب الناس " .			
٥. فوضوى يضايق الاخرين .			
٦. يشعر بالنقص .			
٧. يسرق بالتعاون مع الاخرين .			
٨. منشغل بعالمه الخاص " يسرح " .			
٩. خجول ومنطوى .			
١٠. منسحب يفضل الانشطة المنفردة .			
١١. ينتمى لجماعة " شلة " او مجموعة من المشاغبين .			
١٢. كلامه مكرر يعيد ويزيد ما يقوله .			
١٣. ضعيف التركيز لايستطيع الانتباه لفترة طويلة .			
١٤. ضعيف الثقة بالنفس .			
١٥. غير منتبه لما يقوله الاخرون .			
١٦. كلامه غير مترابط ما يقوله ليس له معنى واضح .			
١٧. كثير الشجار .			
١٨. يدين بالولاء للأصدقاء المنحرفين .			
١٩. تحدث له نوبات غضب حادة .			
٢٠. يهرب من المدرسة عادة بصحبة اخرين .			
٢١. مفرط الحساسية أحاسيسة تجرح بسهولة .			

		٢٢. خائف قلوق بصفة عامة .
		٢٣. غير مسئول ولايعتمد عليه .
		٢٤. له اصدقاء سوء " وهم من يعانوا عادة من مشاكل " .
		٢٥. متوتر غير قادر على الاسترخاء .
		٢٦. غير مطيع صعب السيطرة عليه .
		٢٧. مكتئب وحزين دائما .
		٢٨. غير متعاون فى المواقف الاجتماعية .
		٢٩. سلبى قابل للايحاء سهل الانقياد .
		٣٠. مفرط النشاط دائم الحركة .
		٣١. سهل التشتت يسهل تلهيته عن المهمة الموكلة اليه .
		٣٢. مخرب لممتلكاته و/ أو ممتلكات غيره .
		٣٣. عنيد يتعمد فعل عكس ما يطلب منه .
		٣٤. وقح يرد الكلمة بالكلمة .
		٣٥. متبلد كسول يتحرك ببطء .
		٣٦. نعسان " مش مصحصح " غير يقظ ذهنياً .
		٣٧. عصبى منزعج سريع النرفزة غير مستقر المزاج يصدم من اقل شئ .
		٣٨. سريع الغضب حاد المزاج سهل اغضابه .
		٣٩. يعبر عن افكار غريبة بعيدة الاحتمال .
		٤٠. مجادل ومقاوح يناقش بغضب وحدة .
		٤١. متجهم لايحب الاندماج مع الاخرين دائم العبوس " مكشر".
		٤٢. لحوح وزنان لايقبل ان يرفض له طلب .
		٤٣. يتجنب النظر الى الاخرين فى عيونهم .
		٤٤. يجيب بدون أن يفكر .
		٤٥. لايستطيع أن يعمل مستقلاً يحتاج لمساعدة الاخرين

		وانتباههم بصفة دائمة .
		٤٦. يتعاطى العقاقير مع اخرين .
		٤٧. مندفع يبدأ العمل قبل أن يفهم ولايتوقف للتفكير .
		٤٨. يمضغ أشياء غير صالحة للأكل .
		٤٩. يحاول السيطرة على الاخرين بالتهديد والوعيد .
		٥٠. يشاكس الاطفال الأخرين ليلفت انتباههم "يبدو انه لايعرف الطريقة الصحيحة للتقرب منهم ".
		٥١. يسرق من الناس خارج المنزل .
		٥٢. يعبر عن معتقدات من الواضح انها غير حقيقية " ضلالات"
		٥٣. يقول ان أحد لايحبه او يحبها .
		٥٤. يعبر بصراحة عن عدم احترامه للقيم الاخلاقية والقوانين .
		٥٥. يتفاخر ويتباهى .
		٥٦. بطئ وغير دقيق فى أعماله .
		٥٧. يظهر القليل من الاهتمام بما يحيطه من اشياء .
		٥٨. لاينهى الاعمال يستسلم بسهولة ويفتقد المثابرة .
		٥٩. ينتمى لمجموعة ترفض الانشطة المدرسة مثل الفرق الرياضية والنوادى ترفض ان تساعد الاخرين .
		٦٠. غشاش .
		٦١. يسعى لمصاحبة من هم اكبر منه سناً واكثر منه خبرة .
		٦٢. يعرف ما يحدث لكنه لايثير اهتمامه .
		٦٣. يرفض ان يترك جانب امه " او راعيه " .
		٦٤. يصعب عليه الاختيار لايستطيع ان يقرر .
		٦٥. يغيظ الاخرين .
		٦٦. شارد الذهن ينسى ابسط الاشياء .
		٦٧. يتصرف على أنه او أنها اصغر سناً غير ناضج " طفولى"

٦٨. لديه مشاكل فى تتبع الاتجاهات .

٦٩. يكذب ليحمى اصدقاءه .

٧٠. يخاف ان يجرب اشياء جديدة لخوفه من الفشل .

٧١. أنانى لايحب المشاركة طماع .

٧٢. يشرب الخمور فى صحبة اخرين .

٧٣. كتبه المدرسية قذرة وغير مرتبة .

٧٤. لايستجيب للمديح الموجه من الكبار .

٧٥. غير محبوب من الاخرين " وحيد " بسبب سلوكه العدوانى.

٧٦. لايستخدم اللغة للأتصال .

٧٧. متسرع يريد الاشياء فى التو واللحظة .

٧٨. يرفض التوجيه ولايفعل ما يقال له .

٧٩. يلوم الاخرين وينكر اخطاءه الشخصية .

٨٠. يعجبه ويسعى لمصاحبة الاصدقاء الخشنين .

٨١. العقاب لايغير سلوكه او سلوكها .

٨٢. يتضايق بشدة متململ لايطيق الجلوس مستقراً .

٨٣. متعمد القسوة تجاه الاخرين .

٨٤. يشعر أنه لايستطيع النجاح .

٨٥. يصف الاشياء الخيالية كالحقيقية ولايستطيع التفريق بين الواقع والخيال .

٨٦. لايحتضن او يقبل افراد عائلته " غير عاطفى " .

٨٧. يهرب من المنزل .

٨٨. يعجب بصراحة بالاشخاص الخارجين عن القانون .

٨٩. يكرر ما يقال له او مايسمعه من الاخرين كالببغاء .

II Children's Anxiety Scale

مقياس-ق-للأطفال

الاسم :..... **الجنس:**.....

السن :..... **المدرسة :**.....

الصف الدراسى:.....

التعليمات :

العبارات الى حتقراها دلوقت حاجات بتحصل كتير ذيك المطلوب منك أنك تشوف الحاجات دى بتحصل لك أم لا أذا كانت بتحصل لك ارسم دائرة حول كلمة **نعم** ، وازا كانت عمرها ما حصلت لك ارسم دائرة حول كلمة **لا** ولا تترك اى عبارة دون اجابة وشكرا.

المظاهر الجسمية	المظاهر الفسيولوجية	المظاهر الحركية	المظاهر الانفعالية	المظاهر العقلية	المظاهر الاجتماعية	الدرجة الكلية

١-	ساعات كثيرة احس بصداع فى دماغى	نعم	لا
٢-	باعرق بسرعة	نعم	لا
٣-	حاسس ان نشاطى بقى غير الاول	نعم	لا
٤-	انا عصبى	نعم	لا
٥-	دايما فكرى مشغول ببكرة	نعم	لا
٦-	احب اقعد لوحدى كثير	نعم	لا
٧-	لما يكون بالى مشغول معرفش ابلع الاكل او ابلعة بصعوبة	نعم	لا
٨-	وجهى يحمر بسرعة	نعم	لا

٩-	مبقاش لى نفس العب مع اصحابي زى الاول	نعم	لا
١٠-	مش حاسس أنى مرتاح	نعم	لا
١١-	مش عارف اركز في درسى	نعم	لا
١٢-	انا مليش اصحاب كتير	نعم	لا
١٣-	ساعات كتير ماما او بابا يقولوا لى أن وجهك اصفر	نعم	لا
١٤-	لما أفكر فى حاجة مهمة قلبى يدق بسرعة قوى	نعم	لا
١٥-	بقيت اتعب من اقل مجهود	نعم	لا
١٦-	حاسس أني زهقان	نعم	لا
١٧-	حاسس أن مستواى في المدرسة بقى أقل من الأول	نعم	لا
١٨-	أصحابي بيزعلوا منى علشان مبرضاش العب معاهم زى الأول	نعم	لا
١٩-	صحتي مش كويسة دلوقتى	نعم	لا
٢٠-	ساعات باحس أنى مش عارف اتنفس وأني مخنوق	نعم	لا
٢١-	حركتي قلت عن الأول	نعم	لا
٢٢-	ساعات بأحلم احلام وحشة بالليل	نعم	لا
٢٣-	كل مافتح كتاب الاقى نفسى سرحت	نعم	لا
٢٤-	خسرت اصحاب كثير فى الايام الأخيرة	نعم	لا
٢٥-	ساعات كثير مبيبقاش لى نفس للأكل	نعم	لا
٢٦-	ساعات كثير بيجينى اسهال	نعم	لا
٢٧-	ساعات يلاحظ علي الناس بعض الحركات العصبية	نعم	لا
٢٨-	أنا دائما محتار	نعم	لا
٢٩-	بقيت أنسى كل اللى اذاكره	نعم	لا
٣٠-	ساعات بابقى مش عايز اشوف حد	نعم	لا
٣١-	وزنى نقص في الأيام الاخيرة	نعم	لا
٣٢-	ساعات احس بزغللة في عيني	نعم	لا
٣٣-	ساعات احس اني مش قادر اقعد على كرسي لمدة طويلة وعايز اتحرك من مكان لمكان	نعم	لا
٣٤-	أنا دايما مهموم (شايل الهم بدرى)	نعم	لا
٣٥-	كل اللى يشوفني يقول لي اني كنت أحسن من كدة في المذاكرة	نعم	لا
٣٦-	باحس انى متضايق من رأى الناس في الآن	نعم	لا

III Children's Depression Inventory

مقياس (د) للصغار (CDI)

إختبار الإكـتـئاب للأطفـال

الأســـــــــم : ..... **تاريخ اليوم :** / /

المستوى التعليمى : ..... **تاريخ الميلاد:** / /

أحيانا مايشعر الصغار والشباب ببعض المشاعر ويفكروا فى بعض الأفكار وفى هذه الورقة بعض المشاعر والأفكار مكتوبة فى صورة مجموعات ، كل مجموعة بتتكون من ثلاث عبارات ، عليك ان تختار عبارة واحدة من كل مجموعة من العبارات بتشوف انت ان العبارة دية بتوصفك خلال الأسبوعين الأخيرين ، ثم ضع علامة × فى الصندوق المجاور للعبارة اللى أنت اخترتها ، وبعد كده انتقل لمجموعة العبارات اللى بعدها ، وهكذا حتى تنتهى من الورقة .

خلى بالك ما فيش اجابة صح او اجابة غلط ولكن المطلوب منك هــو أن تختار عبارة واحدة من كل مجموعة ، وتكون العبارة دية هى اللى أنت بتشوف أنها بتوصف حالتك ، وعلى سبيل المثال شوف مثلا المجموعة دية :-

- ☒ - أنا أقرأ كتب طول الوقت .
- ☐ - أنا أقرأ الكتب احيانا .
- ☐ - أنا عمرى ما قريت كتب .

اذا كانت العبارة الأولى تنطبق عليك وبتوصفك فى الأسبوعين الأخيرين ضع علامة × فى الصندوق المجاور لها تماما زى المثال اللى فوق .

إفتكر انك تختار العبارة اللى بتوصف مشاعرك وأفكارك في الأسبوعين الأخيرين .

١- ☐ - بأبقى حزين احيانا .
- ☐ - بأبقى حزين فى أوقات كثيرة .
- ☐ - بأبقى حزين طول الوقت .

٢- ☐ - مافيش حاجة حاتمشى كويس بالنسبة لى أبدا .
☐ - أنا مش متأكد من أن الأشياء والظروف حتبقى كويسة بالنسبة لى.
☐ - الأشياء والظروف حاتبقى كويسة بالنسبة لى.

٣- ☐ - أنا بأعمل اغلب الحاجات بطريقة كويسة .
☐ - أنا بأعمل حاجات كثيرة بطريقة غلط .
☐ - أنا بأعمل كل حاجة بطريقة غلط .

٤- ☐ - فية حاجات كثيرة بتسلينى .
☐ - بعض الحاجات والأشياء بتسلينى .
☐ - مافيش حاجة بتسلينى .

٥- ☐ - فى كل الأوقات أنا وحش أو مش كويس .
☐ - فى أوقات كثيرة بأكون وحش أو مش كويس .
☐ - أحيانا بأكون وحش أو مش كويس .

٦- ☐ - أحيانا بأفكر فى أشياء وحشة (مش كويسة) بتحصل لي .
☐ - أنا قلقان ومشغول من أن بعض الأشياء اللى مش كويسة حاتحصل لى.
☐ - أنا متأكد أن أشياء فظيعة حاتحصل لى .

٧- ☐ - أنا بأكره نفسى .
☐ - أنا لا أحب نفسى .
☐ - أنا بأحب نفسى .

٨- ☐ - كل الحاجات الوحشة أو اللى مش كويسة بتكون بسببى أنا .
☐ - كثير من الحاجات الوحشة أو اللى مش كويسة بتكون بسببى أنا .
☐ - مش دايما الحاجات الوحشة أو اللى مش كويسة بتكون بسببى أنا .

٩- ☐ - أنا مابفكرش فى أنى أموت نفسى .
☐ - أنا بأفكر فى أنى أموّت نفسى لكن مش حاأعمل كدة .
☐ - أنا عايز أموّت نفسى

١٠- ☐ - يوميا بأشعر بأنى عايز أعيط (أبكى) .
☐ - فى أوقات كثيرة بأشعر بأنى عايز أعيط .
☐ - أحيانا بأشعر بأنى عايز أعيط .

١١- ☐ - فية أشياء بتضايقنى طول الوقت .
☐ - فية أشياء بتضايقنى طول اوقات كثيرة .
☐ - فية أشياء بتضايقنى أحيانا .

١٢- ☐ - أنا بأحب أكون مع الناس .
☐ - فى أوقات كثيرة أنا ما أحبش أكون مع الناس .
☐ - أنامش عايز أكون مع الناس أبدا .

١٣- ☐ - أنا ما أقدرش (لا أستطيع) أن أقرر أو أحدد رأى فى الأشياء .
☐ - من الصعب علىّ أنى أقرر أو أحدد رأى فى الأشياء .
☐ - أنا بأقرر أو أحدد رأى فى الأشياء بسهولة .

١٤- ☐ - أنا شكلى كويس .
☐ - فية بعض الحاجات مش كويسة فى شكلى .
☐ - أنا شكلى مش كويس أو وحش .

١٥- ☐ - يجب على أن أدفع نفسى طول الوقت علشان أعمل واجبات المدرسة .
☐ - يجب على أن أدفع نفسى أكثر من مرة علشان أعمل واجبات المدرسة .
☐ - واجبات المدرسة مش مشكلة كبيرة بالنسبة لى .

أفتكر أنك تصف حالك فى الأسبوعين الأخرين .

١٦- ☐- كل ليلة بيبقى صعب على أنى أنام .
☐- فى ليالى كثيرة بيبقى صعب على أنى أنام .
☐- أنا بأنام كويس جدا .

١٧- ☐- بأشعر أحيانا بأنى مجهد أو تعبان .
☐- بأشعر فى أوقات كثيرة بأنى مجهد أو تعبان .
☐- بأشعر طول الوقت بالتعب والأجهاد .

١٨- ☐- فى غالبية الأيام بيبقى ماعنديش نفس للأكل .
☐- فى أيام كثيرة بيبقى ماعنديش نفس للأكل .
☐- أنا بأكل كويس جدا .

١٩- ☐- أنا مش قلقان من أى ألام أو أوجاع .
☐- فى مرات كثيرة بأبقى قلقان من بعض الألام والأوجاع .
☐- طول الوقت بأبقى قلقان من بعض الألام والأوجاع .

٢٠- ☐- أنا لا أشعر بالوحدة .
☐- فى أوقات كثيرة بأشعر بالوحدة .
☐- طول الوقت بأشعر بالوحدة .

٢١- ☐- أنا عمرى ما شعرت بالمتعة فى المدرسة .
☐- أحيانا بأشعرت بالمتعة فى المدرسة .
☐- فى أوقات كثيرة بأشعرت بالمتعة فى المدرسة .

٢٢- ☐- أنا عندى أصحاب كثيرة .
☐- أنا عندى بعض الأصحاب ولكن باتمنى يكون عندى أصحاب أكثر .
☐- أنا ماعنديش ولا صاحب .

٢٣- ☐ - عملى - شغلى - المدرسى كويس .

☐ - عملى المدرسى مش كويس زى ما كان قبل كدة .

☐ - عملى المدرسى وحش قوى فى مواد كنت دايما كويس فيها .

٢٤- ☐ - أنا لايمكن أن أكون كويس مثل بقية زملائى .

☐ - لو أردت ، فأنى أقدر أكون كويس مثل باقى زملائى .

☐ - أنا كويس زى باقى زملائى .

٢٥- ☐ - فى الحقيقة ما فيش حد بيحبنى .

☐ - أنا مش متأكد أن فية حد بيحبنى .

☐ - أنا متأكد من أن بعض الأشخاص بيحبونى .

٢٦- ☐ - أنا فى العادة بأعمل اللى بيطلب منى .

☐ - فى أغلبية الأوقات أنا مش بأعمل اللى بيطلب منى .

☐ - أنا عمرى ما عملت اللى بيطلب منى .

٢٧- ☐ - أنا بأنسجم مع الناس .

☐ - فى أوقات كثيرة أنا بأتورط فى خناقات .

☐ - طول الوقت أنا بأتورط فى خناقات .

النهايـــــة

وشكرا للإجابة على الأسئلة

المجموع ☐

التطبيـــق فردى ☐ جماعى ☐

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مراجع باللغة العربية

- د/غريب عبد الفتاح غريب – ١٩٨٨–مقياس(د) للصغار ماریاکوفـاکس دار النهضة العربية – القاهرة
- د/مايسة النيال و د/مدحت عبد الحميد –١٩٩١الفروق بين القلق و الاكتئاب بين مرض روماتيزم القلب و الاسوياء من الاطفال , المؤتمر السـنوى الرابـع للطفل المصري –القاهرة– مصر.

" جامعة عين شمس "
الكلية :

شكـــــــــــــــــر

أشكـــر الســـادة الأســـاتذة الـذين قامـوا بالاشـــــراف وهـــم :

١) . . . . . .
٢) . . . . . .
٣) . . . . . .
٤) . . . . . .

ثم الأشخـاص الذيـن تعـاونوا معـى فـى البحـــث وهـــم :

١) . . . . . .
٢) . . . . . .
٣) . . . . . .

وكـــذلك الهيئـــات :

١) . . . . . .
٢) . . . . . .
٣) . . . . . .

ARABIC SUMMARY

الملخص العربي

تعتبر حلقة والديرز أكثر بروزا في الطفولة ،عندما يكون حجم البلعوم الأنفي غير مكتمل النمو بعد،ولكن يقل هذا البروز تلقائيا مع التقدم في العمر.

وقد يتسبب تضخم اللوزتين و/أو لحمية البلعوم الأنفي في الاختلال الوظيفي لقناة أستاكيوس ،التهاب الأذن الوسطى ،التهاب الجيوب الأنفيه ،انسداد مجرى التنفس العلوي،انقطاع التنفس الانسدادي أثناء النوم،تغيرات في الصوت،تغيرات في نمو الوجه و مشاكل في البلع كذلك التأثير على نوعية الحياه بشكل عام. ولقد أجريت هذه الدراسه على ٨٣ طفلا،تراوحت أعمارهم بين ٧-١٢ عاما،يعانون درجات مختلفة من انسداد مجرى التنفس العلوي-نتيجة التهاب اللوزتين و لحمية البلعوم الأنفي المزمن- لمدة أكثر من عام- في طريقهم لاستئصال اللوزتين و اللحميه.

وتم اختيار المرضى من عيادة الأنف و الأذن و الحنجره بجامعة عين شمس ومستشفى مدينة نصر للتأمين الصحي،بالاضافه الى ٣٠طفلا عاديا كعينه ضابطه. تم اجراء فحص كامل،وفحص للأنف والأذن والحنجره لكل الأطفال في المجموعتين،بالاضافة الى أشعة جانبيه للبلعوم الأنفي . كذلك تم التشخيص النفسي لكل الأطفال تبعا لنظام دي اس ام-٤(١٩٩٤) وتم تطبيق الاختبارات النفسية الخاصة المستخدمة في مركزالطب النفسي بكلية الطب –جامعة عين شمس و هي:

- القائمة المعدلة للسلوك المشكل.

- اختبار الاكتئاب للأطفال.

- اختبار القلق للأطفال

قبل وبعد ٥-٦ شهور بعد عملية استئصال اللوزتين واللحميه ، و من خلال هذه الدراسه كان هناك فرق ذو دلاله احصائيه في حالات التبول اللارادي لصالح عينة الدراسة،تناسبت مع شدة و طول فترة الانسداد في مجرى التنفس العلوي.

كما أن حالات نقص الانتباه كانت أكثر في عينة البحث و اظهرت ارتباطا بشدة المرض و سجلت أعلى معدل في نقص الانتباه عند ٣-٤ سنوات من الاصابة بالتهاب اللوزتين و اللحمية.

و حقق القياس النفسي باستخدام القائمة المعدلة للسلوك المشكل درجات أعلى ذات دلالة احصائية في المرضى الذين يعانون من تضخم اللحمية في مقياس نقص الانتباه و فرط الحركة مؤكدا بذلك النتائج الاكلينيكية.

و كان هناك فرق ذو دلالة احصائية في حالات اضطراب التحدي المتعارض التي تم تشخيصها اكلينيكيا في عينة البحث ، و كانت الحالات أكثر شيوعا مع تضخم اللوزتين و اللحميه.

أما اضطراب السلوك فلم يظهر فرق ذو دلالة احصائية بين المجموعتين، و لم يظهر مقياس العدوان الاجتماعي فرقا بين مجموعة البحث و العينة الضابطة ، أما مقياس اضطراب السلوك فكان أعلى في عينة البحث ، و أظهر زيادة في الدرجات مع شدة

الانسداد في مجرى التنفس العلوي ، أوضحت النتائج أن كلا من اضطراب التحـــدي المتعارض و اضطراب السلوك قد زاد مع طول فترة التهاب اللوزتين واللحمية المزمن .

و لم يكن هناك زيادة ذات دلالة احصائية في حالات الاكتئاب في عينة البحث ، أمـــا القياس النفسي للاكتئاب فقد أظهر فروقا ذات دلالة احصائية . و كلما ازدادت فـترة الانسداد في مجرى زادت نسبة حدوث الاكتئاب . كذلك لم تكن هناك فروقـــا ذات دلالة احصائية في نسبة حدوث القلق بين مجموعة البحث و العينة الضابطة .

و حقق القياس النفسي للقلق درجات أعلى في عينة البحث ، و قد تناســـب القلـــق عكسيا مع مدة التهاب اللوزتين المزمن.

و بعد اجراء عملية استئصال اللوزتين و لحمية البلعوم الأنفي كان هنـــاك نقـــص في حالات التبول اللارادي خصوصا في الحالات التي تعاني من تضخم شديد بـاللوزتين و لحمية البلعوم الأنفي، و كذلك أصبح عدد حالات اضطراب نقص الانتباه أقل شيوعا.

- و لم يكن هناك تحسن ذو دلالة احصائية في اضطرابات السلوك التمزقي بعـــد اجـــراء الجراحة ، و لكن كان هناك تحسن ذو دلالة احصائية في مقياس اضطراب الســلوك في القائمة المعدلة للسلوك المشكل. و لم يظهر تغير في معدلات القلق و الاكتئاب بعـــد الجراحة، كذلك باستخدام القياس النفسي.

و من هذا العمل يمكن الاستنتاج بأن الانسداد في مجرى التنفس العلوي في الاطفـــال يتميز بوجه خاص بزيادة في اضطرابات السلوك الخارجية- كنقص الانتبـــاه و فـــرط

الحركة و المعارضة . على العكس مما سبق ذكره في الابحاث و الدراسات الاخرى من تأثير الانسداد في مجرى التنفس السفلي – في حالات الامراض الانسـدادية الرئويـة المزمنة و الربو – في الكبار و الاطفال حيث تكون المشكلات النفسية الذاتية كالقلق و الاكتئاب هي الاكثر شيوعا.

" جامعة عين شمس"
الكليـــة:

رسالـــة ماجســـتير / دكتـــوراه

أسم الطـــالب: .....
عنوان الرسالة: .....

أســم الدرجـــة : (ماجستيـــر / دكتـــوراه)

لجنـــة الإشـــراف

١- الاسم/ ..... ٢- الوظيفة/.....
١- الاسم/ ..... ٢ الوظيفة/.....
١- الاسم/ ..... ٢ الوظيفة/.....

تاريخ البحـــث : ١٨ / ٤ / ١٩٩٨

الدراســـات العليـــا

ختـــم الإجـــازة :
/ ١٩٩

أجيزت الرسالـــة بتاريخ /
٢١ / ١ / ٢٠٠١

موافقة مجلس الكلية
١٧ / ٤ / ٢٠٠١

موافقة مجلس الجامعة
/ / ١٩٩

"جامعة عين شمس"
الكلية:

صفحة العنوان

أسم الطالب : .....
الدرجة العلمية : .....
القسم التابع له : .....
أسم الكلية : .....
الجامعة : .....
سنة التخرج : .....
سنة المنح : .....

شروط عامة

يوضع شعار الجامعة على الغلاف الخارجي.

الجوانب النفسية لإلتهاب اللوزتين و لحمية البلعوم الأنفي المزمن في الطفولة المتأخرة

رسالة مقدمة توطئة للحصول على
درجة الدكتوراه في دراسات الطفولة
من الطبيبة

نشوة نصر السيد سليمان

تحت إشراف

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د. حنان الجمل
مدرس بمعهد دراسات الطفولة
جامعة عين شمس

٢٠٠١

الجوانب النفسية لإلتهاب اللوزتين و لحمية البلعوم الأنفي المزمن في الطفولة المتأخرة

رسالة مقدمة توطئة للحصول على
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