"SIGNIFICANCE OF CORTICAL MASTOIDECTOMY IN TUBOTYMPANIC DISEASES"

By

Dr. WINSON IDICULA

Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH,
TAMAKA, KOLAR, KARNATAKA

In partial fulfilment
of the requirements for the degree of
Master of Surgery

in

OTORHINOLARYNGOLOGY

under the guidance of

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Date Signature of the candidate

Place **Dr. WINSON IDICULA**

List of abbreviations (in alphabetical order)

1	**CSOM**	Chronic Suppurative Otitis Media
2	**CHL**	Conductive Hearing Loss
3	**EAC**	External Auditory Canal
4	**HF**	High Frequency
5	**IAC**	Internal Auditory Canal
6	**IDL**	Indirect Laryngoscopy
7	**LF**	Low Frequency
8	**MHL**	Mixed Hearing Loss
9	**MRSA**	Methicillin Resistant Staphylococcus Aureus
10	**MSSA**	Methicillin Sensitive Staphylococcus Aureus
11	**PNS**	Paranasal Sinus
12	**TM**	Tympanomastoidectomy
13	**TP**	Tympanoplasty
14	**TTD**	Tubotympanic Disease

ABSTRACT

Background and objectives

CSOM is very common in rural India. The gold standard for treatment of all tubotympanic diseases has been tympanoplasty with cortical mastoidectomy irrespective of the stage of the disease or the presence of associated complications including mastoiditis. There are two schools of thought regarding the treatment of an uncomplicated CSOM tubotympanic disease. While some otologists suggest adjuvant cortical mastoidectomy with tympanoplasty in all cases of tubotympanic disease, others advocate tympanoplasty alone in an uncomplicated tubotympanic disease. The drawbacks of adjuvant cortical mastoidectomy to tympanoplasty include increased risk of complications, increased consumption of resources and time and increased postoperative morbidity. To keep these drawbacks to a minimum, some surgeons prefer tympanoplastic surgeries alone in an uncomplicated tubotympanic disease. However opinion on this matter varies as, some ENT surgeons prefer tympanoplasty with cortical mastoidectomy as a routine treatment in all tubotympanic disease. Our objective was to evaluate the significance of cortical mastoidectomy as an adjunctive to tympanoplasty in tubotympanic disease with no signs of associated complications.

Materials and Methods

An observational study with historical controls was done on 100 patients with 60 patients included in the historical control group and 40 patients in the trial group. The study period was from October 2009-September 2010 with a minimum period of 12 months follow-up postoperatively. Success was defined as, subjective resolution of all symptoms of tubotympanic diseases and successful graft uptake showing healthy

neotympanum, as visualized with an otoscope, 12 months after surgery. Failure was defined as persistence or recurrence of symptoms or failure of graft uptake or reperforation of the neotympanum during the follow-up period.

Results and interpretation

At 12 months postoperatively, in the group where only tympanoplasty was done (trial group), the surgical rate of success was 80%. The surgical success rate in the group which underwent tympanoplasty with cortical mastoidectomy (historical controls) after a minimum follow-up of 1year was 76.6%. Five patients in the historical control group and 1 patient in the trial group developed postaural wound infection during the follow-up period. The students t test was applied to our results and a '***p***' value greater than 0.05 (***p***>0.05) was obtained. Thus a statistically insignificant difference was seen between the two groups suggesting that we join the list of researchers who are not in favour of adjuvant cortical mastoidectomy in the treatment of uncomplicated tubotympanic disease.

Conclusion

To conclude, in patients in whom there are sign of mastoid infection or other associated complications, cortical mastoidectomy may be indicated but not as routine in all patients with tubotympanic disease.

Key Words: tympanoplasty, cortical mastoidectomy, tubotympanic disease

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					ED	**DH**	**H**	**GI**	**TI**	**P**	**F**	**ED**			**PZ**				**MEM**		**ETP**		**SOD**					**COMPLICATIONS**	**RESULTS**
												M	**MP**	**PU**	**S**	**M**	**L**	**ST**	**N**	**D**	**PA**	**B**	**A**	**Q**	**I**		**PWI**	
1	Saraswathamma	30	F	419473	+	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	+	SS
2	Anjamma	41	F	601913	+	+	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
3	Sunitha	23	F	261771	+	+	-	-	-	-	-	-	+	-	-	+	-	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
4	Sridevi	22	F	495062	+	+	-	-	-	-	-	+	-	-	-	+	-	-	-	+	+	-	+	-	-	CSOM & TM	-	SS
5	Geetha M.N	32	F	429251	+	+	+	-	-	-	-	-	+	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	+	SS
6	O.K Srinivas	25	M	407097	+	+	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	+	-	-	CSOM & TM	-	SS
7	Lakshmi Devamma	35	F	453461	+	+	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
8	Ambika	19	F	396287	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
9	Ambika	24	F	410524	+	+	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
10	S.V Baghyasri	20	F	307113	+	-	-	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	+	-	-	CSOM & TM	-	SS
11	Dhanalakshmi	35	F	426587	+	+	+	-	-	-	-	+	-	-	-	+	-	-	+	-	-	+	+	-	-	CSOM & TM	-	SS
12	Sabeena Banu	48	F	580182	+	+	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
13	Lakshmi Kumari	22	F	425643	+	-	+	-	-	-	-	-	-	-	+	-	-	+	+	-	+	-	+	-	-	CSOM & TM	+	SF
14	Nagaveni	19	F	451552	+	+	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	-	-	+	CSOM & TM	-	SS
15	Munirathnamma	26	F	592546	+	-	-	-	-	-	-	+	-	-	-	+	-	-	-	+	+	-	+	-	-	CSOM & TM	-	SF
16	Laghumamma	35	F	440815	+	+	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SF
17	Nagaveni	26	F	451235	+	+	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
18	Chandrakala	25	F	410770	+	+	-	-	+	+	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
19	Jayalakshmi	20	F	487133	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
20	Fathima	45	F	526923	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
21	Santhosh	15	M	434015	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
22	Sushmitha Y.M	14	F	515791	+	+	-	-	-	-	-	+	-	-	-	+	-	-	-	+	+	-	-	+	-	CSOM & TM	-	SF
23	Guttalappa	35	M	480103	+	+	-	-	+	-	-	+	-	-	-	-	+	-	+	-	+	-	-	+	-	CSOM & TM	-	SS
24	Range Gowda	36	M	517771	+	+	-	-	+	-	-	-	-	-	+	-	-	-	-	+	+	-	-	+	-	CSOM & TM	-	SS
25	Ramesh Kumar	35	M	538403	+	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
26	Chandrashekar	35	M	577960	+	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
27	Mottappa	30	M	450143	+	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-	CSOM & TM	-	SF
28	Narayana Murthy	48	M	453962	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	-	+	-	CSOM & TM	-	SF
29	Munivenkatamma	40	F	445663	+	+	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM & TM	-	SS
30	G.Kala	35	F	395582	+	+	-	-	-	-	-	-	-	+	-	-	-	+	-	+	+	-	+	-	-	CSOM & TM	-	SS
31	Lavanya	23	F	508735	+	+	-	-	+	-	-	-	-	-	-	+	-	-	-	+	+	-	-	+	-	CSOM & TM	-	SS

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32	Gowramma	30	F	391975	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	-	+	+	-	-	CSOM & TM	-	SS
33	Sarula	37	F	392778	+	-	-	-	+	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SF
34	Shylaja	26	F	435674	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	+	-	-	-	+	CSOM & TM	-	SS
35	Syed Haleema Sadiya	19	F	435544	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	+	-	+	-	-	CSOM & TM	-	SF
36	Manjula	23	F	479551	+	+	-	-	-	-	-	-	+	-	+	-	-	-	+	-	+	-	-	+	-	CSOM & TM	-	SS
37	Gowri	40	F	475545	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
38	Venkata Lakshmamm	25	F	433246	+	+	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM & TM	-	SF
39	Choukath Ali	25	M	451061	+	+	-	-	+	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
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41	Rathnamma	40	F	619844	+	+	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
42	Manohar	18	M	605041	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
43	Hassan	37	M	399345	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
44	Ramachandran Reddy	40	M	415878	+	-	-	-	-	-	-	-	-	+	+	-	-	-	+	-	+	-	-	+	-	CSOM & TM	-	SF
45	Muninarayanaswamy	33	M	439876	+	+	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
46	Muniraju	26	M	397599	+	-	-	-	-	-	+	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
47	Anji	22	M	543728	+	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	+	SS
48	Moorthy	16	M	599454	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	-	-	CSOM & TM	-	SF
49	Amravthi	48	F	522435	+	-	-	-	-	-	+	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
50	Reshmi	32	F	398792	+	-	-	-	-	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	CSOM & TM	+	SF
51	Sunandamma	40	F	622123	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
52	Dibjoth	23	M	507281	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM & TM	-	SS
53	Deena	20	F	554211	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM & TM	-	SF
54	Madhusudhan	19	M	555522	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	+	+	-	-	CSOM & TM	-	SS
55	Agnes	25	F	502281	+	+	+	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	-	-	+	CSOM & TM	-	SS
56	Manoj	35	F	565655	+	-	-	+	-	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
57	Deepthi	23	F	413648	+	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
58	Feba	24	F	436542	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SF
59	Varun	25	M	512613	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS
60	Rajesh	49	M	477563	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM & TM	-	SS

Trial group																													
Tympanoplasty (TP)																													
Snl	**Name**	**age**	**sex**	**Hospital No**	**symptoms**							**SIGNS**															**DIAGNOSIS & TREATMENT**	**FOLLOW UP**	
					ED	**DH**	**H**	**GI**	**TI**	**P**	**F**	**ED**			**PZ**				**MEM**		**ETP**		**SOD**						**COMPLICATIONS**	**RESULTS**
												M	**MP**	**PU**	**S**	**M**	**L**	**ST**	**N**	**D**	**PA**	**B**	**A**	**Q**	**I**			
1	Shakunthala	22	F	555783	+	+	-	-	-	-	+	+	-	-	-	-	+	-	-	+	+	-	+	-	-	CSOM + TP	**PWI**	SS
2	Ravanamma	28	F	555975	+	-	-	-	+	-	+	-	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM + TP	-	SS
3	Babhi	26	F	593911	+	-	-	-	-	-	+	+	-	-	-	+	-	-	+	-	+	-	+	-	-	CSOM + TP	-	SF
4	Imran Pasha	33	M	557323	+	+	-	-	+	-	+	-	-	-	+	-	-	-	-	+	+	-	+	-	-	CSOM + TP	-	SS
5	Subhamma	60	F	557040	+	-	-	-	-	-	+	+	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM + TP	-	SS
6	Asha B	24	F	591684	+	-	-	-	-	-	+	+	-	-	-	-	+	-	-	+	-	+	-	-	+	CSOM + TP	-	SF
7	Murthy	20	M	612369	+	-	-	-	+	-	+	-	-	+	+	-	-	-	+	-	+	-	-	-	+	CSOM + TP	-	SS
8	Rahul	18	M	556096	+	-	+	-	-	-	+	+	-	-	-	-	-	+	-	+	+	-	+	-	-	CSOM + TP	-	SS
9	Thippeswamy	55	M	518957	+	-	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	-	-	+	-	CSOM + TP	-	SF
10	B M pushpa	26	F	557887	+	+	-	-	+	-	+	-	+	-	+	-	-	-	+	-	+	-	+	-	-	CSOM + TP	-	SS
11	Srinivas	19	M	560066	+	+	-	-	-	-	+	+	-	-	-	-	+	-	+	-	+	-	-	+	-	CSOM + TP	-	SS
12	Amala	24	F	562079	+	+	-	-	-	-	+	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM + TP	-	SS
13	Shilpa	28	F	616341	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM + TP	-	SS
14	Menaka	18	F	551024	+	+	+	-	+	-	+	-	+	-	-	-	-	+	-	+	+	-	+	-	-	CSOM + TP	-	SS
15	Janardhan	24	M	629328	+	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	+	-	-	-	-	CSOM + TP	+	SF
16	Varamahalakshmi	18	F	562340	+	+	-	-	-	-	+	+	-	-	-	-	+	-	-	+	+	-	+	-	-	CSOM + TP	-	SS
17	Harsha Kala	25	M	609393	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	-	-	-	CSOM + TP	-	SS
18	Narasimappa	45	M	602927	+	+	-	-	+	-	+	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
19	Manohar	19	M	605041	+	+	-	-	-	-	+	+	-	-	-	+	-	-	-	+	+	-	+	-	-	CSOM + TP	-	SS
20	Sujatha	25	F	558542	+	+	-	+	-	-	+	+	-	-	-	-	-	+	+	-	+	-	-	-	-	CSOM + TP	-	SS
21	ManjunathReddy	25	M	547209	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
22	Nazia Sulthana	17	F	565145	+	+	-	-	-	-	+	+	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM + TP	-	SF
23	Radhamma	45	F	601628	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
24	Subramani	25	M	566313	+	+	-	-	-	-	+	+	-	-	-	-	+	-	-	+	+	-	+	-	-	CSOM + TP	-	SS
25	Rathnamma	45	F	601913	+	+	-	-	-	-	+	-	+	-	+	-	-	-	+	-	+	-	+	-	-	CSOM + TP	-	SF
26	Nageen Taj	16	F	577761	+	+	-	-	-	-	+	-	-	-	-	-	+	-	+	-	+	-	+	-	-	CSOM + TP	-	SF
27	Murthy	16	M	599454	+	+	-	-	-	-	+	+	-	-	-	+	-	-	-	+	+	-	+	-	-	CSOM + TP	-	SS
28	Bharathy	22	F	519003	+	+	-	-	+	-	+	+	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
29	Ganesh	15	M	484035	+	-	-	-	-	-	+	-	+	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
30	Shafiulla	20	M	565060	+	-	-	-	-	-	+	-	+	-	-	-	+	-	-	+	+	-	-	+	-	CSOM + TP	-	SS
31	Ramesh	18	M	563522	+	+	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	-	+	-	-	CSOM + TP	-	SS
32	Harish	23	M	585321	+	-	-	-	-	-	+	+	-	-	-	-	-	+	+	-	+	-	-	-	-	CSOM + TP	-	SF
33	Manjunath	30	M	561008	+	-	-	-	-	-	+	-	-	-	+	-	-	-	+	-	-	+	+	-	-	CSOM + TP	-	SS
34	Nandish	29	M	552662	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	+	-	-	CSOM + TP	-	SS
35	Manjunatha	25	M	584148	+	-	-	-	-	-	+	+	-	-	-	+	-	-	-	+	+	-	-	-	-	CSOM + TP	-	SS
36	Srinath	20	M	595620	+	+	-	-	-	-	+	+	-	-	-	-	-	+	+	-	+	-	-	+	-	CSOM + TP	-	SS
37	Gopalakrishnan	34	M	590244	+	+	-	-	-	-	+	-	+	-	-	-	-	+	+	-	+	-	-	-	-	CSOM + TP	-	SS
38	Gurupadappa	26	M	555710		-	+	-	-	-	+	-	-	-	+	-	-	-	+	-	+	-	-	-	-	CSOM + TP	-	SS
39	Munirathnamma	26	F	592546		+	-	-	-	-	+	-	-	-	-	-	-	+	-	+	+	-	-	+	-	CSOM + TP	-	SS
40	Dasarathan K	54	M	392831		+	-	-	-	-	+	+	-	-	-	-	+	-	+	-	+	-	-	-	-	CSOM + TP	-	SS

INTRODUCTION

Since prehistoric times chronic suppurative otitis media (CSOM) has been an important cause of middle ear disease.[1] Incidence of CSOM is high in developing countries. Poor living conditions, overcrowding, poor hygiene and poor nutrition have all contributed to the increased incidence of CSOM.[1]

Otitis media is defined as "an inflammation of the middle ear without reference to aetiology or pathogenesis".[2] As the mastoid air cell system and eustachian tube are anatomically related to the middle ear, the term otitis media also implies inflammation of the entire middle ear cleft.[2] Accordingly otitis media is more correctly conceived of as an inflammatory disorder of the entire tympanomastoid compartment.[2] This can be further divided into tubotympanic (non cholesteatoma) and atticoantral (cholesteatoma) disease[3].

The principal symptoms of tubotympanic diseases are hearing loss and aural discharge.[1] The aim of treatment in tubotympanic disease is to provide subjective resolution of symptoms, closure of the perforations, improvement of hearing, and prevention of complications.[3] Many factors influence the final outcome of surgery. These factors can generally be grouped into "mastoid" and "non mastoid" factors. The "non mastoid" factors that can impair successful graft uptake are general debility, technical errors, and most importantly eustachian tube dysfunction. Failure of mastoid pneumatization and mastoiditis are the mastoid factors causing graft failure.[4]

Tympanomastoidectomy has been the treatment of choice for all tubotympanic disease unresponsive to medical therapy[3]. However, the significance of cortical mastoidectomy in the surgical treatment of tubotympanic disease has come under

scrutiny, especially when the disease is in inactive or quiescent stage.[5] Some authors suggest that myringoplasty is more than sufficient for repair of tympanic membrane perforations, regardless of the status of the middle ear and mastoid, while others advice tympanomastoidectomy, only if the mastoid appears diseased.[4]

Wullstein in 1953 first used the term tympanoplasty for reconstruction of the middle ear hearing mechanism that had been impaired or destroyed by chronic ear disease.[2] In fact it was the introduction of tympanoplasty that inspired otologists to abandon radical mastoidectomy and replace the same with cortical mastoidectomy in uncomplicated tubotympanic disease. But the addition of mastoidectomy to tympanoplasty carries several disadvantages such as: increased risk of damage to the incus, dura, sigmoid sinus, and facial nerve; prolongation of surgery; and higher morbidity due to bone drilling, especially in the hands of an inexperienced surgeon[5]. Furthermore, tympanoplasty alone is both time saving and less expensive when compared to tympanomastoidectomy.

According to a study conducted by Ramakrishna and colleagues in 2011, success rate with regard to graft uptake was 93.55% in tympanomastoidectomy and 96.75% in tympanoplasty alone.[3] Even in this 21st century the importance of mastoidectomy in uncomplicated tubotympanic disease is still a subject of much debate among many well trained and experienced otologists.[3]

Mastoidectomy as an adjuvant to tympanoplasty is justified in complicated tubotympanic disease. However the use of mastoidectomy to treat chronic ear infection in an uncomplicated case of tubotympanic disease remains an issue of debate. Some authors suggest that mastoidectomy is justified in cases of CSOM which have been refractory to maximal antibiotic therapy.[3]

Ruhl and Pensak states that lack of an aerating mastoidectomy at the time of initial tympanoplasty may be a significant source of failure in patients with tubotympanic disease.[6] Based on the theory of mastoid acting as a reservoir of air, many otologists advocate mastoidectomy, as an effective means of re-pneumatizing mastoid and eradicating mastoid source of infection.[4]

According to some authors irrespective of mastoidectomy, tympanoplasty alone can produce successful closure of tympanic membrane perforations and elimination of chronic ear discharge.[3] Hence forth it is suggested that mastoidectomy is not only unnecessary when treating tubotympanic disease, but it increases patients risk with little or no significant clinical advantage.[7] Therefore an attempt is made here to determine whether tympanoplasty alone is sufficient for a successful graft uptake in an uncomplicated tubotympanic disease.

AIMS AND OBJECTIVES

To evaluate the role of adjunctive cortical mastoidectomy in the treatment of tubotympanic disease.

REVIEW OF LITERATURE

Moritz (1952), Zollner (1953, 1955) and Wullstein (1953, 1956) in Germany introduced the fundamental techniques and basic concepts of modern reconstructive middle ear surgery in tubotympanic disease.[8] Tympanoplasty is defined as an operation performed to 'eradicate disease in the middle ear and to reconstruct the hearing mechanism, without mastoid surgery, with or without tympanic membrane grafting.[8] Tympanoplasty is the end result of 100 years of research and perseverance to develop surgical procedures on the middle ear to improve hearing.[8] Wullstein classified tympanoplasty into 5 types depending on the degree of ossicular disruption.

Type I- Reconstruction of the tympanic membrane (ossicular chain intact and mobile).

Type II- Malleus handle absent- Reconstruction of the tympanic membrane over the malleus remnant and long process of incus.

Type III- Malleus and incus absent- Reconstruction of the tympanic membrane over an intact and mobile stapes (myringostapediopexy) with stapes acting as a columella.

Type IV- Mobile stapes footplate exteriorized with reconstruction of the tympanic membrane as a round window baffle.

Type V- Stapes fixed, fenestration.

The invention of monocular operating microscope by Nylen in 1921 and then the invention of binocular operating microscope in 1922 by Holmgren improved the tympanoplasty operation being done more precisely.[8] Tympanomastoidectomy operations were designed not only to eradicate disease from the middle ear and mastoid but also to restore hearing mechanism.[8]

The surgical treatment for CSOM was not pertinent to one single operation but had much more than what was actually done.[8] Every case was different from the

other and depending upon the extent of pathology, treatment protocol was decided on whether disease excision required a purely transcanal operation, or whether in addition, some form of mastoidectomy was needed together with a tympanoplasty.[8] Diamont was the first to make a systematic study of the mastoid air cell volume in normal and diseased temporal bones.[4] He found that mastoid sizes ranged from 0 to 30cm^2, with a mean of 12.3cm^2 for adults using mastoid radiographs.[4] Intact canal wall mastoidectomy (combined approach tympanoplasty) was introduced in the late 1950s by Jansen.[1]

Holmquist reported the significance of reduced mastoid air cell volume in tympanic membrane reconstruction.[9] He showed that for successful myringoplasty a well pneumatized mastoid air cell volume was very necessary and that if mastoid air cell volume was less than 5 cm^2 then the success rate following tympanic membrane closure falls to less than 22%.[4] Degree of mastoid pneumatization correlated directly with successful closure of tympanic membrane perforation. Studies show successful closure of tympanic membrane rose to 57% when mastoid air volume improved to 5 to 10 cm^2 and about 71% success when the mastoid air cell volume was greater than 10 cm^2.[4] This clinical findings suggested that small mastoid air cell size had an adverse effect in tympanoplastic surgery.[4]

Boyle's law states that post-operative well- pneumatized mastoid helps in maintaining the pressure-buffering effect of the mastoid air cell system.[6] Holmquist and Bergstrom for the first time proposed the importance of mastoidectomy surgeries in improving the outcome of tympanoplasty.[6] In cases with eustachian tube dysfunction or a small mastoid air cell, creation of an aerated mastoid enhances success rates.[6] Even though some authors accept the theory proposed by Holmquist

and Bergstrom, there is no solid evidence to prove that mastoidectomy yields better surgical results than tympanoplasty alone.[6]

Benjamin stated that mastoidectomy was not necessary for successful repair of simple tympanic membrane perforations.[10] However, mastoidectomy plays a key role during the clinical course as it reduces the number of patients requiring future surgery and also helps in decreasing disease progression.[10] Combining mastoidectomy with tympanoplasty in simple perforations with no active evidence of infection remains the main stay treatment option and hence reduces the need for future surgery.[10]

According to Bhat, addition of cortical mastoidectomy to tympanoplasty for the surgical treatment of quiescent-stage, tubotympanic disease was not beneficial in terms of hearing improvement or disease eradication, over a short term follow-up period.[5]

Studies conducted by Mishiro et al in 2001 showed no significant difference in tympanic membrane closure rates between groups in whom tympanoplasty alone and tympanoplasty combined with mastoidectomy was done.[11] Balyan also stated that there was no significant difference in the graft success rate between these two types of surgeries for tubotympanic diseases.[7]

Despite the long-standing practice of performing mastoidectomy for tubotympanic disease, there is no convincing data in literature to indicate that mastoidectomy is necessary for the treatment of CSOM.[12]

ANATOMY OF THE EXTERNAL AND MIDDLE EAR

DEVELOPMENT OF MIDDLE EAR

The ear is the first organ of special sense to become differentiated in man. The external and middle ear are however not completely formed at birth.

During the early stage of foetal development, there appears on the lateral aspect of head process a series of six visceral arches. These mesenchymal arches form ridges in the overlying ectoderm and corresponding projections in the endoderm of the pharynx. The ectodermal furrows form the visceral clefts and the endodermal furrows form the pharyngeal pouches.

The tympanic membrane has three layers: outer epithelial layer from the ectoderm of the visceral cleft, a middle fibrous layer from the mesoderm between the first visceral cleft and tubotympanic recess and an inner mucosal layer from a part of tubotympanic recess.

The middle ear cleft is developed from the endoderm of the tubotympanic recess, which is pushed out from the first pharyngeal pouch to approach the surface between the first and second visceral arches.

Towards the end of second foetal month, the development of eustachian tube is clearly seen as a relatively direct extension from the primitive pharynx. The middle ear now at this stage is only a potential cavity filled solidly by mesenchyma, in which the ossicles are embedded. At the eighth foetal week the embedded ossicles in the mesenchyma are all well defined. The mastoid antrum appears as a dorsal expansion of the middle ear cavity. The malleus and incus develops from the two swellings that appear at the dural end of the Meckel's cartilage. The mesoderm of the second visceral arch forms the hyoid bar, from which the head, neck and crura of the stapes,

the styloid process and the stylohyoid ligament develop. The footplate of stapes is derived partly from the otic capsule.

Between the third and seventh foetal month, the gelatinous tissue of the middle ear cleft gets gradually absorbed, and the primitive tympanic cavity develops into the cleft, of an endodermal lined fluid pouch from the eustachian tube. The tympanic cavity gives rise to four primary sacs which buds out from it. They are: saccus anticus – smallest and forms the anterior pouch of Von Troltsch. Sacccus medius forms the attic and breaks into anterior, posterior and medial compartments. Saccus superior develops into posterior pouch of Von Troltsch and inferior incudal space. The saccus posticus extends along the hypotympanum to form the round window niche, sinus tympani, and greater portion of the oval window niche.

THE EXTERNAL EAR

The auricle

The pinna acts to focus and aid in the localization of sound. The lateral surface of the pinna is dominated by concavities, in particular the concha. The smaller superior portion is the cymba concha and is the direct lateral relation to the suprameatal triangle of the temporal bone. The curved rim is the helix, which often has a small prominence (Darwin's tubercule) at its posterosuperior aspect. Anterior and parallel with the helix is another prominence, the antihelix. The skins of the lateral and medial surfaces of the pinna possess hair and both sebaceous and sudoriferous glands; however, the attachment of the skin differs, being tightly bound down to the peichondrium on the lateral aspect and only loosely attached on the medial aspect.

Sensory innervation of the auricle

The greater auricular nerve derived from the cervical plexus C2,3 supplies the medial surface and posterior portion of lateral surface of the pinna. The superior portion of medial surface of the pinna is supplied from the lesser occipital nerve derived from the cervical plexus C2,3. The vagus nerve as shown in figure 1 gives off the auricular branch which supplies the concha and antihelix. The third branch of the trigeminal nerve (mandibular) supplies the tragus, crus of helix and adjacent helix through the auriculotemporal nerve. The facial nerve probably supplies a small region in the root of concha.

The external auditory canal

The lateral one-third of the EAC comprises a continuation of the cartilage of the pinna and is deficient superiorly at the incisura terminalis; the extra cartilaginous endaural incision for access to the underlying temporal bone capitalizes on this gap.

The remaining medial two-thirds of the approximately 2.5cm length of the canal are bony. The isthmus, the narrowest portion of the EAC, lies just medial to the junction of the bony and cartilaginous canals. The skin of the cartilaginous canal has a substantial subcutaneous layer, replete with hair follicles, sebaceous glands and cerumen glands. The skin of the osseous canal, in contrast is very thin and its subcutaneous layers bereft of the usual adnexal structures. Accordingly, the absence of hair serves to distinguish the bony and cartilaginous canals.

Vascular supply

Two branches of the external carotid artery, the posterior auricular artery and the superficial temporal artery, are the sources of arterial blood supply to the pinna and EAC. The posterior auricular artery, as it courses superiorly on the mastoid portion of the temporal bone, supplies the skin of the pinna and the skin and bone of the mastoid; its stylomastoid branch enters the fallopian canal to supply the inferior segment of the facial nerve. Anteriorly, a few twigs of the superficial temporal artery provide additional supply to the pinna and EAC. The veins accompanying the arteries drain into internal jugular vein by either the facial or external jugular vein.

THE MIDDLE EAR CLEFT

The middle ear cleft as shown in figure 2 consists of the tympanic cavity, the eustachian tube and the mastoid air cell system. The tympanic cavity is an irregular, air-filled space within the temporal bone between the tympanic membrane laterally and the osseous labyrinth medially.

The tympanic membrane

It is slightly oval in shape, being broader above than below, forming an angle of 55 degree with the floor of the meatus. Its longest diameter from posterosuperior to anteroinferior is 9-10 mm, while perpendicular to this the shortest diameter is 8-9mm. Most of the circumference is thickened to form a fibrocartilagenous ring, the tympanic annulus, which sits in a groove in the tympanic bone, the tympanic sulcus. From the superior limits of the sulcus, the annulus becomes a fibrous band which runs centrally as anterior and posterior malleolar folds to the lateral process of the malleus,

the handle of which is clearly visible within the tympanic membrane. The tympanic membrane is separated into superior pars flaccida (shrapnell's membrane) and a pars inferior by the anterior and posterior tympanic stria, which runs from the lateral process of the malleus to the anterior and posterior tympanic spines respectively. Shrapnell's membrane serves as the lateral wall of Prussack's space.

Both the pars tensa and pars flaccida comprise three layers. There is an outer epithelial layer, the epidermis, which is continuous with the skin of the external meatus; middle, mainly fibrous layer, the lamina propria; and an inner mucosal layer continuous with lining of the tympanic cavity. The lamina propria of the pars tensa has radially oriented fibres in the outer layers and circular, parabolic and transverse fibres in the deeper layers. In the pars flaccida, the lamina propria is less marked and the orientation of collagen fibres seems random.

The arterial supply of the tympanic membrane arises from branches supplying both the external auditory meatus and the middle ear. The epidermal vessels originate from the deep auricular branch of the maxillary artery coming from the external auditory meatus, whereas the mucosal vessels arise from the anterior tympanic branches of the maxillary artery, the stylomastoid branch of the posterior auricular artery and probably the middle meningeal artery. Branches of the auriculotemporal nerve, the auricular branch of the vagus and the tympanic branch of the glossopharyngeal nerve supply the tympanic membrane.

Pathways of migratory movement

The pathways of epithelial migration on tympanic membrane have been elucidated using modern methods of photographic otoscopy. The epithelium near the region of the handle of malleus moves upwards as far as the lateral process and then

moves posterosuperiorly like pars flaccida. The normal rate of migration is about 0.1mm /day.

The tympanic cavity

The tympanic cavity is notionally divided into three compartments: the epitympanum (upper), the mesotympanum (middle) and hypotympanum (lower). The epitympanum or attic, lies above the level of malleolar fold and is separated from the mesotympanum and hypotympanum by a series of mucosal membranes and folds.

The lateral wall

The lateral wall of the tympanic cavity is formed by the bony lateral wall of the epitympanum superiorly, the tympanic membrane centrally and the bony lateral wall of the hypotympanum inferiorly. Three holes are present in the bone of the medial surface of the lateral wall of the tympanic cavity. The petrotympanic fissure is a slit about 2mm long which opens anteriorly just above the attachment of the tympanic membrane. It receives the anterior malleolar ligament and transmits the anterior tympanic branch of the maxillary artery to the tympanic cavity. The chorda tympani, which carries taste sensation from the anterior two-third of the same side of the tongue and secretomotor fibres to the submandibular gland, enters the medial surface of the fissure through a separate anterior canaliculus (canal of Huguier) which is sometimes confluent with the fissure. It then runs posteriorly between the fibrous and mucosal layers of the tympanic membrane, across the upper part of the handle of the malleus and then continues within the membrane, but below the level of the posterior malleolar fold.

The roof

The roof of the epitympanum is the tegmen tympani, a thin bony plate that separates the middle ear space from the middle cranial fossa.

The floor

The floor of the tympanic cavity may consist of compact or pneumatized bone and separates the hypotympanum from the dome of the jugular bulb. At the junction of the floor and the medial wall of the cavity there is a small opening that allows the entry of glossopharyngeal nerve into the middle ear from its origin below the base of the skull.

The anterior wall

The anterior wall of the tympanic cavity is rather narrow as the medial and lateral walls converge. The lower-third of the anterior wall consists of a thin plate of bone covering the carotid artery as it enters the skull and before it turns anteriorly. This plate is perforated by the superior and inferior caroticotympanic nerves carrying sympathetic fibres to the tympanic plexus, and by tympanic branches of the internal carotid artery. The middle third comprises the tympanic orifice of the Eustachian tube, which is oval and 5x2mm in size. The upper-third is usually pneumatized and may house the anterior epitympanic sinus, a small niche anterior to the ossicular heads, which can hide residual cholesteatoma in canal wall up surgery.

The medial wall

The medial wall separates the tympanic cavity from internal ear. The promontory is a rounded elevation occupying much of the central portion of the medial wall. It covers part of the basal coil of the cochlea and usually has small

grooves on its surface containing the nerves which form the tympanic plexus. Behind and above the promontory is the oval window; a nearly kidney-shaped opening that connects the tympanic cavity with the vestibule, but which in life is closed by the footplate of the stapes and surrounding annular ligament. Its size naturally varies with the size of the footplate, but on average it is 3.25mm long and 1.75mm wide.

The round window niche lies below and a little behind the oval window niche from which it is separated by a posterior extension of the promontory called the subiculum. Occasionally another bridge of bone the ponticulus leaves the promontory above the subiculum and runs to the pyramid on the posterior wall of the cavity. The round window niche is most commonly triangular in shape, with anterior, posterosuperior and posteroinferior walls.

The facial nerve canal (or Fallopian canal) runs above the promontory and oval window in an anteroposterior direction. It has a smooth rounded lateral surface that often has microdehisence and when the bone is thin and the nerve is exposed by disease, there are two or three straight blood vessels clearly visible along this line of nerve. The facial nerve canal is marked anteriorly by the processus cochleariformis, a curved projection of bone concave anteriorly, which houses the tendon of tensor tympani muscle as it turns laterally to the handle of malleus. Behind the oval window, the facial canal starts to turn inferiorly as it begins its descent in the posterior wall of the tympanic cavity. The region above the level of facial nerve canal forms the medial wall of the epitympanum. The dome of the lateral semicircular canal is the major feature in the posterior portion of the epitympanum, lying posterior and extending a little lateral to the facial canal.

The posterior wall

The posterior wall is wider above than below and has in its upper part a large irregular opening the aditus ad antrum that leads back from the posterior epitympanum into the mastoid antrum. Below the aditus is a small depression, the fossa incudis, which houses the short process of the incus and its suspensory ligament. Below the fossa incudis and medial to the opening of the chorda tympani nerve is the pyramid, small hollow conical projection with its apex pointing anteriorly. This houses the stapedius muscle and tendon, which inserts into the posterior aspect of the head of stapes.

The facial recess is a groove which lies between the pyramid and facial nerve and the annulus of the tympanic membrane. The facial recess is therefore, bounded medially by the facial nerve and laterally by the tympanic annulus, with the chorda tympani nerve running obliquely through the wall between the facial nerve and tympanic annulus.

The sinus tympani is a posterior extension of the mesotympanum and lies deep to both the promontory and the facial nerve. This extension of air cells into the posterior wall can be extensive, and is probably the most inaccessibly site in the middle ear and mastoid. The medial wall of the sinus tympani becomes continuous with the posterior portion of the medial wall of the tympanic cavity where it is related to the oval and round window niches and the subiculum of the promontory. On rare occasions it can communicate with the mastoid air cells.

THE CONTENTS OF THE TYMPANIC CAVITY

The Malleus

The malleus in figure 3 is the largest of the three ossicles, measuring up to 9mm in length. It comprises a head, neck and handle or manubrium. The head lies in the epitympanum and this is suspended by the superior ligament, which runs upward to the tegmen tympani. The head of the malleus has a saddle- shaped facet on its posteromedial surface to articulate with the body of the incus by way of a synovial joint. Below the neck of the malleus, the bone broadens and gives rise to the lateral process, the anterior process and the handle. The lateral process is a prominent landmark on the tympanic membrane and receives the anterior and posterior malleolar folds from the tympanic annulus. The chorda tympani crosses the upper part of the malleus handle on its medial surface above the insertion of the tendon of tensor tympani, but below the neck of the malleus itself. The neck of the malleus connects the handle with the head and amputation of the head by cutting through the neck leaves both chorda tympani and tensor tympani intact. A slender anterior ligament arises from the anterior process to insert into the petrotympanic fissure.

The Incus

The incus articulates with the, malleus and has a body and two processes. The body lies in the epitympanum and has a cartilage covered facet corresponding to that on the malleus. The body of the incus is suspended by the superior incudal ligament that is attached to the tegmen tympani. The short process projects backward from the body to lie in the fossa incudis to which it is attached by a short suspensory ligament.

The long process descends in to the mesotympanum behind and medial to the handle of the malleus, and at its tip is small medially directed lenticular process. This is sometimes been called the fourth ossicles because of its incomplete fusion with the tip of the long process, thereby giving the appearance of a separate bone or at least a sesamoid bone. The lenticular process articulates with the head of the stapes.

The Stapes

The stapes is shaped like a stirrup and consists of a head, neck, the anterior and posterior crura and a footplate. The head points laterally and has a small cartilage-covered depression for a synovial articulation with the lenticular process of the incus. The stapedius tendon inserts into the posterior part of the neck and upper portion of the posterior crus. The two crura join the footplate, which usually has a convex superior margin, an almost straight inferior margin and curved anterior and posterior ends. The average dimensions of the footplate are 3mm long and 1.4mm wide and it lies in the oval window where it is attached to the bony margins by the annular ligament.

The Stapedius

A slender tendon emerges from the apex of the pyramid and inserts into the stapes. The muscle is supplied by a small branch of the facial nerve.

The Tensor tympani

This is a long slender muscle arising from the walls of the bony canal lying above the Eustachian tube. Parts of the muscle also arise from the cartilaginous portion of the Eustachian tube and the greater wing of the sphenoid. From its origins, the muscle passes backwards into the tympanic cavity where it lies on the medial

wall, a little below the level of the facial nerve. This enters the processus cochleariformis where it is held down by a transverse tendon as it runs through a right angle to pass laterally and inserts into the medial aspect of the upper end of the malleus handle. The muscle is supplied from the mandibular nerve by way of branch from the medial pterygoid nerve.

The Chorda tympani

This branch of the facial nerve enters the tympanic cavity from the posterior canaliculus at the junction of the lateral and posterior walls. It runs across the medial surface of the tympanic membrane between the mucosal and fibrous layers and passes medial to the upper portion of the handle of malleus above the tendon of tensor tympani to continue forwards and leave by way of the anterior canaliculus, which subsequently joins the petrotympanic fissure.

The Tympanic plexus

The tympanic plexus is formed by the tympanic branch of the glossopharyngeal nerve (Jacobson's nerve) and by caroticotympanic nerves, which arise from the sympathetic plexus around the internal carotid artery. The nerves form a plexus around the promontory and provide the branches to the mucous membrane lining the tympanic cavity, eustachian tube and mastoid antrum and air cells.

THE MUCOSA OF THE TYMPANIC CAVITY

The middle ear mucosa is essentially mucus-secreting respiratory mucosa bearing cilia on its surface. Three distinct mucociliary pathways can be identified– epitympanum, promontorial and hypotympanic, the latter being the largest. Each of these pathways coalesces at the tympanic orifice of the eustachian tube. The mucous

membrane lines the bony walls of the tympanic cavity, and it extends to cover the ossicles and their supporting ligaments. The mucosal folds also cover the tendons of the two middle ear muscles and carry the blood supply to and from the contents of the tympanic cavity. These folds separate the middle ear space into compartments.

THE EUSTACHAIN TUBE

The Eustachian tube (figure 4) can be divided into an osseous intratemporal portion and a cartilaginous portion. Respiratory mucosa lines the entire system. The Eustachian tube lumen is the persistence of the first pharyngeal pouch. In adults it is about 36mm in length, a size that is normally reached by the age of seven. It runs downwards from the middle ear at 45^0 and is turned forwards and medially. The lateral third is bony and arises from the anterior wall of the tympanic cavity. This joins a medial cartilaginous part, which makes up two-thirds of the tubal length, just after its narrowest portion, called the isthmus. The tube is lined with respiratory mucosa containing goblet cells and mucous glands, having a carpet of ciliated epithelium on its floor. The bony portion is about 12mm long and is widest at its oval-shaped orifice in the anterior wall of the tympanic cavity. It runs through the squamous and petrous portions of the temporal bone, gradually tapering to the isthmus, where the diameter is only 0.5mm or less. A thin plate of bone forms the roof, separating the tube from the tensor tympani muscle from above. The carotid canal lies medially and can impinge on the bony eustachian tube. The cartilaginous part of the tube is about 24mm long and consists of the fibrocartilagenous muscles to which is attached the peritubal muscles. The posteromedial wall is composed of cartilage and the anterolateral wall is composed of cartilage and fibrous tissue. The apex of the cartilage is attached to the isthmus of the bony portion, while the wider

medial end protrudes into the nasopharynx, lying directly under the mucosa to form the torus tubarius. In the nasopharynx, the tube opens 1-1.25 cm behind and a little below the posterior end of the inferior turbinate.

THE MASTOID AIR CELL SYSTEM (Figure 5)

The mastoid antrum is an air-filled sinus within the petrous part of the temporal bone. It communicates with the middle ear by way of the aditus and has mastoid air cell arising from its walls. The roof of the mastoid and the mastoid air cell space form the floor of the middle cranial fossa, while the medial wall relates to the posterior semicircular canal. More deeply and inferiorly is the dura of the posterior cranial fossa and the endolymphatic sac. The latter emerges through the operculum on the posterior surface of the petrous bone and derives from the endolymphatic duct which has passed through the vestibular aqueduct.

Allam and schuknecht classify the pneumatic tracts as follows:

A. Perilabyrinthine

1. Supralabyrinthine
    a. Posterosuperior
    b. Posteromedial
    c. Subarcuate
2. Infralabyrinthine
    a. Hypotympanic
    b. Retrofacial

B. Apical

1. Peritubal
    a. Anteroposterior
    b. Anterolateral
2. Apical
    a. Hypotympanum
    b. Peritubal

MASTOID PNEUMATIZATION

The mastoid is pneumatized from two sources – the squamous and petrous portions of the temporal bone. The squamous part is pneumatized by the saccus superior. The petrous part is pneumatized by the saccus medius. The two areas may be completely separated by the petrosquamous lamina (wall of schwartze and eysell or korner's septum). This is of considerable clinical importance because one area may be diseased and the other normal. If the more superficially placed squamous portion contains air, one can easily overlook the pathology in the deeper portion. If the petrosquamous lamina is not recognized and the surgeon seeks the aditus opening, there is great risk of injury to the facial nerve in its pyramidal segment.

The following are theories which explain the mastoid pneumatization:

1. Diamont (1954) – states that the degree of pneumatization is determined by hereditary factors. In the normal infant the mastoid becomes pneumatized. If pneumatization fails as a result of hereditary factors, the bone remains diploic or it may develop into a compact bone by the physiologic process of formation of cancellous bone in the marrow spaces.
2. Reudi (1963) – stated that infection can arrest pneumatization by destroying mucosa replacing it with connective tissue and eventually, sclerotic bone,
3. Tumarkin (1961) – stated that Eustachian tube obstruction with intratympanic vacuum may influence pneumatization

THE MIDDLE EAR SPACES

Planes extended from the tympanic annulus subdivide the tympanic cavity into a mesotympanum, hypotympanum, protympanum and posterior tympanic cavity. The epitympanum lies above the plane of the anterior and posterior tympanic spines.

Anteriorly the mesotympanum is dominated by the bulge of the semi canal of the tensor tympani muscle; the tympanic orifice of the eustachian tube is immediately inferior to this bulge. Posteriorly, the key anatomic features are the pyramidal eminence and lateral to it, the chorda eminence. The chordal eminence houses the iter chordae posterius by which the chorda tympani nerve enters the tympanic cavity.

The medial wall (the surgical "floor" of the middle ear) features 3 depressions: the sinus tympani, oval window niche, and round window niche).The sinus tympani is defined by the ponticulus superiorly, the subiculum inferiorly, the mastoid segment of the facial nerve laterally and the posterior semicircular canal medially; there is substantial variability in the posterior extension of the sinus tympani, ranging from "shallow" to "deep". The oval window niche, occupied by the stapes footplate, is located anterosuperior to the ponticulus. The round window niche can be found posteroinferior to the promontory, the bulge created by the basal turn of the cochlea.

INNER EAR

The bony labyrinth houses the sensory organs and soft tissue structures of the inner ear and consists of the cochlea, three semicircular canals, and vestibule. Its bone has three layers: an inner, or endosteal, layer; an outer, or periosteal, layer; and a middle layer consisting of enchondral and intrachondrial bone. The cochlea spirals $2^{1/2}$ turns about its central axis, the modiolus, and has a height of 5 mm. The base of the cochlea abuts the fundus of the IAC and is perforated (cribrose), allowing for the passage of cochlear nerve fibers. The apex lies medial to the tensor tympani muscle. The osseous spiral lamina winds about the modiolus and, along with the basilar membrane, separates the scala media (the cochlear duct) from the scala tympani. Adjacent turns of the cochlea are separated by an interscalar septum. The three

semicircular canals are the lateral (horizontal), superior (anterior vertical), and posterior (posterior vertical). Each canal has an ampullated limb, measuring 2 mm in diameter, and a nonampullated limb, which is 1 mm in diameter. The ampulla is cribrose for passage of nerve fibers. The nonampullated limbs of the posterior and superior canals fuse to form the crus commune. The ampullated and nonampullated limbs all open into the vestibule. The angle formed by the three semicircular canals is the solid angle, whereas the triangle bounded by the bony labyrinth, sigmoid sinus, and superior petrosal sinus is known as Trautmann's triangle.

The vestibule is the central chamber of the bony labyrinth and measures 4 mm in diameter. Its medial wall is marked by depressions for the saccule (the spherical recess), utricle (the elliptical recess), and cochlear duct (the cochlear recess). Cribrose areas accommodate nerve fibre access to their sensory organs. "Mike's dot" (the macula cribrosa superior) marks the passageway for superior vestibular nerve fibres to the cristae ampullaris of the lateral and superior semicircular canals. There are three fissures of the bony labyrinth. The fissula ante fenestram is an evagination of the perilymphatic space that is invariably found extending anterosuperior to the oval window; in the adult, fibrous tissue and cartilage fill the fissula. The fissula post fenestram is a perilymphatic evagination that extends posterior to the oval window; it is a less constant feature of the temporal bone. Hyrtl's fissure (or the tympanomeningeal hiatus) is a remnant of embryologic development and is rarely present. The membranous (endolymphatic) labyrinth housed within the bony labyrinth consists of the cochlear duct (scala media), the three semicircular ducts and their cristae ampullaris, the otolithic organs (the utricle and the saccule), and the endolymphatic duct and sac.

INTERNAL AUDITORY CANAL

The IAC is the bony channel that shelters the superior and inferior vestibular, cochlear, facial, and intermediate nerves, as well as the labyrinthine artery and vein, as they course from the posterior cranial fossa to the labyrinth. The Porus is the posterior cranial fossa opening of the canal, whereas the canal abuts the bony labyrinth at its fundus. At the fundus, the vestibular, facial, and cochlear nerves are in a constant anatomic relationship that is determined by the horizontal (falciform) crest and the vertical crest ("Bill's bar").

THE PHYSIOLOGY OF HEARING

Acoustic signals are transmitted from the air of the external environment to the fluid of inner ear. The external auditory canal enhances the sound pressure levels at the tympanic membrane over a range of frequencies. The middle ear couples sound signals from the ear canal to the cochlea primarily through the action of the tympanic membrane and the ossicular chain.

Functions of the Middle Ear Cleft

Tympanic membrane

Acoustic energy collected by the large area of the tympanic membrane is applied through the ossicles to the small area of the stapes footplate. The effective ratio of this area is about 14:1. By this effect the amplitude of vibration at the stapes is reduced as compared with that of the membrane, while the forces exerted by the stapes upon the labrynthine fluid are increased in the same proportion.

Ossicles

The malleus handle is attached to the tympanic membrane. As the membrane moves in and out, the malleus and incus moves in and out by rotating about an axis through the anterior ligament of the malleus and the tip of the short process of the incus. This moves the stapes which rocks around an axis passing vertically through the posterior border of the footplate. The movement of the footplate of the stapes, produces vibration of the endolymph and perilymph in the bony labyrinth.

Middle ear muscles

The middle ear muscles include *stapedius* and *tensor tympani*. They contract under a variety of circumstances, including loud sounds, before and during

vocalization, tactile stimulation of the head or face, and fight or flight behavioural responses. Such protective contractions reduce the transmission of low frequency sound through the middle ear but have little effect on high frequency sound. Contraction of the *stapedius* muscle in response to sound is known as the *acoustic reflex*. The reflex is thought to help in speech discrimination (the reflex reduces masking by low frequency sound of high frequency stimuli) and in protecting the inner ear from acoustic trauma of loud continuous sound. Contractions of the *tensor tympani* have also been associated with opening of the eustachian tube, where the inward motion of the tympanic membrane that results from the contraction produces an overpressure in the middle ear that helps open the tube.

Middle ear mucosal folds (Figure 6)

The mesotympanum is separated from the epitympanum and mastoid by a series of mucosal folds and suspensory ligaments known as the tympanic diaphragm. Only two narrow passages known as the anterior and posterior tympanic isthmus breach this diaphragm. Patency of this connection between the epitympanum and mesotympanum is essential for the successful aeration of the middle ear and mastoid. When they are blocked by cholesteatoma, tympanosclerosis or inflammatory oedema, negative pressure develops in the mastoid followed by fluid exudation and chronic infection.

Eustachian tube function: The eustachian tube connects the middle ear cleft with the outside environment and has three important functions. These are

1. Pressure regulation in the middle ear
2. Protection of the middle ear from the pathogens in the nasopharynx
3. Clearance of the middle ear space

Normally the eustachian tube remains closed and opens during deglutition to equalize pressure in the middle ear. During swallowing, the tensor veli palatini and levator veli palatini causes medial superior rotation of the medial lamina, which in turn causes deformation of the fatty tissue surrounding the eustachian tube thus opening the eustachian tube. During each swallow the volume of gas exchange is around 1uL in a normal eustachian tube. This helps in maintaining equal pressure between the middle ear and external ear, thus enabling optimum compliance of the tympanic membrane and round window.

Mastoid air cell system

The anatomic and physiologic properties of the mastoid air cell system contribute to the homeostasis of the middle ear. First, the physical property of mastoid air volume affects compliance of the ear drum: the greater the volume, the more compliant the tympanic membrane. This property is based on Boyle's law, which states that at a constant temperature, pressure multiplied by volume equals a constant (P x V=C). Thus pressure and volume are inversely proportional. Second, the surface area of the mastoid affects mucosal respiration: the greater the surface area, the more efficient the middle ear respiration. Hence a well pneumatized large mastoid increases the respiratory properties of the middle ear by increasing the amount of gas transfer across the middle ear mucosa. The mastoid air cell system acts as a reservoir of air and provides a buffer during transient periods of eustachian tube dyfunction.[22]

The transformer system of the middle ear may be divided into three stages:

1. **Catenary lever action of the tympanic membrane:** Owing to the immobility of the bony annulus, the sound energy applied to the tympanic membrane is amplified at its central attachment, the malleus, and is directed into the ossicular chain for transmission to the perilymph

2. **Ossicular lever action:** The action that results from different lengths of rotating malleus and incus arms around the axis of rotation of the ossicles produces the amplification of sound. The movement of stapes is two thirds that of the manubrium and their lever ratio is 1.5:1.

3. **Hydraulic lever:** The sound pressure collected over the larger area of the tympanic membrane and transmitted to the smaller area results in an increase in force proportional to the ratio of the areas.

By means of this transformer action, amplitude at oval window is greatly reduced as compared with amplitude at tympanic membrane, and force (pressure) at oval window is increases in the same proportion or 18.3 times.

THEORIES OF HEARING

1. Place theory of cochlear action (Helmholtz) postulates that perception of pitch depends upon the selective vibratory action of the basilar membrane. The hair cells and the particular nerve fibres activated by them correspond to the point of maximum displacement of the membrane by the travelling wave.

2. Rutherford's telephone theory suggested that pitch perception is based upon the rate of firing in individual nerve fibres. The latent period of nervous action limits this theory to the perception of frequencies below 1000 Hz only, if the relation between sound wave frequency and nerve impulses has a simple 1:1 ratio.

3. Waver's volley theory combines both place and telephone principles, postulating that high frequencies are perceived by place alone (in basal turn) and low frequencies

(below 1000 Hz) stimulate nerve action potentials at a rate equal to the stimulus frequency.

4. Bekesy's travelling wave theory states that a 'wave' travels along the basilar membrane from base to the apex, reaches a peak, and then decays rapidly. This travelling wave took time to reach its maximum displacement and thereby introduced a phase delay between the stimulus entering the cochlea and the peak of basilar membrane displacement.

5. Eward's wave theory says that basilar membrane gets segmental standing waves which will have nodes and antinodes when stimulated by sound. As the segmentation increases with frequency, so the distance between nodes decreases. It is possible that discrimination of pitch depends on the antinodes patterns produced.

AETIOLOGY & PATHOGENESIS OF CHRONIC OTITIS MEDIA

a) Environment: There is a close correlation between patients with chronic otitis media and socioeconomic status, with the lower groups having a higher incidence of otitis media because of poor general health, poor diet and overcrowding at home.

b) Previous otitis media: Chronic otitis media can occur as a sequela of acute otitis media or otitis media with effusion. Repeated attacks of acute otitis media or otitis media with effusion causes chronic retraction of the tympanic membrane and loss of fibrous tissue layer which predisposes the tympanic membrane for chronic otitis media.

c) Infective: Bacteria in ears with chronic otitis media are secondary invaders of a mucosa which is inflamed, because of other factors, rather than primary cause of the disease. Bacterial invasion can produce substances that affect ciliary function and hence would encourage stasis of secretions in the middle ear.

d) Upper respiratory tract infection: Viral infections of the upper respiratory tract, would also affect the mucosa of the middle ear, making it less resistant to the organisms that are normally present in the middle ear, allowing bacterial overgrowth.

e) Autoimmunity: It seems likely that individuals with established autoimmune disease will have a higher incidence of chronic otitis media.

f) Allergy: Though postulated by some authors as a causative factor it remains to proven that allergic individuals have a higher incidence of chronic otitis media than non allergic subjects.

g) Eustachian tube: Differences exists in the partial pressure of gases in the middle ear and in the surrounding tissues, which result in the continuous absorption of small

amounts of oxygen and nitrogen across the mucous membrane. With the constant absorption of air in the middle ear, a tendency exists for the development of negative pressures within the middle ear cleft. Normally this pressure is equalized via the eustachian tube which opens about 30 times per hour permitting entry of approximate 1µL for each deglutition and 80µL for each yawn. Should eustachian tube obstruction occur, negative pressures will develop leading to transudation of fluid, collapse of the middle ear space, and frequent middle ear infections.

h) Mastoid air-cell system: The association of mastoid pneumatization and chronic middle ear infection is well recognized. The most important association is that the mastoid acts as "air reservoir" for middle ear in case of eustachian tube block. The mastoid air cell system provides a pneumatic reservoir upon which the middle ear can draw air during periods of eustachian tube dysfunction. This mastoid air cell volume may slow or prevent the development of negative middle ear pressures associated with transient eustachian tube dysfunction. In the total absence of eustachian tube function, no amount of air reservoir will prevent the ultimate development of severely negative middle pressures and resultant middle ear disease. However, mastoid size becomes clinically important in ears with partial or intermittent eustachian tube function. In this situation the mastoid air cell volume compensates for the decreased air cell volume in the middle ear. Even a well pneumatized mastoid may be rendered ineffective by inflammatory disease which blocks the aditus-ad-antrum, thereby disconnecting the middle ear and mastoid cavities thereby hampering ventilation of the middle ear.

MATERIALS AND METHODS

This is an observational study with historical controls conducted in the Department of Otorhinolaryngology and Head and Neck Surgery, R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar attached to Sri Devaraj Urs Medical College. A prior clearance from the Institutional Ethical Committee was taken before the commencement of the study. All consecutive patients with tubotympanic disease attending the ENT clinic between October 2009 and September 2010 (one year) were included in the trial group and underwent Tympanoplasty (TP) whereas all patients who underwent Tympanoplasty with Cortical mastoidectomy (TM) for tubotympanic disease between October2007-September2009 (2years) were included as the historical controls. All patients were followed-up for a minimum period of 12 months.

Patients who entered the study were selected on the basis of the following **inclusion criteria**.

a) Patients with a clinical diagnosis of Chronic Suppurative Otitis Media (CSOM) of the Tubotympanic type without complications.

b) Patients of either sex, 18 years of age and above.

The **exclusion criteria** for patients in this study were as follows.

a) Patients with features of atticoantral disease.

b) Patients with positive mastoid reservoir sign.

c) Patients presenting with associated features of complications like vertigo, facial nerve palsy, intracranial or extracranial abscess.

d) Patients with a previous history of surgery of the same ear.

e) Patients with associated sino-nasal diseases.

At the end of the study period of one year (2009-2010) 40 patients were part of the trial group. From the preceding two years (2007-2009) the case records of 60

consecutive patients who were followed up for a minimum of 1 year were included as historical controls.

All patients included in the study were subjected to a detailed Otorhinolaryngological examination to rule out other source of infection in the nose, paranasal sinus and pharynx and associated complications of CSOM. A plain X-ray of the mastoid in lateral oblique view was done to assess the extent of pneumatization and position of dural and sinus plates. Aural swab culture and sensitivity was done in all actively discharging ears and they were treated with culture specific antibiotic for 7 days and 1.5% vinegar aural wash on alternate days for 10 days. Routine haematological and biochemistry investigations were done to assess fitness for surgery.

Definition of a case In this study, a case is defined as a patient who has undergone Tympanoplasty.

Definition of a control In this study, a control is defined as a patient who has undergone Tympanoplasty with Cortical mastoidectomy.

Success is defined as

a) Subjective resolution of all symptoms of tubotympanic disease for a minimum period of 12 months after surgery.

b) Successful graft uptake, showing a healthy neotympanum as visualized with an otoscope, 12 months after surgery.

Failure is defined as

a) Persistence or recurrence of the symptoms during the minimum follow-up period of 12 months.

b) Failure of graft uptake or reperforation of the neotympanum during the follow-up period.

SURGICAL TECHNIQUE

Preoperative Evaluation Patients with history of ottorrhea and decreased hearing are thoroughly examined in the department of Otorhinolaryngology. Features suggestive of complications of CSOM and mastoid reservoir sign were ruled out. Patients with associated nasal allergy or sino-nasal disease are excluded. Using suction, the purulent discharge and squamous debris from the ear canal was cleaned to visualize the tympanic membrane and middle ear mucosa. Assessment of the disease was done using an otoscope. A fistula test was performed by doing seigelization, especially for patients with history of vertigo. Overlying acute infection was treated with systemic antibiotics and 1.5% vinegar ear wash given on alternate days for 10 days.

Instruments A standard Tympanoplasty set of instruments was used as shown in figure 7.

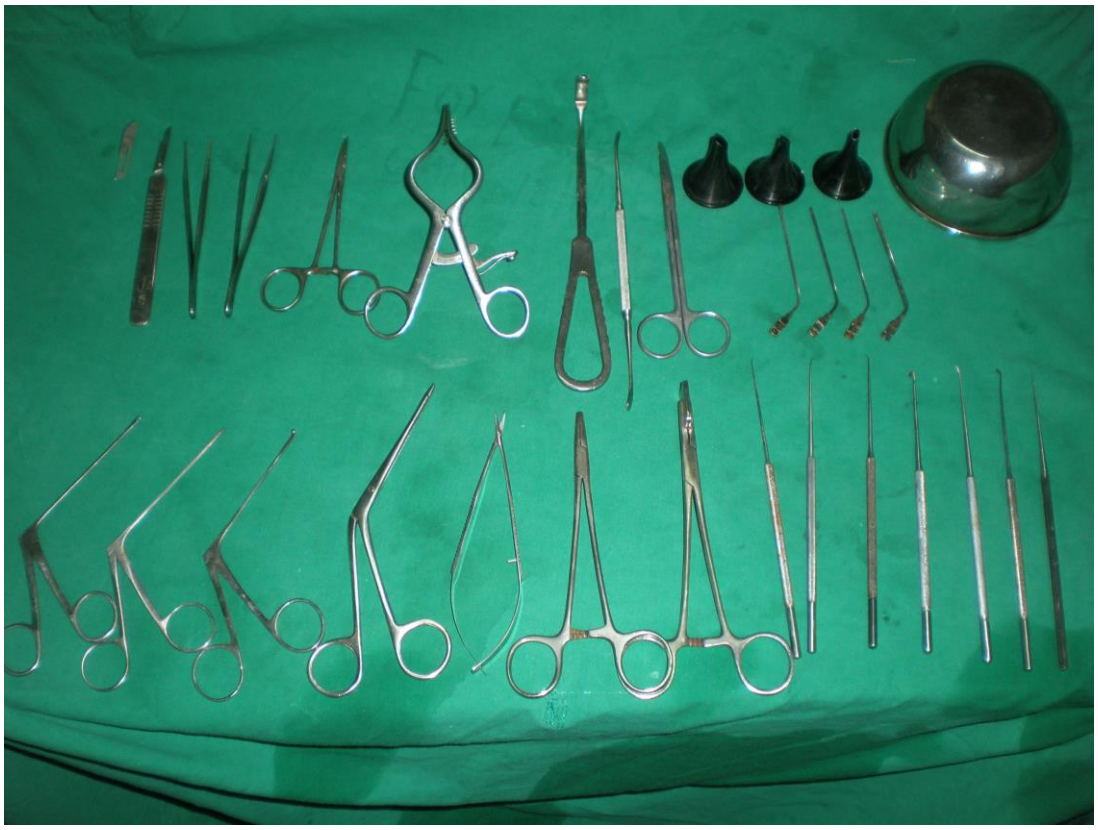


Figure 7: Tympanoplasty instruments

A ZEISS-MOVENA S7 operating microscope was used for all patients of both groups (figure 8).

Figure 8: Zeiss MovenaS7 Operating Microscope

Operation theatre layout: The surgeon is seated comfortably with his back straight in a chair with a good back rest. Scrub nurse stands at the right hand side of the operating table with the instrument trolley. The video monitor (Sony Bravia) is positioned such that it is easily visible to assisting surgeon, students and to the scrub nurse to enable her to foresee the surgical steps and assist actively (figure 9).

Figure 9: Operation theatre layout

Anaesthesia: All cases were operated under local anaesthesia.

Premedication: A mixture of pethidine 1.5mg/kg body wt, promethazine 25mg and atropine 0.6mg were given deep intramuscularly in the gluteal region 45 minutes before surgery. Pethidine is used for its analgesic and sedative properties. Promethazine acts as an antiemetic agent in addition to its sedative properties. Atropine is given to decrease secretions, it also acts as a vagolytic (cardioprotective).

Anaesthetic solution: The local anaesthetic preparation consists of premixed 2% Xylocaine with adrenaline in a concentration of 1:100,000. A mixture of 4% topical Xylocaine with adrenaline was used to anaesthetise the middle ear mucosa.

Operative Techniques

Preparation To isolate the surgical field, hair about one inch above and behind the auricle on the side of surgery, is shaved. Two long plastipore adhesive tapes are placed one inch above the hair lines to prevent the hair falling in the surgical field. The patient lies supine with the head turned away from the surgeon, close to the edge of the table, so that the operating ear faces upwards. The operative site is painted with povidone iodine solution and surgical spirit. The patient is then draped with sterile surgical towels (figure 10). The nose and mouth are left uncovered to enable the patient to breathe freely and to monitor facial movements.

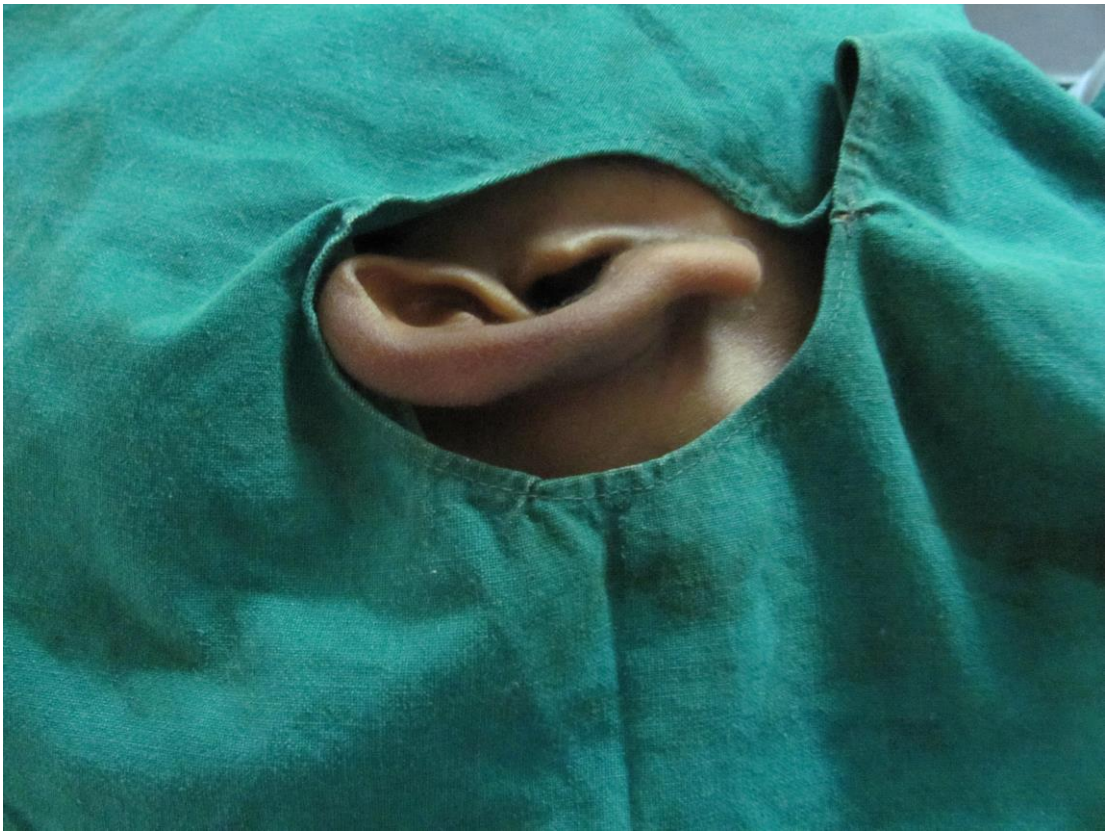


Figure 10: Draping the ear

Using 2ml disposable syringe, 1ml of above solution is infiltrated slowly at 3, 6, 9, and 12 o'clock position of external auditory canal at bony cartilaginous junction. Half ml of the solution is injected each in the helico-tragal junction and anterior to tragus. Post auricular infiltration consists of injecting along the post auricular sulcus, in the temporalis region and under the periosteum over the mastoid cortex.

Surgical Steps

Canal incisions and elevation of posterior meatal skin flap: The external auditory canal and the tympanic membrane are exposed using Shea's aural speculum. Using Rosen's round knife a circumferential incision is made from 12'o'clock to 6'0'clock position about 6mm from the fibrous annulus (figure 11). Now using 15 number blade vertical incisions are made and a rectangular posterior meatal skin flap is elevated laterally up to bony cartilaginous junction.

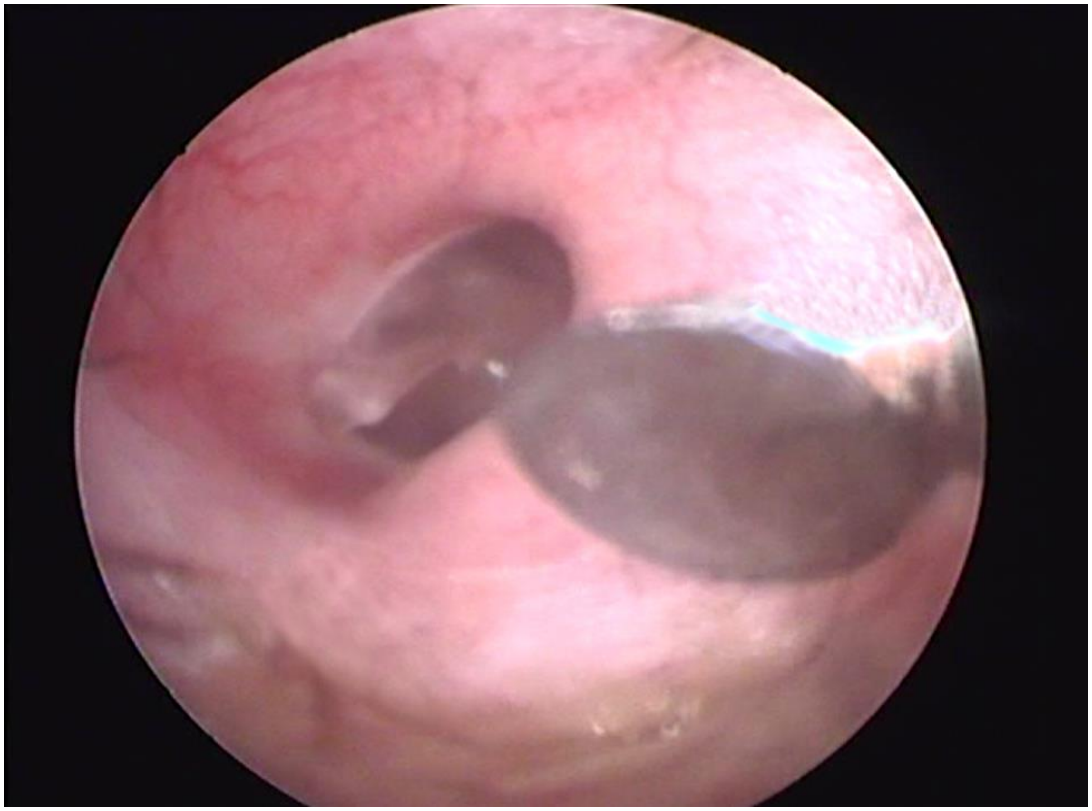


Figure 11: Canal incision using Rosen's round knife

Post-aural incision and temporalis fascia harvest: The post auricular incision is made about 5mm behind the post-aural sulcus as described by William Wilde (figure 12). The plane of temporalis fascia is identified and is elevated from the underlying muscle by injecting saline underneath the fascia. The temporalis fascia measuring 1" X 3/4" in size is harvested leaving around half an inch of fascia near the temporalis muscle attachment along the linea temporalis (figure 13).

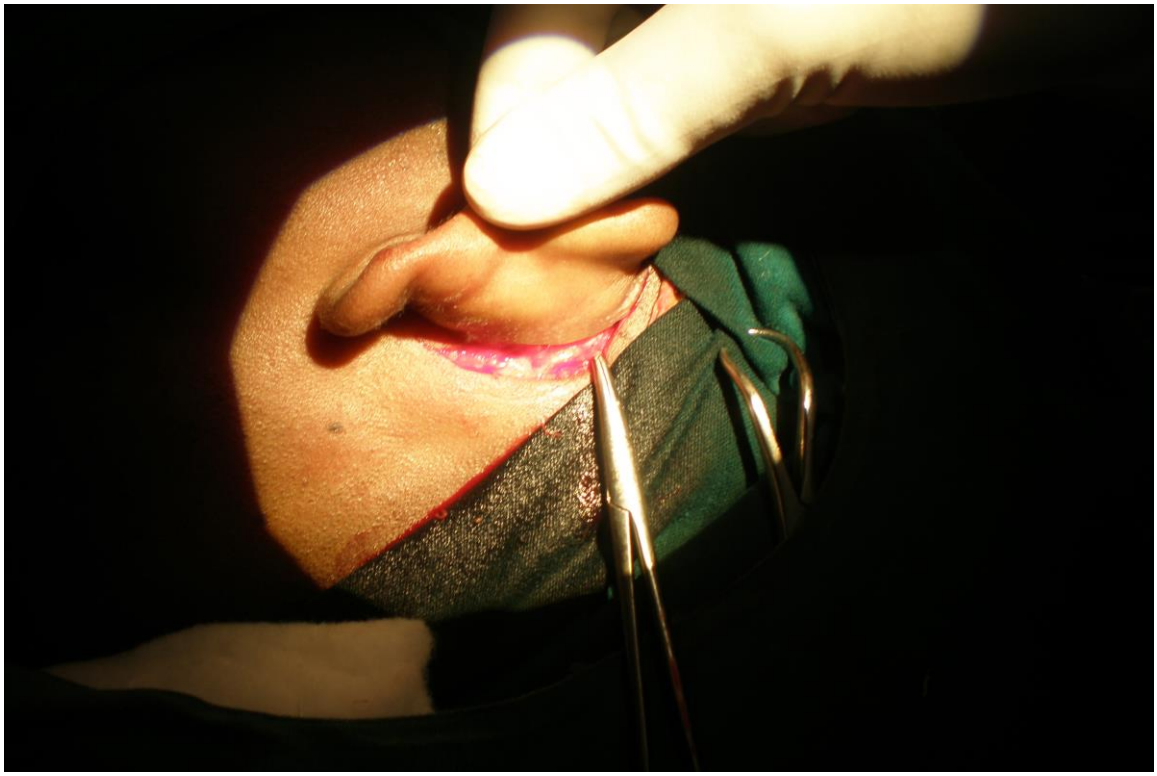


Figure 12: William Wilde's postaural incision

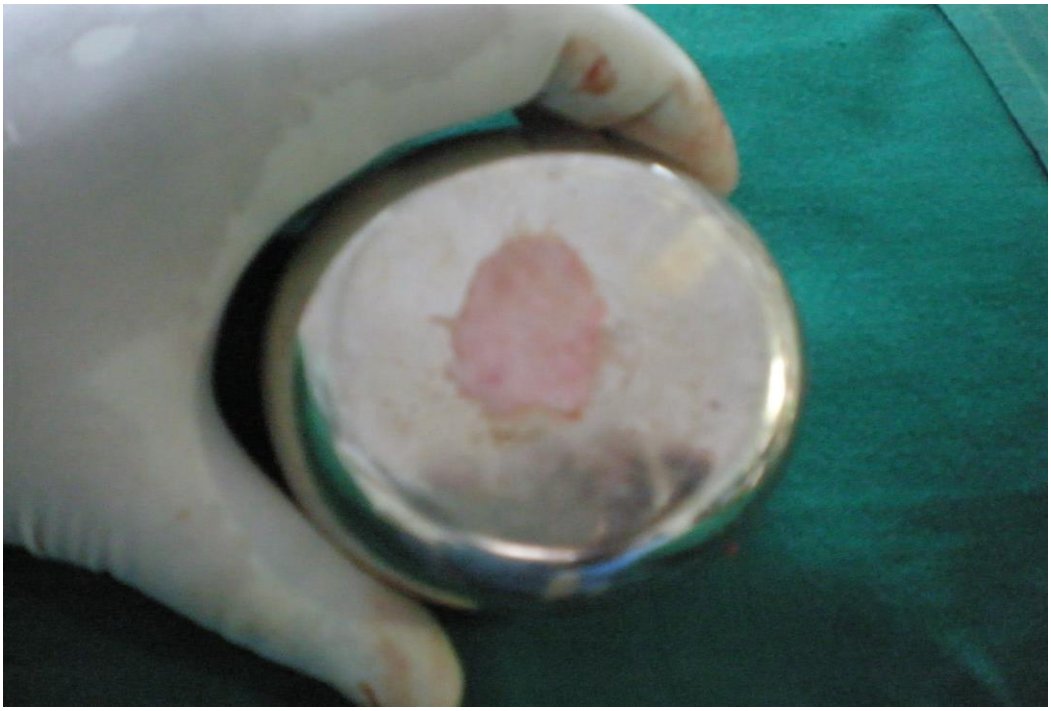


Figure 13: Harvested temporalis fascia graft

Subcutaneous tissue incision and exposure of middle ear: The curvilinear post-aural incision of William Wilde is deepened down to the mastoid bone (post-aural approach). The post-aural subcutaneous tissue with periosteum is separated from the posterior bony canal wall. The cartilaginous canal along with the posterior meatal skin flap is separated from its attachments at spine of Henle, tympanomastoid suture and tympanosquamous suture by sharp dissection using a 15 number blade. The posterior meatal skin flap is now retracted laterally with a Plester's canal wall retractor to expose the canal and middle ear (figure 14).

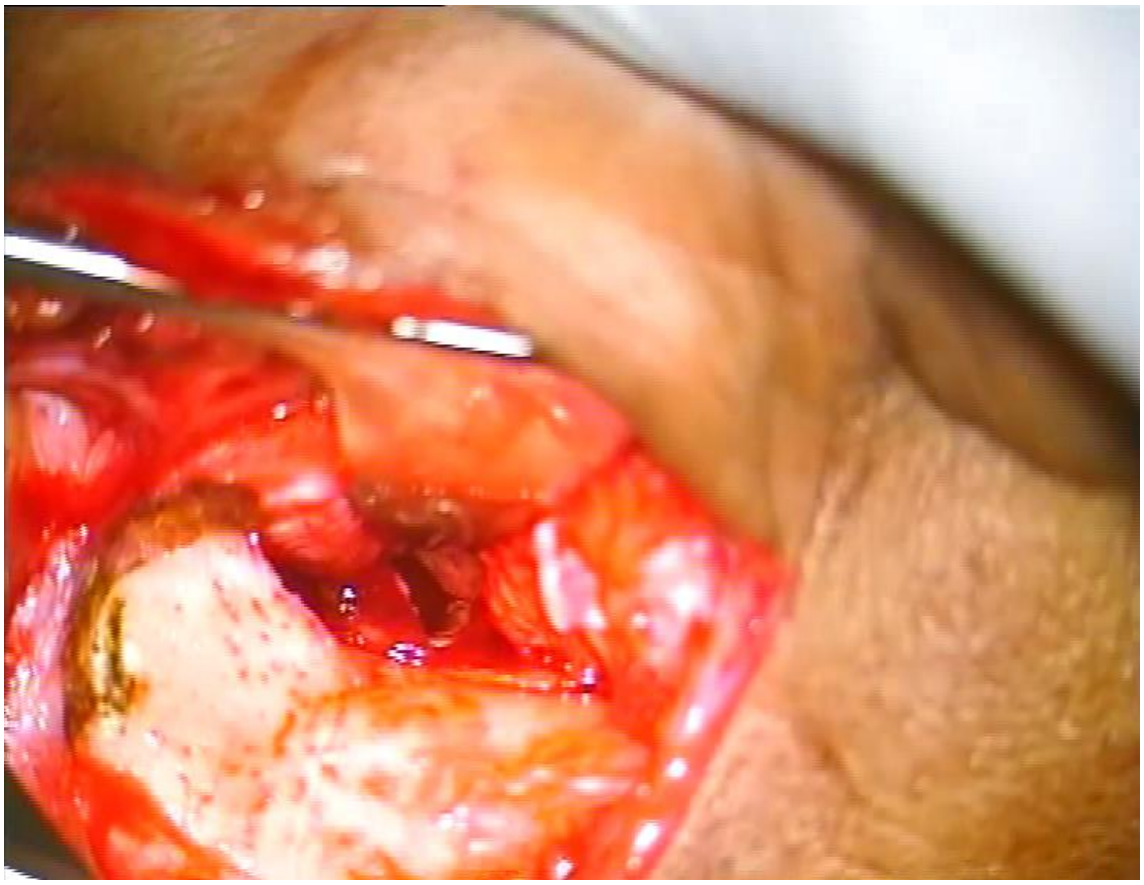


Figure 14: Posterior meatal skin flap retracted

Superior and Inferior Tympanomeatal incision: Superior and inferior meatal incision is made using Plester's flap knife. This incision joins the previously made superior and inferior meatal skin flap.

Scraping of undersurface of Tympanic Membrane and Freshening the Margins of Perforation: The mucosa in the undersurface of the remnant tympanic membrane is scraped through the perforation using Plester's flap knife. The margins are excised by small sickle knife and microscissors.

Elevation of Anteriorly Based Tympanomeatal Flap: The semi-circular tympanomeatal flap is now elevated first from the posterior bony canal wall in a lateral to medial fashion till the fibrous annulus is reached using oval angulated canal elevator.

Assessment of Ossicular Chain: The incudostapedial joint is visualized by drilling the bony overhanging of the posterior bony rim and posterior part of lateral attic wall. The status of the ossicular chain is evaluated for continuity and mobility by palpating each ossicle individually with a smooth curved pick and eliciting the round window reflex.

Preparation of Graft Bed: The external auditory canal is thoroughly irrigated with normal saline to remove all free squamous debris and bacteria. Complete haemostasis is achieved in the middle ear, external ear and subcutaneous tissue before placement of temporalis fascia. Gelfoam soaked in drops containing a mixture of Neomycin, Polymyxin-B, Bacitracin and Hydrocortisone was placed in the middle ear.

Underlay Temporalis Fascia Grafting with Exteriorization of Malleus Handle: Underlay technique implies placing of the graft medial to remnant of tympanic membrane. The dried temporalis fascia is cut according to the size and shape of canal. A slit is made in the temporalis fascia graft about 6 to 7 mm from the tip of the graft for exteriorization of malleus handle. The temporalis fascia is then rehydrated by dipping it in saline and then immediately placed under the already elevated tympanomeatal flap. The malleus handle is exteriorized through the slit made in the temporalis fascia.

Repositioning of Posterior Meatal Flap: The Plester's retractor is now removed and the posterior meatal flap is repositioned over the temporalis fascia lying over the posterior bony canal wall. The edges are straightened and the flap is positioned properly over the temporalis fascia.

Canal Pack: The external auditory canal is packed with gelfoam soaked in drops containing Neomycin, Polymyxin-B sulphate, Bacitracin and Hydrocortisone

followed by cotton impregnated with the same ointment which will keep the gelfoam in place.

Closure of Wound: The postaural periosteum is sutured using 2-0 plain catgut (cutting needle). The same suture material is used for subcutaneous tissue suturing. Skin is closed with 3-0 mersilk (cutting needle) and a standard mastoid dressing is applied. Patient is then shifted to the ward.

Post-operative care: A broad spectrum oral antibiotic such as ciprofloxacin 500mg twice a day is given along with oral antihistamines and analgesics for three days. Patients are discharged on the 3rd post-operative day after change of mastoid dressing. All patients were instructed to avoid soiling the mastoid dressing and are advised to continue ciprofloxacin upto 7th post-operative day.

Follow-up: The patient is reviewed on the 7th post-operative day for suture and canal pack removal. The patient is re-examined at weekly intervals for one month and again after 3 months from the date of surgery. At each follow-up, the following factors are assessed, subjective resolution of the symptoms, persistence of discharge and status of the graft.

Complications of Tympanoplasty Faulty surgical techniques, traumatic handling of tissues, poor knowledge, anomalies of local anatomy, improper placement of graft and infections can lead to complications. Complications that occur during and after tympanoplasty are discussed below.

Bleeding Excessive bleeding can occur during surgery. This can be due to inadequate preoperative preparation of the patient, using less than required amounts of vasoconstrictor such as adrenaline in the local anaesthetic infiltration, local inflammation, poor handling of tissues and poor control of pre-existing Hypertension.

Bleeding can usually be controlled by cauterization or by placing an adrenaline soaked cotton ball over the bleeding site followed by gentle pressure over it.

Stapes footplate fracture/Subluxation Excessive manipulation of stapes while removing the polypoidal mucosa can result in stapes fracture or subluxation resulting in iatrogenic sensorineural hearing loss and vertigo.

Facial nerve injury Failure to recognise a dehiscent/anomalous facial nerve and inadvertent injury to the nerve can cause facial nerve palsy.

Postoperative Infection Primary post-operative infection of wound or the fascia graft can occur rarely. This can be prevented by routine broad spectrum antibiotic prophylaxis, middle ear packing with gelfoam soaked in antibiotic-steroid solution and intermittent saline wash to clear the bone dust, blood clots and squamous debris.

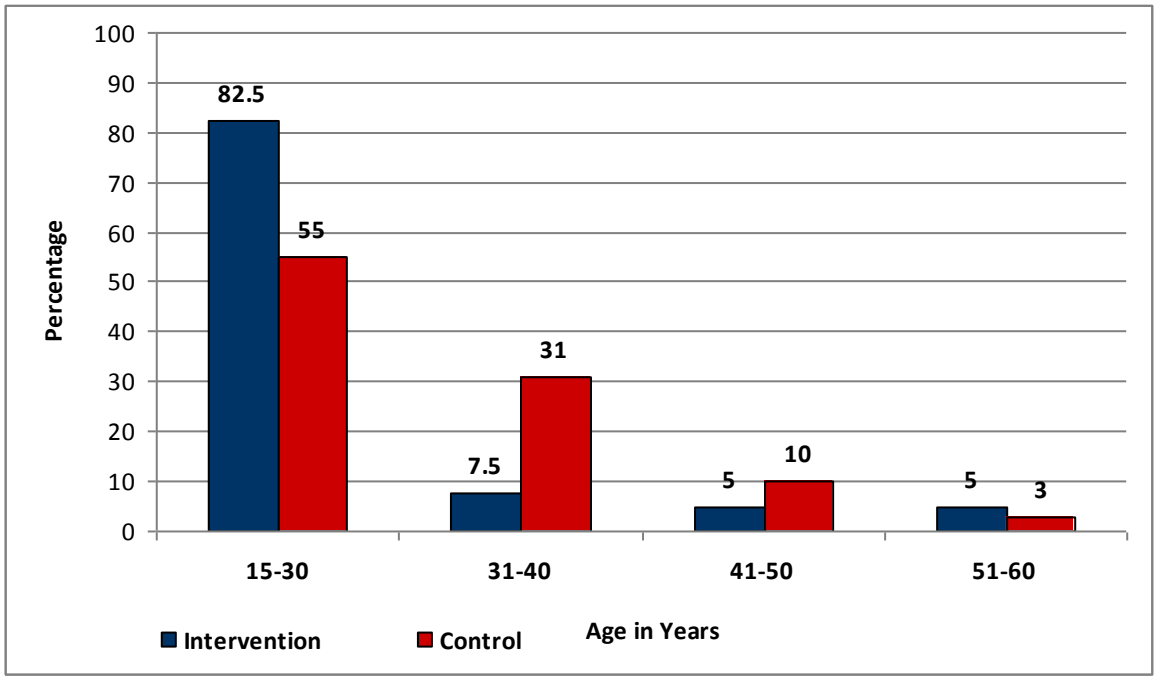
OBSERVATIONS AND RESULTS

AGE DISTRIBUTION

The age distribution in the trial group and control group is shown in Table 1. The age of the patients in the study sample varied from 18yrs to 60 years. The mean age in the trial group and control group was 33 years and 32.4 years respectively. Most patients in both groups belonged to the age group of 18 – 30 years.

Table 1: Age distribution

AGE DISTRIBUTION	TRIAL GROUP (n=40)	Control group (n=60)
18-30 years	33 (82.5%)	33 (55%)
31-40 years	03 (7.5%)	19 (31%)
41-50 years	02 (5.0%)	06 (10%)
51-60 years	02 (5.0%)	02 (3.0%)
Total	**40**	**60**
Mean Age	33	32.4



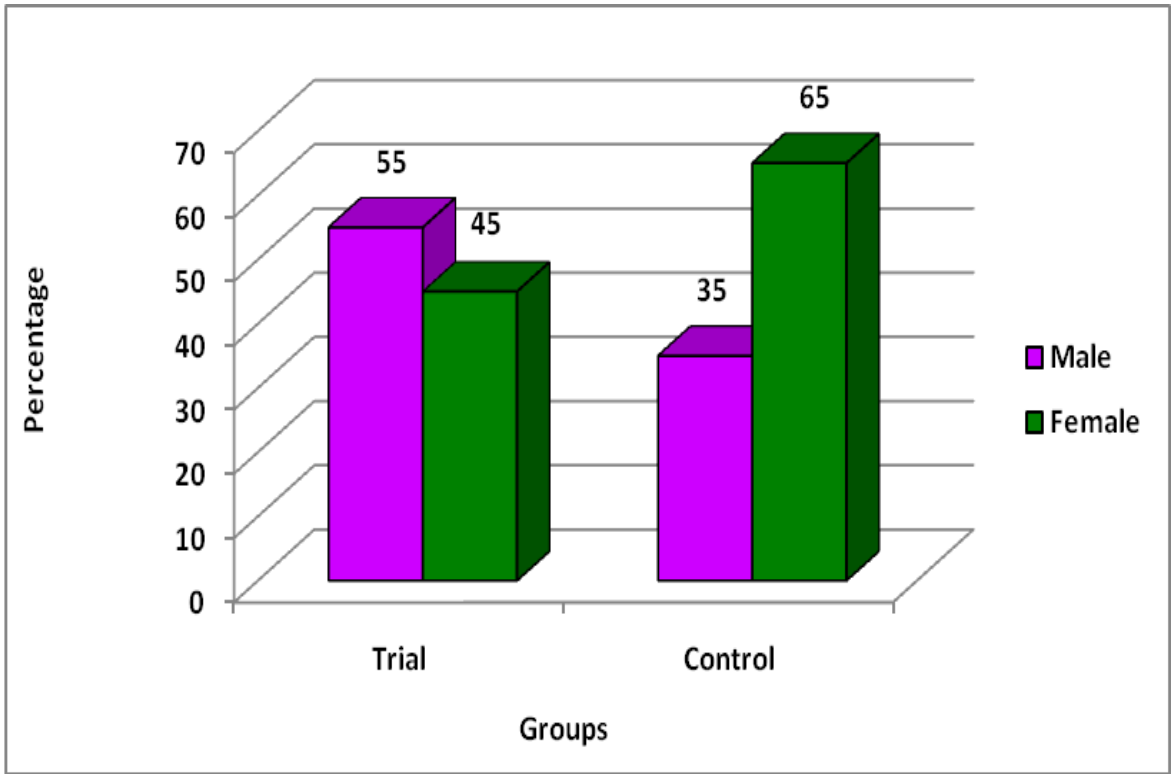
Graph 1: Age Distribution

SEX DISTRIBUTION

There were 43 male patients and 57 female patients in the study. The trial group had 22 (55%) males and 18 (45%) females, whereas the control group had 21(35%) males and 39 (65%) females as shown in table 2.

Table 2: Sex Distribution

Sex	Trial group (n=40)	Control group (n=60)	Total
Male	22 (55%)	21 (35%)	43
Female	18 (45%)	39 (65%)	57
Total	**40**	**60**	**100**



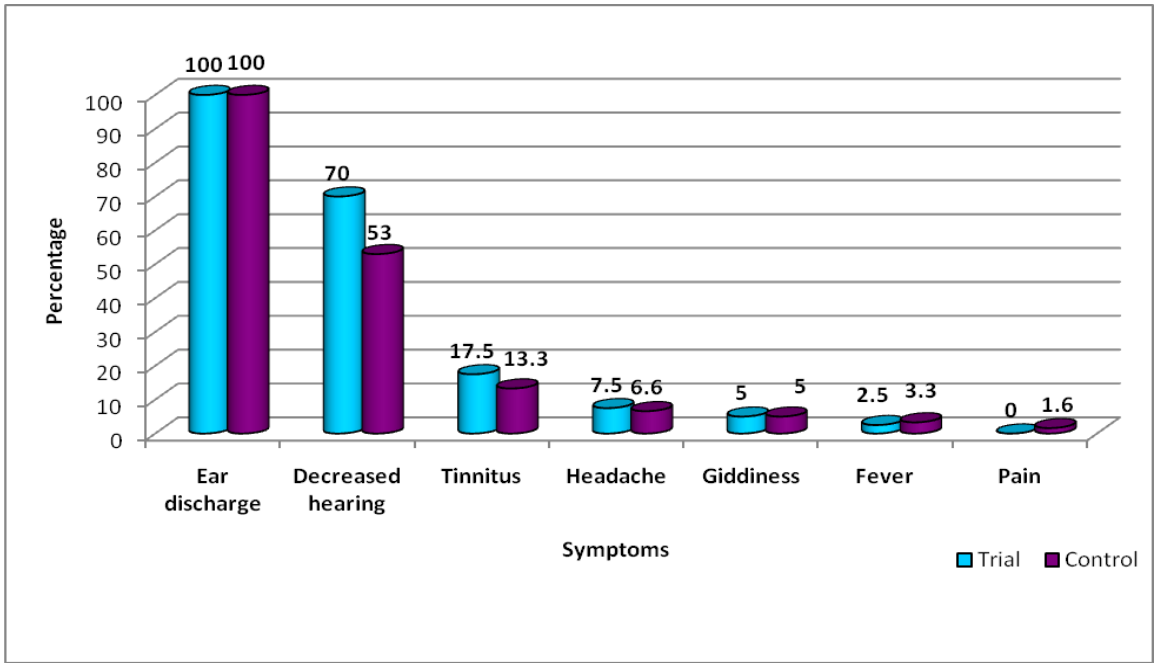
Graph 2: Sex Distribution

SYMPTOMS

All patients were evaluated for aural discharge, decreased hearing, pain, tinnitus, vertigo, and headache. All the patients in both the trial group and control group had ear discharge. Decreased hearing was found in 28 (70%) patients in trial group and 32 (53%) patients in the control group. The third most common symptom in both the groups was tinnitus with 7 (17.5%) patients in the trial group and 8 (13.3%) patients in the control group. The frequency of symptoms in the trial group and control group are given in Table 3.

Table 3: Symptoms

SYMPTOMS	Trial group (n=40)	Control group (n=60)
Ear discharge	40 (100%)	60 (100%)
Decreased hearing	28 (70%)	32 (53%)
Tinnitus	7 (17.5%)	8 (13.3%)
Headache	3 (7.5%)	4 (6.6%)
Giddiness	2 (5%)	3 (5%)
Fever	1 (2.5%)	2 (3.3%)
Pain	0	1 (1.6%)



Graph 3: Symptoms

CLINICAL EXAMINATION

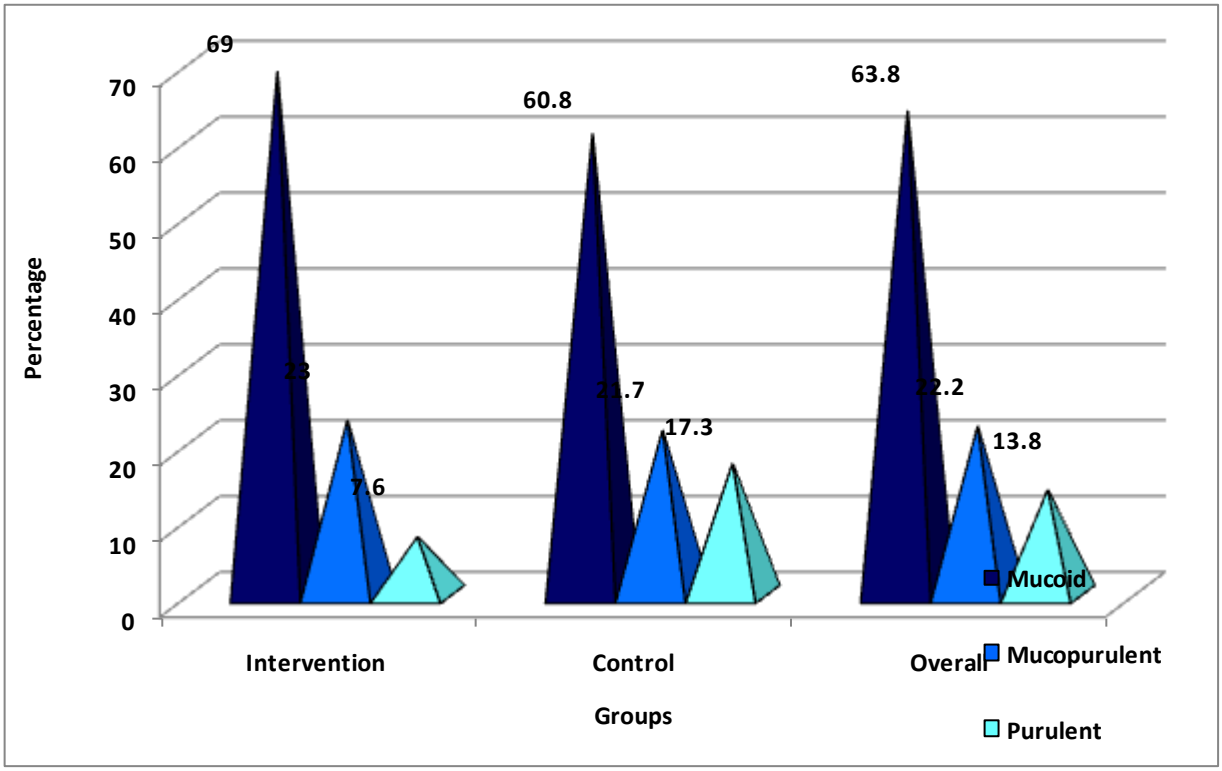
All patients were examined by an ENT surgeon in the ENT OPD of R L Jalappa Hospital. In patients with active discharge, aural swab was sent for culture and sensitivity after noting the type of discharge. Subsequently aural toileting was done and the size of tympanic membrane perforation and status of middle ear mucosa was noted. The findings on clinical examination are shown in table 4 to table 7.

Type of Discharge

In the trial group, 26 (65%) patients had active ear discharge and among them 18 (69%) patients had mucoidal ear discharge, 6 (23%) patients had mucopurulent ear discharge and 2 (7.6%) patients had foul smelling purulent discharge. In the control group 46 (76%) patients presented had active ear discharge on examination. Among them 28 (60.8%) patients had mucoidal ear discharge, 10 (21.7%) patients had mucopurulent ear discharge and 8 (17.3%) patients had purulent ear discharge.

Table 4: Type of Discharge

Type of Discharge	Trial Group (n=40)	Historical Controls (n=60)	Total
Mucoid	18 (69%)	28 (60.8%)	46 (63.8%)
Mucopurulent	6 (23%)	10 (21.7%)	16 (22.2%)
Purulent	2 (7.6%)	8 (17.3%)	10 (13.8%)
Total	**26**	**46**	**72**



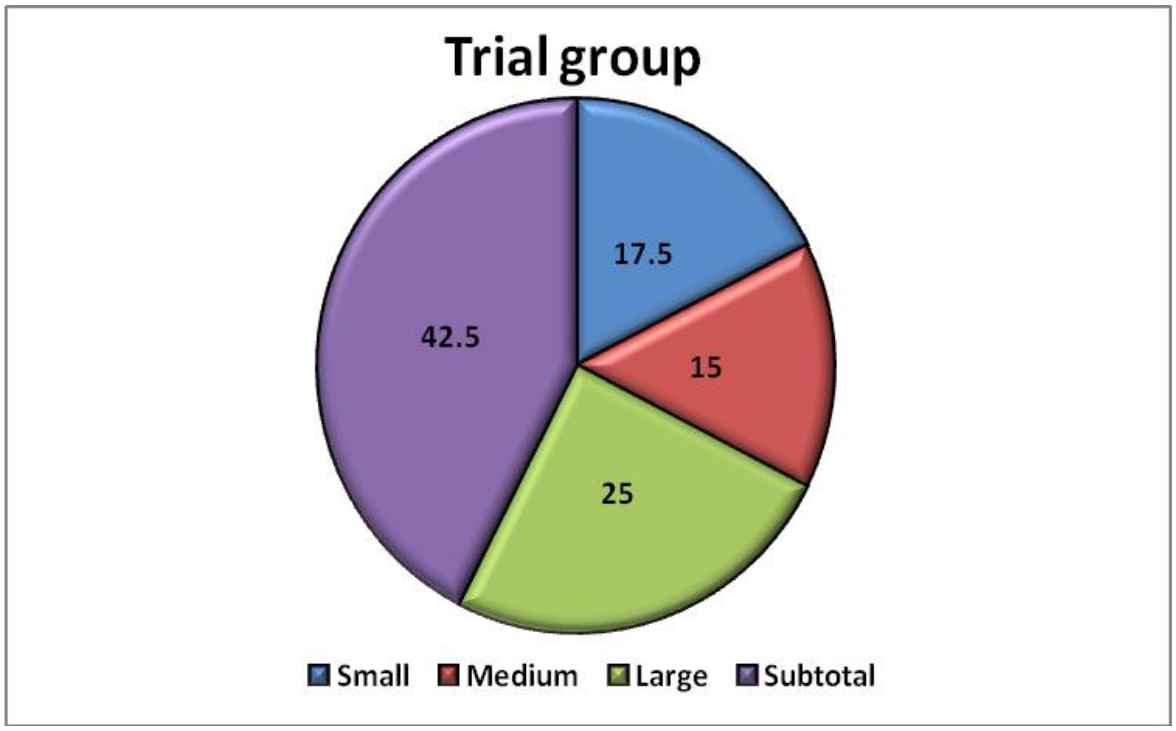
Graph 4: Type of Discharge

SIZE OF PERFORATION

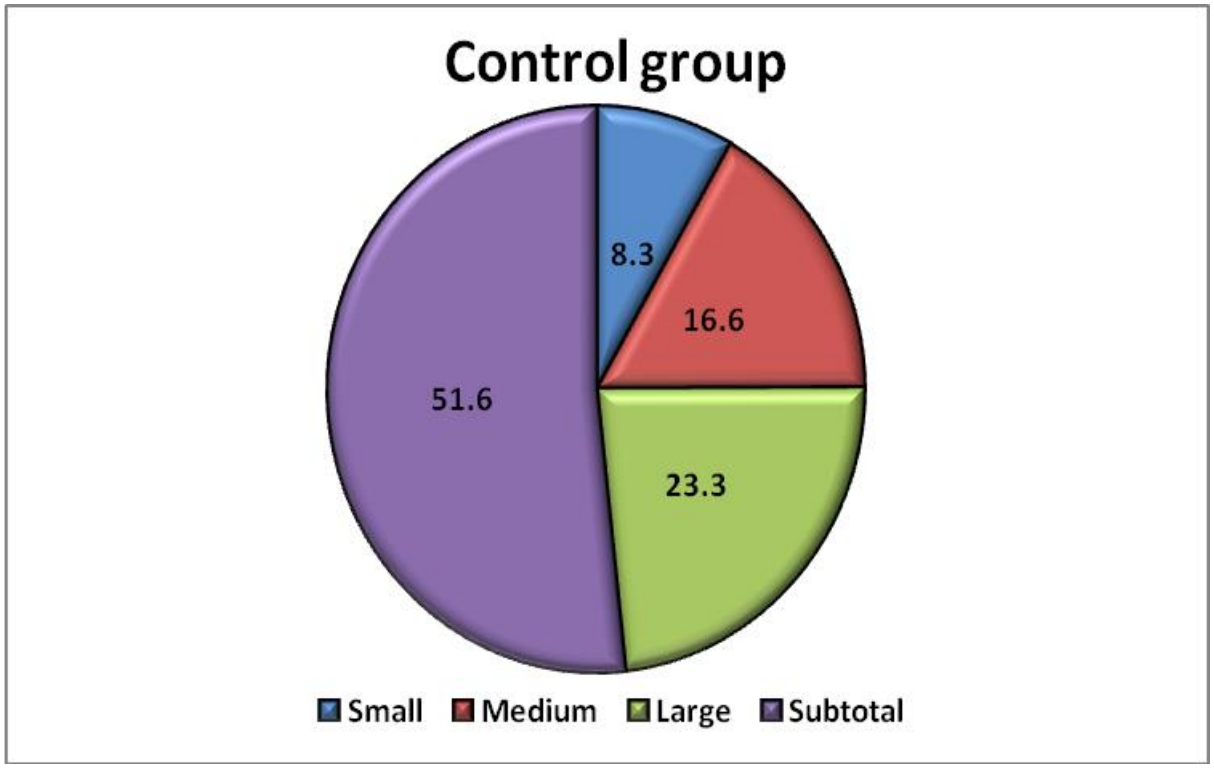
Of the total 40 patients in the trial group, 7 (17.5%) patients had small perforation, 6 (15%) patients had medium perforation, 10 (25%) patients had large perforation and 17 (42.5%) patients had subtotal perforation (table 5.1). In the control group, the following distribution of perforation was found preoperatively, 5 (8.3%) patients had small perforation, 10 (16.6%) patients had medium perforation, 14 (23.3%) patients had large perforation and 31 (51.6%) patients had subtotal perforation (table 5.2).

Table 5: Perforation Size

Perforation size	Trial Group (n=40)	Historical Controls (n=60)	Total
Small	7(17.5%)	5(8.3%)	12
Medium	6(15%)	10(16.6%)	16
Large	10(25%)	14(23.3%)	24
Subtotal	17(42.5%)	31(51.6%)	48
Total	**40**	**60**	**100**



Graph 5.1: Size of perforation



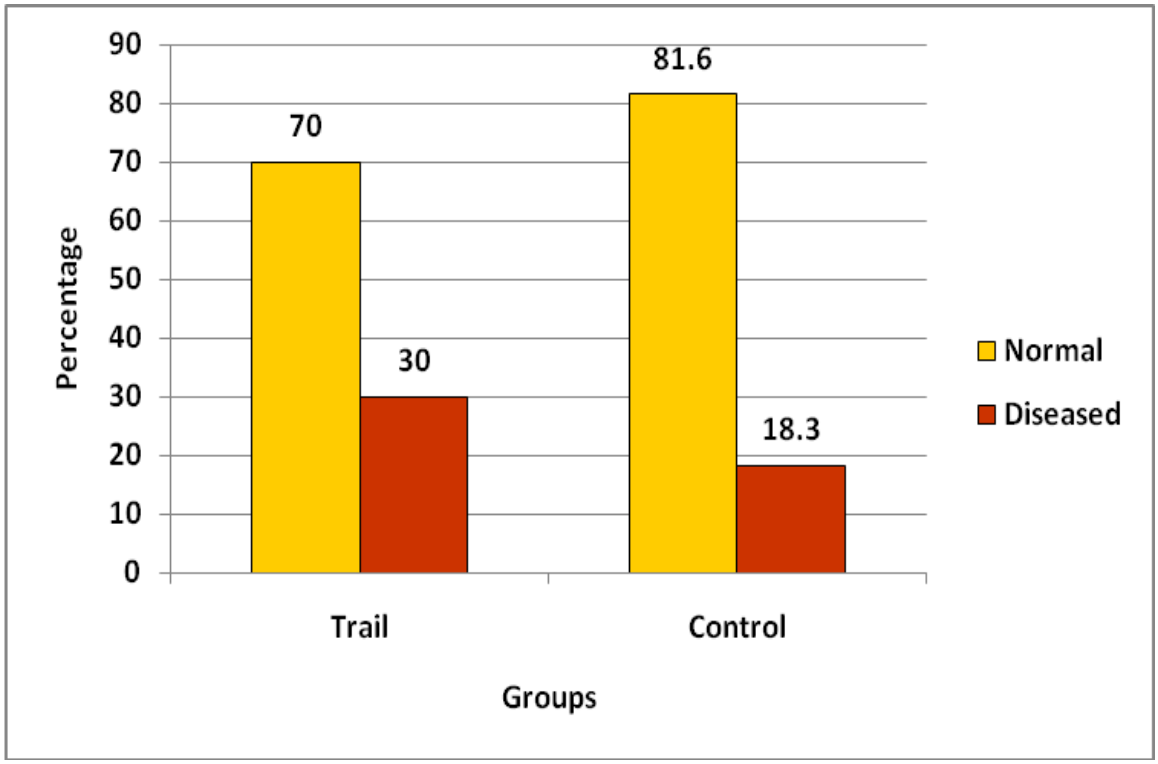
Graph 5.2: Size of perforation

MIDDLE EAR MUCOSA

Majority of patients in both the groups had normal middle ear mucosa with 28 (70%) patients in the trial group and 49 (81.6%) patients in the control group having normal middle ear mucosa. Twelve (30%) patients in the trial group and 11 (18.3%) patients in the control group had diseased middle ear mucosa.

Table 6: Condition of Middle Ear Mucosa

Middle Ear Mucosa	**Trial Group (n=40)**	**Historical Controls (n=60)**	**Total**
Normal	28 (70%)	49 (81.6%)	77
Diseased	12 (30%)	11 (18.3%)	23
Total	**40**	**60**	**100**



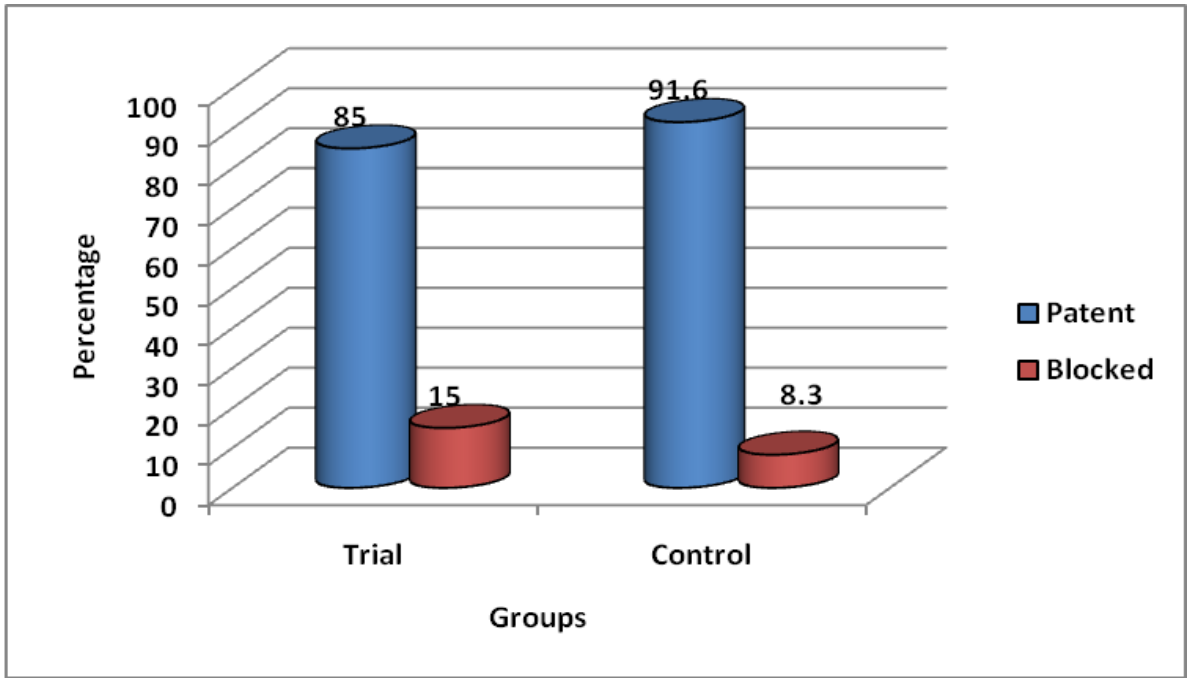
Graph 6: Condition of Middle Ear Mucosa

EUSTACHIAN TUBE PATENCY

Eustachian tube patency was assessed by asking the patient of bitter taste in the mouth after instilling Ciprofloxacin ear drops into the diseased ear canal. All 40 (100%) patients had patent eustachian tube in the trial group. On the other hand in historical controls 58 (96.6%) patients had patent eustachian tube.

Table 7: Eustachian Tube Patency

Eustachian Tube Patency	**Trial Group (n=40)**	**Historical Controls (n=60)**	**Total**
Patent	38 (85%)	55 (91.6%)	93
Blocked	2 (15%)	5 (8.3%)	7
Total	**40**	**60**	**100**



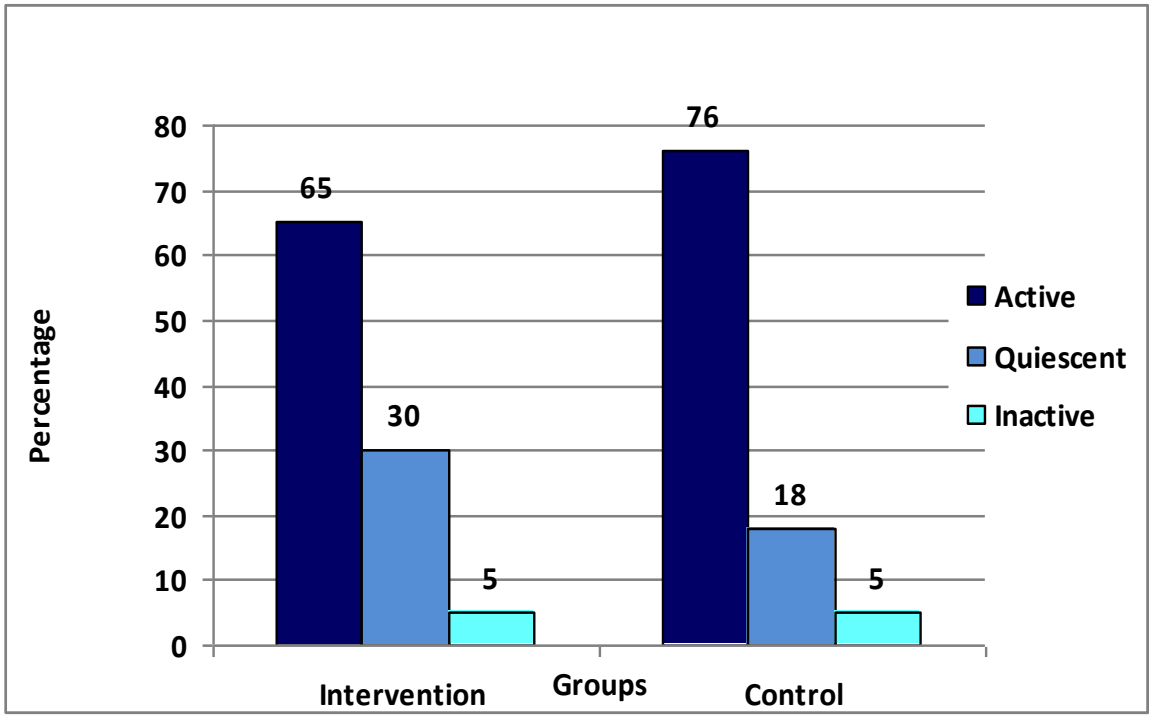
Graph 7: Eustachian Tube Patency

STAGE OF DISEASE

All 100 patients were grouped on the basis of stage of disease. Patients with aural discharge at the time of presentation were included in the *active stage* group, all the patients with no evidence of discharge at the time of presentation but with history of aural discharge in the previous six months were included in the *quiescent stage* group and those patients with no history of discharge during the past six months were said to be in the *inactive stage.*

Table 8: Stage of Disease

Stage of Disease	Trial Group (n=40)	Historical controls (n=60)	Total
Active	26 (65%)	46 (76%)	72
Quiescent	12 (30%)	11 (18.3%)	23
Inactive	2 (5%)	3 (5%)	5
Total	**40**	**60**	**100**



Graph 8: Stage of Disease

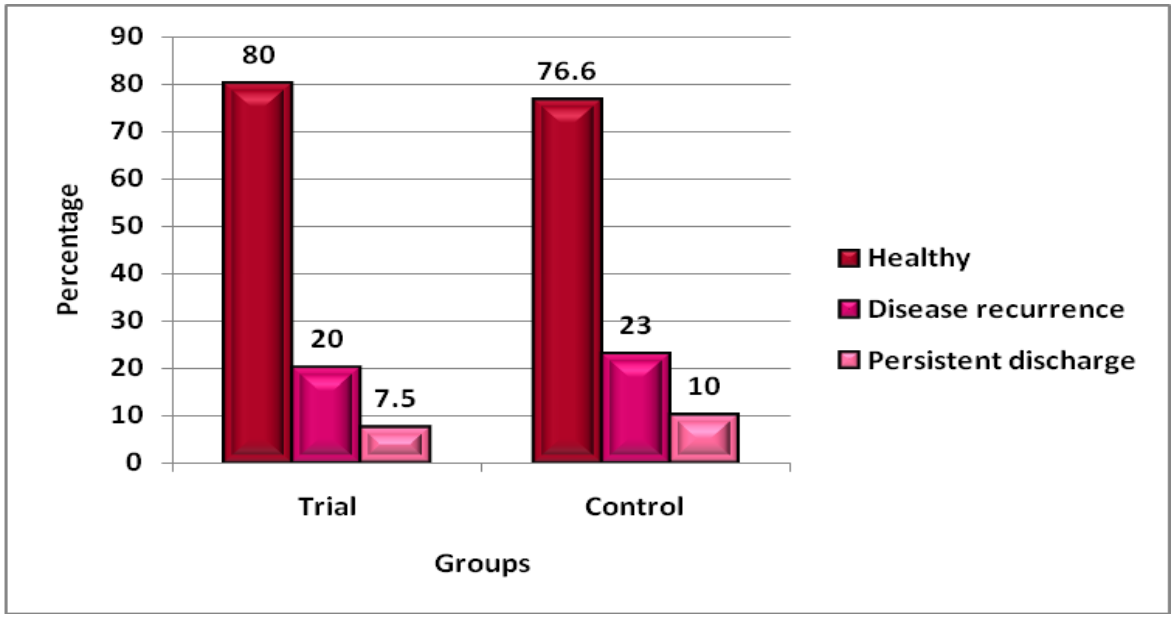
In the trial group 26 (65%) patients belonged to the active stage, 8 (20%) patients in the quiescent stage and 2 (5%) patients in the inactive stage. In the control group 46 (76%) patients were in the active stage of disease, 11 (18.3%) patients were in the quiescent and 3 (5%) patients in the inactive stage.

FOLLOW-UP FINDINGS

Patients grouped in the trial group underwent only tympanoplasty while patients included in the control group underwent tympanoplasty with cortical mastoidectomy. Patients in the trial group were followed up for a minimum of 12 months. Sixty patients that were randomly included in the control group, who had completed more than 1 year post-operatively were recalled for follow-up. Patients of both groups were assessed as to the presence of symptoms associated with CSOM and the status of the graft. The follow-up findings are shown in Table 9.

Table 9: Follow-up findings

Findings	Trial group (n=40)	Historical controls (n=60)
Status of the graft	Healthy n=32 (80%)	Healthy n=46 (76.6%)
Recurrence of the disease	8 (20%)	14 (23%)
Persistence of discharge	3 (7.5%)	6 (10%)



Graph 9: Follow-up findings

In the trial group, 32 (80%) patients had a healthy neotympanum after twelve months follow-up. In 8 (20%) patients recurrence of the disease was seen with reperforation of the neotympanum. All 8 patients had subtotal perforation with 3 (7.5%) patients having persistent discharge.

In the control group (tympanoplasty with cortical mastoidectomy) 49 (81.6%) patients were found to have successful graft uptake with healthy neotympanum. However, 14 patients had recurrence of disease of which 8 patients had subtotal perforation and 6 patients had a large perforation. Persistent discharge was seen in 6 (10%) patients.

COMPLICATIONS

There were no major complications in any patient in our study. Postaural infection was the only complication encountered in this study. A total of 6 patients developed post aural infection. Five (8.3%) of these patients were in the control group and 1(2.5%) patient was in the trial group. For all patients culture and sensitivity of the postaural discharge was done and treated with specific antibiotics. The graft was not disturbed in any of these patients and all patients responded favourably to specific antibiotics.

Table10: Complications

Complications	Trial Group (n=40)	Historical Controls (n=60)
Postaural wound infection	1(2.5%)	5(8.3%)

RESULTS

In this study the key result being assessed was the rate of successful graft uptake following tympanoplasty.

Patients were declared a surgical success when

1. Subjective resolution of all symptoms of chronic otitis media for a minimum period of 12 months.

2. Successful graft uptake after a minimum period of 12 months showing a healthy neotympanum as visualized with an otoscope.

Patients were declared a surgical failure when

1. Persistence or recurrence of the symptoms during the follow-up period.

2. Failure of graft uptake or reperforation of the neotympanum, during the follow-up period.

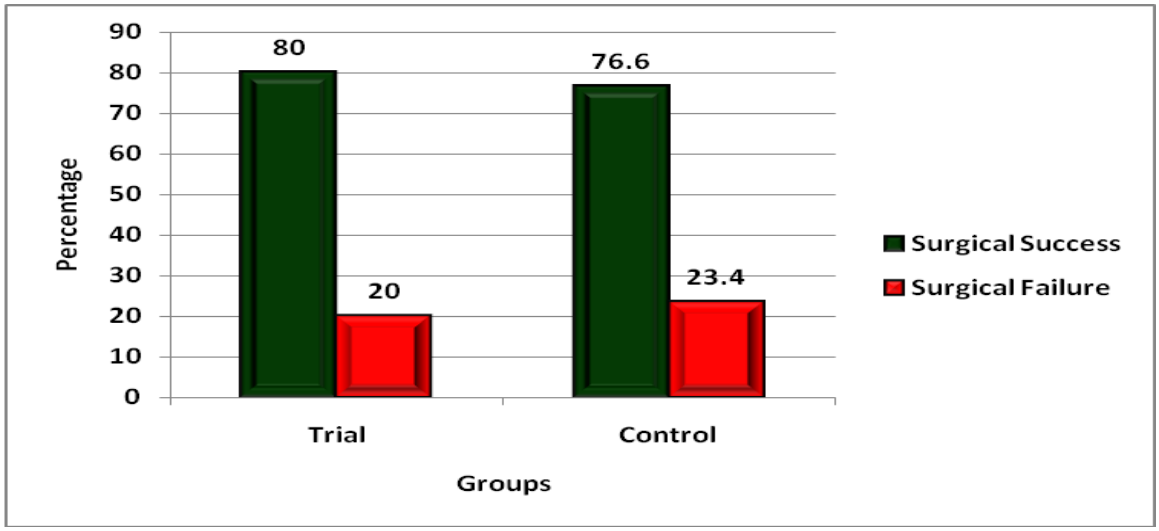
In the group where only tympanoplasty was done (trial group), the surgical rate of success following a minimum follow-up period of 1year was 80%. The surgical success rate in the control group after a minimum follow-up of 1year was 76.6%.

The results of our study are shown in Table 11.

Table 11: Results of Surgery

	Surgical Success	**Surgical Failure**	**Total**
Trail Group (n=40)	32 (80%)	8 (20%)	40
Historical Controls (n=60)	46 (76.6%)	14 (23%)	60
Total	**78**	**22**	**100**

In the group where only tympanoplasty was done (trial group), the surgical rate of success following a minimum follow-up period of one year was 80%. The surgical success rate in the control group after a minimum follow-up of one year was 76.6%.



Graph 10: Results of Surgery

The above results were subjected to statistical analysis using the chi-square test

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

Where

O= Observed value

E= Expected value

$$E = \frac{\text{Row total x Column total}}{\text{Total number}}$$

$X^2 = 0.205128+(-0.07272727)+(-0.0136752136)+0.484848484848)$

$X^{2=} 0.603$

Degree of freedom = (no: of rows-1)(no: of rows-1)

Degree of freedom = (2-1)(2-1)

Degree of freedom=1

When the chi-square and degree of freedom values were applied to the chi-square table a ***'p'*** value of 0.45 was obtained. Since the ***'p'*** value is greater than 0.05 ($p>0.05$) the difference in the results of the trial group and historical controls was not statistically significant. Hence these results indicate that there is no significant benefit in using cortical mastoidectomy as an adjunct to tympanoplasty in tubotympanic disease.

DISCUSSION

Tubotympanic type of CSOM is a common disease in our country causing persistent or intermittent discharge, impaired hearing and thereby affecting the quality of life. Traditionally all cases of uncomplicated tubotympanic disease are treated by tympanomastoidectomy because, a well aerated mastoid is considered as a necessary factor for successful graft uptake. But the advantages and disadvantages of adding mastoidectomy to tympanoplasty in tubotympanic disease has been the focus of much controversy and debate.

There are a number of studies in literature highlighting the role of mastoidectomy in the surgical treatment of tubotympanic disease. They suggest that in a well pneumatized mastoid, significant changes in the middle ear pressure would have little or no effect on the middle ear and tympanic membrane owing to the buffering action of mastoid air cell system. Thus failure to create a pneumatized air cell system in a patient with tubotympanic disease can increase the chance of surgical failure.

The results of a few studies advocating cortical mastoidectomy as an adjuvant to tympanoplasty in tubotympanic disease are shown in table 12.

Table 12:
Studies supporting mastoidectomy in tubotympanic disease

Author	Year	Sample size	Study design	Follow-up	Results (Successful graft uptake)
Holmquist & Bergstrom[12]	1978	31	Retrospective	6 months	TP – 50% TM – 83%
Jackler & Schindler[4]	1984	48 (3groups based on mastoid size)	Retrospective	8 years	Small mastoid-84.6% Medium mastoid-85.7% Large mastoid-100%
Lau & Tos[13]	1986	229	Retrospective	11 years	TM Reperforation 12% Reoperation 16%
Vartiainen & Kansanen[14]	1992	221 (both dry and discharging ears)	Retrospective	6.3 years	Single stage TM – 73%
Ruhl & Pensak[6]	1999	135	Retrospective	8 year	TM - 90.4%
Krishnan[15]	2002	120 (dry 76, quiescent 44, TM 76, TP 44)	Prospective, comparative	3 years	Quiescent ears: TP -50% TM - 80% Dry ears: TP – 78% TM - 100%
Nayak DR[16]	2003	20+20 (3 groups based on mastoid size)	Prospective, controlled	20.4 months	TP- 60% TM- 100%

Holmquist & Bergstrom[12] in his series of 31 patients had a surgical success rate of 83% for TM and 50% for TP. They had a follow-up period of 6 months.

Jackler[4] in their study of 48 patients with tympanic membrane perforation reported a success rate of 84.6%-100% depending on the mastoid size, with TM after 8 year follow-up.

Lou and Tos[13] in 1986 divided 229 ears with chronic granulating otitis media into 3 groups. Group I (23%) underwent TP, Group II (38%) underwent canal wall up TM and Group III (39%) underwent canal wall down TM. However, they did not mention the exact reperforation rate for each group but reported a 12% overall graft failure rate after the first operation.

Vartiainen & Kansanen[14] in 1992 did a retrospective study on both dry and discharging ears with a mean follow-up of 6.3 years. He concluded a 73% success rate with single stage TM.

Ruhl & Pensak[6] series consisted of 135 patients who underwent TM. He achieved a good outcome of 90.4% after a eight year follow-up.

Krishnan[15] in 2002 grouped 120 ears into dry and quiescent depending upon the stage of the disease. In 76 ears TM was done and 44 ears TP was done. After a follow-up of 3 years TM showed 80% and 100% successful graft uptake for quiescent and dry ears, whereas TP showed only 50% and 78% successful graft uptake for quiescent and dry ears.

Nayak DR's[16] series done in 2003 on 40 patients with a mean follow-up of 20.4 months reported a 100% success with TM and 60% with TP.

From the above studies it is evident that those patients who underwent TM had better results (average 94.3%) when compared to TP alone (average 62.8%). Large mastoid and dry ear further augments the results in both the groups.

The clinical studies not supporting mastoidectomy in uncomplicated tubotympanic disease are discussed below.

Table 13:

Studies not supporting mastoidectomy in tubotympanic disease

Author	Year	Sample size	Study design	Follow-up	Results (Successful graft uptake)
Pratt[17]	1976	50(TM 18, TP 32)	Retrospective	2 years	84% in either group
Balyan[7]	1997	81 discharging ears (TM 28, TP 53)	Retrospective	34 months	TP- 90.5% TM- 85.7%
Mishiro[10]	2001	251 (TM 147, TP 104; both dry and discharging ears)	Retrospective comparative	31.7 months	TP - 93.3% TM - 90.5%
McGrew[9]	2004	484 (TM 144, TP320)	Retrospective comparative	31.7 months	TP- 90.6%% TM -91.6%
Mutoh[18]	2007	18+31 (both dry and discharging ears)	Retrospective comparative	16.8 months	MRSA group: TP- 62.5% TM- 90% MSSA group: TP- 80% TM- 72.7%
K V Bhat[5]	2009	68	Prospective controlled	3-6 months	TP- 75.7% TM- 74.2%
Our study	2009 / 2010	No. of cases-40 No. of controls-60	Prospective with Historical controls	12 months	TP- 80.0% TM- 76.6%

Pratt[17] in 1976 compared TM and TP in 50 ears with a perforation of the tympanic membrane and no cholesteatoma through a retrospective study. After 2 years follow-up he reported, 42(84%) patients had a successful surgical result. In eight (16%) patients failure of the graft occurred and incidentally all eight patients had undergone mastoidectomy. Thus he concluded that addition of adjuvant mastoidectomy does not prevent failures when done with tympanoplasty.

Similarly, Balyan[7] studied patients with CSOM (active stage) treated by means of TP and TM, and patients with current dry perforation with a history of CSOM treated with TP alone. They found no significant difference in graft failure rates or hearing results compared with the literature, or any difference in outcome measures whether or not drainage was present. In fact the success rate following TP was 90.5% while the success rate following TM was 85.7% after 34 months of follow-up.

Another study by Mishiro[10] to determine whether mastoidectomy is helpful when combined with TP for tubotympanic disease showed almost similar result. A retrospective analysis of 251 ears operated by a single surgeon over an 11-year period was performed. Tympanoplasty with Mastoidectomy was done in 147 patients while another 104 patients were operated without mastoidectomy. Graft success rates were 90.5% and 93.3% respectively. No statistically significant difference in the graft success rates or post-operative air-bone gap was found between the two groups. The authors concluded that mastoidectomy was not helpful in tympanoplasty even if the ear was discharging.

Mc Grew[9] in his study of 484 patients reported 91.6% success rate with TM and 90.6% success rate with TP after a mean follow-up of 31.7 months. He concluded that even though mastoidectomy was not necessary for the successful repair of tympanic membrane, the procedure impacted the clinical course in patients by reducing the number of future procedures and decreasing disease progression.

Mutoh[18] did a retrospective comparative study on 49 patients (18 dry & 31 discharging) in the year 2007. After a mean follow-up of 16.8 months, he reported that TM was superior to TP only in MRSA- infected ears.

A single-blinded, randomized, controlled study within a referral hospital by Bhat[5] found no statistically significant differences in hearing improvement, tympanic membrane perforation closure, graft uptake or disease eradication with or without mastoidectomy at three and six months post-operatively.

An analysis of studies not favouring adjuvant mastoidectomy shows that the average success rates for TP without adjuvant mastoidectomy was 85.6% and for those with adjuvant mastoidectomy was 83.1%. This statistically insignificant difference between the two groups of patients indicates that adjuvant mastoidectomy contributes little to the results of surgery.

In our study, all patients with tubotympanic disease who underwent tympanoplasty with mastoidectomy during the period October2007-September2009 were included in the historical controls and all patients with tubotympanic disease who underwent tympanoplasty between October 2009 and September 2010 (one year) were included in the trial group. All patients were followed-up for a minimum period of one year.

Successful tympanoplasty was observed in 80% of the patients who underwent tympanoplasty alone while in the group in which adjuvant cortical mastoidectomy was done, the success rate was 76.6%.

The students t test was applied to our results and a ***'p***' value greater than 0.05 (***p***>0.05) was obtained. Thus a statistically insignificant difference was seen between the two groups suggesting that we join the list of researchers who are not in favour of adjuvant cortical mastoidectomy in the treatment of uncomplicated tubotympanic disease.

The role of cortical mastoidectomy in the repair of tympanic membrane perforation has long been debated. Mastoidectomy was regarded as a means of surgically creating an air reservoir in case of eustachian tube dysfunction in an uncomplicated tubotympanic disease. Although there are numerous studies favouring and contradicting the addition of mastoidectomy to tympanoplasty there is no scientific data till date to prove that TM yields better surgical results than TP in cases of uncomplicated tubotympanic disease.

CONCLUSION

Opening the mastoid antrum may be considered a good practise in the treatment of Chronic Suppurative Otitis Media provided there are features suggestive of a mastoid reservoir of infection, aural polyps or other associated complications of Chronic Suppurative Otitis Media. However, the role of adjunctive cortical mastoidectomy in the treatment of uncomplicated tubotympanic disease is insignificant. A meticulously performed tympanoplasty with complete removal of disease from the middle ear under strict aseptic precautions can provide favourable results similar to a tympanoplasty with cortical mastoidectomy.

Hence we do not suggest cortical mastoidectomy as a routine adjunctive procedure in the management of tubotympanic disease.

SUMMARY

Since prehistoric times chronic suppurative otitis media has been an important cause of middle ear disease. Incidence of chronic suppurative otitis media is high in developing countries, especially in the rural population[1]. Otitis media is defined as "an inflammation of the middle ear without reference to aetiology or pathogenesis"[2]. The principal symptoms of tubotympanic disease are aural discharge and hearing loss. The treatment objectives in tubotympanic disease are to provide subjective resolution of symptoms, closure of the perforation, improvement of hearing and prevention of complications[3]. Degree of mastoid pneumatization and mastoiditis are among several factors that can influence the surgical outcome[4].

The significance of cortical mastoidectomy as an adjuvant to tympanoplasty in the treatment of uncomplicated tubotympanic disease has come under scrutiny[5]. There are several authors who advocate adjuvant cortical mastoidectomy[4,6,12,13,14,15,16] and an equally large number of otologists are not in favour of adjuvant cortical mastoidectomy.[5,7,9,10,17,18]

The purpose of this study was to evaluate the significance of adjunctive cortical mastoidectomy in the treatment of uncomplicated tubotympanic disease. In this observational study 40 patients were enrolled for tympanoplasty alone (trial group) and another 60 patients from medical records (historical controls) who had undergone tympanoplasty with cortical mastoidectomy (control group) were included. The study period was 1 year (October 2009 - September 2010) with a minimum follow-up period of 1 year.

Success was defined as, subjective resolution of all symptoms of tubotympanic diseases and successful graft uptake showing healthy neotympanum, as

visualized with an otoscope, 12 months after surgery. Failure was defined as persistence or recurrence of symptoms or failure of graft uptake or reperforation of the neotympanum during the follow-up period. The trial group of patients underwent a standardised tympanoplasty by postaural approach and underlay grafting using temporalis fascia.

Most of our patients from either group belonged to the age group of 18 to 30 years of which 57% were females. All patients complained of ear discharge of which 70% also complained of decreased hearing. In both the groups, more than 60% of the patients had mucoid type of ear discharge. Middle ear mucosa was normal in 70% of the patients in the trial group and 81.6% patients in the control group. Eustachian tube patency was assessed in both the groups and in majority (93%) of the cases eustachian tube was found to be patent. At the time of presentation 72% patients had active discharge from the ear and 23% were in the quiescent stage of disease.

The two groups were analysed and compared with respect to; graft uptake. In the trial group successful graft uptake was observed in 32 (80%) patients and in the control group successful graft uptake was seen in 46 (76.6%) patients. These results suggest that adjunctive cortical mastoidectomy carries an insignificant role in the treatment of uncomplicated tubotympanic disease.

Although it is postulated to have disease lurking behind, in the mastoid air cells, the conclusion that there is no significant difference in the rate of re-perforation of the tympanic membrane among the two groups, suggests that; if a meticulous tympanoplasty is performed with particular care to remove all disease from the middle ear, the results are similar. Thus the possible complications associated with cortical mastoidectomy, longer operating time and greater costs can be avoided.

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ANNEXURE

PROFORMA OF THE CASE SHEET

I PERSONAL DETAIL

Name Age Sex | M | F |

Address DOA DOD

Occupation Hospital no: | | | | | | |

II PRESENTING COMPLAINT

Ear discharge : Y/N | R | L | B | Duration Pain in the ear: Y/N | R | L | B | Duration

Decreased hearing: | R | L | B | Y/N Duration Giddiness Y/N Fever Y/N

Headache Y/N Tinnitus | R | L | B | Y/N Duration

Facial weakness | R | L | B | Y/N Duration

III HISTORY OF PRESENT ILLNESS

Ear discharge

Onset: insidious/sudden Type: intermittent/continous Quantity: copious/scanty

Quality: watery/serosanginous/mucoidal/mucopurulent/purulent

Odor: odorless/foulsmelling Consistency: sticky/nonsticky

Aggravating factors: cold/head bath/pharyngitis/tonsillitis

Relieving factors:

Pain in the ear

Onset: insidious/sudden Type: dull/throbbing/sharp intermittent/continous

Aggravating factors:Y/N If yes swallowing/yawning and chewing/pulling pinna or pressing tragus

Relieving factors:Y/N If yes: expulsion of discharge

Hard of hearing

Onset: insidious/sudden Progressive/Non progressive Fluctuant/Non fluctuant

Associated symptoms

Itching in the ear: Y/N Fever: Y/N Headache: Y/N

Tinnitus:Y/N If yes subjective/objective

Type-intermittent/continuous roaring[LF]/whistling[HF]

Aggravating factors: smoking/yawning/blowing

Relieving factors: By putting pressure on the side of the neck Y/N

Vertigo: Y/N If yes Nature occasional/continuous/postural/nausea/vomitting

Precipitating factors

Aggravating factors

Relieving factors

IV PAST HISTORY

Similar complaints in the past: Y/N

Previous ear surgery: Y/N If Yes

Diabetes mellitus, Hypertension, Tuberculosis, Bronchial asthma

V FAMILY HISTORY

VI PERSONAL HISTORY

Loss of appetite: Y/N Disturbed sleep: Y/N

Bowel and bladder disturbances: Y/N Habituated smoking: Y/N

Alcohol: Y/N

EXAMINATION

VII GENERAL PHYSICAL EXAMINATION

Temperature Pulse BP

Built: Poor/ medium/wellbuilt Pallor: Y/N Icterus: Y/N

Clubbing: Y/N

Koilonychia: Y/N Nutritional status: Poor/Satisfactory Lymphadenopathy: Y/N

Oedema: Y/N

VIII E.N.T Examination

Sl No:	Ear	Rt	Lt
1,	Pinna: deformity scar	Y/N Y/N	Y/N Y/N
2,	Mastoid area: swelling tenderness fistula scar	Y/N Y/N Y/N Y/N	Y/N Y/N Y/N Y/N
3,	Postauricular area: obliterated accentuated scar	Y/N Y/N Y/N	Y/N Y/N Y/N
4,	Preauricular area		
5,	External auditory canal: stenosis discharge : mucus mucopurulent pus copious scanty bloodstained foul smelling Polyp Granulation Sagging of postero-superior meatal wall	Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N	Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N
6,	Tympanic membrane:Color-greyish white congested dull white blue dark blue Surface- bullae calcareous deposits Position- bulging retracted Mobility Epithelialised margins of perforation	Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N	Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N Y/N
7,	Middle ear mucosa- red and velvety pale and edematous / smooth and pink	Y/N Y/N Y/N	Y/N Y/N Y/N
8,	TFT: Rinne's test Weber's test ABC decreased	 +ve/-ve R/L/C Y/N	 +ve/-ve R/L/C Y/N

9,	Eustachian tube patency: Bitter taste following instillation of Ciprofloxacin Ear drops	Y/N	Y/N
10,	Vestibular function tests Spontaneous Nystagmus Fistula test Positional test	Y/N Positive/negative Positive/negative	Y/N Positive/negative Positive/negative
11,	Facial Nerve weakness	Y/N	Y/N

IX NOSE AND PNS

External nose and vestibule

Anterior Rhinoscopy

Posterior Rhinoscopy

Tenderness of PNS: Y/N If yes

X EXAMINATION OF THROAT

Oral cavity and Oropharynx

IDL

Neck

XI SYSTEMIC EXAMINATION

Cardio vascular system

Respiratory system

Abdomen

Central nervous system

XII CLINICAL DIAGNOSIS

Tubotympanic disease active/quiescent/inactive with CHL/MHL

XIII INVESTIGATIONS

X-Ray of both mastoids: lateral oblique view- pneumatic/sclerotic/diploiec

Swab for culture sensitivity

Pure Tone Audiometry: CHL/MHL

Pure tone average:

Operative findings: Middle ear mucosa

Mastoid mucosa

Condition of ossicular chain: continuous/discontinuous

Patency of aditus: patent/blocked

Operative procedure: Myringoplasty ☐ TympanoplastyI ☐

TympanoplastyII ☐ TympanoplastyIII ☐

Adjuvant cortical mastoidectomy ☐

KEY TO MASTER CHART

ED - Ear Discharge

DH - Decreased Hearing

H - Headache

GI - Giddiness

TI - Tinnitus

P - Pain

F - Fever

PZ - Perforation Size

MEM - Middle Ear Mucosa

ETP - Eustachian Tube Patency

SOD - Stage of Disease

M - Mucoid

MP - Mucopurulent

PU - Purulent

S - Small

M - Medium

L - Large

ST - Subtotal

N - Normal

D - Diseased

PA - Patent

B - Blocked

A - Active

Q - Quiescent

I - Inactive

PWI – Postaural wound infection

M – Male

F - Female