1 DISSERTATION ON
EFFECT OF CHRONIC SINUSITIS ON MIDDLE EAR FUNCTION

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2 CERTIFICATE

*This is to certify that this dissertation entitled "*EFFECT OF CHRONIC SINUSITIS ON MIDDLE EAR FUNCTION*" submitted by* ***Dr. P. THIRUNAVUKARASU,*** *appearing for* ***M.S., Branch-IV (Oto-rhino-Laryngology)*** *Degree examination in March 2008, is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulation of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.*

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4 CONTENTS

INTRODUCTION

A wide variety of diseases of the nose and paranasal sinuses may affect the function of the eustachian tube and consequently that of the middle ear. The common cold, allergic rhinitis and acute and chronic sinusitis may lead to the obstruction of the eustachian tube orifice with all the attendant consequences for the middle ear.

If pathological changes occur in the nasal or paranasal sinus mucosa and the nature of secretions alter, the normal secretion routes may undergo significant changes and the two major routes may join before they reach the eustachian tube orifice, and one or both routes may form whorls around or even in the orifice itself. Abnormal infected secretions may then move directly over the eustachian tube orifice causing congestion and obstruction of the orifice due to the inflammation of its lymphoreticular tissue with slowing down of the muco ciliary clearance and may lead to impeded ventilation and/or ascending infection of the middle ear.

5 AIMS OF THE STUDY

1. To study the effect of chronic sinusitis on middle ear pressures.
2. To study the effect of chronic sinusitis on hearing thresholds.
3. To study the correlation of location of sinusitis(anterior omc vs posterior omc vs pan sinusitis)on middle ear functions
4. To evaluate eustachian tube patencies by valsalva maneuver and eustachian tube catheterisation.
5. To evaluate the effect of mastoid pneumatisation in buffering the negative pressures created.

6 HISTORICAL PERSPECTIVE

Many historical figures have made invaluable contributions to our understanding of the eustachian tube. The most notable were eustachius, valsalva, toynbee, politzer, rich, perlman and ingelstedt. During the approximately 2, 400 years since alcmaeon of sparta first mentioned the tube, which was latter definitively described by bartolomeus eustachius as the auditory tube and which now bears his name, much has been written about its anatomy, function and dysfunction. Alcamaeon thought that the tube that connected the nasal airway and the ear enabled goats to breathe through their ears and noses. the existence of the tube from the nasopharynx to the middle ear was vaguely known to the ancients, such as aristotle, celsus and even vesalius, during the renaissance, but alcamaeon and other contemporaries of eustachius paid little attention to it.

BARTOLOMEUS EUSTACHIUS

Eustachius was born in Italy cirCA 1510 and died in 1574. he was relatively unrecognized in his time because his fine collection of anatomical plates remained unprinted and forgotten in the vatican library until discovered in the early 1700s and presented by pope clement to his physician maria lancisi who published them. Eustachius not only discovered the tube but also described the cochlea, pharyngeal musculature, optic nerves, thoracic duct, adrenal glands and abducens nerve and gave the first accurate description of the uterus.

Eustachius published the first detailed description of the auditory tube in 1562 in his thesis EPISTOLA DE AUDITUS ORGANIS. He wrote;

“From the cavity of the petrous bone, there in which the auditory passage called concha such a passage toward the nasal cavity is perforated. Others would perhaps think that this passage, about which this dissertation is being written, ends in that place; this is not so, however, for it is augmented by a substance of different nature and is carried on between two muscles of the pharynx and it ends in either cavity of the nose near the internal part of the root of the apophysis of the bone that is shaped like the wings of the bat, and is inserted in a thick investment of the palate near the root of the uvula. Its substance, where it touches the extremity of the fissure which is common to the temporal and wedge-shaped bones, is cartilaginous, and quite thick; but the substance of the opposite part is not exactly cartilaginous, but is somewhat membranous and becomes thinner gradually; but the internal end of the passage facing the middle of the nasal cavity has a strong cartilage which is very thick and is covered by the mucous membranes of the nares, and is seen at the end of the same meatus as if it were a guardian. It is not round, but is somewhat depressed and makes two angles. It is as large as writing cane, but is twice as large at the end as at the beginning, which is equally invested by a mucous membrane, which is, however, thinner. ” According to POLITZER, Eustachius compared the tube to a writing pen and wrote;

“It originates at the anterior course of the base of the skull, and takes an anterior course toward the pterygoid process of the sphenoid bone. It consists of two parts: the first solidly connected with the temporal bone, close to the tympanic cavity; the second soft, partly ligamentous, partly cartilaginous, directed toward the nasopharynx. Cross sections of the tube are not perfectly round and the inner part is twice as wide as the outer.

Also, the inner part adjacent to the nasopharynx is lined with mucous membrane and seems to possess a sphincter at its end. The mucous lining is continuous with the nasal mucosa.”

ANTONIO MARIA VALSALVA

A century later, Antonio Maria Valsalva (1666-1723) was born in Imola, Italy. He became notable for his description of the aortic “sinus of Valsalva, ” but he is even more famous for his Treatise on the Human Ear. His description of the Eustachian tube is classic, in which he detailed the cartilaginous, membranous, and osseous parts of the tube. He discovered, and named the dilator tubae of the tensor veli palatini muscle and made note of the insertion of some fibers of the tensor tympani into the membranous portion of the tube. He recorded his thoughts on the acoustic functions of the Eustachian tube and supported concept of drainage of purulent material from the middle ear. His observations on the function of the Eustachian tube resulted in Valsalva’s maneuver, which he used in clinical practice and which has persisted to this day. In addition to treatment of middle- ear effusion and negative pressure the maneuver is used as an inflation test for the patency of the Eustachian tube. Most likely, the maneuver was described much earlier, such as by Arab physicians of the eleventh century and some of the early Italian anatomists

JOSEPH TOYNBEE

During the nineteenth century, otologists continued the work of Eustachius. Joseph Toynbee (1815–1866) was an early English clinician who published a textbook on otologic diseases. He was a pioneer in the field of aural pathology and described a method for removing temporal bones from cadavers; he performed over 2,000 dissections of the ear. Among his contributions, he studied the muscles that open the Eustachian tube. He is credited with the eponymous test, the Toynbee test.

ADAM POLITZER

The most famous otologist of the nineteenth century was Adam Politzer (1835–1920), who is universally acknowledged as the father of modern otology. Politzer will be remembered for, among other important contributions, his method of inflating the Eustachian tube–middle ear for treatment of middle- ear diseases. Related to the role of the Eustachian tube in the pathogenesis of middle-ear effusion, his hydrops ex vacuo theory is still considered to be a valid explanation. He wrote: “It is beyond doubt that sometimes in excessive swelling of the tubal mucous membrane and impermeability of the Eustachian tube there occurs in consequence of the of consecutive rarefaction of the air in the tympanum, a transudation of serous fluid”. His method politzerization was recommended to restore middle-ear pressure not only was this Viennese clinician a pioneer in otology, but Professor Politzer was also a scholar who could speak many languages. Related to the Eustachian tube, he translated ancient Egyptian writings that may have been some of the earliest descriptions of the auditory tube. In one such text, the following was stated: “Man has two vessel-strands leading to the right ear, filled with Pneuma, ‘BREATH OF LIFE’; two similar strands leading to the left ear conduct the ‘BREATH OF DEATH’ However, Politzer made no conclusion from these passages that the Egyptians preceded Eustachius in describing the auditory tube. Adam Politzer remains a giant among clinicians and investigators who have helped us understand the role that the Eustachian tube plays in middle-ear disease.

THE PIONEERS

Arnold Rice Rich

During the first half of the twentieth century, Arnold Rice Rich, a professor of pathology at Johns Hopkins Medical School, distinguished himself by performing elegant physiologic experiments in which he assessed the function of the muscles of the Eustachian tube and surrounding structures, such as the tensor veli palatini, tensor tympani, levator veli palatini, and salpingopharyngeus. He was the first investigator to attribute the sole active dilator function of the Eustachian tube to contraction of the tensor veli palatini muscle.

Henry B. Perlman: In the mid portion of the twentieth century, Henry B. Perlman, a professor of otolaryngology at the University of Chicago, made notable contributions to the understanding of the patulous Eustachian tube, which could be caused by denervation of the tensor veli palatini muscle.

7 REVIEW OF LITERATURE

Honjo and colleagues examined 131 years of 83 patients 10-20 years of age and found that sinusitis was associated with 49% of refractory otitis media cases.
Nakano y and soto y have correlated the size of the mastoid air cell system with prognosis in otitis media with effusion in children and found it to be inversely related.
Stoikes and dutton found that post operatively patients who had undergone endoscopic sinus surgery had relief of their otological symptons related to eustachian tube dysfunction.
Cinnamunu, sade j developed a model to evaluate how pressure homeostasis of the middle ear can be maintained. They found that the worst model for adapting to these changes was a middle ear space with a small mastoid
Politzer more than 100 years ago suggested that abnormal function of the eustachian tubeis the most important factor in the pathogenesis of middle ear disease.
Sade suggested that the retraction of the pars tensa serves as a buffering agent of middle ear negative pressures.
Bayramoglu et al stressed on the small mastoid cellular system allowing for less efficient gas exchange between the middle ear cleft and micro circulation of the mucosa.
Klein jo and bluestone elaborated on the unphysiological pressures that can develop in the nasopharynx and adversely affect the middle ear and eustachian tube when the nose and nasopharynx have inflammation or obstruction.
Bluestone et al have elucidated on the inflammative swelling of the eustachian tube mucosa in allergic individuals and subsequent eustachian tube dysfunction development.
Shure et al have stated the use of visually inspecting the tympanic membrane as one of the simplest and oldest ways to assess how the eustachian tube functions and provides a presumptive evidence of eustachian tube dysfunction.
Jerger has elaborated in the use of an immitance instrument to obtain a tympanogram as an excellent way of determining the middle ear system and in assessing eustachian tube function.
Elner et al reported that 86% of the otologically normal adults could perform the valsalva maneuver while 79% perform the toynbee.
Holmquist studied eustachian tube function in adults before and after tympanoplasty and reported that the operation had high rate of success in good eustachian tube function.
Palva and karja studied the eustachian tube patency in chronic ears and found no differences between the ears that failed tympanoplasty and those that succeeded.

8 ANATOMY OF THE EUSTACHIAN TUBE

In the adult, the length of the Eustachian tube has been reported to be as short as 30 mm and as long as 40 mm, but the usual range of length reported in the literature is 31 to38 mm. 27, 36–40 The study by Sudo and colleagues, in which they constructed a wire frame model from their temporal bone histologic specimens, found the average length of the cartilaginous, junctional, and osseous portions from the temporal bone specimens to be 23. 6, 3.0, and 6. 4 mm, respectively, for a total length of 33 mm. 1 It forms a 42 +/- 9–degree angle with a parasagittal plane through the medial pterygoid plate. The Eustachian tube in the adult begins in the nasopharynx and passes posteriorly and laterally through the petrous temporal bone. The tube does not take a straight course from the nasopharynx to the middle ear but rather a slowly curving inverted S course. Speilberg found that in adults the tube makes two curves from the tympanic cavity, arching downward and forward across the space between the anterior canal wall and the bony external auditory meatus in the condyle of the mandible. Before the pharyngeal orifice, it makes another slight curve downward and forward. Additional observations Support Speilberg's observations.

The nasopharyngeal end of the Eustachian tube lies about20 mm above the plane of the hard palate. The cartilage protrudes into the nasopharynx; this protrusion is known as the torus tubarius A thick layer of epithelium continuous with the soft tissue lining of the nasopharynx covers it. An observer viewing the nasopharynx endoscopically cannot see the torus tubarius but can see the mucosa overlying the cartilage. Any inferences drawn from observing motion in this area must consider this point of the anatomy.

The lumen is lined with PSEUDOSTRATIFIED COLUMNAR EPITHELIUM of the CILIATED type, which sweeps material from the middle ear to the nasopharynx. The mucosa is continuous with the lining of the tympanic cavity at its distal end, as it is with the nasopharynx at its proximal end. Associated with these ciliated epithelial cells are goblet cells that comprise about 20% of the cell population. Tos and Bak-Pedersen studied temporal bones from premature and newborn infants, children, and adults who were free of signs of otitis media and made 30, 000 to 60, 000 counts of goblet cells in different portions of the Eustachian tube, for example, pharyngeal to tympanic ends and the lateral, medial, floor, and roof of the mucosa of the lumen. They found very low densities in all parts of the tube in premature infants, increasing in the pharyngeal portions gradually through childhood and attaining a very high density in the adult. A similar density was reported between the lateral and medial walls, but it was lower in the roof and higher in the floor of the tube.

The fat tissue located in the inferolateralportion of the Eustachian tube, termed OSTMANN'S FAT PAD, most likely aids in closing the tube. As described above, after birth, the fat pad increases in volume, primarily in height (in association with an increase in the tubal height) but not in width, thus making a greater mass in the child Takasaki and colleagues assessed temporal bones from individuals 3 months to 88 years and reported that contraction of the tensorveli palatini muscle moves the lateral lamina of the Eustachian tube cartilage in an inferolateral direction to the superior aspect of the tubal lumen and that Ostmann's fatty tissue will limit the opening of the tubal lumen, especially in the inferior aspect Orita and colleagues, from studies of 25 temporal bone specimens(33 gestational weeks to 38 years), reported differences in the location of the fatty tissue between infants and children versus adults; there was more tissue at the narrowest portion of the tube in adults compared with infants and young children, which they postulated may be related to better protective function.

There are four muscles associated with the Eustachian tube: the TENSOR VELI PALATINI, the TENSOR TYMPANI, the LEVATOR VELI PALATINI, and the SALPINGOPHARYNGEUS. Each at one time or another has been directly or indirectly implicated in tubal function.

At rest, the Eustachian tube is passively closed. It opens during such actions as swallowing, yawning, or sneezing, thereby permitting the equalization of middle ear and atmospheric pressures. Most anatomic and physiologic evidence supports active dilation induced either solely by the tensor veli palatini muscle with assistance from the levator veli palatini muscle. Closure of the tube has been attributed to passive reapproximation of tubal walls by extrinsic forces exerted by the surrounding deformed tissues, by the recoil of elastic fibers within the tubal cartilage, or by both mechanisms. Other experimental and clinical data suggest that, at least for certain abnormal populations, the closely applied internal pterygoid muscle may assist tubal closure by an increase in its mass within the pterygoid fossa; this increase applies medial pressure to the tensor veli palatini muscle and consequently to the lateral membranous wall of the Eustachian tube. The closely applied internal pterygoid muscle may assist tubal closure by an increase in its mass within the pterygoid fossa; this increase applies medial pressure to the tensor veli palatini muscle and consequently to the lateral membranous wall of the Eustachian tube.

Five arteries constitute the blood supply to the Eustachian tube: the ascending palatine artery, the pharyngeal branch of the internal maxillary artery, the artery of the pterygoid canal, the ascending pharyngeal artery, and the middle meningeal artery. The venous drainage is via the pterygoid venous plexus.

An extensive lymph network is maintained in the tunica propria of the submucosa of the Eustachian tube and is more abundant in the cartilaginous portion than in the osseous portion. This network drains into either the retropharyngeal nodes medially or the deep cervical nodes laterally. Early investigators, such as Gerlach, described a lymphoid mass within the tube of a6-month-old infant. However, Wolff, in an examination of 250 subjects, and Aschan, in a histologic study of 39 Eustachian tubes, failed to find such a structure. Further, in a study of the developmental anatomy of the tubal system, Rood and Doyle failed to find this lymphoid mass and concluded, like Wolff and Aschan that this tubal tonsil was a rare pathologic abnormality.

THE MIDDLE EAR

The middle ear is an irregular, laterally compressed, gas-filled space lying within the petrous portion of the temporal bone between the external auditory canal and the inner ear. This cavity can be considered to be divided into three parts superoinferiorly in relation to the tympanic membrane. The middle ear in infants is smaller than in adults. The epitympanum, or attic, refers to that space lying above the superior border of the tympanic membrane. The mesotympanum lies opposite the membrane, and the Because of these dimensions, the middle ear has hypotympanum lies below the membrane. At birth, the cavity and associated structures are of adult size. The vertical and anteroposterior diameters measure about 15mm, whereas the transverse diameter measures 4 mm at the epitympanum, 2mm at the mesotympanum, and 6mm at the hypotympanum been termed a cleft, or narrow box. Superiorly, the cavity is bounded by a thin plate of bone, the tegmen tympani, which extends forward to cover the semi canal of the tensor tympani muscle and posteriorly to cover the attic, thereby isolating the middle ear from the middle cranial fossa. Anteriorly, the floor of the middle-ear cavity is raised to become continuous with that of the bony portion of the Eustachian tube. Superiorly and beneath the tegmen tympani lies the Eustachian tube by an cylindric semicanal for the tensor tympani muscle, which is separated from upwardly concave thin bony septum, the cochleariform process. This process enters the middle ear along its laterally. Superomedial margin to end just above the oval window, at which point it flares. This termination of the cochleariform process serves as a pulley about which the tendon of the tensor tympani muscle makes a right-angled turn to proceed laterally to its insertion on the muscular process of the malleus. The middle ear is bounded medially by the lateral surface of the bone covering the labyrinth of the inner ear. A bulbous, hollowed prominence formed by the outward projection of the basal turn of the cochlea occupies the position between the oval and round windows. This structure, the promontory, is crosshatched by the various branches of the tympanic plexus of nerves. The lateral wall

is formed by the tympanic membrane, the tympanic ring, and a portion of the squamous temporal bone called the scutum. The posterior border of the middle ear is demarcated by the anterior wall of the mastoid cavity, the pyramidal prominence, and is connected to the mastoid antrum by the aditus ad antrum.

MASTOID AIR CELL SYSTEM

The mastoid refers to that portion of the petrous temporal bone that lies posterior to the middle-ear cavity. In the adult, the mastoid mastoid is extended exteriorly and interiorly to form a process to which the sternocleidomastoid muscle is attached superficially. The mastoid is filled with a system of interconnected cells. The mastoid antrum is posterior and continuous with the epitympanum and is the largest of these gas-filled spaces. The antrum serves as a patent communication between the middle ear and the mastoid gas cells. In the young infant, the mastoid process is small and the degree of pneumatization is low. This small gas cell system may be related, at least in part, to the susceptibility of infants to high negative middle ear pressure and, subsequently, otitis media. Also, incomplete development of this gas cell system has been associated with frequent bouts of otitis media in infants and young children. By between 5 and 10 years of age, the process of pneumatization is, for the most part, complete.

PHYSIOLOGY OF THE EUSTACHIAN TUBE

All land-living vertebrates – reptiles, birds and mammals – have developed some form of middle ear transformer mechanism to convert airborne sound waves into pressure changes suitable for transmission within the fluids of the inner ear. Despite divergent evolution of the various orders, the middle ear mechanism has achieved a remarkable homology with tympanic membrane coupled by one or more ossicles to a mobile platform in contact with the perilymph. The middle ear is gas filled and so there is minimal frictional resistance to the movement of the ossicles, and the middle ear gas pressure is approximately equal to external pressure, which allows optimal transfer by the tympanic memrane.
The middle ear is, in effect, a gas pocket. Biological gas pockets are found in birds as respiratory air sacs and in fish as buoyancy bladders and elsewhere as in the paranasal sinuses and the pleural cavities. Some biological gas pockets are quipped with elaborate mechanisms to maintain their total pressure or even to increase it. Fish possess an organelle called the rete mirabiule, which can actively pump gas from the circulation into the buoyancy bladder to equilibrate its pressure with that of the surrounding water.
All biological gas pockets face two special problems: the need to overcome shrinkage or reduced pressure because of a net loss of gases into the surrounding circulation, and the need to keep the inside of the sac clean. In the middle ear these two problems have been overcome by ‘gas inhalation’ or ventilation through the Eustachian tube and by a mucociliary transport mechanism to clear mucus and debris from the middle ear towards the nasopharynx.
In the middle ear lumen the gases ‘strive’ to reach equilibrium with their corresponding gas pressures in the surrounding capillaries. The middle ear oxygen, carbon dioxide and water vapour are in equilibrium with the blood leaving the middle ear, as they move relatively quickly across the various barriers. Assuming that the venous blood draining the middle ear has a gas composition similar to the of mixed venous blood, then a steady state situation would finally evolve in which the total middle ear gas pressure would be lower than that of the atmosphere by about 56 mmHg (760–704 = 56). This would result in a large pressure gradient across the tympanic membrane, which would compromise its impedance matching properties. For the middle ear to reach a total pressure equal to atmospheric, some compensation is needed and this is provided by the periodic supply of gas from the nasopharynx through the Eustachian tube.
Not enough is known about the qualitative and quantitative physiological variables that govern gas transfer from the nasopharynx through the Eustachian tube into the middle ear. These are important if

we wish to identify and compare ventilation in healthy and diseased ears. The equivalent of ventilation in the lung (tidal volume X respiratory rate) for the middle ear is the ventilation volume multiplied by the number of gas admissions per unit of time. The human Eustachian tube is 3-4 cm long and can be thought of as two cone-like structures fused together by a narrow ring, the isthmus.
The medial cone-like structure that joins the nasopharynx is collapsible and, indeed, is collapsed most of the time. This section can be thought of as the 'Eustachian tube bellows'. The lateral cone that joins the middle ear is bony and rigid and is an extension of the middle ear itself. The 'bellows' and the lateral cone meet at the isthmus, which is a 1-2 mm long segment of about 0.6-1.2 mm diameter. The bellows are actively opened by the tensor palati muscle during swallowing, yawning of movements of the jaw. The bellows open for nearly 0.2 seconds once every 1-2 minutes, that is for a total of about 3-4 minutes per 24 hours – a rather short time. In general, gas flows from one place to another in accordance with the total pressure difference existing between the two places. This is in contrast to the diffusion of individual gases within a mixture, which move according to partial pressure differences between one place and another. When passing through the Eustachian tube, gas flow is hampered by the isthmus. The Eustachian tube is therefore not so much a tube as a bellows at the end of which is the narrow isthmus. Once gas passes the isthmus it is in the middle ear. The structure of the Eustachian tube is such that, normally, the mucociliary clearance and ventilation functions do not interfere with one another. Mucus streams along the floor of the tube and, indeed, only the floor is paved with ciliated cells. Air flows about it, provided the Eustichian tube is not blocked with mucus. The amount of gas that will pass through the isthmus is a function of the pressure difference between the nasopharynx and the middle ear, the time the isthmus is opened and the length and diameter of the isthmus.
The negative pressure difference created between the middle ear and the nasopharynx by the loss of 1-2µl per 1-2 minutes depends of the size of the middle ear cavity, which in turn depends on the volume of the mastoid cavity. In a normal cellular mastoid, which has been estimated to have an average volume of about 12 ml, the negative pressure difference developed when 1-2 ml, the negative pressure difference developed when 1-2µl gas are lost is about 1-2 mm H_2O. This pressure difference is so small that it is questionable whether gas can pass passively through the 1 mm diameter opening of the 1-2 mm long isthmus during 0.2 seconds. The volume of the narrow hole that constitutes the isthmus is also about 1-3µl. Consideration should therefore be given to the possibility that air normally passes through the Eustachian tube not passively down a pressure gradient, but by some active mechanism.

The mastoid as a physiological buffer for the middle ear

The effect of a gas balance deficit in respect of the total pressure of an air pocket such as the middle ear depends on the amount lost in relation to the initial size of the gas pocket. Although the human middle ear proper gas volume is about 0.5 ml, the volume of the entire middle ear cleft differs greatly among individuals as it includes mastoid cavities of variable volume. The mastoid pneumatic system varies from about 1 ml to 30µl. Therefore, a given gas deficit (or excess) will manifest itself differently in well pneumatized and in poorly pneumatized mastoids.
A 20 µl volume change will result in a 100 mm H2O pressure change in a 2µl middle ear cleft, but will exert only a 10 mm H2O pressure change in a 20µl middle ear cleft. The pressure difference may be pathological in the small volume middle ear but non-significant in the other. It is not surprising, therefore, that most ears afflicted with the 'chronic ear syndrome 'have a hypopneumatized, acellular or sclerotic mastoid. Ears with well pneumatized mastoids rarely exhibit any of the 'chronic otitis media' conditions and probably rarely develop a negative middle ear pressure. The explanation for the protective effect of an adequately pneumatized mastoid is quite simple: its volume serves as a pressure buffer.
Hellstrom and Stenfors showed that when pressure was exerted in the bulla of rats the pars flaccida retracted or ballooned to a degree that was proportional to the pressure exerted in the bulla. The

correlation between the size of the mastoid pneumatization and middle ear pressure was also nicely demonstrated when detailed observations showed that well pneumatized mastoids are associated with a pars flaccida that is not usually retracted, whereas a retracted pars flaccida was usually associated with a scerotic mastoid. This suggested the association of a non-pneumatized mastoid system with a negative middle ear pressure.

The effect of mastoid pneumatization on middle ear total pressure changes has also been demonstrated during anaesthesia with nitrous oxide. This gas diffuses quickly into the middle ear, and it is found that the pressure in human middle ears increases more rapidly and attains higher values in those with small mastoids than in those with large ones. The observation shows that extensive pneumatization could buffer pressure changes, whereas small mastoid air volumes could not.

Mastoid cavities develop only after birth and grow almost to reach their adult size by the age of 5 years. Their final size is achieved when the skeleton matures, that is, between 15 and 20 years of age. Infants who lack adequate mastoid pneumatization do seem to suffer from acute suppurative otitis media and chronic secretory otitis media much more frequently than adults, although as the mastoid volume increases they ‘grow out’ of this problem. The reason some children suffer for a longer time than others probably depends on a slow rate of the development of the air-cell system. Ears with recurrent acute suppurative otitis media and chronic secretory otitis media can be shown radiologically to have delayed mastoid pneumatization. Histologically the primitive mesenchyme, which in development fills the middle ear and mastoid, seems to linger for longer in the mastoids of those ears with recurrent acute suppurative and secretory otitis media. In some patients mastoid pneumatization develops late and is incomplete. These individuals constitute the group most at risk of the entire chronic ear syndrome.

THE REACTION OF THE MIDDLE EAR TO NEGATIVE PRESSURES

When a negative pressure develops in the middle ear, the atmospheric pressure on the outside compresses the tympanic membranes at its weakest points, which therefore retract. The pars flaccida retracts first because of its elasticity. Its sensitivity to pressure is high and it will retract in response to even minor changes. The amount of negative pressure that is damped by retraction of the pars flaccida depends on the buffering action of the mastoid cavity. Hellstrom and Stenfors found that in rats the retracted or ballooned pars flaccida may change the middle ear volume by up to 0. 5 per cent in the face of a 50 mmH2O pressure gradient. When the pars flaccida had retracted to its maximum, a further decrease in middle ear pressure caused the relatively non-flexible pars tensa to retract. If the pressure continues to decrease after the final retraction of the pars flaccida and pans tensa, the Eustachian tube may open when gas from the nasopharynx enters the middle ear and abolishes the negative pressure. This is what happens during aircraft descent, but if equilibration does not occur and the negative pressure continues to increase then, when it reaches 50-90 mm H_2O, a transudate from the blood vessels lying in the middle ear fills the cavity. This transudate by itself diminishes the middle ear volume and prevents a further decrease in its pressure. These changes constitute barotrauma.

In humans the volume displaced by total retraction of the pars flaccida is about 0. 05 ml. Total retraction will therefore damp pressure differences of about 50 mm H_2O in a small middle ear cleft whose entirevolume is 1 ml. In a normal middle ear cleft of about 10 ml, only
5 mm H_2O will be compensated by a total retraction of the pars flaccida. It seems likely that regular physiological pressure fluctuations are of the order of 5mm H_2O, and that such small pressure changes are readily equalized by retraction of the pars flaccida. If negative pressure persists, then the more rigid tympanic membrane starts to retract. This compensating mechanism may not neutralize the entire negative pressure, but usually keeps it a few mm H_2O below atmospheric, as has been measured by Buckingham and Ferrer and Sade et al. It further gas depletion continues there are several possible consequences. Damage to the collagenous skeleton of the pars tensa will convert it to an elastic membrane with properties similar to the pars flaccida. The new structure of the pars tensa will allow for further retraction and deep atelectatic pockets. It is the ear with the sclerotic mastoid that normally

succumbs to consistent negative pressures.
The volume of the middle ear may be reduced by swelling of the mucosa or engorgement of the blood vessels. Middle ear gas volume is also reduced by ‘flooding’ the middle ear space by an effusion. This is usually seen in acute and chronic secretory otitis media, but also in 20 percent of atelectatic middle ears. Under these conditions the middle ear cavity can be partly or even entirely filled with an effusion, leaving little or no gas pocket free to develop a negative pressure. Indeed, at times so much exudate is formed that the pressure becomes positive – as seen when the exudate spills out through the edge of a myringotomy.
Thus, although atelectatic ears and those with a secretory otitis media may develop significant negative pressure during their formation, reduction of the middle ear volume in one way or another leaves a pressure that is only a few mm H2O below that of the atmosphere.
The middle ear exudate in acute and chronic secretory otitits media can be viewed as a mechanism protecting the tympanic membrane from prolonged negative pressure. The price of this protection is a relatively small conductive hearing loss.
A prolonged and uncompensated negative pressure can damage middle ear structures. Changes in the collagenous layer of the tympanic membrane have been mentioned already, but the ossicular chain, especially the long process of the incus, can also disintegrate.

9 SURGICAL ANATOMY

The introduction of functional endoscopic sinus surgery (FESS) by Messerklinger and Wigand radically changed the way otolaryngologists treat sinusitis. For safe surgery, a clear understanding of sinus surgical anatomy is fundamental. This article takes the reader through endoscopic sinus surgery, emphasizes the relevant surgical anatomy, and briefly discusses pertinent anatomical variations.

ETHMOID SINUS

Of all the paranasal sinuses, the ethmoid sinus is the most complex and is aptly referred to as a labyrinth. The anterior cells of this sinus first appear in the third fetal month as pits of the lateral nasal wall adjacent to the middle meatus. At birth the anterior ethmoid measures 2 x 2 x 5 mm, and the posterior ethmoid measures 2 x 4 x 5 mm; they are difficult to view with routine radiography. The ethmoids attain adult size by the twelfth year, expanding into adjacent areas beyond its capsule.

In the adult, the ethmoids are pyramidal in shape, with the base located posteriorly. They measure 4 cm to 5 cm anterior to posterior, 2.5 cm in height, 0.5 cm wide anteriorly, and 1.5 cm wide posteriorly. The roof of the sinus, the fovea ethmoidalis, usually extends 2 to 3 mm above the more medial cribriform plate.

The entire ethmoid bone is a paired scaffolding held together by two lamina cribrosa. Each lamina cribrosa is separated in the midline by an upward projection, the crista galli. The ethmoid labyrinth forms the greater portion of the ethmoid bone.

Laterally, the lamina papyracea forms the bony division from the orbit. Any dehiscence of this lamina forms a potential pathway for spread of sinus infections into the orbit. Medially, it is bounded by the middle, superior, and if present supreme turbinates. Posteriorly and inferiorly, the ethmoid clefts open into the corresponding nasal passages and finally into the choanae. The anterior roof is bounded by the

downward extension of the frontal bone.

The complex ethmoid labyrinth is divided into a series of lamellae, which are relatively constant and can help guide the surgeon during the surgery. These lamellae are the uncinate process, the bulla ethmoidalis, the ground lamella of the middle turbinate, and the lamella of the superior turbinate. Rarely, there is a fifth lamella known as the supreme turbinate. The ground lamella of the middle turbinate is a constant and well - developed lamella. It divides the ethmoid into anterior and posterior portions. The anterior cells are smaller and more numerous than the posterior cells.

ANTERIOR ETHMOID

Most sinus disease involves the anterior ethmoids. Several important anatomic landmarks are associated with this structure.

UNCINATE PROCESS

The uncinate process is a thin and almost sagittally oriented bone. It runs in a sickle - shaped curve from anterosuperior to posteroinferior. Its anatomy is better appreciated by medializing the middle turbinate. It is approximately 3 to 4 mm wide and 1.5 to 2 cm in length. Its posterior margin is sharp and concave. It is anterior and parallel to the anterior surface of the ethmoid bulla. The hiatus semilunaris occupies the space between the posterior aspect of the uncinate and the anterior surface of the ethmoid bulla. The lateral surface of the uncinate forms the medial surface of the infundibulum. Posteriorly and inferiorly, the uncinate attaches to the ethmoidal process of the inferior turbinate bone. The posterior - superior attachment is to the lamina perpendicularis of the palatine bone. The ascending anterior convex margin contacts the lateral nasal wall, which may extend up to the lacrimal bone. The uppermost segment of the uncinate process runs a variable course. It is hidden by the insertion of the middle turbinate. It can extend to the base of the skull or turn laterally to insert into the lamina papyracea and may turn frontally and fuse with the insertion of the middle turbinate.

There are almost always defects between the uncinate and the inferior turbinate. These defects are covered with dense connective tissue and nasal mucosa and are known as the anterior and posterior fontanelles. Accessory maxillary ostia are often observed in the posterior fontanelle region.

The uncinate is usually oriented at 140° to the lateral nasal wall and the lamina papyracea, but the orientation can vary significantly. Extensive nasal polyps within the infundibulum can displace the

uncinate medially. It can be displaced laterally, as seen in maxillary sinus hypoplasia. Rarely, the uncinate can be pneumatized. Orbital injury can occur if lateral displacement of the uncinate (with accompanying atelectasis of the infundibulum) is not appreciated during the infundibulotomy.

ETHMOID BULLA

The ethmoid bulla is one of the largest and most consistent anterior ethmoid air cells. It is created by the pneumatization of the bulla lamella. Occasionally, the ethmoid bulla is poorly developed or absent. It is located in the middle meatus, directly behind the uncinate process, and in front of the ground lamella of the middle turbinate. Superiorly, the anterior wall of the ethmoid bulla can extend to the skull base to form the posterior limit of the frontal recess. If this division is absent, there is direct communication between the frontal recess and the sinus lateralis, normally located above the bulla. Posteriorly, the bulla can fuse with the ground lamella.

Variations include a highly pneumatized bulla lying in the lower aspect of the middle meatus. In this position, the ethmoid bulla can narrow the infundibulum. If the ethmoid bulla is not pneumatized, a bony projection known as the torus lateralis forms from the lamina papyracea.

HIATUS SEMILUNARIS

The hiatus semilunaris is a sagittal cleft between the posterior border of the uncinate process and the anterior surface of the ethmoid bulla. The middle meatus communicates with the infundibulum through this area, designated as the hiatus semilunaris inferior. The hiatus semiulnaris superior is the cleft formed between the posterior wall of the ethmoid bulla and the ground lamellae of the middle turbinate.

ETHMOID INFUNDIBULUM

The ethmoid infundibulum (often simply called the infundibulum) is a funnel - shaped, three - dimensional space located in the anterior ethmoidal region. It is bounded medially by the uncinate and laterally by the lamina papyracea. The frontal process of the maxilla and lacrimal bone forms its anterior - superior

boundary. The lumen of the infundibulum appears as a V-shaped structure in axial CT. The posterior border of the infundibulum is bounded by a portion of the anterior ethmoid bulla and tapers in this direction. The ethmoid infundibulum can measure 4 cm in length and 5 mm to 12 mm in depth. The ethmoidal infundibulum communicates with the middle meatus through the hiatus semilunaris.

Superiorly the infundibulum is closely related to the frontal recess, which depends greatly on the attachment of the uncinate process. The uncinate usually bends laterally to attach to the lamina papyracea; in this case, the infundibulum and frontal recess are separated so the frontal recess opens into the middle meatus medial to the ethmoidal infundibulum between the uncinate process and the middle turbinate. Less commonly, the uncinate can attach to the ethmoid roof or insert into the middle turbinate; in this case, the frontal recess opens directly into the infundibulum.

OSTIOMEATAL UNIT

Naumann described this functional unit. The term osteomeatal unit refers collectively to the uncinate process, infundibulum, and anterior ethmoid cells. This area also contains the ostia of the anterior ethmoid, maxillary, and frontal sinuses.

FRONTAL RECESS AND SINUS

Frontal sinus development begins in the fourth fetal month. Development is usually completed before 20 years of age. The frontal sinus originates from the anterosuperior pneumatization of the frontal recess into the frontal bone.

The adult sinus measures 28 mm in height, 24 mm in width, and 20mm in depth. It drains into the ostiomeatal unit through the nasofrontal duct. The frontal sinus is shaped like a funnel with its narrow end toward the duct ostium. The frontal recess lies inferior to the nasofrontal duct ostium. This recess is bounded by the middle turbinate medially, the lamina papyracea laterally, the agger nasi anteriorly, and the ethmoid bulla posteriorly. The frontal ostium is usually found in the most anterosuperior part of the frontal

recess.

The ground lamella of the ethmoidal bulla is an important structure in relation to the frontal recess. It separates the frontal recess from the lateral sinus if the bulla lamella ascends to the roof of the ethmoid. Frequently the bulla lamella is incomplete, in which case the frontal recess may communicate posteriorly with the lateral sinus. Depending on the position of the uncinate process, the frontal recess may open into the middle meatus medial to the uncinate process or directly into the ethmoidal infundibulum.

There is significant variation in regard to the nasofrontal duct. This variation is better understood when one takes the agger nasi cell, frontal cells, and supraorbital ethmoid cells into account. Secretions from frontal sinus enter the middle meatus by passing posterior and medial to the agger nasi cell. If agger nasi cells are extensively pneumatized, the frontal recess can be narrowed; this narrowing can predispose a patient to frontal sinusitis with minimal mucosal derangement. Cells may develop in the frontal bone near the frontal sinus, known as the bulla frontalis. These cells open into the frontal recess. Supraorbital ethmoid cells are another anatomic variation caused by pneumatization of the frontal bone by ethmoid air cells. Stammberger believes these supraorbital cells develop as an extension of the posterior frontal recess.

AGGER NASI

The term agger nasi means nasal eminence. This area if found just anterior to the middle turbinate's insertion into the lateral nasal wall. When the agger nasi is pneumatized by an anterior ethmoid cell, it forms an agger nasi cell. It arises from the superior aspect of the infundibulum of the frontal recess. it is bounded anteriorly by the frontal process of the maxilla, anterolateral by the nasal bones, superiorly by the frontal recess, inferomedially by the uncinate process, and inferolaterally by the lacrimal bone. The superior aspect of the cell serves mainly as the anterior border of the frontal recess and to a lesser degree as the anteromedial floor of the frontal sinus.

POSTERIOR ETHMOID

The posterior ethmoid sinus is a collection of one to five cells that drain into the superior and supreme meati. It is bounded anteriorly by the ground lamella of the middle turbinate, posteriorly by the anterior wall of the sphenoid sinus, laterally by the lamina papyracea, medially by the superior and supreme turbinates, and superiorly by the ethmoid roof. The meati are located along the medial surface.

The behaviour of the most posterior cells of the posterior ethmoidal sinus is of great importance to the surgeon. Onodi found that when the most posterior cell is highly pneumatized, it can extend posteriorly along the lamina papyracea into the anterior wall of the sphenoid sinus. In this case, the optic nerve may be adjacent to the posterior ethmoid cell. To avoid injury, dissection should be medial and inferior. The internal carotid artery may also impinge the lateral wall of the posterior ethmoidal cells. Yellow orbital fat may be seen through the lamina papyracea in some cases.

MAXILLARY SINUS

At birth, the maxillary sinus has a volume of 6 mL to 8 mL. In the adult, it is triangular in shape. it measure 25 mm along the anterior limb of its base, 34 mm deep, and 33 mm high. The floor of the sinus will be usually 4 mm to 5mm below the floor of the nose in the adult. The sinus is bounded superiorly by the orbital roof. The hard palate, alveolus, and dental portion of the maxilla comprise the inferior boundary. The zygomatic process forms the lateral boundary. A thin plate of bone separates the sinus from the infratemporal and pterygopalatine fossae posteriorly. The uncinate process and inferior turbinate form the medial boundary.

The natural ostium is located in the superior aspect of the medial wall of the sinus and drains into the hiatus semilunaris; it is an elliptical structure measuring between 1 mm to 20 mm in diameter. The ostium is seen behind the lower attachment of the uncinate process and above the superior portion of the anterior superior aspect of the inferior turbinate.

Infraorbital ethmoid cells, also known as Haller cells, are the most common anatomic variation. They arise from the anterior ethmoid in 88% of individuals or from the posterior ethmoid in 12%. These cells develop into the floor of the orbit above the natural ostium of the maxillary sinus. They are easily seen on coronal CT. When a Haller cell becomes diseased, the natural ostium of the maxillary sinus may become obstructed.

Other variations may occur. Another variation is hypoplasia or atelectasis of the maxillary sinus. Uncinectomy is difficult in these cases because of the risk of orbital injury from lateral displacement of the uncinate. Accessory maxillary sinus ostia are another common variation. They can occur in 20% to 50% of patients. These are located in the infundibulum or the membranous meatus inferior to the uncinate but above the insertion of the inferior turbinate.

SPHENOID SINUS

There is minimal development of the sphenoid sinus until 3 years of age. After this period, the sphenoid sinus begins to pneumatize the sphenoid bone. Sometimes these sinuses are highly pneumatized to form prominent lateral recesses. The sphenoid sinus develops into adulthood to measure 20mm.

The right and left sphenoid sinuses are separated by the intersinus septum. Occasionally this septum may be asymmetric. It frequently deviates laterally and superiorly, inserting into the bony prominence over the optic nerve or internal carotid artery. Thus, sphenoid septum manipulation must be done with caution to avoid visual or hemorrhagic complications.

The ostia of the sphenoid sinus are usually located in the sphenoethmoidal recess, medial to the superior turbinate. The ostia may be slitlike, oval, or round in shape. The average distance from anterior nasal spine to the sphenoid ostium is 7 cm.

Image - guidance systems are helpful to confirm the location of the sphenoid during surgery.

Other anatomic relationships are important for sphenoid surgery. The vidian nerve passes along the floor of the sphenoid. On the lateral wall, two bulges may be produced by the optic nerve and carotid artery; these bulges may be covered only by thin bone and in some cases are dehiscent. The anterior wall of the sphenoid is not always located immediately posterior to the most inferior posterior ethmoid; the inferior aspect of an Onodi cell may be 1. 5 cm superior to the anterior wall of the sphenoid sinus. When entering the sinus, the surgeon should work in an inferior and medial direction to avoid injuring the lateral wall of the sinus. The sinus mucosa is typically left in situ to avoid injuring a dehiscent internal carotid artery.

10 SURGICAL PHYSIOLOGY

The physiological role of the paranasal sinuses is uncertain. They are a continuation of the respiratory cavity and are covered by respiratory mucosa. They share certain features with the nose but the responses are much less marked on account of relatively poorly developed vasculature and nerve supply. In man they assume importance in the diseased state.

The development of the sinuses takes upto 25 years, the ethmoids and maxillary sinuses are rudimentary at birth, the frontal sinuses develop after the age of 6 years but may be completely absent: the sphenoid sinus differs considerably in the degree of development. It holds true that whatever physiological role they play, it is not essential and of only minor importance.

MUCOSA

The mucosa runs in continuity from the nose and is respiratory in type: however, there are differences between the nose and the sinuses. Goblet cells and cilia are less numerous in sinus mucosa but increase in frequency near the ostia: the blood supply is less well developed with no cavernous plexus, which gives the mucosa a pale almost translucent appearance. As the nerve supply is less well developed. The sinus mucosa is able to give only a weak vasomotor response and increase mucus production on parasympathetic stimulation.

DRAINAGE

Mucociliary clearance in the maxillary sinus is spiral, towards the natural ostium (Toremalm, Mercke and Reimor, 1975). Drainage of the frontal and sphenoid sinuses is downwards, aided by gravity. Ciliary motion remains normal if the blood supply is adequate. If the blood supply is impaired then ciliary activity is reduced and stasis of secretions results. The secretions join the nasal mucus in the middle meatus and may contribute to both the total amount and effectiveness of the nasal mucus.

OXYGEN TENSION

The Po_2 is lower in the maxillary sinuses than in the nose and it is lower still in the frontal sinuses. The oxygen tension drops further if the ostium becomes blocked.

OSTIUM SIZE

Blockage of the natural sinus ostium results in a reduction of ventilation and stasis of secretions. If the ostium size is below 2. 5 mm. it predisposes to the development of disease. (Aust, Drettner and Hemmingsson, 1976).

PRESSURE CHANGES

The pressure in the maxillary sinus varies with respiration but lags behind by 0. 2s. There is little fluctution when the nose is patent, and the variation of pressure during quiet respirations is + / -4 mmH_2O which reaches 17 - 20 mmH_2O on exercise. Pressure fluctuations are much more marked if the nose is blocked. Barotrauma is five times less common than in the ear and is most frequently seen in the maxillary sinuses, particularly in divers.

11 PATHWAYS OF SECRETION TRANSPORT

Following the pioneering work on animal studies by Hilding senior Dr Messerklinger made the observation that the secretions of the various sinuses do not reach their respective ostia and nasopharynx by random routes, but instead follow very definite pathways which seem genetically pre determined at the lateral nasal wall two major routes of mucociliary transport can usually be identified.
The first route combines secretion from the frontal and maxillary sinuses and the anterior ethmoidal complex. These secretions usually join in or near the ethmoidal infundibulum and from there passes over the free rear margin of the uncinate process and along the medial face of the inferior turbinate towards the nasopharynx. At this point the secretions pass anteriorly and inferior to the eustachian tube orifice. Active transport continues upto the borderline of the ciliated and the squamous epithelium in the nasopharyn. From this point the secretions are moved by gravity and assisted ultimately by the swallowing mechanism.
The second route combines the secretions from the posterior ethmoid complex and the sphenoid sinus. The secretions from these two sinuses meet in the spheno ethmoid recess and are then transported towards the nasopharynx posteriorly and superiorly to the eustachian tube orifice.. **the eustachian tube is thus like a breakwater between these two pathways.**

12 CLASSIC TESTS FOR EUSTACHIAN TUBE PATENCY

Prior to the 1960s, most tests of the pressure regulation function of the Eustachian tube were, in reality, only assessments of the tubal patency. The classic methods of Valsalva, Politzer, and Toynbee for assessing the Eustachian tube are still in use today, asis catheterization of the Eustachian tube. But of these tests, the Toynbee, albeit crude, provides some insight into the patient's Eustachian tube regulatory function. These tests are traditionally used when the tympanic membrane is intact, but some are used when the eardrum is not intact, such as Valsalva's test.

VALSALVA TEST

The effect of high positive nasopharyngeal pressures at the proximal end of the Eustachian tube system can be evaluated qualitatively by Valsalva's test. The test results are considered to be positive (normal) when the Eustachian tube and middle ear can be inflated by a forced expiration (with the mouth closed and the nose held by the thumb and forefinger). The amount of overpressure thus created is variable and may be as much as 2,000 mm H_2O. When the tympanic membrane is intact, the overpressure in the middle ear can be observed as a bulging tympanic membrane by visual inspection of the tympanic membrane with a pneumatic otoscope or, more precisely, with the aid of the otomicroscope and a nonmagnifying Bruening or Siegle otoscope. The tympanic membrane moves inward when positive canal pressure is applied, but outward mobility in response to applied negative canal pressure is decreased or absent if positive pressure is present within the middle ear. Elner and colleagues found that 85% of 101 adults with normal ears had positive results on Valsalva's test.

POLITZER TEST

Politzer's test is performed by compressing one nares into which the end of a rubber tube attached to an air bag has been inserted while compressing the opposite nares with finger pressure. The subject is asked to repeat the letter K or is asked to swallow to close the velopharyngeal port). When the test result is positive, the overpressure that develops in the nasopharynx is transmitted to the middle ear, thus creating positive middle-ear pressure. Assessment of the middle-ear pressure and the significance of the test results are the same as with Valsalva's test in that a normal result indicates only tubal patency.

EUSTACHIAN TUBE CATHETERISATION

Transnasal catheterization of the Eustachian tube with the classic metal cannula has been used to assess tubal function for more than a century. Cannulation can be performed by blindly rooting for the orifice of the tube, by indirect visualization with a nasopharyngoscope, or by use of the transoral right angle telescope. Successful transferring of applied positive pressure from the proximal end of the cannula into the middle ear signifies only tubal patency. Catheterization of the Eustachian tube has been used for over 100 years to insufflate medications

TOYNBEE TEST

In performing the Toynbee test, the subject is asked to swallow when the nose is manually compressed This maneuver usually creates a positive pressure within the nasopharynx, followed by a negative pressure phase. Change in middle-ear pressure is assessed on the Toynbee test in the same way that it is assessed on Valsalva's test. If negative pressure is present within the middle ear, the tympanic

membrane will be retracted and will not move inward to applied positive pressure with the pneumatic otoscope. It will move outward to applied negative pressure if the pressure applied exceeds the negative pressure within the middle ear. The test results are usually considered positive when there is an alteration in the middle-ear pressure. Negative middle-ear pressure after the Toynbee test or only momentary negative middle-ear pressure followed by normal middle-ear gas pressure usually indicates good tubal function because it shows that the Eustachian tube can open actively (the tensor veli palatini muscle contracts) and that the tubal structure is sufficiently stiff to withstand nasopharyngeal negative pressure. In the study by Elner and colleagues, the results of the Toynbee test were positive in 79% of normal adults

TYMPANOMETRY

Determination of middle-ear pressure and acoustic immittance using electroacoustic impedance equipment were introduced by Metz about 50 years ago. These same techniques have been used to perform tympanometry, which is the measurement of the acoustic driving-point immittance as a function of the static pressure in the canal. If low-frequency tones are used for the measurement, the static pressure that produces themaximal acoustic immittance is approximately equal to the gas pressure in the middle ear. Bylander used tympanometry with a pressure chamber to evaluate Eustachian tube function in normal children. Shupak and colleagues also used tympanometry inside a pressure chamber to assess the ability of naval scuba divers to equilibrate negative middle-ear pressure. Using an immittance instrument to obtain a tympanogram is an excellent way of determining the status of the tympanic membrane–middle-ear system, and it can be helpful in assessing Eustachian tube function. A resting pressure that is highly negative is associated with some degree of Eustachian tube obstruction, but the presence of normal middle-ear pressure does not necessarily indicate normal Eustachian tube function; a normal tympanogram is obtained when the Eustachian tube is patulous. However, unlike the otoscopic evaluation, tympanometry is an objective way of determining the degree of middle-ear negative pressure.

13 MATERIALS AND METHODS

STUDY POPULATION:

Over a two year period ranging from November 2005 to November 2007 forty patients were selected from the outpatient department of upgraded institute of otorhinolaryngology, Madras Medical College. A detailed clinical history was taken regarding the duration of sinusitis. As a majority of the population present with acute signs and symptoms medical management of sinusitis was resorted to. following a 3 to 4 week course of antibiotics a computed tomography of the paranasal sinuses was done along with diagnostic nasal endoscopy with combination of zero and thirty degree rigid hopkins nasal endoscope. Otoendoscopy was performed along with eustachian tube patency tests like valsalva maneuver. for patients not able to summon a good valsalva maneuver, eustachian tube catheterisation was done along with politzerisation. pure tone audiometry, impedance audiometry and x ray mastoids(both) laws view was also taken.

INCLUSION CRITERIA

AGE - 20 – 40 years
SEX – an equal proportion of males and females
DURATION OF SINUSITIS – a period greater than 3 months
DIAGNOSTIC MODALITY – chronic sinusitis proven by a combination of clinical history, CT paranasal sinuses and diagnostic nasal endoscopies.

EXCLUSION CRITERIA

1. patients with Csom or Asom or a previous history of Csom/Asom.
2. cases of allergic rhinitis, nasal polyposis and nasal masses
3. cases with adenoidal hypertrophy and other nasopharyngeal masses like carcinomas, angiofibromas and antro choanal polyps.
4. patients with grossly deviated nasal septum.
5. patients with history suggestive of GERD.
6. patients with history of irradiation to the head and neck.
7. patients with history of surgeries like adenoidectomy, cleft palate surgery, maxillectomy, palatal resection, nasal surgeries and ear surgeries including tympanostomy tube placement.
8. pregnant woman.
9. smokers.

DIAGNOSTIC NASAL ENDOSCOPY-

A diagnostic nasal endoscopy was done on an outpatient basis with emphasis on the following points-

1. abnormalities or structural variants in the lateral nasal wall that predispose to sinusitis
2. presence of frank mucopus.
3. closing and opening of the eustachian tube orifice when the patient is asked to swallow.
4. structural changes in the eustachian tubal eminence.
5. post nasal discharge from the sinuses and its relation to the eustachian tube orifice.

OTOENDOSCOPY –

An otoendoscopic evaluation was done with use of rigid hopkins zero degree endoscope and the tympanic membrane analysed for the following features

1. apparent shortening of the handle of the malleus.
2. exaggeration of the prominence of the short process of malleus and anterior and posterior malleolar folds.
3. distortion of the cone of light reflex.
4. movement of the tympanic membrane on performing the valsalva maneuver.

EUSTACHIAN TUBE CATHETERISATION

After anaesthetesizing and decongestion of both nasal cavities using a combination of 2%xylocaine and adrenaline an eustachian tube catheter is introduced along the floor of the nasal cavity. At the post nasal space the catheter is turned 90 degrees medially and pulled forward to hitch against the posterior end of the septum. Following this the catheter is turned 180 degrees laterally to enter the eustachian tube orifice. Following this the catheter is attached to a pneumatic bulb or a politzer bag and movements of the tympanic membrane noted via an otoscope or otoendoscope upon insufflation.

CT PARANASAL SINUSES-

A computed tomography of the paranasal sinuses with both axial and coronal views were taken after 3 weeks of antibiotics to document the presence of sinusitis.

X RAY BOTH MASTOIDS LATERAL OBLIQUE VIEW-

Was taken to note the pneumatisation of the mastoid.

PURE TONE AUDIOMETRY-

Taken to note to aasess hearing and note if any elevation is present in the air conduction and or bone conduction thresholds.

IMPEDANCE AUDIOMETRY-

Taken to assess the type of curve, the mastoid volumes and the stapedial reflexes.

14 OBSERVATIONS AND RESULTS

MALE PATIENTS	FEMALE PATIENTS
20	20

NUMBER OF SUBJECTS WITH REGARDS TO DURATION

< 3 YEARS	22
> 3 YEARS	18

INCIDENCE OF EUSTACHIAN TUBE DYSFUNCTION SYMPTOMS

BLOCKED SENSATION	14 CASES	35%
POPPING SENSATION	4 CASES	10%
HEARING LOSS	1 CASE	2. 5%
AUTOPHONY	1 CASE	2. 5%
DIZZINESS	0 CASE	0%

INCIDENCE OF RETRACTED TYMPANIC MEMBRANE

GRADE 1	20 CASES	50 %
GRADE 2	15 CASES	37. 5 %
GRADE 3/4	4 CASES	10 %

DISTRIBUTION OF SINUSITIS :

ANTERIOR OMC	22 CASES	55 %
POSTERIOR OMC	3 CASES	7. 5 %
PANSINUSITIS	15 CASES	37. 5 %

CELLULARITY OF MASTOIDS:

PNEUMATISED	30 CASES	75%

SCLEROSED	10 CASES	25 %

TYMPANOMETRY CURVES:

TYPE A	25 CASES	62. 5 %
TYPE B	1 CASE	2. 5 %
TYPE C	14 CASES	35 %

STAPEDIAL REFLEXES:

STAPEDIAL REFLEX PRESENT	39 CASES	97. 5 %
STAPEDIAL REFLEX ABSENT	1 CASE	2. 5 %

MIDDLE EAR VOLUMES:

NORMAL	37 CASES	92. 5 %
HIGH	0 CASES	0 %
LOW	3 CASES	7. 5 %

RESULTS OF PURE TONE AUDIOMETRY:

NORMAL HEARING	38 CASES	95 %
CONDUCTIVE LOSS	2 CASES	5 %
SENSORINEURAL LOSS	0 CASES	0 %

THRESHOLD ELEVATION AMONG PATIENTS WITH NORMAL HEARING

AIR CONDUCTION	25 CASES	65 %
BONE CONDUCTION	0 CASES	0 %

LOCATION OF SINUSITIS IN RELATION TO TYPE C CURVES

LOCATION	NUMBER	C CURVES	PERCENTAGE
ANTERIOR OMC	22 CASES	4	18.18%
POSTERIOR OMC	3 CASES	1	33.3%
PAN SINUSITIS	15 CASES	9	60%

LOCATION OF SINUSITIS WITH RELATION TO THRESHOLD ELEVATION

LOCATION	NUMBER	THRESHOLD ELEVATION	PERCENTAGE
ANTERIOR OMC	22	12	54.5%
POSTERIOR OMC	3	2	66.6%
PAN SINUSITIS	15	11	73.3 %

PERIOD OF SINUSITIS IN RELATION TO C CURVES :

< 3 YEARS	22 CASES	5 TYPE C CURVES	22.7%
> 3 YEARS	18 CASES	9 TYPE C CURVES	50%

MASTOID CELLULARITY WITH RELATION TO TYPE C CURVES:

CELLULARITY	NUMBER	TYPE C CURVES	PERCENTAGE
PNEUMATISED	30 CASES	8 CASES	26.6%
SCLEROSED	10 CASES	6 CASES	60%

VALSALVA POSITIVE	25 CASES	62.5 %
VALSALVA NEGATIVE	15 CASES	37.5 %

EUSTACHIAN TUBE CATHETERISATION(N = 15 CASES)

POSITIVE	6	40 %
NEGATIVE	9	60 %

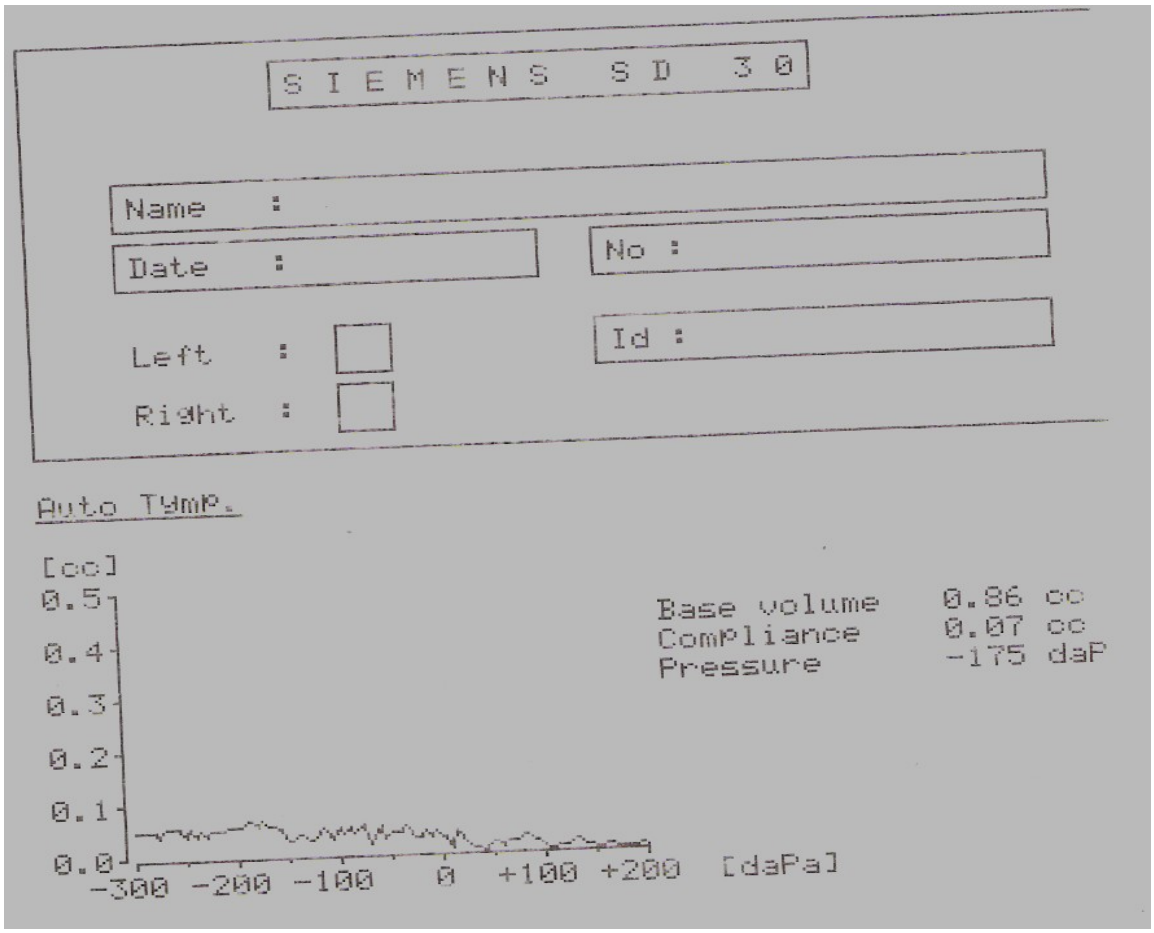
15 DISCUSSION

Of The Forty Patients screened for sinusitis a majority (55%) had disease confined to the anterior osteomeatal complex which includes the maxillary sinus, the frontal sinusitis and the anterior ethmoids followed closely by the sinusitis involving all the sinuses. the least incidence involved sinusitis changes of only the posterior osteomeatal complex comprising the sphenoid and the posterior ethmoid complex.
The major complaint relating to an Eustachian tube pathology was a fullness of the ears (35%) followed by a popping sound experienced by 10% of the patients. However 50% of the cases experienced no symptoms pertaining to Eustachian tube dysfunction.
About 50% of the patients had grade 1 retraction as defined by Sade et al followed by 37.5 having a grade 2 retraction and only about 10% having a grade 3 / grade 4 retraction. A single patient had a bulging tympanic membrane with air bubbles with features suggestive of secretory otitis media.
Impedance audiometry was taken and three parameters assessed which included jerger curves, middle ear volumes and stapedial reflexes.
Impedance curves were classified according to jerger type 1, type 2 and type 3 curves. In our study a majority of cases displayed type A curve (62.5%) while 35% displayed a type C curve indicative of high negative middle ear pressures indicative of a probable Eustachian tube dysfunction. A single patient had a type B curve indicative of an effusive process in the middle ear. huang xk and zhan ys in a similar study had an abnormal tympanogram in 42% of their cases.
Mastoid ear volumes were found to be within the normal range in 92.5% of the cases and low volumes were found in 7.5 of the cases probably reflecting the fact that mastoid ear volumes are pre determined in the first few years of life when the middle ear cleft is developing and sinusitis in adult life may not have an impact on the mastioid ear volumes.
Stapedial reflexes both ipsilateral and contralateral were measured and found normal in 97.5% of the cases and absent in one case (2.5%). the one case with absent reflex had type. As curve with moderate conductive hearing loss. This finding illustrates that the hearing loss in a pure sinusitis case is not severe enough to cause any changes in stapedial reflex patterns.
Pure tone audiometry was taken or all the patients. The predominant finding was of normal hearing (95%) followed by 5% (2cases) with conductive hearing loss and zero cases of sensorineural hearing loss in the speech frequencies of 500-2000hz. Among the cases with normal hearing a significantly high proportion (65%) had elevated air conduction thresholds implying a silent middle ear dysfunction however no elevation in bone conduction thresholds are noted.
The location of sinusitis from the study appears to play a vital role in tympanometric and pure tone audiometric findings cases of pan sinusitis and posterior osteomeatal complex sinusitis consume a majority of the proportion of type C curves and a majority of hearing threshold elevations reflecting the fact that mucopus secretions from the infected sinuses especially from the posterior group traverse the Eustachian tube ostia there by causing a long standing inflammation, mucosal swelling, patency disturbance and there by dysfunction.
The period of sinusitis also seems to play a significant role with a long course of the disease in the para nasal sinuses contributing to deterioration of middle ear function.
60% of the sclerosed mastoids had type C curves on impedence audiometry while 26.6% of pneumatised mastoids had type C curves, implying poorly pneumatised mastoids cope poorly with the negative pressures generated in Eustachian tube dysfunctions.
Of the 15 cases negative with the valsalva maneuver Eustachian tube catheterization was positive in 6 cases and negative in 9 cases reflecting that the 6 cases may not have had an adequate effort and the nine cases had a block in their Eustachian tubes.

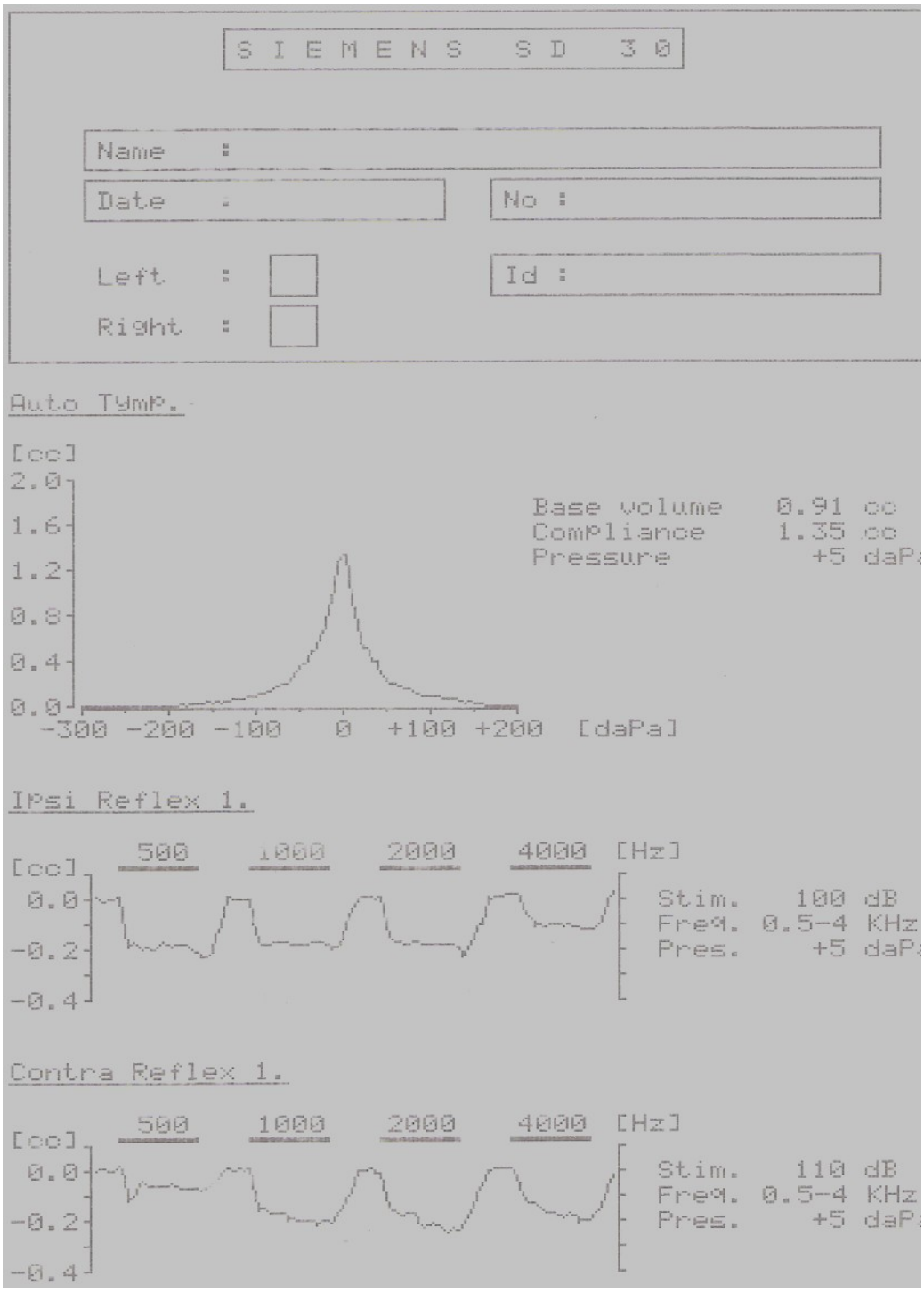
16 CONCLUSION

- The damage of middle ear function becomes more serious with the extent of lesion and with the longer course of the disease in the para nasal disease.
- Though hearing was unaffected in the study group a significantly high (65%) had elevated air conduction thresholds.
- Sinusitis involving the posterior groups were more likely to cause middle ear dysfunction.
- Middle ear volumes largely remained normal in the study groups.
- Patients with sclerosed mastoids are more likely to adjust poorly with negative pressures.

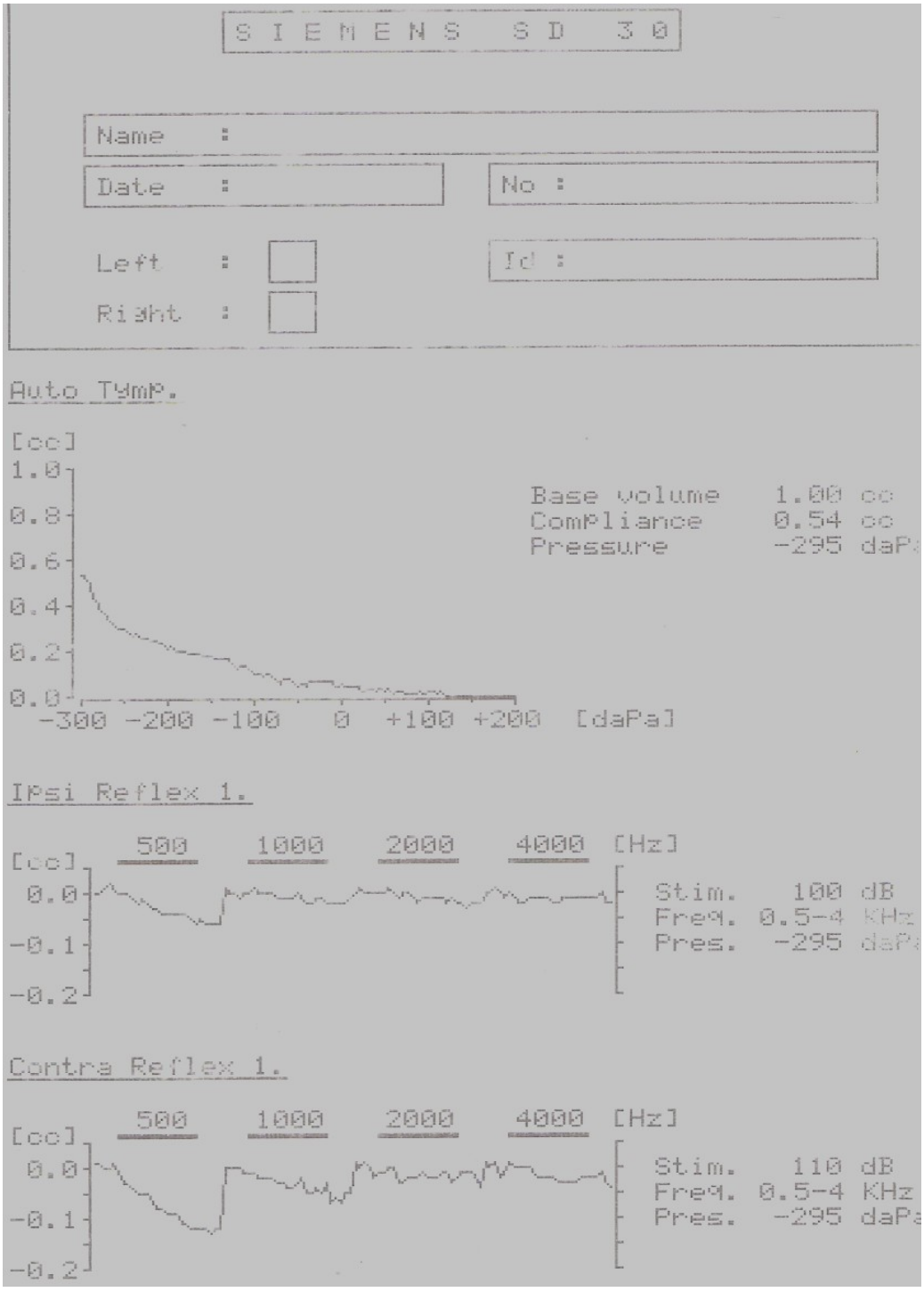
TYPE B
TYMPANOGRAM



TYPE – A
TYMPANOGRAM



TYPE – C
TYMPANOGRAM



17 PROFORMA

NAME-

IP/OP NO-

AGE-

SEX-

ADDRESS-

OCCUPATION-

CHIEF COMPLAINTS-

- HEADACHE
- FACIAL HEAVINESS
- POST NASAL DISCHARGE
- NASAL STUFFINESS/NASAL OBSTRCTION
- RECURRENT SNEEZING/WATERY NASAL DISCHARGE
- TASTE AND SMELL ALTERATIONS
- PURULENT RHINORRHEA
- EAR DISCHARGE
- HARD OF HEARING
- OTALGIA
- POPPING SENSATION IN THE EARS
- FULLNESS OF EARS
- FLUCTUATING HEARING LOSS
- TINNITUS
- DIZZINESS

PAST HISTORY-

- HISTORY OF PREVIOUS NASAL SURGERIES, EAR SURGERIES, MAXILLECTOMIES, CLEFT PALATE REPAIR.
- PAST HISTORY OF IRRADIATION TO THE HEAD AND NECK

CLINICAL EXAMINATION-

- ANTERIOR AND POSTERIOR RHINOSCOPY EXAMINATIONS

- PARANASAL SINUS TENDERNESS
- OTOSCOPY EXAMINATION OF EAR IN ADDITION TO OTO ENDOSCOPIC EVALUATION

DIAGNOSTIC NASAL ENDOSCOPY

- FIRST PASS DOCUMENTING THE NASAL FLOOR, INFERIOR MEATUS, SEPTUM AND THE EUSTACHIAN TUBE ORIFICE
- SECOND PASS DOCUMENTING THE SPHENO ETHMOIDAL RECESS
- THIRD PASS DOCUMENTING THE MIDDLE MEATUS

OTOENDOSCOPY-

- DOCUMENTING THE TYMPANIC MEMBRANE AND ITS APPEARANCE

18 BIBLIOGRAPHY

1. Honjo et al. Recent advances in otitis media. 5th international symposium, 1993
2. Nakano y, acta otolarngol 1990
3. Sato y acta otolaryngol,
4. Stoikes , effect of fess on eustachian tube. American journal rhinol 2005
5. Sade j, pressure homeostasis of middle ear, otol neuro otol 2003
6. Sade j, american journal otol 2000
7. Bayramoglu et al, int. Journal ped. Otorhinolaryngol 1997
8. Klein jo, bluestone et al, ann otol rhinol 1974
9. Bluestone et al, cd, pead 1978
10. Shure et al pead north american 1974
11. Elner, acta otolaryngol 1971
12. Holmquist , et function and tympanoplasty acta otolaryngol 1969
13. Palva a , et patency in chronic ears acta otolaryngol 1974
14. Huang , zhan. Influence of sinusitis on middle ear. Pubmed 2000 apr
15. Leo g, piacentini . Sinusitis and etd in children. Ped allergy immunol 2007
16. Udi cinnamon passive and dynamic properties of eustachian tube . Neuro otol 2004
17. Sade j .middle ear and auditory tumor otolaryngol head and neck surgery 1997
18. sade j , et lumen . Ann otolrhinolaryngol 1989
19. Mover – lev h , quantitative estimation of gas loss in middle ear . Respire physiol 1998
20. Buckinham middle ear pressures in eustachian tube malfunctions laryngoscope 1973
21. Bluestone cd . Assessment of eustachian tube function
22. Bluestone cd anatomy of eustachian tube
23. Bunne M . Variability of et function laryngoscope 2000
24. Mawson diseases of the ear 5 th edition.
25. Stammberger endoscopic sinus surgery
26. Ap singh functional endoscopic sinus surgery