DISSERTATION ON

COMPARATIVE STUDY BETWEEN CONVENTIONAL ADENOID CURETTAGE AND ENDOSCOPIC ASSISTED ADENOID CURETTAGE

Dissertation submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the

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CERTIFICATE

I certify that the Dissertation titled **COMPARATIVE STUDY BETWEEN CONVENTIONAL ADENOID CURETTAGE AND ENDOSCOPIC ASSISTED ADENOID CURETTAGE** submitted by Dr.JAGAN A S, for Degree of Master of Surgery (Otorhinolaryngology) to The Tamilnadu Dr.M.G.R. Medical University, Chennai is the result of original research work undertaken by him in the department of ENT AND HEAD & NECK SURGERY, Thanjavur Medical College, Thanjavur during his Post Graduate Course from 2014 – 2017. This is submitted as partial fulfilment for the requirement of M.S. Degree Examination – Branch IV(Otorhinolaryngology) to be held in April 2017.

Prof. Dr. Gandhi M.S.,D.L.O
Head of the Department,
Department of Otorhinolaryngology,
Thanjavur Medical College,
Thanjavur.

Prof. Dr. Vanithamani M.S.,Mch
Dean,
Thanjavur Medical College,
Thanjavur - 613004

DECLARATION

I hereby declare that the dissertation titled **COMPARATIVE STUDY BETWEEN CONVENTIONAL ADENOID CURETTAGE AND ENDOSCOPIC ASSISTED ADENOID CURETTAGE ,** a clinical study submitted by me is a result of original work carried out by myself under the guidance of **Prof.Dr.G.GANDHI, M.S.,D.L.O., Head of the Department Otorhinolaryngology and Head and Neck, Thanjavur** Medical College, Thanjavur. I further declare that the result of research has not been submitted previously by myself or other persons in any conferences or journals.

Place : Thanjavur. **Dr. A.S. JAGAN**

Date :

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Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

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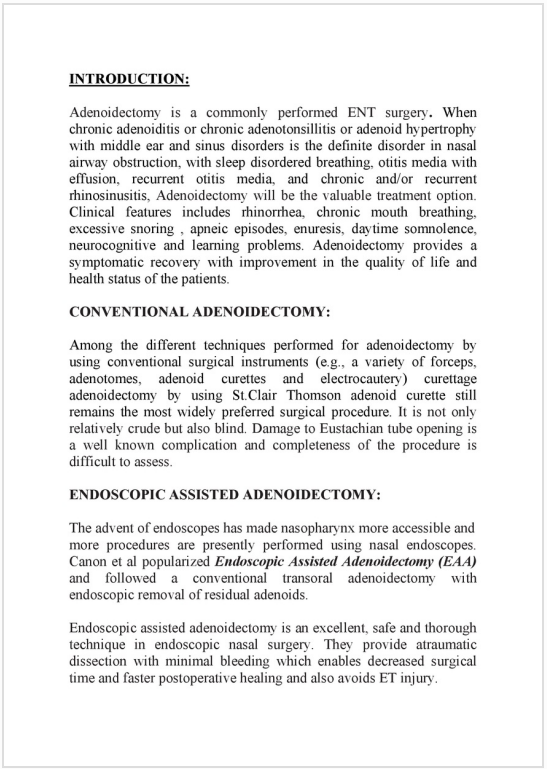


INTRODUCTION:

Adenoidectomy is a commonly performed ENT surgery. When chronic adenoiditis or chronic adenotonsillitis or adenoid hypertrophy with middle ear and sinus disorders is the definite disorder in nasal airway obstruction, with sleep disordered breathing, otitis media with effusion, recurrent otitis media, and chronic and/or recurrent rhinosinusitis, Adenoidectomy will be the valuable treatment option. Clinical features includes rhinorrhea, chronic mouth breathing, excessive snoring , apneic episodes, enuresis, daytime somnolence, neurocognitive and learning problems. Adenoidectomy provides a symptomatic recovery with improvement in the quality of life and health status of the patients.

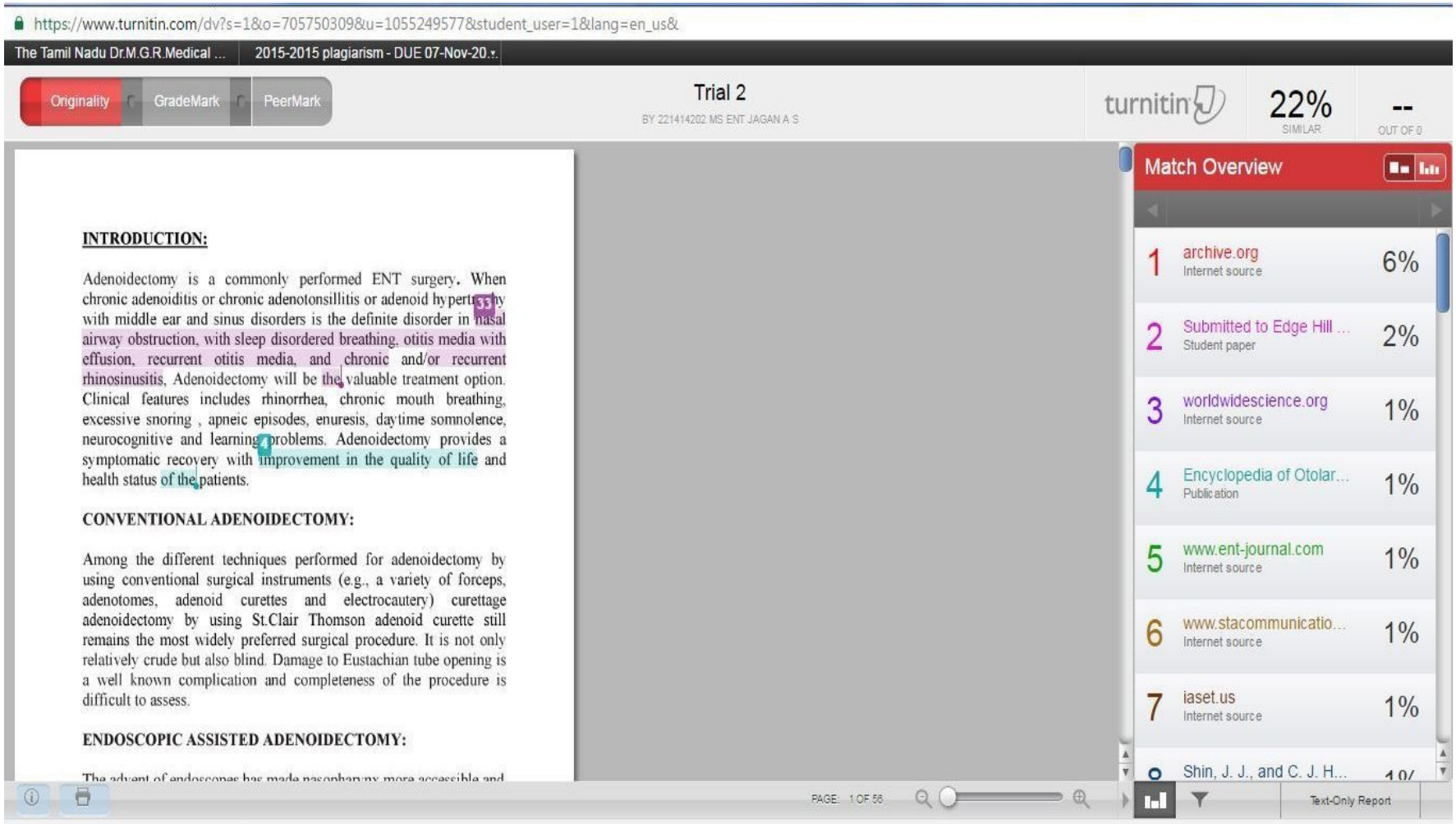
CONVENTIONAL ADENOIDECTOMY:

Among the different techniques performed for adenoidectomy by using conventional surgical instruments (e.g., a variety of forceps, adenotomes, adenoid curettes and electrocautery) curettage adenoidectomy by using St.Clair Thomson adenoid curette still remains the most widely preferred surgical procedure. It is not only relatively crude but also blind. Damage to Eustachian tube opening is a well known complication and completeness of the procedure is difficult to assess.

ENDOSCOPIC ASSISTED ADENOIDECTOMY:

The advent of endoscopes has made nasopharynx more accessible and more procedures are presently performed using nasal endoscopes. Canon et al popularized ***Endoscopic Assisted Adenoidectomy (EAA)*** and followed a conventional transoral adenoidectomy with endoscopic removal of residual adenoids.

Endoscopic assisted adenoidectomy is an excellent, safe and thorough technique in endoscopic nasal surgery. They provide atraumatic dissection with minimal bleeding which enables decreased surgical time and faster postoperative healing and also avoids ET injury.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CONTENTS

S.NO	TITLE	PAGE.NO
1	INTRODUCTION	1
2	AIM OF THE STUDY	3
3	REVIEW OF LITERATURE	4
4	SUMMARY OF PUBLICATIONS	54
5	MATERIALS AND METHODS	58
6	OBSERVATIONS AND RESULTS	65
7	DISCUSSION	77
8	CONCLUSION	79
9	ANNEXURES	
	BIBILIOGRAPHY PROFOMA ABBREVIATIONS MASTER CHART	

INTRODUCTION

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When both these methods are combined and ***endoscopic assisted adenoidectomy*** performed, advantages of both techniques should get pooled, thereby increasing the precision in removing intranasal, choanal and laterally based adenoidal tissue without leaving residue and the ensuing complete adenoid removal is expected to provide an improvement in the elimination of symptoms and an advantage in decreasing the risk of a recurrence. Furthermore, a finer control of adenoid resection under direct vision decreases the risk of damaging neighbouring structures and reduces intraoperative bleeding and causes early post operative recovery.

AIMS AND OBJECTIVE

1. To compare the post operative tissue remnant.
2. To compare the post operative Eustachian tube scarring.
3. To compare post operative snoring assessment.
4. To compare the operating time between the two techniques.

REVIEW OF LITERATURE

HISTORY:

In 1724, **SANTORINI** described about nasopharyngeal lymphoid aggregates and named it as ***LUSHKA TONSIL***.

In 1868 **WILHELM MEYER** described it as *NASOPHARYNGEAL VEGETATION*, which he then coined the term ***ADENOID*** for it.[1]

In 1885, **GOTTSTEIN** described the first adenoid curette.[1]

In 1806, the first endoscope was developed by **PHILIPP BOZZINI,** for the examination of the canals and cavities of the human body.

In 1945, **KARL STORZ** started producing instruments for ENT specialists.

DEVELOPMENTAL ANATOMY OF NASOPHARYNX AND ADENOID :

The development of buccal cavity is mainly from ventral growth of the upper pharyngeal arches. The pericardial area and buccophayrngeal membrane come to lie on ventral surface of the embryo due to the rostral growth of the embryo and the formation of the head fold. Further expansion of the forebrain dorsally, and the bulging of the pericardium ventrally, together with enlargement of the facial processes laterally, means that the buccopharyngeal membrane becomes depressed at the base of a hollow forming the stomodeum or primitive buccal cavity. By the end of 4^{th} week the buccopharyngeal membrane breaks down thus communication is created between the stomodeum and the cranial end of the foregut (future nasopharynx and oropharynx respectively).

ADENOID:

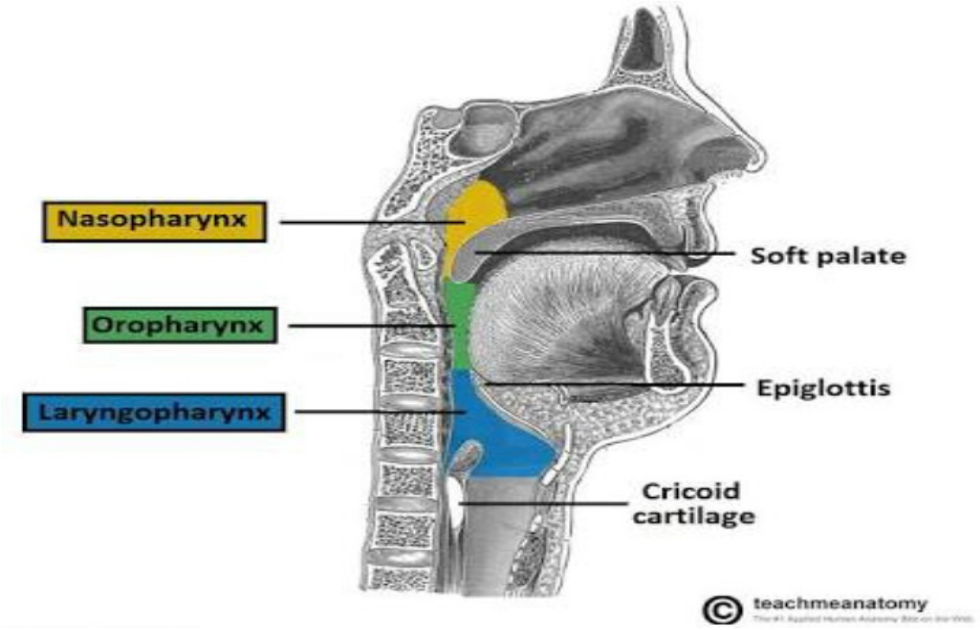
The adenoid is a lymphoid organ that essentially develops from three sources: first, an epithelial component arising from the lining of the primitive oronasal cavity; second, this epithelium grows into, and is enveloped by a connective tissue or mesenchymal stroma; third, the region is infiltrated by lymphoidal cells. The resultant organ is composed of resident population of lymphoid cells in association with a more or less elaborated epithelial framework (the crypts)

that has grown into, and been enveloped by, mesenchymal tissue. The adenoid develops in close association with mucous glands. As early as the third month of development, glandular primordia are visible as solid buds or cords of cells surrounded by blood vessels and an increasing number of discrete lymphoid cells in a loose mesenchyme. During the fourth month, the lymphatic vessels appear and glandular primordia increase in number and complexity as they branch and acquire lumen and show evidence of secretary activity. Infiltration by lymphoid cells is intense. In the fifth month, pharyngeal crypts appear as 12 shallow sagittal folds or plicae . These folds are covered with pseudostratified ciliated epithelium with goblet cells. Discrete lymph follicles organize around the glandular ducts, and lymphocytes penetrate the epithelium and into the adenoid crypts and lumen of the nasopharynx. The adenoid folds deepen during the sixth month and form fully developed tonsil during the seventh month. The alveoli of the glands come to lie deep to the lymph follicles. The peculiarly dilated glandular ducts pass through, or next to, the lymphoid follicles. Further evidence of function of these tissues was reported in a study which, demonstrated that IgA, IgG and IgM were all present in epiphyaryngeal tissues taken from 5 to 16 week old human embryos.[2,3]

ANATOMY OF NASOPHARYNX

BOUNDARIES :

The nasopharynx lies superior to the soft palate and posterior to the posterior nares. The posterior nasal apertures (Choanae) are thus divided medially by the free posterior edge of the septum. Their roof is formed by the body of the sphenoid bone and vaginal process of the medial pterygoid plate. Their lateral walls are formed by the medial pterygoid plates and the floor by horizontal plate of the palatine bone. Each of posterior nares measure 25 mm vertically and 12mm transversely.Middle and inferior nasal conchae posterior end extend into choana. The cavity of nasopharynx cannot collapse unlike cavity of oropharynx and laryngopharynx. This is due to rigidness of nasopharyngeal wall. The nasopharynx and the oropharynx communicates through the isthumus, lying behind soft palate. During swallowing,the soft palate constricts with contraction of palatopharyngeus muscle leading to prevention of regurgitation of fluids ino nasal cavity .[4]

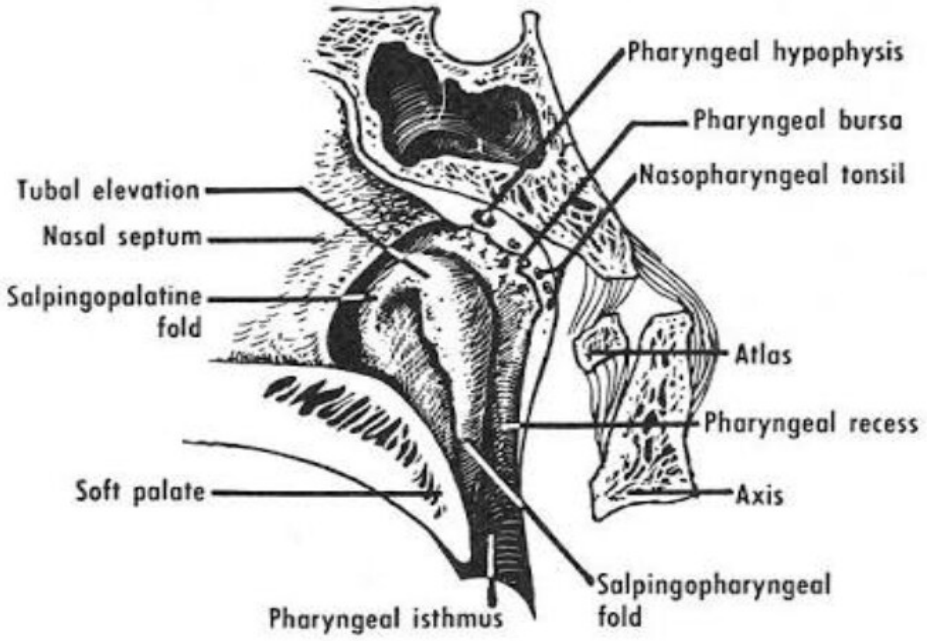


PIC.1.picture showing division of pharynx

The nasopharynx is bounded by lateral walls, open anteriorly and inferiorly and covered posteriorly and superiorly. Body of sphenoid and basilar part of occipital bone form the superior and posterior walls of nasopharynx. The superior and posterior wall is continuous. These two, together are termed as basisphenoid. Posterior wall on passing further down is formed by mucosa covering superior constrictor muscles. The lymphoid mass, the adenoid, lies in the mucosa of the upper part of the roof and posterior wall in the midline. Adenoid also called as LUSCHKA tonsil. The lateral walls of the nasopharynx projects a number of important structures. On either side there is opening of the Eustachian tube, situated 10-12 mm behind and a little inferior to the level

of the posterior end of the inferior turbinate. The shape of the tubal aperture is approximately triangular, and is bounded superior and posteriorly by the tubal elevation which consists of mucosa overlying the protruding pharyngeal end of the cartilage of the Eustachian tube. The salpingopharyngeal fold is a vertical mucosal fold that descends from the tubal elevation behind the aperture and covers salpingopharyngeus and in front of the aperture it is covered by a a smaller salpingopalatine fold extending from the anterosuperior angle of the tubal elevation to the soft palate. On entering the soft palate the levator veli palatini produces an elevation of the mucosa immediately below the tubal opening. A small lymphoid tissue, lies immediately back to the opening of nasopharyngeal end of Eustachian tube, called tubal tonsil, also called as GERLACH'S TONSIL.[5]

A variable depression in the lateral wall behind the tubal elevation is the pharyngeal recess (Fossa of Rosenmuller). The pharyngeal recess corresponds to sinus of Morgagni and continues laterally above superior constrictor muscle. The inferior wall of the nasopharynx is open and is covered partly by soft palate muscles.



PIC.2. Picture showing structures in lateral wall of nasopharynx.

NONTONSILLAR MICROSTRUCTURE OF NASOPHARYNX

The nasopharyngeal epithelium anteriorly is ciliated pseudostratified respiratory type of epithelium containing goblet cells. Its surface has openings for the ducts of mucosal and submucosal seromucous glands. The respiratory epithelium changes to non-keratinized stratified squamous epithelium posteriorly, which continues into the oropharynx and laryngopharynx. There is short microvilli instead of cilia in the transitional zone between the two types of epithelium consists of columnar epithelium. This zone meets the nasal septum superiorly and crosses the orifice of the Eustachian tube laterally and

passes posteriorly to the meeting point of the soft palate and the lateral wall. Tubal orifice has numerous mucous glands around it. In infancy the nasopharyngeal epithelium is ciliated columnar but in adults most of the epithelium has undergone squamous metaplasia, leaving areas of columnar epithelium only in relation to the fossa of Rosenmuller.

INNERVATION AND VASCULAR SUPPLY OF NASOPHARYNX:

The pharyngeal branch from pterygopalatine ganglion supplies much of the mucosa of the nasopharynx behind the pharyngotympanic tube. The maxillary nerve is thought to transmit the principal sensory supply from the pharyngotympanic tube and middle ear cavity, presumably through its pharyngeal branch. Sphenopalatine nerve, a branch from otic ganglion innervates the pharyngeal opening of pharyngotympanic tube, and also by the pharyngeal plexus. The levator veli palatini muscle receives its innervations from the nucleus ambigus through the vagus nerve. Vagus nerve through its pharyngeal branch form pharyngeal plexus which supplies salphingopharyngeus muscle and its contraction assists in opening the Eustachian tube. The pharyngeal artery usually arises from the inferior aspect of the internal maxillary artery, it courses posteriorly where it ascends on the bony wall, passes beneath the sphenopalatine ganglion and maxillary nerve

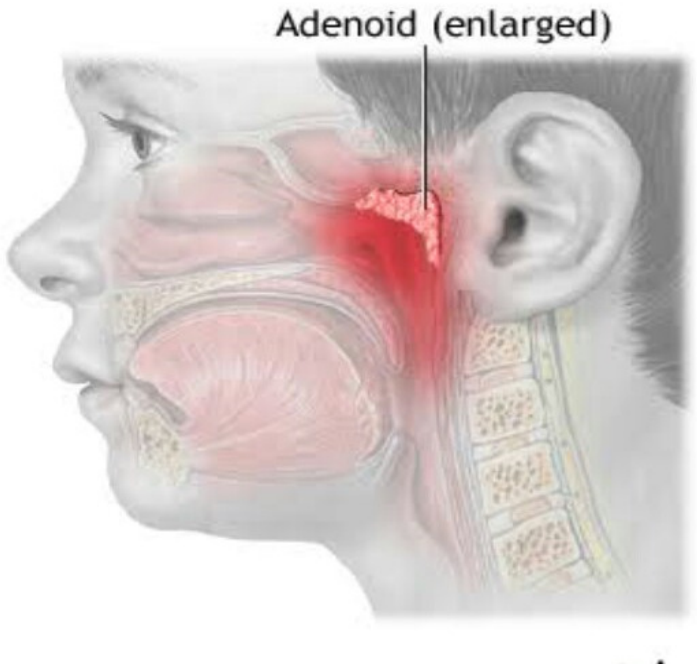
branches, and then turns medially, running close to the vidian nerve and giving a branch to the pterygoid canal before it enters the pharyngeal canal 4mm medial to the pterygoid canal. Distally it passes into the soft tissues of the nasopharnx.[6]

LYMPHATIC DRAINAGE OF NASOPHARYNX :

The nasopharynx has an extensive sub mucosal lymphatic plexus. The first order drainage sites are the retropharyngeal nodes situated in the retropharyngeal space between the posterior nasopharyngeal wall, pharyngobasillar fascia and the prevertebral fascia. The node of Rouviere forms the main and constant lateral group. It lies anterior to the atlas bone at the lateral border of the longus muscle, anteromedial to the internal carotid artery. Efferent vessels then drain to the upper most deep internal jugular chain at the skull base in the retrostyloid parapharyngeal space compartment deep to the upper end of the sternomastoid muscle. The nodes then drain downwards posteriorly to the accessory nerve group and anteriorly to the jugulodigastric group.[7]

ANATOMY OF ADENOID (PHARYNGEAL TONSIL) :

The pharyngeal, palatine and lingual tonsils together form the so called Waldeyer's ring .The ring is a group of subepithelial lymphoid aggregates at opening of oropharynx and nasopharynx to external environment. The adenoid is composed of mucosa associated lymphoid tissue(MALT) located at the junction of the roof and posterior wall of the nasopharynx. During the early years of life, it is truncated pyramid in shape, often with a vertically oriented median cleft, thus its apex points towards the nasal septum and its base at the junction of the roof and posterior wall of the nasopharynx.



PIC.3. Picture showing location of adenoid in nasopharynx

The free surface of the nasopharyngeal tonsil is marked by folds that radiate forwards and laterally from a median blind recess, the pharyngeal bursa (bursa of Luschka), which extends backwards and up.

After birth the adenoid initially grows rapidly, but by 8-10 years of age it usually undergoes a degree of involution. The size of the adenoid is largest at 5 years, which may account for the frequency of the nasal breathing problems in preschool children, and the incidence of adenoidectomy in this age group.

VASCULAR SUPPLY AND LYMPHATIC DRAINGE OF ADENOID:

The arterial supply of the nasopharyngeal tonsil is derived from,

Ascending pharyngeal artery : A branch of external carotid artery which is a slender vessel arising from the medial surface of the external carotid artery near the origin of that artery. It anastomoses with the ascending palatine branch of the facial artery and the ascending cervical branch of the vertebral artery.

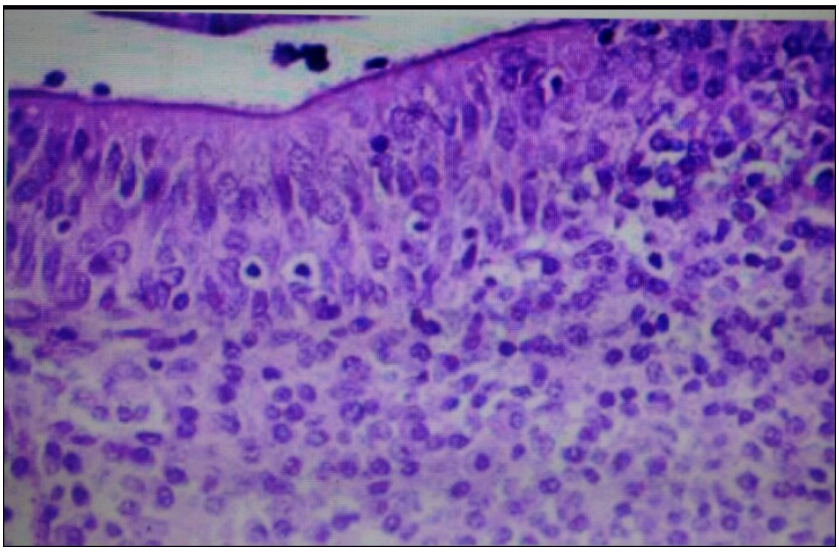
Ascending palatine artery : It's a branch of facial artery, arising close to the origin of facial artery. It anastomoses with the fellow opposite branch and greater palatine branch of maxillary artery. Tonsillar branch of facial artery, Pharyngeal branch of maxillary artery and the artery of the pterygoid canal. In addition, the basisphenoid artery, which is a branch of hypophyseal arteries which supplies the bed of the nasopharyngeal tonsil is considered as a possible cause of persistent post adenoidectomy haemorrhage in some patients.[8]

Numerous communicating veins drain the nasopharyngeal tonsil into the internal submucous and external pharyngeal venous plexuses. They emerge from the deep lateral surface of the tonsil and join the external palatine (paratonsillar) veins, and pierce the superior constrictor either to join the pharyngeal venous plexus, or to unite to form a single vessel that enters the facial or internal jugular vein. They may also connect with the pterygoid venous plexus. Lymphatic drainage is either directly or indirectly by means of the retropharyngeal nodes to the upper deep cervical lymphnodes.

MICROSTRUCTURE OF THE ADENOIDS :

The adenoid is covered laterally and inferiorly mainly by ciliated respiratory epithelium which contains scattered small patches of non keratinized stratified squamous epithelium. Its superior surface is separated from the periosteum of

the sphenoid and occipital bones by a connective tissue hemicapsule to which the fibrous frame work of the tonsil is anchored. The latter consists of a mesh of collagen type III (reticular) fibres which supports a lymphoid parenchyma similar to that in the palatine tonsil.



PIC.4.Histological picture of adenoid tissue

The nasopharyngeal epithelium lines a series of mucosal folds around which the lymphoid parenchyma is organized into follicles and extra follicular areas. Internally, the adenoid is subdivided into four to six lobes by connective tissue septa, which arise from the hemicapsule and penetrate the lymphoid parenchyma. Seromucous glands lie within the connective tissue, and their ducts extend through the parenchyma to reach the nasopharyngeal surface.

PHYSIOLOGY OF NASOPHARYNX

Nasopharynx serves as a pathway for air passage. It is separated completely from oropharynx by Passavant's ridge,which is a prominence by contraction of superior constrictor's upper fibres, against soft palate.

The mucous secretion comes from the nose through the nasopharynx into oropharynx. Normally,the mucous secretion is thin.When the mucous amount is reduced it is thickened. Normal mucous secretion is insensitive. When it is thickened it becomes sensitive and is called as post nasal drip. Enlargement of adenoid tissue further limits the nasopharyngeal airway and may result in obstruction.

ROLE OF NASOPHARYNX IN SPEECH :

Speech is one of the most complex of voluntary activities and makes use of the mechanisms of breathing, swallowing and hearing. Respiration provides the power supply. Phonation is the process by which that power is used to produce articular speech, and the whole of the vocal tract above the larynx plays a part in its production. The nasopharynx serves in resonation while using some vowels. Velopharyngeal closure depends on adequate functioning of soft palate.

ROLE OF NASOPHARYNX IN MIDDLE EAR FUNCTION

The functioning of Eustachian tube depends on the proper functioning of medial end of Eustachian tube which is present in lateral wall of nasopharynx.. The eustachain tube has at least three physiologic functions –

1) Protecting the middle ear from nasopharyngeal pressure changes and secretions.

2) Draining middle ear secretions into the nasopharynx, and

3) Ventilating the middle ear to equilibrate pressure with ambient atmospheric pressure and to replenish absorbed oxygen.

Normally the walls of Eustachian tube remains collapsed. Active contraction of dilator muscles leads to opening of Eustachian tube leading to equalisation of air pressure between middle ear and external environment. A gradient - driven, transmucosal exchange of gases between the middle ear and blood also participates in the regulation of middle ear pressure.

Those with Eustachian tube dysfunction are highly susceptible for middle ear infections. In children Eustachian tube is smaller and horizontally aligned. As flow through a tube is inversely proportional to the fourth power of the radius, ventilatory function is considerably reduced in child, causing greater incidence of middle ear disease.

Understandably ventilation of the middle ear of the child is requisitely sensitive to inflammatory oedema of the lining epithelium of the Eustachian tube. The tendency toward negative middle ear pressure in children improves with maturation, paralleling the decreased incidence of otitis media in adolescence as compared with infancy.

PHYSIOLOGY AND IMMUNOLOGY OF THE ADENOIDS

Immunology of adenoids and tonsils are discussed together, because of the similar functions. They are predominately B-cell organs. B lymphocytes comprise 50% to 65% of all adenoid and tonsillar lymphocytes. T-cell lymphocytes comprise approximately 40% of adenoid and tonsillar lymphocytes and 3% are mature plasma cells. Conversely, 70% of the lymphocytes in peripheral blood are T-cells. The immuno reactive lymphoid cells of the adenoids and tonsils are found in four distinct areas; the reticular cell epithelium, extra follicular area, the mantle zone of the lymphoid follicle and the germinal center of the lymphoid follicle. There is an ample evidence that the adenoids and tonsils are involved in inducing secretory immunity and regulating secretory immunoglobulin production. They contain a system of channels covered by specialized endothelium that can mediate antigen uptake in a fashion similar to Payer's patches of epithelium in the bowel. Both the

adenoids and tonsils are favourably located to mediate immunologic protection of the upper aero digestive tract as they are exposed to airborne antigens. Both organs, specifically the tonsils, are particularly designed for direct transport of foreign material from the exterior to the lymphoid cells. This is in contrast to lymph nodes, which depend on antigenic delivery through afferent lymphatics. The crypts are covered by stratified squamous epithelium, which are ideally suited for trapping foreign material and transporting it to the lymphoid follicles.

The tonsils and adenoids rank among the secondary lymphatic organs. Intra tonsillar defence mechanisms eliminate weak antigenic signals. Only when additional higher antigenic concentrations are presented does proliferation of antigen - sensitive B cells occur in the germinal centers. Low antigen doses affect the differentiation of lymphocytes to plasma cells, where as high doses produce B-cell proliferation. The generation of B-cells in the germinal centers of the tonsils and adenoids is considered by Seigel to be one of the most essential functions.

Immunoglobulins (Igs) produced by the adenoid include IgG, IgA, IgM and IgD.[9] IgG appears to pass into the nasopharyngeal lumen by passive diffusion. The tonsil produces antibodies locally as well as Bcells that migrate to other sites around the pharynx and periglandular lymphoid tissues to produce antibodies.

T-cell functions such as interferon production and presumably production of other important lymphokines have been shown to be present in tonsils and adenoids. The role played by tonsillar and adenoid T cells in tumour response is still unknown.

The human tonsils and adenoid are immunologically active between the ages of 4 and 10 years. Involution of tonsils begins after puberty, but involution of adenoids starts by 8 years of age. This involution results in a decrease of B-cell population and a relative increase in the ratio of T to B cells. Although the over all Immunoglobulin producing function is affected, considerable B-cell activity is still seen in clinically healthy tonsils even at 80 years of age. The situation is different in disease associated changes, such as when recurrent tonsillitis and adenoid hyperplasia are observed. Inflammation of the reticular crypt epithelium results in shedding of immunologically active cells and decreasing antigen transport function with subsequent replacement by stratified squamous epithelium. These changes lead to reduced activation of the local B-cell system, decreased antibody production, and an overall reduction in density of the B-cell and germinal centers in extrafolliuclar areas.

In contrast to recurrent tonsillitis, the changes are less pronounced in adenoid hyperplasia where the immunoregulatory conditions necessary for maintenance of the B cell population are well preserved. The reason for this is most likely that the reticular epithelium is less affected in inflammation of adenoids than in tonsils. There are conflicting reports regarding the immunologic consequences of tonsillectomy and adenoidectomy, yet it is clear that no major immunologic deficiencies result from these procedures. Orga showed a three to four fold drop in titers in children previously immunized with live poliovirus vaccine. Children who were previously immunized orally with live poliovirus vaccine dropped their titers three to four fold after tonsillectomy and adenoidectomy. Attempts to vaccinate seronegative children subjected to tonsillectomy and adenoidectomy have resulted in delayed and lowered nasopharyngeal secretory immune responses as measured by IgA antibodies to the poliovirus. Fortunately, poliovirus epidemics are no longer an annual threat. It is clear that the adenoids and tonsils are active immunologic organs that generally reinforce the mucosal immunity of the entire upper aerodigestive tract. The immunologic role of these organs should be considered before a patient undergoes adenoidectomy or tonsillectomy. Nonetheless, clinical consideration still forms the actual basis of surgery.

CLINICAL MICROBIOLOGY OF NASOPHARYNX AND ADENOIDS

The nasopharynx is accessible to the introduction of many types of organisms. The establishment of the normal flora in the upper respiratory tract is initiated at birth. Certain organisms such as Lactobacilli and anaerobic Streptococci establish themselves at an early date and reach high numbers within a few days. Actinomyces, Fusobacterium, and Nocardia are acquired by age of 6 months. After that time, Bacteroides, Leptotrichia, Propionobacterium, and Candida are also established as part of the oral flora. Fusobacterium populations reach high numbers after dentition and reach maximal numbers at age of one year. Healthy children upto 5 years of age can harbour known aerobic pathogens. The frequency of pathogens decreases with age, which may be due to an increased immunity.

Bacterial interference between various organisms may be responsible for maintenance of the normal equilibrium in the nasophrynx.[10] The normal flora seems to convey to the individual an efficient resistance barrier against many pathogens. The balance between these organisms can be disrupted by antimicrobial therapy. Certain antibiotics have been shown to suppress Beta-hemolytic Streptococci and promote the emergence of gram negative enteric organisms. Bacterial interference has been shown to exist between alpha and

beta hemolytic Streptococci and between beta-hemolytic Streptococci and gram negative bacteria recovered from tonsillar regions. The lack of interfering strains may explain the increased susceptibility of certain individuals to beta-hemolytic Streptoccci. The nasopharyngeal culture is of little value in determining the cause of otitis media, largely because of the multiplicity of pathogens present. The nasopharynx harbours the same potential pathogens in healthy as well as in sick children. Thus, potential pathogen recovered from a nasopharyngeal culture have generally been regarded as part of normal flora.

In one study it was found that there were substantial differences in the type and number of aerobic bacteria, found in non diseased and diseased adenoids. The core samples of normal adenoids revealed that 75% of children who were relatively free of upper respiratory disease, otitis media, and symptoms of adenoid obstruction had either no bacterial growth on culture or bacteria that are considered to be part of the normal flora and not pathogenic. This compares with only 45% of children who had chronic adenoid infection and 39% who had obstructive adenoid hypertrophy; the bacteria found in these children were more likely to be beta-lactamase producers.

In one of the studies it was reported that in children having adenoid hypertrophy and chronic adenotonsillitis, both harbour aerobes and anaerobes. The aerobic organisms most frequently isolated in both groups of children were

alpha- and beta-hemolytic Streptococci, Staphylococcus aureus, beta hemolytic Streptococci, Hemophillus species. The predominant anaerobic organisms in both groups were Bacteroides species, Fusobacterium species, anaerobic gram positive cocci.

UPPER AIRWAY OBSTRUCTION AND THE PHARYNGEAL LYMPHOID TISSUE :

Chronic adenotonsillar hypertrophy is a common cause of upper airway obstruction in children and in severe cases may result in cor pulmonale, pulmonary vascular hypertension, and alveolar hypoventilation, all of which may be reversed by adenotonsillectomy. At present, upper airway obstruction due to hypertrophy of tonsils and adenoids with consequent hypoventilation and cor pulmonale has come to be called the "cardiopulmonary syndrome". This syndrome is characterized by stridor and snoring that worsen when the patient is supine or sleeping, as well as lethargy and somnolence. In addition to markedly enlarged tonsils and adenoids on physical examination, auscultation of the chest will be abnormal.

Advanced cases may also show signs of right heart failure with hepatomegaly and peripheral venous distensions. Most important in the syndrome, abnormal

findings will revert to normal or near normal following removal of the obstructing tonsils and adenoids.

Obstructive sleep apnea is a common finding in children with a history of adenotonsillar hypertrophy and adenotonsillar hypertrophy is the most common cause of sleep apnea in children.[11] Nocturnal enuresis is another indicator of severe underlying airway obstruction in children. In a study it was described enuresis as related to chronic adenotonsillar hypertrophy. A proposed cause of enuresis is poor nocturnal regulation of antidiuretic hormone release that is related to disorders of rapid eye movement (REM) sleep. Due to abnormality in regulating the release of growth hormone, some child may possibly undergo failure to thrive. OSAS seems to be increasing due to adenoid hypertrophy. In infants those having nasopharyngeal narrowing, adenoidectomy alone can be curative. Polysomnography is useful to define the severity of obstructive sleep apnea.

Failure to thrive is a common complication of childhood obstructive sleep apnea. According to Marcus et al, failure to thrive could be a byproduct of the higher caloric expenditure caused by the increased effort required to breathe during sleep. This effect may explain the rapid catch-up in growth, and subsequent normal growth curves, following adenoidectomy in infants with adenoid hypertrophy and failure to thrive.

ADENOID HYPERTROPHY AND SINUS DISEASE:

Adenoid hypertrophy as a cause of sinus disease has been debated for years. It is difficult to explain how a posterior nasopharyngeal obstruction can produce obstruction of the osteomeatal complex. However, adenoid enlargement may mimick sinusitis by causing stagnation of secretions from nose thereby causing nasal symptoms. In such cases adenoidectomy plays a major role. However in some patients adenoid enlargement may be due to chronic adenoiditis caused by chronic sinusitis.[12]

CRANIOFACIAL GROWTH AND ADENOID HYPERTROPHY :

In 1872, Tomes proposed that maxillofacial abnormalities may develop in chronic cases of mouth breathing. Mouth breathing leads to displacement of the mandible and tongue downward and backward and potential postural changes of the head and neck that may secondarily affect dental occlusion and jaw growth. In a study it was demonstrated that the classic stigmata of adenoid facies in children with chronic nasopharyngeal obstruction from adenoid hypertrophy. These consisted of longer anterior face height with a tendency toward a retrognathic mandible compared with controls. The postural changes of chronic mouth breathing disturb the normal equilibrium of forces excreted

on the teeth and orofacial soft tissues by the tongue and lip, resulting in an open bite, a protrusive maxilla, and a buccal posterior crossbite. The severity of adenotonsillar hypertrophy and resulting airway obstruction as measured by surgical findings and lateral cephalometric radiographs is directly related to the degree of maxillofacial growth disturbance and malocclusion. Adenotonsillectomy has been shown to reverse some of these findings. Maxillofacial growth disturbances in children who are mouth breathers also may be multifactorial in etiology.

DISEASES OF MIDDLE EAR AND ADENOID :

The age at which incidence peaks for recurring middle ear effusions correspond to the period of maximum lymphoid hyperplasia in the nasopharynx. There are at least two possible mechanical factors responsible for this. The first factor is the effect of direct closure of the Eustachian tubal orifice by excessively enlarged adenoids. The second factor, obstruction of lymphatics draining the middle ear and Eustachian tube may be of greater importance. As adenoid is adjacent to Eustachian tube, its infection may cause dysfunction of Eustachian tube. Thus adenoidectomy plays a vital part in normal functioning of Eustachian tube

In a study it was showed that growth of the adenoid outstrips that of the nasopharynx in children aged between three and five years of age causing maximum airway compromise. The effect of enlarged adenoids in causing acute otitis media in young children has been demonstrated, and between abnormality of the vomero- ethmoid suture in children with otitis media with effusion. Enlarged adenoid causes impedence in mucocilliary flow thus due to mucus stagnation pathogens may grow[13] causing retrograde infection of middle ear via Eustachian tube. This is further confirmed by checking the organism from the pus taken from middle ear and nasopharynx,which will be same. The lateral position of the hypertrophied adenoids, with abundant on the Eustachian tube orifice, is a contributing factor in the final otologic outcome of patients requiring pressure equalization tubes insertion for otitis media. In addition to being bacteriological reservoir, hypertrophic adenoid serves as a mechanical barrier to the Eustachian tube lumen, causing middle ear under pressures and subsequent effusion formation. Of the many positive mechanisms explaining adenoidal involvement in middle ear infection, three are compelling :

(1) obstruction of the Eustachian tube, both mechanical and functional,

(2) obstruction of the nasal airway leading to Eustachian tube reflux and

(3) a reservoir for pathogenic bacteria

DIAGNOSIS AND INVESTIGATION OF PATIENTS WITH DISEASES OF THE ADENOIDS :

HISTORY AND PHYSICAL EXAMINATION :

1) Nasal obstruction :

This is the most frequently reported symptom. Nasal obstruction may be associated with other symptoms like mouth breathing, snoring, rhinorrhea, apneic episodes while sleeping, hyponasal voice, day time somnolence and enuresis.

2) Throat pain / dysphagia :

In most of the cases adenoiditis is usually associated with tonsillitis. The patient may complain of throat pain and dysphagia. Pain may radiate to the ears or may occur in the neck due to enlargement of the jugulo digastrics lymphnodes. Swallowing is acutely sore and solid food is refused at the height of the inflammation although fluids are usually accepted.

3) **Otalgia/Otorrhoea :**

Very young children will not complain of pain but will be irritable and may bang the head on the cot sides. Conversely some children may become very quiet and refuse food, not sleep well or be inconsolable. If the ears discharge it is usually muco purulent.

SIGNS :

There is a characteristic facial appearance in children with adenoid hypertrophy.

The so-called "adenoid facies" has classical appearance that include :

a) open mouth

b) crowding of upper teeth

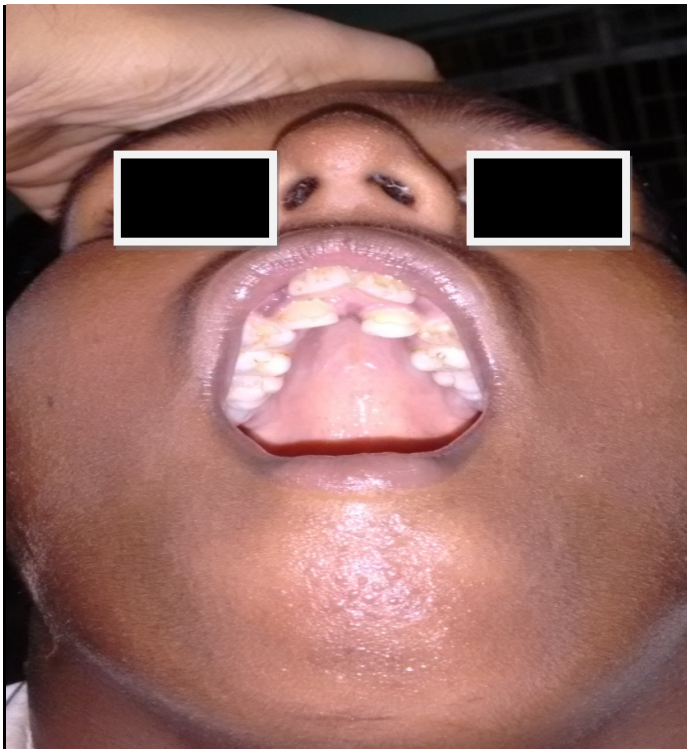
c) Short upper lip

d) High arched palate

e) Pinched nose

f) hypoplastic maxilla

g) narrow alveolus



PIC.5.Picture showing adenoid facies

Anterior rhinoscopy :

Using thudicum speculum, nasal cavity examination may be normal or congested mucosa or secretion may be present or inferior turbinate hypertrophy may be present.

Posterior rhinoscopy :

Posterior rhinoscopic examination is difficult in children.But if possible can show enlarged adenoids

Examiantion of the throat :

Tonsils are hyperaemic and enlarged,if adenoiditis is associated with tonsillitis.

Examination of neck :

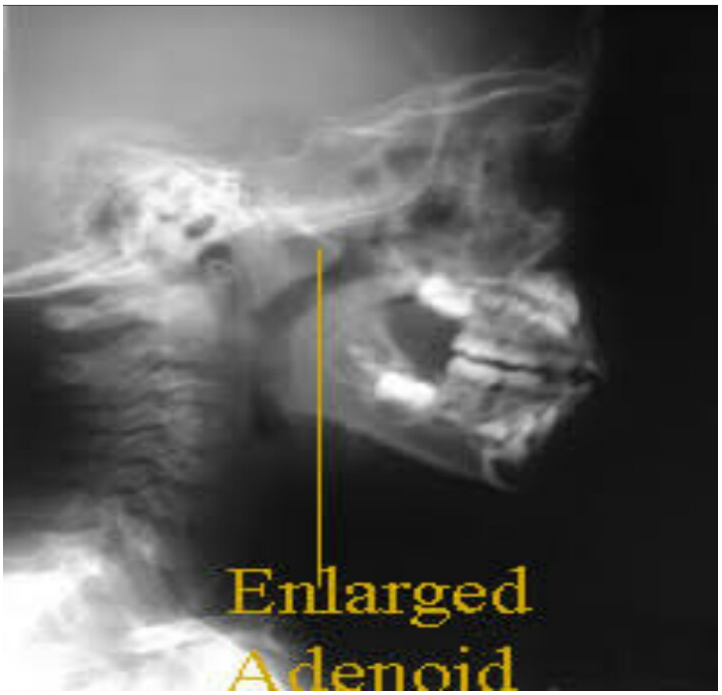
Jugulo digastric lymph nodes are palpable and tender if associated tonsillitis is present.

Examination of the ear :

In some of the patients associated otitis media will be present. In such patients the tympanic membrane may be hyperaemic, and bulges out if middle ear filled with exudate. In suppurative stage, otoscopy shows a small perforation in the parstensa, just large enough to let the middle ear drain.

DIAGNOSTIC ASSESSMENT OF ADENOIDS :

1) Lateral projections of the neck (plain x-ray) using low-dose film (soft tissue technique) will visualize the posterior wall and roof of the nasopharynx. It is helpful in assessing adenoid hypertrophy.



PIC.6.X ray neck lateral view showing adenoid enlargement

Lateral neck radiography is not necessary for the evaluation of all patients with suspected adenoid hypertrophy. Patients with significant obstructive symptomatology and obvious tonsillar hypertrophy most likely require surgical intervention, and lateral neck radiography would not alter the decision to proceed with surgery. Patients with significant nasal obstruction and insignificant tonsillar hypertrophy should undergo radiologic assessment of the nasopharynx. Patients with significant nasal obstruction and evidence of allergic rhinitis may have concurrent under lying adenoid hypertrophy and would benefit from lateral neck radiography.

2) With CT scanning, major studies in the examination of the nasopharynx were made possible. Scans of the nasopharynx are characteristically performed in the axial plane. Important processes visualized include adenoidal pads, the torus tubaris and the Eustachian tube.

3) Flexible endoscopic nasopharyngoscopy may also be valuable in the assessment of adenotonsillar disease. With appropriate topical anesthesia, pediatric endoscopes, and reassurance from the physician, this procedure is generally tolerated well by children. The presence of adenoid tissue obstructing the posterior nasal choana will be apparent by this technique. The presence of adenoiditis may also be diagnosed by the presence of purulent secretions covering the adenoid pad. Wormald, Prescott, Wang and others demonstrated the efficacy of flexible nasopharyngoscopy compared with lateral neck radiography and clinical symptomatology in the assessment of adenoid hypertrophy in children.

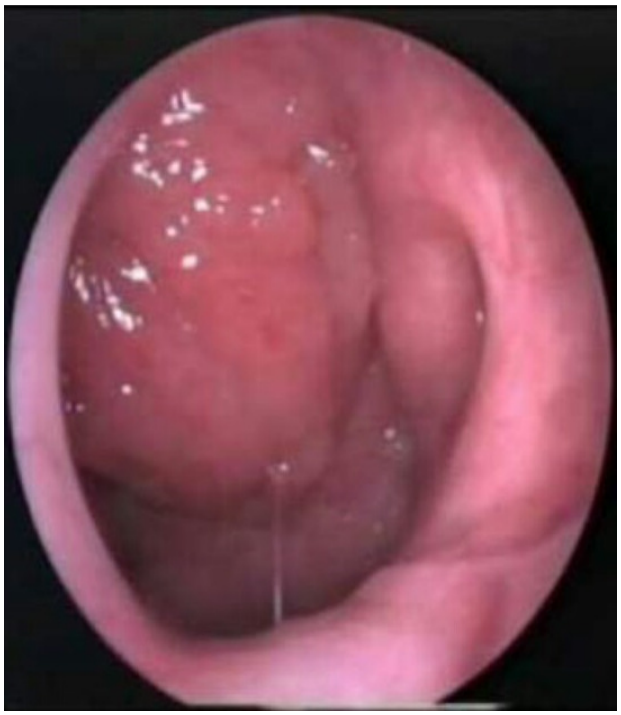
Clinical grading of adenoid size by Clemens et al.[14]

Grade I - adenoid tissue filling one-third of the vertical portion of the Choana.

Grade II - Adenoid tissue filling from one-third to two-thirds of the Choana.

Grade III - from two-thirds to nearly complete obstruction of the choana.

Grade IV - complete choanal obstruction.



PIC.7.Endoscopic view of enlarged adenoid

4) Rhinomanometry has also been demonstrated to correlate with the presence of nasal obstruction secondary to adenoid hypertrophy.[15] However, this test is not well tolerated by children, is difficult and time-consuming to administer, and is probably not of clinical benefit in its current form.

ADENOIDECTOMY :

A Danish otolaryngologists, Wilhelm Meyer, identified the adenoids and their significance more than a century ago. From that time surgical removal of adenoid has been one of the most common procedures. He did first adenoidectomy in 1876. He recommended curettage through the nose, assisted by the finger in the nasopharynx.

Adenoidectomy is a commonly performed and crucial procedure in the field of otorhinolaryngology. In 1994, an estimated 140,000 U.S. children under the age of 15 years had adenoidectomies and an estimated 286,000 had adenotonsillectomy.

ADENOIDECTOMY INDICATIONS :

Adenoid hypertrophy or chronic adenoiditis may cause significant problems requiring adenoidectomy in situations in which the tonsils themselves are not diseased and are not contributing to symptomatology. Patients with chronic adenoid hypertrophy causing craniofacial morphology problems, excessive snoring, or possibly quality of life issues are candidates for adenoidectomy. Patients with a history of chronic recurrent sinusitis may also benefit from adenoidectomy. Patients with chronic sinusitis and significant adenoid

hypertrophy may initially benefit form adenoidectomy rather than undergoing more extensive sinus surgery. In addition, patients with chronic purulent rhinitis secondary to chronic adenoiditis may also respond to adenoidectomy if they have not responded well to appropriate medical therapy.

Patients with hyponasal speech (rhinolalia clausa) are also candidates for adenoidectomy. Although surgical intervention should be considered in cases of severe nasal obstruction related to adenoid hypertrophy, there is evidence that alternative medical therapy exists to manage adenoid hypetrophy.

In one of the study it was demonstrated that aqueous nasal beclomethasone therapy led to significant improvement of nasal obstruction secondary to adenoid hypertrophy, which was confirmed by pre and post management flexible nasopharyngoscopy. In addition, patients with underlying inhalant allergies may benefit from antihistamine therapy and possibly from allergic immunologic desensitization therapy.

Conservative adenoidectomy should be performed in patients with a cleft palate or submucous cleft palate, leaving the lower portion of the adenoid pad intact to decrease the risk of post-operative velopharyngeal insufficiency.

Surgical extirpation of the adenoids may remove a nasopharyngeal nidus of contaminated tissue that secondarily acts as a source of infection in the middle ear, or adenoidectomy may simply remove an anatomic obstruction of the Eustachian tube. The actual size of the adenoid pad has not necessarily been implicated in the etiology of chronic otitis media with effusion.

Based on current evidence, adenoidectomy should be considered in children undergoing ventilation tube replacement who have symptomatology suggestive of chronic nasal obstruction or adenoid hypertrophy that confirmed by nasopharyngoscopy or nasopharyngeal radiography. Patients who require subsequent sets of ventilation tubes also may be candidates for adenoidectomy, regardless of adenoid size or symptomatology.

PREOPERATIVE ASSESSMENT :

Preoperative assessment in patients undergoing adenoidectomy is crucial and may reveal potential problems that may complicate either surgery or the patients postoperative course.[16] It is crucial to elicit the existence of any coagulation abnormalities. A family history of coagulation disorders or easy bruising may be warning sign of underlying bleeding disorder warranting further haematologic evaluation.

In one of the study the risk of postoperative haemorrhage in children undergoing adenoidectomy and adenotonsillectomy recorded. In this study 2.5% of the children had at least one abnormality on preoperative coagulation screening, which consisted of protrhombin time, partial thromboplastin time, bleeding time, and platelet count. It was suggested that although haematologic disorders were diagnosed infrequently by preoperative coagulation screening, the coagulation profile may detect patients who are more likely to bleed postoperatively.

It is apparent that patients who have an obvious family or clinical history of excessive bleeding or an underlying hematologic disorder require close monitoring of their coagulation profiles and consultation with a hematologist.

Patients with obvious severe airway obstruction secondary to adenotonsillar hypertrophy may require polysomnography, chest radiography, electrocardiography, and possible cardiology consultation.

ANAESTHESIA FOR ADENOIDECTOMY :

General anaesthesia is the anaesthetic method of choice for children undergoing adenoid surgery.

PREMEDICATION:

Good anaesthesia depends on good premedication and preparation of patients. Premedication are tailored according to condition of the patients. Most of the children require no premedication.Only atropine is given in children with severe respiratory obstruction ,sleep disorders and maxillofacial abnormalities.Younger children can be given trimeprazine or atropine.Since the curettage can cause bradycardia, atropine or gycopyrollate can be given if no other vagal block is given.

INDUCTION :

Patient can be anaesthetised with nasotracheal or orotracheal intubation. Nasotracheal intubation has no difficulty but can injure the adenoid tissue ,so it is not preferred in adenoidectomy.So,the preferred route of anaesthesia is orotracheal intubation.There can be compression of plastic tube by tongue blade.After introduction of Doughty tongue blade and flexometallic intubation tubes, orotracheal intubation has become easy and safe.

Endotracheal anaesthesia provides better control of the patients airway and allows the anaesthetist to assist spontaneous respirations when required. The endotracheal tube (cuffed) also prevents aspiration of blood or debris during

the surgical procedure and allows ready suctioning of secretions from the pharynx as needed. A wide choice of anaesthetics is available, and patients can also be managed in lighter planes of anaesthesia to allow for more rapid recovery.

Extubation of the children is the critical step in the procedure. Anaesthetist are provided with the choice of deep extubation or awake intubation. Both method has its own complications. In deep extubation, there can be laryngospasm, aspiration and hypoxia. In case of awake intubation, there can be straining, coughing and chance of increased bleeding. Awake extubation is done in patients induced with thiopentone and halothane and those with difficult airway. In such cases muscle relaxants are used during extubation. Deep extubation can be tried in patients who are induced with trimeprazine and halothane. Intra operatively complete hemostasis is attained and during extubation the mouth, pharynx are cleared of blood clots. Blood clot left in oropharynx may lodge over the laryngeal inlet and cause respiratory arrest known as Coroners clot. Close monitoring of the child is done till it has got good cough reflex and good response to commands.

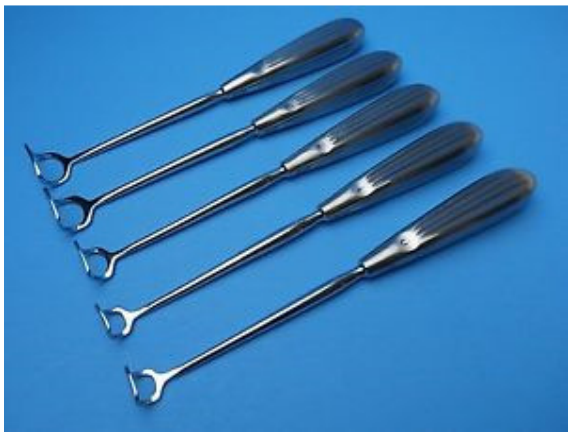
TECHNIQUE OF CONVENTIONAL ADENOIDECTOMY

General anaesthesia is given through Oro-tracheal intubation. In Rose position, Boyle Davis mouth gag with Doughty tongue blade is applied. A Blair type of head drape can then be used, particularly to protect the patients eyes and sterile drapes are placed over the patients chest. The actual surgery is not sterile, but it should be performed with sterile instruments and ancillary equipments to prevent needless exogenous contamination or infection.

In the Rose's position (position for tonsillectomy) the neck is extended but some find complete removal of adenoid difficult. Hence they use neutral supine position. After positioning the patient, Boyle Davis mouth gag with appropriate tongue blade is applied. The soft palate is retracted or relaxed. Digital palpation of adenoid in the nasopharynx is performed.[17] Extent of adenoid, its proximity to Eustachian tube orifice ascertained. Digital dissection of the adenoid from Eustachian tubal orifice is done. Adenoid tissue pushed to midline. Using St.Clair Thompson adenoid curette, the adenoid tissue is curetted. Adenoid curette of appropriate size is chosen. Large curettes can injure the tubal orifice. Small curettes can leave back the remnant tissue. The adenoid curette is brought behind the soft palate and brought to the adenoid till the nasal septum is felt. By gentle swinging movement forwards, the adenoid tissue is removed.

Remnant adenoid attached to mucosa are removed with forceps without injuring the posterior pharyngeal wall. Pack is placed in post nasal space and waited to attain hemostasis.

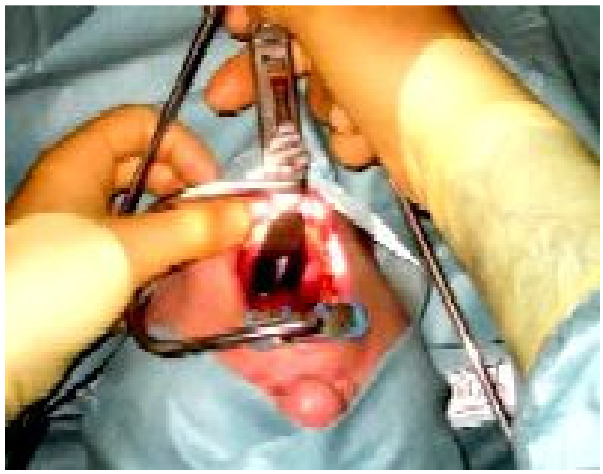
PIC.8.Picture showing St.Clair Thompson Adenoid curette



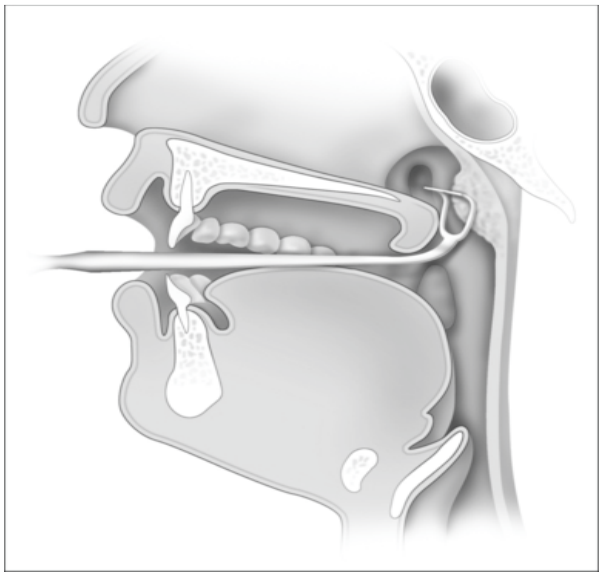
PIC.9.Picture showing different sizes of adenoid curette

Bleeding is usually controlled by pack for 5 minutes.If even after 5 minutes bleeding continues some resort to re packing for another 5 minutes. If bleeding persists some use laryngeal mirror for visualisation and use cautery directly. Some use post nasal pack and left in place for 24 hours. Hence the child is extubated only when complete hemostasis is attained.

Other techniques employed are removal of the adenoid by LaForce adenotome and curretting the remainsts of adenoid by Barnhill currette.



PIC.10.Picture showing position for adenoidectomy



PIC.11.Picture showing technique of adenoid curettage

COMPLICATIONS OF ADENOIDECTOMY :

The complications of adenoidectomy include:

1. bleeding;

2. velopharyngeal dysfunction

3. airway obstruction, due to:

    - retained swab;

    - nasopharyngeal blood clot;

4. infection;

5. dental trauma;

6. cervical spine injury (particularly in Down syndrome);

7. regrowth of the adenoid.

BLEEDING:

Reactionary haemorrhage is bleeding within 6 to 20 hours of adenoidectomy.[18] It is rare following adenoidectomy. Even if it occurs, they are managed by post nasal pack. Some keep it for four hours others keep in situ for twenty four hours. Some even admit in intensive care after post nasal packing and monitor with antibiotics. Percentage of reactionary hemorrhage decreases with adenoidectomy done under direct vision and with powered instruments.[19]

Unusual bleeding after adenoidectomy should raise the suspicion of bleeding disorders and should be evaluated by expert haematologist. Bleeding can also arise from aberrant ascending pharyngeal artery.[20]

VELOPHARYNGEAL DYSFUNCTION:

Velopharyngeal insufficiency following adenoidectomy is rare accounting for 1 in 1500 to 1 in 10,000 adenoidectomies. Hence patients subjected to adenoidectomy should be checked for sub mucosal cleft palate and bifid uvula.[21] They may have regurgitation of fluids and hypernasal speech. Sub mucosal cleft palate or bifid uvula are managed with adenoidectomy under

direct vision with endoscope and precise excision. In cases where swallowing and speech are severly affected, reconstructive surgery required.[22]

RETAINED SWAB:

Retained swabs in the pharynx either in nasopharynx or laryngopharynx are considered as negligence. Operative field should be thoroughly checked for any retained swabs before removing the mouth gag and reversing from anaesthesia.

NASOPHARYNGEAL BLOOD CLOT:

Blood may get collected and get clotted in nasopharynx during the procedure. Thorough suctioning is done before removing the mouth gag and reversing from anaesthesia. Otherwise, blood clot may fall over the larynx to cause acute respiratory obstruction. This is called as ***coroners clot.***[23]

INFECTION:

It is uncommon to have infection of nasopharynx following adenoidectomy. Retropharyngeal abscess and mediastinal abscess are rare occurrences after adenoidectomy following trauma during adenoidectomy.[24]

DENTAL TRAUMA:

Damage to the upper teeth is common during adenoidectomy as with tonsillectomy. It is due to damage caused by the mouth gag. Care should be taken to avoid injury to teeth. It is considered negligent if injury occurs to children in whom secondary dentition has erupted. Injury can be prevented if mouth gag is applied with gag resting lateral to upper incisors. In case of loose tooth present pre operatively, consent is obtained to remove the tooth in anaesthesia to prevent aspiration of the tooth.

CERVICAL SPINE INJURY:

Overuse of diathermy either to remove adenoid or to control bleeding can lead to non-traumatic Atlanto axial joint dislocation called as ***Grisel syndrome***.[25]

It is also seen in Downs syndrome children who are more prone for atlanto axial joint instability. Pre operative radiography of neck is taken to evaluate the joint condition and caution is executed while positioning the neck.

REGROWTH OF THE ADENOID:

On follow up it was found that most of the patients developed no regrowth of the adenoid. It was formulated that adenoid tissue occupying forty percent of choana is sufficient to cause symptoms and requires re-surgery.[26]

DEATH:

Death due to isolated adenoidectomy without tonsillectomy or without anaesthetic complication has not been accounted.

ENDOSCOPY ASSISTED ADENOID CURETTAGE:

In 1806, the first endoscope was developed by **PHILIPP BOZZINI,** for the examination of the canals and cavities of the human body.[27]

In 1945, **KARL STORZ** began producing instruments for ENT specialists.[28]

PROCEDURE:

In endoscopy assisted curettage, till intubation every step is similar to the conventional method. The position of the patient is in supine position with head ring support. Boyle Davis mouth gag and correct sized tongue blade was introduced and fixed. The 0^{o} 2.7mm/4mm Hopkin's rigid endoscope is introduced intranasally and the nasopharynx is visualised for adenoid hypertrophy. St.Clair Thompson adenoid curette was introduced orally and reached upto the superior end of adenoid by visualising with the help of endoscopy. Then the adenoid mass is curetted under visualisation without injuring the adjacent structures. Bleeding is arrested by suctioning through other nostril and postnasal pack kept.[29]

OTHER METHODS OF ADENOIDECTOMY :

ENDOSCOPIC POWER ASSISTED ADENOIDECTOMY USING MICRODEBRIDER.

The original design **"*VACCUM ROTATORY DISSECTOR*"** was introduced by URBAN in 1969. It was used in acoustic neuroma and in arthroscopy. SETILIFF and PARSONS first used in nasal surgery in 1994.

In the endoscopic technique, the endoscope was used along with a micro-debrider (Stryker : Hummer model) in the oscillating mode with saline irrigation using speeds up to 2400 rpm to curette and shave off the adenoid tissue using adenoidectomy blades. Bipolar cautery was used to stop bleeding from the raw surface of the adenoid bed. The procedure was visualized using 2.7mm and 4mm nasal endoscopes using the contralateral nostril as the conduit. When it was not possible to introduce the scope from the opposite side, an angled 45-70 degree scope was introduced through the oral cavity and working end of the instruments seen.

Angled microdebrider blades are introduced through oral cavity after retacting the soft palate. Hemostasis attained with guaze pack and can use bipolar diathermy.[30,31]

TELESCOPIC ASSISTED RADIOFREQUENCY ADENOIDECTOMY.

This technique also begins as conventional method, but the head end of the opertaing table is 10° to 20° below the horizontal plane. The nasopharynx was visualized with Hopkins II 90° and /or 120° telescope. (Karl Storz), which was administered trans orally. An adenoidal radio frequency currette which was connected to radio frequency apparatus was gently introduced against the

posterior end of the septum with the adenoids or its main part in its cage. The power was adjusted to 7W in a fully rectified mode. The currette was activated for 2 to 3 seconds and gently swept downwards over a period of 3 to 5 seconds, shaving the adenoids. No packing was required at the end of the procedure.

This method allows removal of huge adenoids completely in a precise, easy, and cost effective manner. It also provides a clear visualization that helps complete removal of adenoids, reduction of unnecessary trauma, and effective control of bleeding.[32]

ENDOSCOPIC TRANSNASAL ADENOID ABLATION.

The procedure was performed under general anaesthesia. The nasal mucosa was decongested. A 0° endoscope was introduced into the nasal cavity and advanced into the nasopharynx. The suction diathermy was then introduced on the ipsilateral side and used at a setting of 30W monopolar coagulation to ablate the adenoid tissue. A similar procedure was performed on the contralateral side. Any bleeding was controlled with the use of the same instruments. This technique begins the same way as conventional adenoidectomy. The overbent cannula which is seated in the hand piece, with attached suction tube is introduced into the nasopharynx under direct mirror visualization. The foot

pedal switch is depressed to activate the oscillating blade. Resection of the adenoid is performed by ossilating blade under mirror visualization. This technique has advantages like greater speed, greater control of the breadth and depth of the resection and lesser blood loss.

SUMMARY OF PUBLICATIONS

1. In the study, " **Comparison of Endoscopic-Assisted Adenoidectomy(EA) with Conventional Method(CA)"** EA was performed in 16 patients (9 boys, 7 girls, mean age 8.2 years, age range 5-13 years) and CA was performed in 16 patients (8 boys, 8 girls, mean age 8.3 years, range 5-13 years) for a total of 32 patients. There was no statistical difference in terms of age and sex. Results of the study were evaluated using the various parameters. There was a greater incidence of higher grade of residual adenoid tissue after 3 months of surgery in CA as compared to EA ($p < 0.05$). The mean blood loss in CA was 33cc, and in EA, it was 38cc ($p > 0.05$). The mean operative time in CA was 9 minutes while in EA it was 14 minutes ($p < 0.05$). No postoperative complications in the form of postoperative bleeding, velopharyngeal insufficiency, atlantoaxial dislocation, Eustachian tube scaring, etc. were observed in either group.

2. In the study "**ENDOSCOPIC ASSISTED ADENOIDECTOMY VERSUS CONVENTIONAL CURETTAGE ADENOIDECTOMY: A META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS"** the meta-analysis demonstrated that compared with the conventional adenoidectomy, endoscopic assisted adenoidectomy had a shorter operative time(SMD -1.09;95% CI-1.29 to -0.90; p<0.00001), less blood loss(MD -19.74; 95% CI -22.75 to -16.73;p<0.00001). Thus endoscopic assisted method has advantages over conventional method with regard to total operative time, blood loss and complications.

3. Conventional Versus Endoscopic-Assisted Adenoidectomy:

A Comparative Study by Ihsan Allawi Hussein Saad AL-Juboori College of Medicine, University of Babylon, Hilla, Iraq.After a short period of follow –up ,the time taken in Group A (conventional surgery) varied from 10-35 minutes [with a mean= 32.25 minutes](95% CI 20.004-26.496) (P <0.001) and in Group B (endoscopic method) from 20-90 minutes [with a mean of 42.75 minutes] (95% CI 43.097-15.604) .The resection was almost complete in group B whereas in group A four cases (20%) had residual adenoid tissue(95% CI _0.3895-_ 0.0141)(P<0.035).associated trauma in Group A found in 3 cases (15%) all are minor involving the uvula and posterior pharyngeal wall whereas

in Group B trauma occurs in 5 cases (25%),3 are minor involving septal mucosal injuries while 2 cases (10%) required nasal packing(P >0.05). Velopharngeal dysfunction developed in 2 cases (10%) of Group A and 3 cases(15%) of Group B ,all are resolved spontaneously within a week and not required any treatment (95% CI _0.0155-0.255)(P>0.05).Infection complicated 1 cases (5%) of Group A and 1 case of Group B(95% CI _0.1479-0.3479) (P>0.05) .Retained swab complicated 1 case (5%) of Group A whereas none in Group B.The overall complication rate in Group A was 55% whereas in Group B 45%.

4. In the study "***Transoral Endoscopic Adenoidectomy"by Amr El-Badrawy and Mosaad Abdel-Aziz"*** they studied 300 children who underwent transoral endoscopic adenoidectomy using the classic adenoid curette and St Claire Thomson forceps with a 70◦ Hopkins 4-mm nasal endoscope introduced through the mouth and the view was projected on a monitor. Telephone questionnaire was used to follow-up the children for one year. Flexible nasopharyngoscopy was carried out for children with recurrent obstructive nasal symptoms to detect adenoid rehypertrophy. No cases presented with postoperative complications. Only one case developed recurrent obstructive nasal symptoms due to adenoid regrowth and investigations showed that he had nasal allergy which may be the cause of recurrence.They concluded that Transoral endoscopic

adenoidectomy is the recent advancement of classic curettage adenoidectomy with direct vision of the nasopharynx that enables the surgeon to avoid injury of important structures as Eustachian tube orifices, and also it gives him the chance to completely remove the adenoidal tissues.

MATERIAL AND METHODS

TOPIC:

COMPARATIVE STUDY BETWEEN CONVENTIONAL ADENOID CURETTAGE AND ENDOSCOPIC ASSISTED ADENOID CURETTAGE.

SELECTION OF SUBJECTS :

Patients between 5 to 15 years of age , of both the sexes

DATA COLLECTION :

Relevant and detailed history, clinical examination ,x ray neck soft tissue lateral view or nasal endoscopic examination .

DESIGN OF STUDY

Prospective randomised study

PERIOD OF STUDY: 1 year (2015 -2016)

ETHICAL CLEARANCE:

CONSENT:

Individual/ parental written and informed Consent

ANALYSIS:

Statistical analysis

INCLUSION CRITERIA

1.Cases of chronic adenotonsillitis and Adenoid hypertrophy.

2.Any sex of weight more than 20 kgs.

3.Hemoglobin>10%

EXCLUSION CRITERIA :

The patients with

1. Bleeding disorders.
2. Eustachian tube pathology.
3. Middle ear infections.
4. Cleft palate.
5. Immunodeficiency state.
6. Comorbid conditions.

INVESTIGATION :

- Routine blood examination
- Bleeding time and clotting time
- Urine routine

- Chest X ray.
- Electrocardiogram
- Diagnostic nasal endoscopy

METHOD:

Fifty consecutive cases between the ages of 5-15,admitted in Thanjavur medical college and hospital and requiring adenoidectomy fulfilling the inclusion and exclusion criteria were included in the study.

All subjects including their parents were counselled about the nature of the study and informed and written consent taken. On enrolment, the subject underwent a baseline evaluation including symptom analysis and nasal endoscopy.

All symptoms recorded and their severity graded with Visual Analog Scale(VAS).

The grade of adenoid hypertrophy was assessed using the scale described by ***Clemens and Mcmurray*** where adenoids are graded according to the extent of vertical height of choana filled by adenoid tissue,

Grade I –filling upto one third of height,

Grade II- up to two third,

Grade III –more than two third but not completely filling choana and

Grade IV - complete choanal obstruction .

All the cases were randomized into two groups consecutively.

Group A consisted of cases undergoing ***Conventional Adenoidectomy using Curettage method*** and ***Group B*** undergoing ***Endoscopic Assisted Adenoid curettage***.

All surgeries were performed by a single surgeon under supervision. General anaesthesia was used using oro-tracheal tube and a laryngeal pack. In the conventional technique, adenoidectomy was done using the adenoid curette.

In endoscopic assisted curettage, the 0^{o} 2.7mm/4mm Hopkin's rigid endoscope is introduced intranasally and the nasopharynx is visualised for adenoid hypertrophy. St.Clair Thompson adenoid curette was introduced orally and reached upto the superior end of adenoid by visualising with the help of endoscopy. Then the adenoid mass is curetted under visualisation without injuring the adjacent structures. Bleeding is arrested by suctioning and postnasal pack was kept.

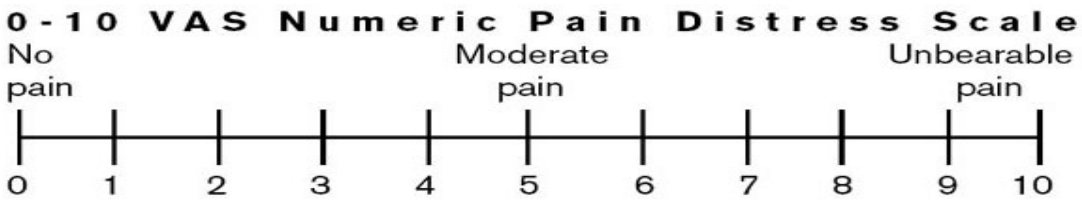
PARAMETERS COMPARED:

The intra-operative parameters studied were operative time, completeness of removal of adenoid and Eustachian tube injury . Post-operative parameters included assessment of post-operative Symptomatic relief and recovery time. During each followup patient evaluated for symptom relief and remnant adenoid tissue

Intra operative time was defined as the time taken for completion of the procedure from the time patient was handed over by the anaesthetist and included preoperative endoscopic examination to assess adenoid grade, operative steps, packing ,securing the bleeding and post operative endoscopy. The measurement ended when the patient was handed back to the anaesthetist. In cases where tonsillectomy was also combined, the time taken for tonsillectomy and haemostasis was deducted.

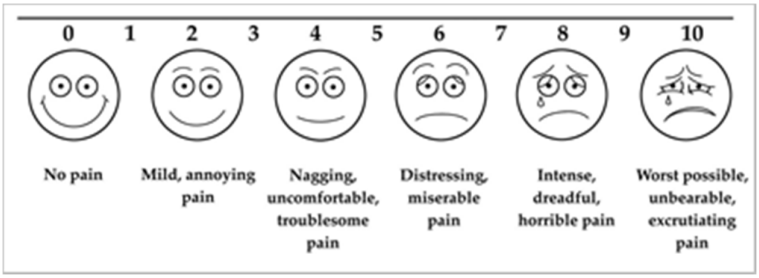
The completeness of adenoid removal was assessed by nasal endoscopy at the end of the procedure in both groups. ***A less than 20% residual adenoid was regarded as complete removal, 20-50% as mild residue indicating partial removal and more than 50% residual as significant residue indicating sub-optimal removal.***

Post operatively, the patient was assessed for post operative symptomatic relief using visual analog scale (VAS) and is compared with preoperative symptoms.



PIC.12. Visual analogue scale

A six point faces scale was used (where 0= no pain and 10= intolerable pain).



PIC.13.Pain score scale

The recovery time was defined as the number of days taken to return to normal activity as gauged by the patient / parents during the routine post operative follow-up visit at seven days. Patient advised post operative follow up in post operative first week, third week, second month. In each visit, symptom relief and nasal endoscopy to assess remnant, neighbouring structure injury and any complication.

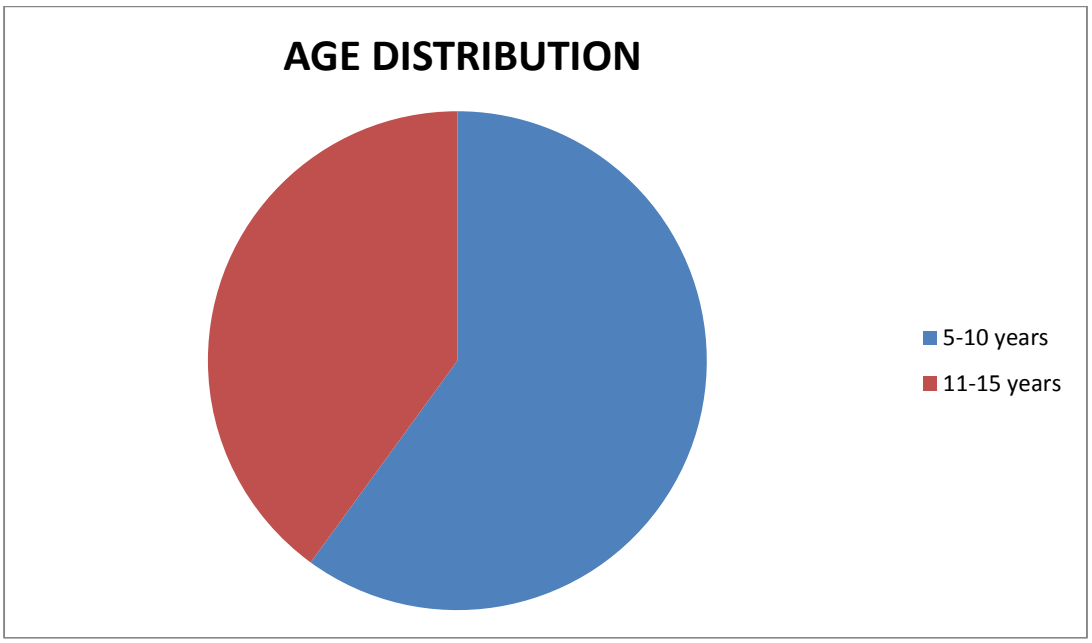
The data so obtained was compared in each group and the mean in two groups statistically analysed using the ***paired t test for significance.***

RESULTS AND OBSERVATION

AGE DISTRIBUTION

Age group	Number of Patients
5 - 10 years	30
11 – 15 years	20

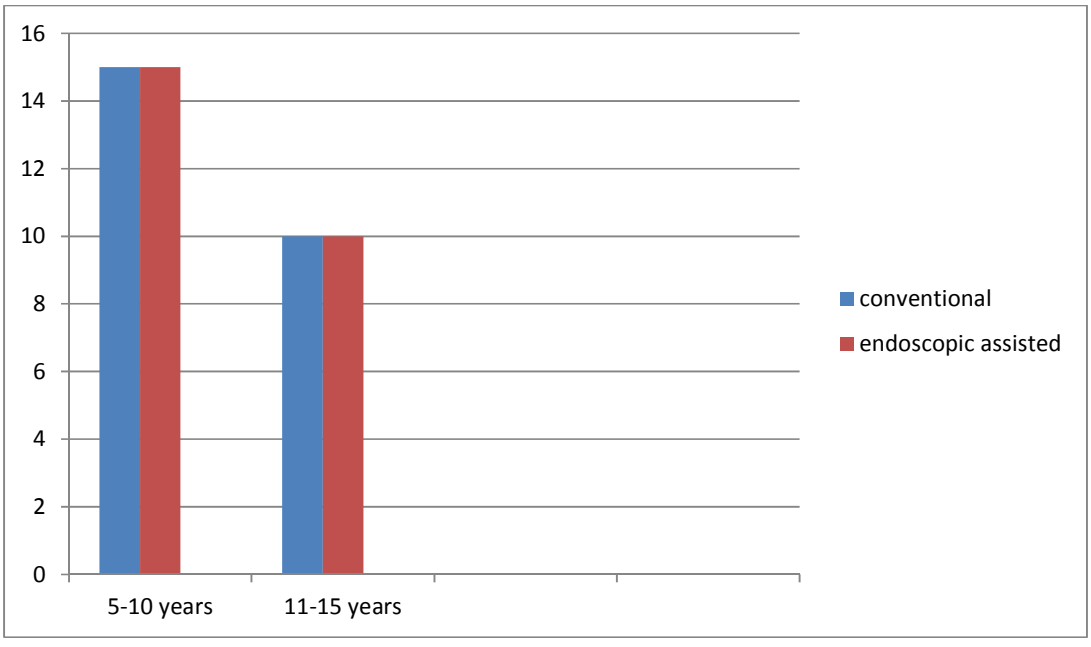
Out of 50 patients included in the group 30(60%) falls between 5 – 10 years of age and 20(40%) falls between 11 – 15 years of age.



GROUP WISE AGE DISTRIBUTION

AGE IN YEARS	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
5-10 years	15	15
11-15 years	10	10

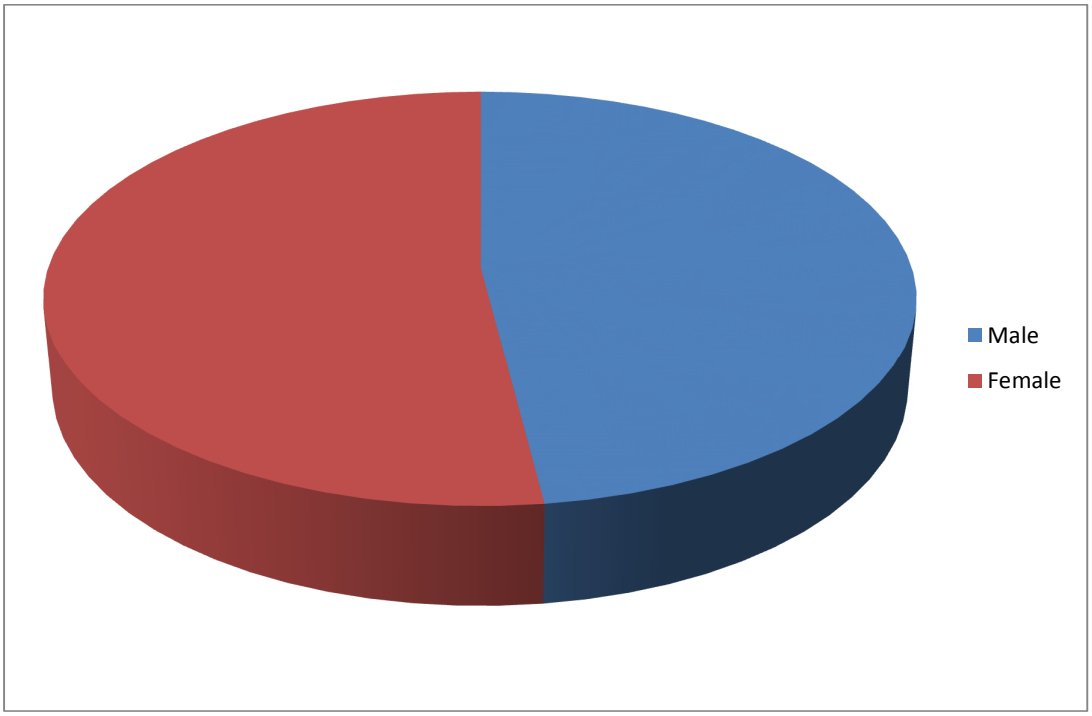
Out of 30 children in age group 5-10 years 15 undergone conventional method and 15 undergone endoscopic assisted curettage and out of 20 children in age group 11-15 years 10 undergone conventional method and 10 undergone endoscopic assisted curettage.



SEX DISTRIBUTION

SEX	NUMBER OF PATIENTS
MALE	24
FEMALE	26

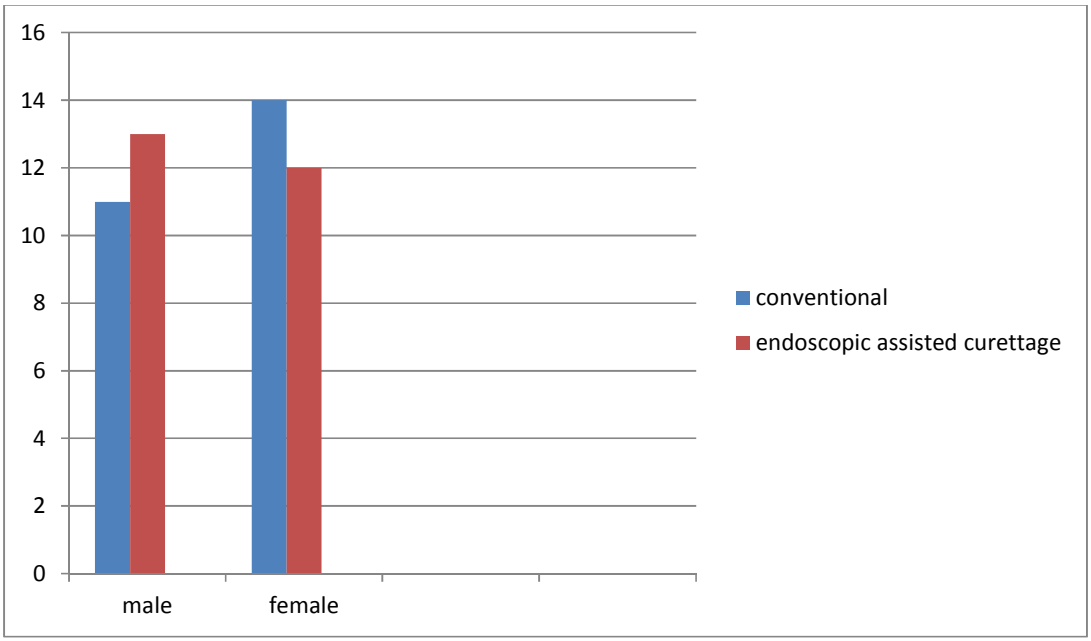
Out of 50 patients 24 were males (48%) and 26 were females (52%)



GROUP WISE SEX DISTRIBUTION

SEX	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
MALE	11	13
FEMALE	14	12

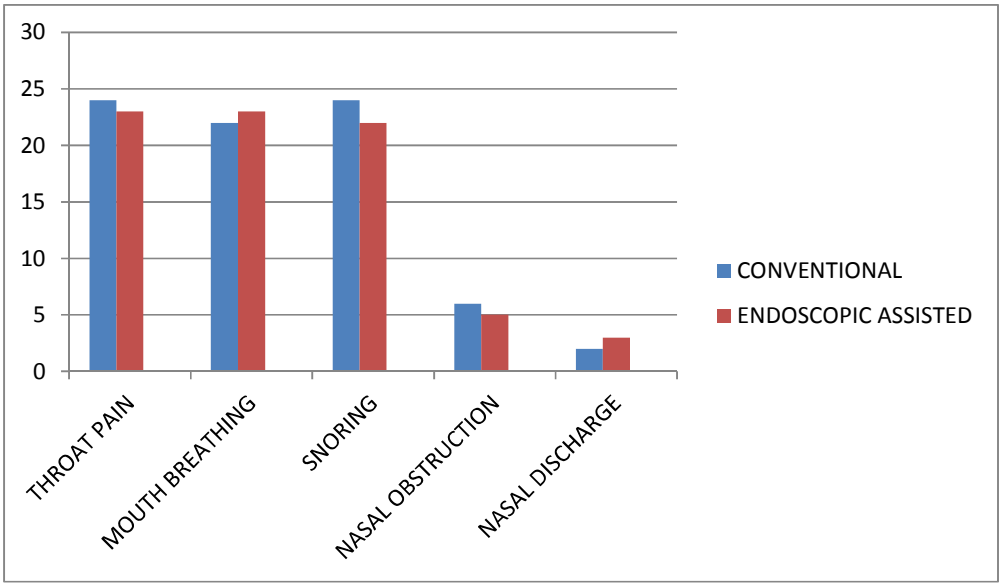
Out of 24 males 11 underwent conventional method and 13 underwent endoscopic assisted curettage and out of 26 females 14 underwent conventional and 12 underwent endoscopic assisted curettage.



SYMPTOM PROFILE OF PATIENTS

SYMPTOMS	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
THROAT PAIN	24	23
MOUTH BREATHING	22	23
SNORING	24	22
NASAL OBSTRUCTION	6	5
NASAL DISCHARGE	2	3

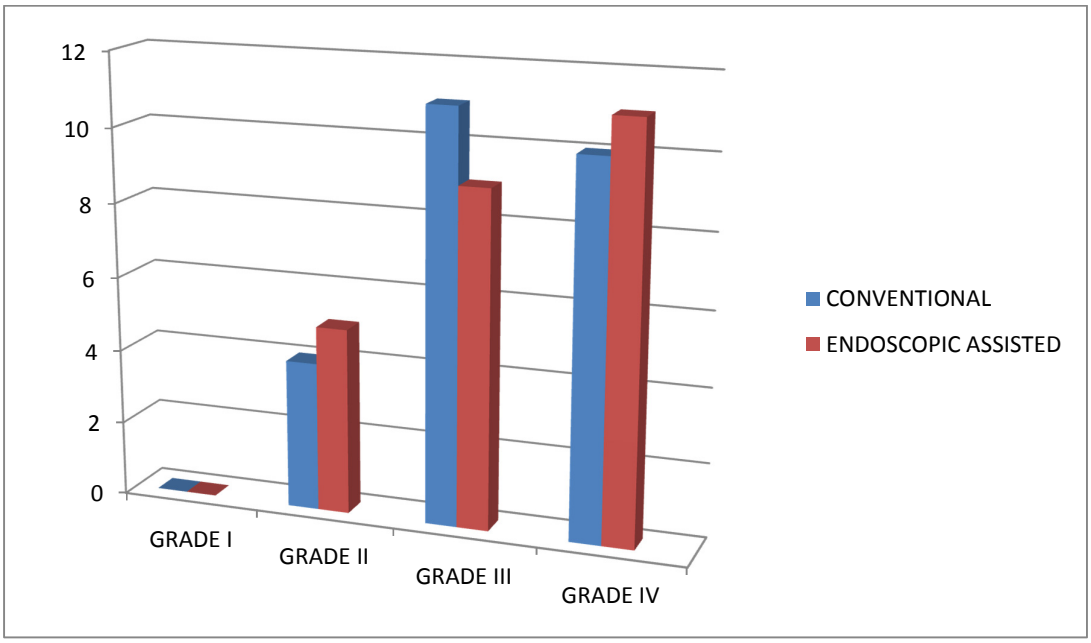
Among 50 patients, throat pain accounted for 94% and mouth breathing 90%, snoring 92% and that of nasal obstruction 22% and nasal discharge 10%



GRADES OF ADENOID

GRADE	CONVENTION CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
I	0	0
II	4	5
III	11	9
IV	10	11

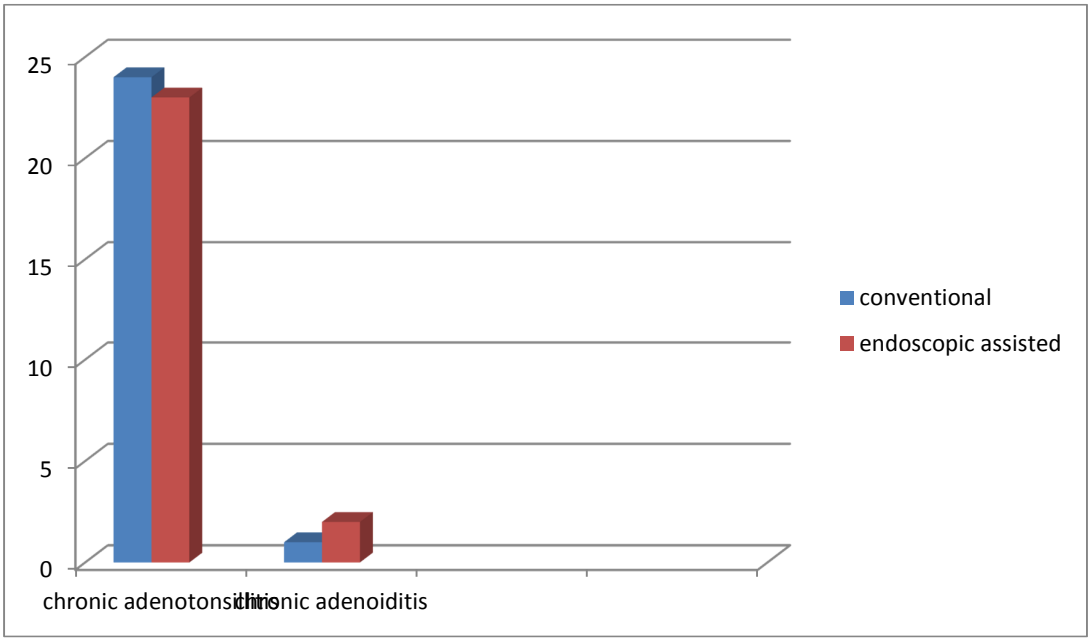
Out of 50 cases 9 patients had grade II (18%), 20 had grade III (40%) and 21 had grade IV (42%) and none of them had grade I



DIAGNOSIS

DIAGNOSIS	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
CHRONIC ADENOTONSILLITIS	24	23
CHRONIC ADENOIDITIS	1	2

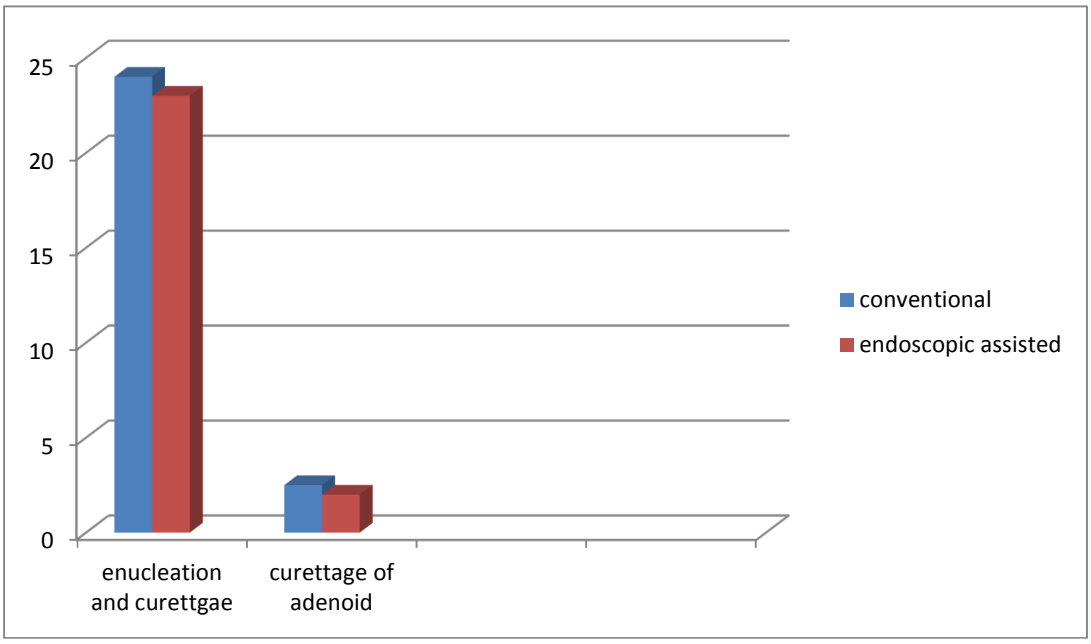
Out of 50 patients 47(94%) had chronic adenotonsillitis and in which 24 underwent conventional method and 23 underwent endoscopic assisted curettage and those who had chronic adenoiditis is alone 3(6%) cases.



PROCEDURE DONE

PROCEDURE	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
ENUCLEATION OF TONSIL AND CURETTAGE OF ADENOID	24	23
CURETTAGE OF ADENOID	1	2

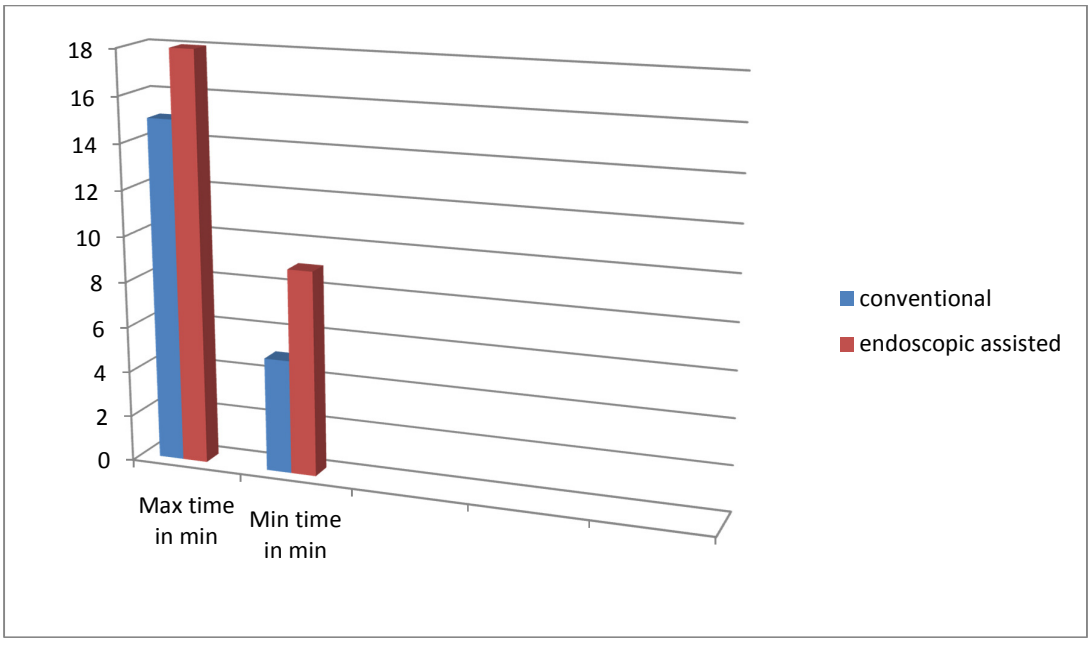
Out of 50 patients 47 (94%) underwent enucleation and curettage and 3 (6%) underwent curettage of adenoids alone.



OPERATIVE TIME

OPERATIVE TIME IN MINUTES	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
MAXIMUM	15	18
MINIMUM	5	9

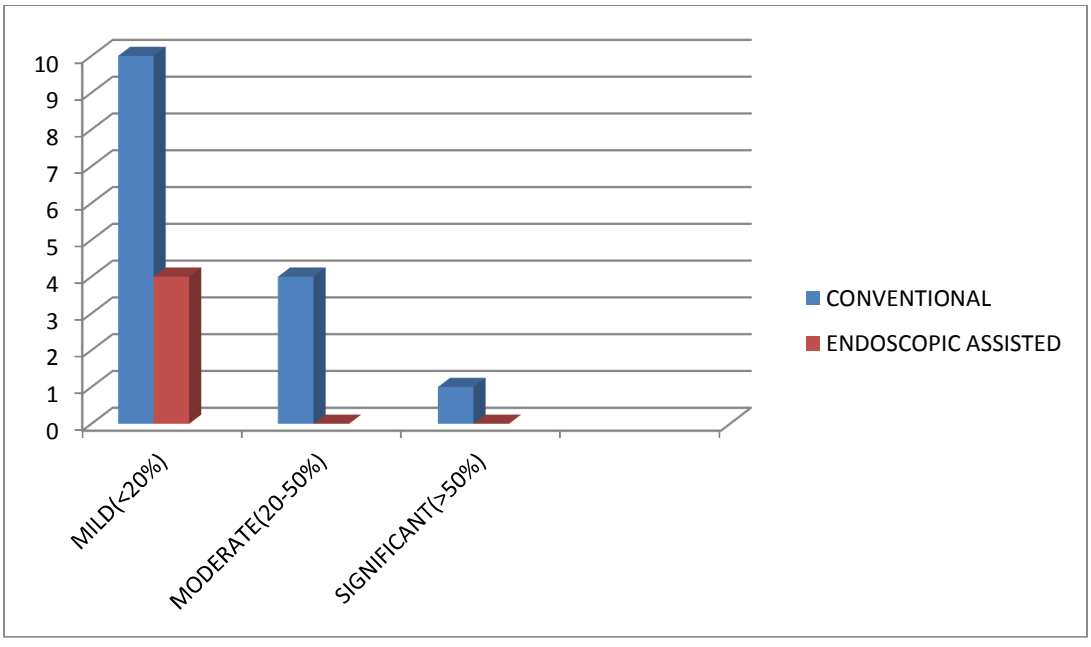
Operating time was maximum in endoscopic assisted adenoid curettage reaching upto 18 minutes whereas in conventional method the maximum time is upto 15 minutes. The difference in operating time is significantly better in conventional method when compared to endoscopic assisted adenoid curettage($p<0.05$).



TISSUE REMNANT

REMNANT	CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
MILD (<20%)	10	4
MODERATE (20-50%)	4	0
SIGNIFICANT (>50%)	1	0

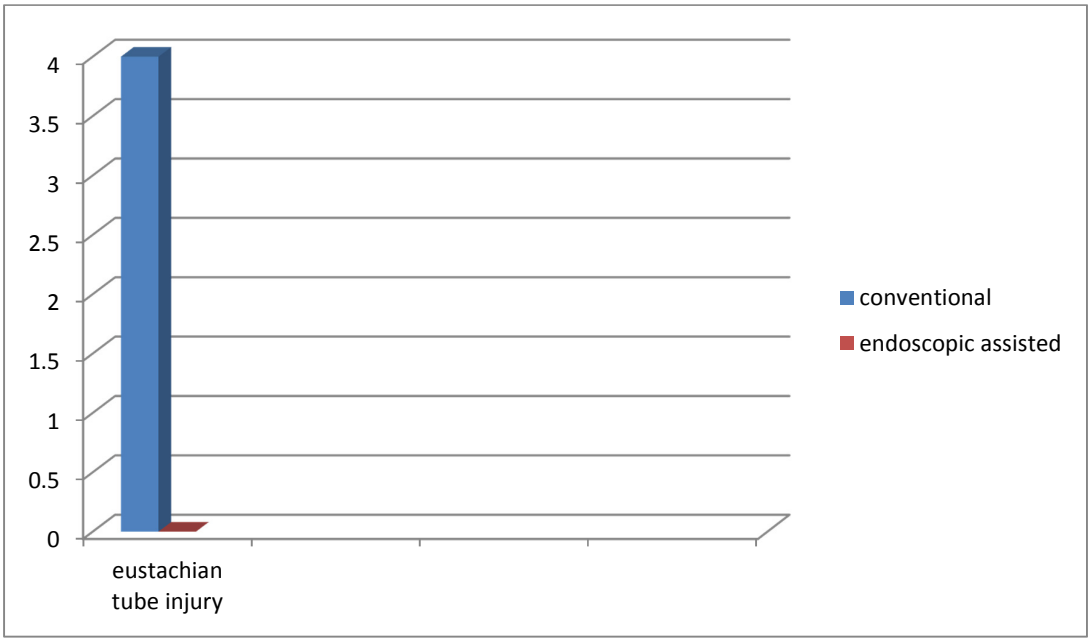
Out of 25 cases underwent conventional curettage 15 cases had remnant tissues postopertively and only 4 cases in endoscopic assisted curettage and that too of <20% tissue remnant. Thus endoscopic assisted method is significantly better when compared with conventional method.($p<0.05$)



EUSTACHIAN TUBE INJURY

CONVENTIONAL CURETTAGE ADENOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
4	0

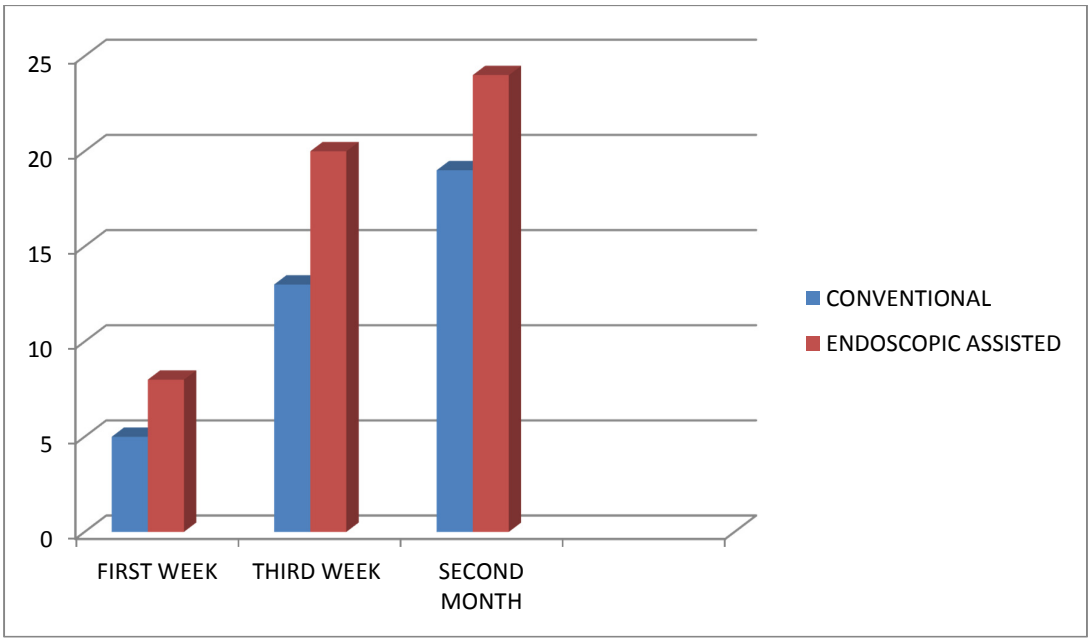
Out of 50 cases only 4 cases had Eustachian tube injury that too in conventional method group. Endoscopic assisted method is significantly better than conventional method($p<0.05$)



POSTOPERATIVE SYMPTOMATIC RELIEF

SYMPTOMATIC RELIEF PERIOD	CONVENTIONAL CURETTAGE ADNOIDECTOMY	ENDOSCOPIC ASSISTED ADENOIDECTOMY
FIRST WEEK	5	8
THIRD WEEK	13	20
SECOND MONTH	19	24

24(96%) out of 25 cases in endoscopic assisted adenoid curettage were free of symptoms in second month whereas 19(76%) out of 25 cases in conventional method were free of symptoms in second month. Endoscopic assisted method is significantly better($p<0.05$).



DISCUSSION

The present study is to compare the conventional adenoid curettage method with endoscopic assisted adenoid curettage. The groups were evenly matched to the type of surgery done and randomisation was done to enable a more thorough comparison.

In the present series mouth breathing and snoring formed the predominant indication in both our group.

Regarding the operative time, though the steps of adenoidectomy would only take 4-5 minutes, we felt that the operative time should include all steps including preoperative endoscopy, packing and securing haemostasis. The overall operative time is more in the endoscopy assisted technique due to the handling of endoscopes and precise tissue removal. Conventional curettage is a relatively blind method and hence it took less time compared to endoscopy assisted curettage.

On comparing the post operative tissue remnant, as the endoscopic assisted technique has advantage of direct visualisation the remnant tissue after surgery is seldom present. On the contrary, in the conventional method there is possibility of leaving remnant tissue thereby increasing the rate of recurrence of the symptoms. In our study, only 4 out of 25 cases undergone endoscopy

assisted method had <20% remnant tissue. On conventional technique, 10 out of 25 cases had <20% remnant tissue, 4 out of 25 cases had 20-50% remnant tissue and 1 out of 25 cases had >50% remnant tissue.

Following adenoidectomy, injury to neighbouring structure that is Eustachian tube is not very common. In our study 4 out of 25 cases who had undergone conventional method, Eustachian tube injury is observed postoperatively whereas no such incidence occurred in any of the cases underwent endoscopy assisted technique.

To compare the postoperative symptomatic relief, Visual Analog Scale (VAS) and six point faces pain scale were used. Symptomatic relief in endoscopy assisted group were faster when compared with those who had conventional curettage.

CONCLUSION

The advanced technique of endoscopic assisted adenoid curette was found to be a safe and useful tool for adenoidectomy.

Endoscopy assisted curettage scored on completeness of resection, accurate removal without injuring nearby structure and a quicker symptomatic relief.

In our Indian scenario, it is to be kept in mind that the availability of endoscopes is a factor in choosing the method of surgery.

In cases like submucosal cleft palate and craniofacial anamolies, accurate removal by endoscopy assisted curettage enable the surgeons to carefully excise the adenoid tissue and the velopharyngeal sphincter untouched.

To conclude, endoscopy assisted curettage needs to be acknowledged as a safe alternative to the conventional adenoid curettage.

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PROFORMA

Case No. :

Name :

IP/OP No. :

Age :

Sex :

Address :

D.O.A. :

D.O.S. :

D.O.D. :

Religion :

Occupation :

Diagnosis :

CHIEF COMPLAINTS :

1) Nasal obstruction
2) Mouth breathing
3) snoring
4) Throat pain
5) Nasal voice

HISTORY OF PRESENTING ILLNESS :

1) Nasal obstruction :

Duration

Onset

Constant / Intermittent

Aggravating factors/ Relieving factors

Mouth breathing

Snoring

2) Mouth breathing :

3) Snoring

4) Throat pain :

Duration

Onset

Constant / Intermittent

Aggravating factors

Relieving factors

Dysphagia

4) Nasal voice :

Duration

Onset

5) Other symptoms :

Decreased hearing

Ear Pain

Ear Discharge

Recurrent URI

PAST HISTORY :

• Previous h/o any similar complaints in the past

• H/o bleeding disorders

• Previous h/o any surgeries in the past

• Previous h/o medications

FAMILY HISTORY :

H/o any Bleeding disorders

PERSONAL HISTORY :

• Diet

• Appetite

• Sleep

• Bowel & Bladder

GENERAL PHYSICAL EXAMINATION :

• Built

• Nourishment

• Orientation to time, place & person

• Vital data: Pulse ,B.P

Respiratory rate

• Pallor/Icterus/Cyanosis/Clubbing/Pedal oedema/Generalised

Lymphadenopathy

• Systemic examination:

- CVS

- RS

- CNS - Higher mental functions

- Cranial nerves

- Motor functions

- Sensory functions

- P/A

EXAMINATION OF THE NOSE :

• External appearance :

Root

Dorsum

Supratip

Tip

Alae

Collumella

Vestibule

• Anterior rhinoscopy :

Septum

Rt.

Lt.

Floor

Roof

Lateral wall

• Posterior rhinoscopy :

Middle & inferior turbinate

Post nasal discharge

Eustachian tube opening

• Paranasal sinus tenderness : Maxillary

Frontal

Ethmoidal

• Cold spatula test

EXAMINATION OF THE ORAL CAVITY :

• Lips

• Teeth

• Gums

• Buccal mucosa

• Anterior 2/3 of tongue

• Floor of the mouth

• Hard palate

• Soft palate

• Palpation of upper gingivobuccal sulcus

• Oro-antral fistula

EXAMINATION OF THE OROPHARYNX :

• Anterior pillar

• Tonsil

• Posterior pillar

• Posterior pharyngeal wall

EXAMINATION OF THE EAR :

- Preauricular region

- Pinna

- Postauricular region

- External auditory canal

- Tymphanic membrane

- Mastoid tenderness

- Facial nerve

- Tunning fork tests - Rinne's

- Weber's

- Air bone conduction

Rt.

Lt.

EXAMINATION OF THE NECK :

- Lymphnodes / Swellings/ Fistula/Skin of neck

INVESTIGATIONS :

Blood :

Urine :

Hb%

BT:

TC:

ESR:

HIV

HBsAg

Albumin

Sugar

Microscopy

CT:

DC:

Radiology: X-ray - Nasopharynx lateral view

Nasal endoscopy: to assess adenoid grade

FINAL DIAGNOSIS :

TREATMENT :

SURGERY: Type of surgical procedure :

ANAESTHESIA : LA/GA

POSTOPERATIVE COMPLICATIONS :

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR.JAGAN A.S.,** Post graduate in the Department of Otorhinolaryngology, Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date :

Signature of participant

ABBREVIATIONS:

S.NO -SERIAL NUMBER

M-MALE

F-FEMALE

I.P NO- IN PATIENT NUMBER

N.O-NASAL OBSTRUCTION

M.B-MOUTH BREATHING

SN-SNORING

N.D-NASAL DISCHARGE

T.P-THROAT PAIN

CHR.ADE-CHRONIC ADENOIDITIS

CHR T &A-CHRONIC ADENOTONSILITIS

SOM-SEROUS OTITIS MEDIA

CSOM-CHRONIC SUPPURATIVE OTITIS MEDIA

CRS-CHRONIC RHINO SINUSITIS

CA-CONVENTIONAL CURETTAGE ADENOIDECTOMY

EA-ENDOSCOPIC ASSISTED ADENOIDECTOMY

MASTER CHART

S.NO	NAME	AGE	SEX	IP.NO	T.P	M.B	S.N	N.O	N.D
1	PRIYANKA	9	F	6798	8	9	6	0	0
2	MAHESWARAN	6	M	6799	8	8	8	0	0
3	ALISULTAN	10	M	7425	8	6	8	0	0
4	DHARSINI	6	F	10070	9	0	9	5	0
5	DHARSHANA	9	F	10609	8	7	7	0	0
6	ALI ASKAR	10	M	10560	9	8	9	0	0
7	ANBUSELVI	14	F	10834	0	7	9	8	6
8	ASMAR ALI	11	M	11189	8	9	8	0	0
9	RITHESH	7	M	11357	8	9	8	0	0
10	DHAHYA	12	F	11562	9	0	7	7	6
11	ABINAYA	10	F	13409	7	8	8	0	0
12	PUSHPENDRA	9	M	14379	9	9	8	0	0
13	STEPHEN	10	M	14384	9	8	7	0	0
14	SURESH	10	M	16843	8	7	8	0	0
15	GANESH	8	M	19945	8	8	8	0	0
16	VISHAL	11	M	20696	7	9	0	0	0
17	AJAY	7	M	21426	8	8	9	0	0
18	ABDUL HAKIM	9	M	22072	7	9	9	0	0
19	SAISHREE	7	F	22256	9	0	8	7	5
20	HARINI	11	F	22295	8	7	7	0	0
21	SAFIKA BANU	9	F	23977	7	8	6	0	0
22	MAHALAKSHMI	10	F	24596	9	9	7	0	0
23	KANNAN	11	M	25853	8	8	6	0	0
24	SEIK MUHAMMED	10	M	25893	8	8	0	6	0
25	VASUKI	7	F	26138	7	9	8	0	0

S.NO	NAME	AGE	SEX	IP.NO	T.P	M.B	S.N	N.O	N.D
26	RAGAVENDRAN	9	M	28972	8	9	8	0	0
27	APARNA	12	F	29530	7	8	0	8	7
28	ARTHI	13	F	30324	8	8	7	0	0
29	RAJALAKSHMI	11	F	30493	9	8	7	0	0
30	BARANIKA	10	F	31452	9	7	8	0	0
31	SWETHA	15	F	31463	8	7	8	0	0
32	ARULKANI	12	F	31905	8	8	7	0	0
33	APARNA	12	F	31943	0	9	8	0	0
34	SHARMI	11	F	31931	7	7	8	0	0
35	VENTHAN	10	M	32802	7	8	9	6	0
36	RASIYA	13	F	32804	8	7	8	0	0
37	SATHISH	9	M	33070	6	8	7	0	0
38	MOHANRAJ	8	M	34178	9	0	6	5	6
39	JOHN BRITO	9	M	35577	0	9	6	0	0
40	SANTHOSH	9	M	35572	8	8	7	7	0
41	RATHIGA	8	F	37127	9	7	8	0	0
42	SENTHAMIL SANDHYA	11	F	36112	9	7	6	0	0
43	RAVI	8	M	36225	7	8	7	0	0
44	BEER MOHAMMED	10	M	36552	8	0	6	0	0
45	GOPIKA	10	F	38516	8	8	8	6	0
46	VISHNU	12	M	39521	9	7	9	0	0
47	SHRIRAM	13	M	40121	7	8	0	0	0
48	RENU	11	F	40356	7	8	7	0	0
49	MEENA	13	F	40926	7	9	6	0	0
50	SURENDAR	11	M	40997	9	7	8	6	0

S.NO	GRADING	DIAGNOSIS	PROCEDURE	OP.TIME IN MINS	REMNANT	E.T INJ	SYMPTOMS RELIEF		
							1^{ST} WK	3^{RD} WK	2^{ND} MNTH
1	IV	CH.T&A	EAT	16	NIL	NO	YES	YES	YES
2	III	CH.T&A	EAT	12	NIL	NO	YES	YES	YES
3	III	CH.T&A	EAT	13	NIL	NO	NO	YES	YES
4	III	CH.T&A	CAT	9	MILD	NO	YES	YES	YES
5	II	CH.T&A	CAT	8	NIL	NO	NO	YES	YES
6	IV	CH.T%A	EAT	18	NIL	NO	NO	YES	YES
7	IV	CH.AD	CA	13	NIL	NO	NO	YES	YES
8	IV	CH.T%A	EAT	17	NIL	NO	NO	YES	YES
9	III	CH.T%A	CAT	10	MILD	NO	NO	YES	YES
10	II	CH.T%A	EAT	9	NIL	NO	YES	YES	YES
11	III	CH.T%A	CAT	10	MILD	YES	NO	NO	YES
12	IV	CH.T%A	EAT	15	MILD	NO	NO	NO	YES
13	III	CH.T%A	EAT	11	NIL	NO	NO	NO	YES
14	IV	CH.T%A	CAT	12	NIL	NO	NO	YES	YES
15	III	CH.T%A	CAT	6	SIGNIFICANT	NO	NO	NO	NO
16	III	CH.T&A	EAT	13	NIL	NO	NO	YES	YES
17	IV	CH.T&A	CAT	8	MILD	NO	NO	NO	YES
18	IV	CH.T&A	CAT	7	MODERATE	NO	NO	NO	NO
19	III	CH.T&A	EAT	12	NIL	YES	NO	YES	YES
20	II	CH.T&A	CAT	5	NIL	NO	NO	YES	YES
21	II	CH.T&A	EAT	10	NIL	NO	YES	YES	YES
22	IV	CH.T&A	CAT	6	MODERATE	NO	NO	NO	NO
23	III	CH.T&A	EAT	11	NIL	NO	NO	YES	YES
24	III	CH.T&A	CAT	13	MILD	NO	YES	YES	YES
25	IV	CH.AD	EA	16	MILD	NO	NO	NO	YES

S.NO	GRADING	DIAGNOSIS	PROCEDURE	OP.TIME	REMNANT	E.T INJURY	SYMPTOMS RELIEF		
							1^{ST} WK	3^{RD} WK	2^{ND} MNTH
26	III	CH.T&A	CAT	5	MILD	NO	NO	NO	YES
27	II	CH.T&A	EAT	11	NIL	NO	YES	YES	YES
28	III	CH.T&A	CAT	5	NIL	YES	NO	YES	YES
29	IV	CH.T&A	EAT	12	MILD	NO	NO	YES	YES
30	III	CH.T&A	CAT	6	NIL	NO	NO	YES	YES
31	IV	CH.T&A	EAT	14	NIL	NO	NO	YES	YES
32	IV	CH.T&A	CAT	6	MODERATE	NO	NO	NO	NO
33	IV	CH.T&A	EAT	17	NIL	NO	NO	YES	YES
34	III	CH.T&A	CAT	7	NIL	NO	NO	NO	YES
35	IV	CH.T&A	EAT	16	MILD	NO	NO	NO	NO
36	IV	CH.T&A	CAT	8	MILD	NO	NO	NO	YES
37	II	CH.T&A	EAT	9	NIL	NO	YES	YES	YES
38	II	CH.T&A	CAT	8	NIL	NO	YES	YES	YES
39	IV	CH.AD	EA	14	MILD	NO	NO	NO	YES
40	III	CH.T&A	CAT	6	NIL	YES	NO	YES	YES
41	III	CH.T&A	EAT	12	NIL	NO	NO	YES	YES
42	III	CH.T&A	CAT	7	MILD	NO	NO	NO	YES
43	II	CH.T&A	EAT	10	NIL	NO	YES	YES	YES
44	II	CH.T&A	CAT	5	NIL	NO	YES	YES	YES
45	IV	CH.T&A	EAT	17	NIL	NO	NO	YES	YES
46	IV	CH.T&A	CAT	9	MODERATE	NO	NO	NO	NO
47	III	CH.T&A	EAT	15	NIL	NO	NO	YES	YES
48	IV	CH.T&A	CAT	8	NIL	NO	YES	YES	YES
49	III	CH.T&A	EAT	14	NIL	NO	YES	YES	YES
50	IV	CH.T&A	CAT	10	MILD	NO	NO	NO	NO