SENSORINEURAL HEARING LOSS AFTER STAPES SURGERY

(Do stapedectomy procedures cause a degree of sensorineural hearing loss?)

A Meta-Analytic Study

SUBMITTED FOR PARTIAL FULFILMENT OF MASTER DEGREE IN OTOLARYNGOLOGY AND HEAD&NECK SURGERY

By

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Study ID	No. Of ears included	Srgical Technique	Laser	prosthesis	Follow up	No. Of cases with SNHL											
						Guide lines	0.5	1	2	3	4	0.5,1,2 &4	1,2&4	0.5,1,2 &3	0.5,1 &2	Dead ear	Notes
1	40	stapedotomy	Co2	Teflon piston 0.6mm	More than 1 year	yes	7	1	3		7		3				
2	30	stapedotomy	Co2	Teflon piston 0.6mm	Early<3ms	yes	2	0	2		6		4				
					Late more than 1 year		2	0	0		7		3				
3	112	stapedotomy	M	Teflon piston 0.6mm	12 to 34 ms.	yes							9				
	100			Teflon piston 0.4mm									5				
4	30	stapedotomy	M	Teflon loop	More than 1 year	M										1	
5	2527	stapedotomy	Argon laser	Teflon piston 0.4mm	Mean 46 ms.	yes						13					
6	27	stapedotomy	Argon laser	Titanium wire 0.6	More than 1 year	yes						0	0				With Hyaluronate Gel
	32											0	1				Without Gel Hyaluronate
7	135	stapedotomy	Argon laser	Modified cawthrone teflon0.3mm	Average 10ms.	M						0					
8	35	stapedotomy	In 91%	Titanium	More than 1 year	yes	2	1	0	3	6		3				
	183			Teflon wire			3	4	1	4	15		3				
9	314	Stapedotomy with P graft	Motor drill	Teflon piston 0.4mm	3 years	yes					35		14			1	P=Perichondrium V= Vein
	138	stapedotomywith V graft									11		2				
10	30	stapedotomy	M	Teflon piston	M	M	0										
	30			Titanium piston													
11	134	stapedectomy	M	Wire loop or platinum	11.5ys	M							9				
	75	stapedotomy		Teflon piston	6ys								4				
12	117	stapedotomy	Er:YAG	Gold (K-piston) 0.4&0.6mm	Early 1-3ds	yes							20	13		0	Erbium:Yttrium-Aluminum-Garnit Laser
					Late 11ms								8	4		0	
13	451	M	M	M	M	yes							10				

Study ID	No. Of ears included	Srgical Technique	Laser	prosthesis	Follow up	Guide lines	No. Of cases with SNHL										Notes
							0.5	1	2	3	4	0.5,1,2 &4	1,2&4	0.5,1,2 &3	0.5,1 &2	Dead ear	
14	60	Stapedotomy with perioperative coticosteroid	Er:YAG	Gold (K-Piston) 0.4 &0.6mm	Early1-4ds	yes							8				
					Late>6ws								5				
	35	without coticosteroid			Early1-4ds								5				
					Late>6ws								1				
15	60	stapedectomy	M	Wire(Schu-knect)	10ys	M	2 cases sever high frequency SNHL									1	
	55	stapedotomy	drill	Tiflon Piston 0.4mm	5ys		0 no SNHL										
16	34	stapedotomy	Co2	Tiflon Piston 0.4mm	12ms	M						0					SNHL>20db
	316		Non laser	M	17ms							3					
17	100	stapedotomy	M	Teflon piston 0.4mm	>one year	M					6	1					
	100			Teflon piston 0.6mm							10	2					
18	62	stapedotomy	M	Gold (K-Piston) 0.4mm	54ws	M						0					
19	4137	stapedectomy	M	Teflon in 80%	>one year	M									74	25	
20	63	stapedotomy	Co2	Tiflon Piston 0.6mm	>2ys	yes							2				
21	227	stapedectomy	M	M	More than 4 ms.	M	15	7	9	16	31			9			
	323	stapedotomy					20	17	7	15	23			10			
22	228	stapedectomy	M	Stainless steel(Hous) wire	1year	M					12				3		Total **5.5**% at 4khz >15db and **0.9**% at PTA>10db
	120	stapedotomy	drill	Teflon piston 0.4mm							6				0		
23	691	stapedectomy	M	M	Less than 6 weeks	M	16 cases SNHL (41 to 95db)										
	234	stapedotomy															

Study ID	No. Of ears included	Srgical Technique	Laser	prosthesis	Follow up	Guide lines	No. Of cases with SNHL										Notes
							0.5	1	2	3	4	0.5,1,2 &4	1,2&4	0.5,1,2 &3	0.5,1 &2	Dead ear	
24	330	stapedectomy	M	Teflon piston	More than 5 years	M					16				5	1	<60ys. old
	46										0				1	0	>60ys. old
25	35	stapedectomy	Many types	Many types	More than one year	M						1				2	
	22	stapedotomy															
26	100	stapedotomy	Co2	Teflon piston 0.6mm	More than one year	M					6			0			
27	39	stapedectomy	Argon	M	More than 8weeks	M									4		
	61		Non laser												4		
28	56	stapedotomy	Manual perforator	Teflon piston 0.4mm	More than 8weeks	M									0		
29	153	stapedotomy	Co2	M	M	M	0 no cases of SNHL in speech range										
30	317	stapedectomy	M	0.8Teflon	Immediate	M						11					
					Delayed more than 5years							88					
	162	stapedotomy		0.3Teflon	Immediate							0					
					Delayed more than 5years							17					
31	192	stapedotomy	Many types	M	Early <one month	M	3 cases Sever SNHL(50 to 80dB)									3	Total 11(21)
					Late >6 years		8 cases Sever SNHL(50 to 80dB)									7	
32	120132	stapedectomy	M	M	For long time	M	1211 cases SNHL										

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M= Missed

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1	10	Lesions that may cause conductive hearing loss. Quoted from **(Lalwani, 2007)**.
2	41 to 43	Show summary of data collected from the 32 included articles

Introduction

Otosclerosis is a disease of the bone that is unique to the otic capsule. It may cause a conductive hearing loss, a mixed conductive-sensorineural hearing loss, or occasionally a purely sensorineural hearing loss. The most common area of stapedial fixation in otosclerosis is the anterior crus. The process may progress to involve the entire footplate or may continue anteriorly towards the cochlea causing a sensorineural hearing loss **(Ferekidis, 2007)**.

Stapedectomy is one of the standard treatments advocated for the conductive hearing loss of otosclerosis. The technique originally described by **Shea (1958)** consisted of removal of the stapes, including all or part of the footplate and insertion of a prosthesis to reconstitute the ossicular chain. Over the years, technical innovations, primarily in the form of the small fenestra technique and the availability of prosthesis have contributed towards improved outcomes in stapes surgery. Coincident with discussion of the advantages of large versus small oval window fenestra, debate concerning the best method to create the fenestra itself continues. Laser as a tool for performing stapedotomies in otosclerosis surgery has been in use over the last two decades **(Raut and Halik, 2005)**.

Surgery for otosclerosis usually gives remarkably good results, as long as the patients are well selected. Hence, it has been compared to cataract surgery in its high success rate and infrequent complications. It is, however, known that in rare cases things do not turn out as well as expected and that a permanent sensorineural hearing loss can occur. In the past 2 decades, efforts have been concentrated on making stapes

surgery as minimally invasive as possible in the hope of decreasing this complication rate **(Somers et al, 2006)**.

Sensorineural hearing loss was defined as a postoperative bone-conduction threshold that was more than 10 dB worse than preoperative bone conduction average at 1,000, 2,000, and 4,000 Hz **(Massey et al, 2005)**.

Although the complication of SNHL following stapes surgery is rare, it remains a serious side effect. Advocates of the small fenestra technique have suggested that it is a safer technique because it requires less manipulation of the footplate, and this has been seen in some series. There was no statistically significant differences in SNHL between the two techniques (stapedectomy and stapedotomy), with prevalence of significant loss (>10dB) relatively low in both groups. Certainly, the experience of the surgeon with either technique may play the most significant role in avoiding complications **(House et al, 2002)**.

There has been a trend in recent years favoring more conservative treatment of the otosclerotic footplate. Many investigators believe that a small fenestra stapedotomy causes fewer labyrinthine complications including immediate and delayed sensorineural hearing loss, perioperative vertigo and perilymphatic fluid leaks than partial or total removal of the footplate. Many of these proponents report good results but their numbers are quite small. Many use a laser to produce the stapedotomy opening. There is at present still a debate as to the type of laser that should be used and some surgeons are concerned about possible damage to the membranous labyrinth. Other techniques use a small drill or pick to remove a portion of the footplate. Unfortunately stapedotomy techniques do not protect the oval window from the problems already mentioned in relation to an artificial prosthesis **(Hough and Dyer, 1993)**.

Sensorineural hearing loss and/or vertigo are rare but severe complications of stapes surgery for otosclerosis, ranging from 0.2 to 3%. Management of such complications depends on the underlying cause: intravestibular protrusion of the prosthesis, perilymph fistula, labyrinthitis, and reparative granuloma extending into the vestibule. Surgery is mandatory in cases of intravestibular prosthesis or of persistent perilymph fistula. In cases of suppurative labyrinthitis or reparative granuloma extending into the vestibule, prognosis is usually poor, despite aggressive medical therapy or revision surgery. CT scan or magnetic resonance imaging can frequently help to determine the cause of the inner ear complication of stapedectomy **(Ayache et al, 2007).**

Aim of work

To conduct a systematic review of published literature on sensorineural hearing loss as a complication of stapedectomy procedures in order to conclude evidence regarding whether stapedectomy procedures cause a degree of sensorineural hearing loss or not.

Embryology

The maturation of the bony labyrinth plays a role in the pathogenesis of otosclerosis. The otic capsule arises from mesenchyme surrounding the otic vesicle at 4 weeks of embryologic development. At 8 weeks, the cartilaginous framework is begun. At 16 weeks, endochondral bony replacement of this framework begins in 14 identifiable centers. In some people, complete bony replacement does not occur and leaves cartilage in certain locations. One of these regions, the fissula ante fenestram, is anterior to the oval window and is usually the last area of endochondral bone formation in the labyrinth ***(Roland and Samy, 2006)***.

According to temporal bone studies, this region is affected in 80% to 90% of patients with otosclerosis. Areas of predilection for otosclerotic lesions, such as the border of the round window the apical medial wall of the cochlea, the area posterior to the cochlear aqueduct, the region adjacent to the semicircular canals, and the stapes footplate itself (which is derived from otic capsule, as opposed to the superstructure, which is a branchial arch derivative) ***(Roland and Samy, 2006)***.

Epidemiology

Otosclerosis is transmitted in an autosomal dominant fashion with incomplete penetrance (25% to 40%). The degree of penetrance is related to the distribution of lesions in the otic capsule. Some lesions are not located where they can cause clinical symptoms. About 10% of Caucasians have histologic findings of otosclerosis. However, of those with histologic changes, only 12% have clinical symptoms; thus, overall, this represents about 1% of the Caucasian population. In all races, when

one ear is affected, the contralateral ear shows histologic involvement 80% of the time ***(Roland and Samy, 2006)***.

The age at which symptoms become apparent is variable due to the insidious progression of hearing loss, but hearing loss often begins between the ages of 15 and 45 years. The average age at presentation is 33 years. About 60% of patients with clinical otosclerosis report a family history of this condition. Otosclerosis has been reported to advance more rapidly in females than males ***(Roland and Samy, 2006)***.

Histopathology

Otosclerosis usually affects both ears symmetrically. The disease process is probably confined to the temporal bone. The pink swelling of otosclerosis may sometimes even be detected clinically through a particularly transparent tympanic membrane as a well-demarcated and pink focus near the promontory. A characteristic translucency of bone adjacent to the cochlea and anterior to the footplate is identified on a CT scan ***(Michaels, 2006).***

The lesion always commences in the otic capsule tissue anterior to the footplate of the stapes. In this position it does not produce symptoms. These occur when the otosclerosis invades the adjacent stapes footplate and produces fixation of that structure and thus conductive hearing loss. It later spreads widely in the otic capsule and may involve the round window ligament. Blood vessels are prominent and evenly distributed ***(Michaels, 2006)***.

The histological characteristic of otosclerosis is the presence of trabeculae of new bone, mostly of the woven type with marked vascularity. This contrasts with the well-developed lamellar bone under the outer periosteum, the endochondral middle layer and the endosteal

layer of the otic capsule, a sharply demarcated edge between normal and otosclerotic bone being a prominent feature. In most places osteocytes are very abundant within the woven bone ***(Michaels, 2006)***.

The footplate of the stapes is often invaded by otosclerotic bone, and the lower end of the anterior crus of the stapes is sometimes invaded (Fig. 1). Otosclerotic bone sometimes reaches the endosteum of the cochlear capsule. In some cases it may lead to a fibrous reaction deep to the spiral ligament. These changes are probably the basis of the sensorineural hearing loss that is also occasionally found in cases of otosclerosis ***(Michaels, 2006)***.

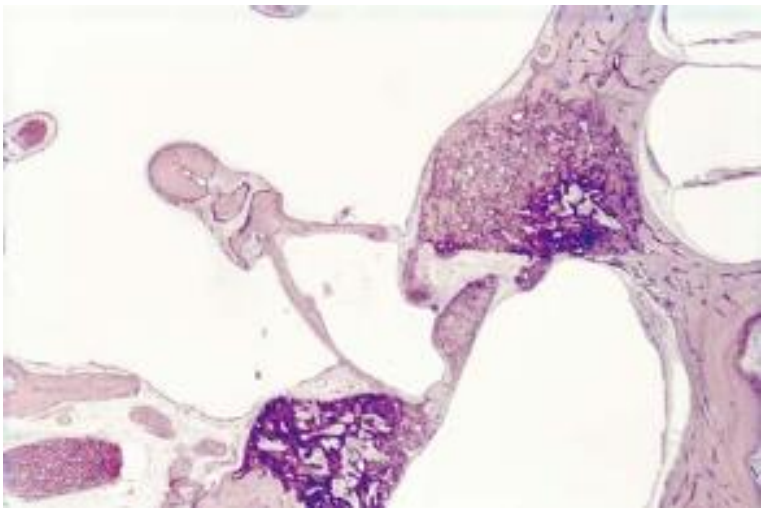

Fig.1. Focus of otosclerosis involving both the anterior (*upper*) and posterior (*lower*) part of footplate of the stapes. The anterior focus has invaded onto the footplate and the anterior crus. This would have produced fixation of the stapes and its attendant conductive hearing loss. Notice that the otosclerotic foci are more darkly staining and vascular than the adjacent normal bone. Quoted from **(Michaels and Hellquist , 2001).**

Clinical picture

Symptoms:

The typical patient with otosclerosis presents with a history of slowly progressive hearing loss that is usually bilateral but asymmetric.