Published in final edited form as:

Clin Otolaryngol. 2017 April ; 42(2): 268–274. doi:10.1111/coa.12712.

The Surgical Challenge of Carotid Artery and Fallopian Canal Dehiscence in Chronic Ear Disease: A Pitfall for Endoscopic Approach

Henrique F. Pauna, MD^{1,2}, Rafael C. Monsanto, MD^{1,3}, Patricia A. Schachern, BS¹, Sady S. Costa, MD, PhD⁴, Geeyoun Kwon, PhD¹, Michael M. Paparella, MD^{1,5}, and Sebahattin Cureoglu, MD¹

¹University of Minnesota, Department of Otolaryngology – Head and Neck Surgery, Minneapolis, Minnesota, USA

²University of Campinas – UNICAMP, Department of Otolaryngology, Head and Neck Surgery, Campinas, São Paulo, Brazil

³Banco de Olhos de Sorocaba Hospital (BOS), Department of Otolaryngology and Head and Neck Surgery, Sorocaba, São Paulo, Brazil

⁴Federal University of Rio Grande do Sul, Department of Ophthalmology and Otolaryngology, Porto Alegre, Rio Grande do Sul, Brazil

⁵Paparella Ear, Head and Neck Institute, Minneapolis, Minnesota, USA

Abstract

Objective—Endoscopic procedures are becoming common in middle ear surgery. Inflammation due to chronic ear disease can cause bony erosion of the carotid artery and Fallopian canals, making them more vulnerable during surgery. The objective of this study was to determine whether or not chronic ear disease increases dehiscence of the carotid artery and Fallopian canals.

Design—Comparative human temporal bone study.

Setting—Otopathology laboratory.

Participants—We selected 78 temporal bones from 55 deceased donors with chronic otitis media or cholesteatoma, and then compared those 2 groups with a control group of 27 temporal bones from 19 deceased donors with no middle ear disease.

Main outcome measures—We analyzed the middle ear, carotid artery canal, and Fallopian canal, looking for signs of dehiscence of its bony coverage, using light microscopy.

Results—We found an increased incidence in dehiscence of the carotid artery and Fallopian canals in temporal bones with chronic middle ear disease. The size of the carotid artery canal

Corresponding author: Sebahattin Cureoglu, M.D., Co-Director, Otopathology Laboratory, Department of Otolaryngology, University of Minnesota, Minneapolis, Minnesota, MMC 2873, 2001 6th Street SE, LRB 210, Minneapolis, MN 55455, Office: 612-626-9875, cureo003@umn.edu.

Conflicts of interest

Authors declare no conflict of interest.

dehiscence was larger in the middle ear diseased groups, and its bony coverage, when present, was also thinner compared to the control group. Dehiscence of the carotid artery canal was more frequently located closer to the promontory. The incidence of Fallopian canal dehiscence was significantly higher in temporal bones from donors older than 18 years with chronic middle ear disease.

Conclusion—The increased incidence of the carotid artery and Fallopian canal dehiscence in temporal bones with chronic middle ear disease elevates the risk of adverse events during middle ear surgery.

Level of Evidence—N/A.

Keywords

Carotid artery canal; Fallopian canal; dehiscence; middle ear surgery; chronic ear disease

INTRODUCTION

The internal carotid artery (ICA) is one of the largest blood vessel neighboring the middle ear cavity. As it climbs through the petrous bone, it passes the middle ear cavity and cochlea anteriorly, and then bends to run medially to the Eustachian tube^{1,2} (Fig. 1). Previous studies have reported dehiscence of the carotid artery canal in 4.9% to 35.2% of human temporal bones with no middle ear disease^{3–5}. In patients with dehiscence of the carotid artery canal, middle ear procedures such as tympanostomies can cause devastating complications, including massive hemorrhage^{6–8}.

The facial nerve (FN) is important for both communication and expression, and impairment of its function may affect the quality of life. After entering the internal acoustic canal, the FN goes into the Fallopian canal, passing through the labyrinthine, tympanic, and mastoid segments toward the stylomastoid foramen⁹. The prevalence of dehiscence in the Fallopian canal is reported to range from 25% to 57%¹⁰. Baxter et al.¹¹, during operative observations, found that 91% of the tympanic segment of the FN was dehiscent. Thus, procedures performed in the middle ear cleft could potentially traumatize the FN at this site of dehiscence.

Endoscopic approaches to the middle ear have become popular. But serious complications can occur, given the close proximity of the ICA to the tympanic membrane^{6,7,12}. In mastoid surgery, variations in vascular structures related to the temporal bone can lead to severe complications during middle ear or skull base surgery, including trauma to the ICA, the jugular bulb, or the sigmoid sinus¹³. Therefore, to avoid damage to those structures, preoperative measurements of surgical landmarks are important¹⁴. Dehiscence of the carotid artery and Fallopian canals increases the risk of complications during otologic surgery via the transcanal, posterior, or endoscopic approach.

Chronic ear disease is usually associated with persistent inflammatory response¹⁵. Many patients have viral or bacterial infections that lead to the production of several cytokines, such as tumor necrosis factor-alpha (TNF- α), interleukin (IL)-1 β , IL-6, and IL-8, all known

to have nonspecific and proinflammatory activity¹⁵ as well as the potential to damage the bony structures of the middle ear¹⁶.

No studies, to the best of our knowledge, have described the incidence and location of ICA and Fallopian canal dehiscence in patients with chronic middle ear disease. In this study, we investigated the dehiscence of the carotid artery and Fallopian canals in temporal bones with chronic middle ear disease compared to non-diseased control group.

MATERIALS AND METHODS

Ethical considerations

The Institutional Review Board of the University of Minnesota approved this study (0206M26181).

Specimens

We obtained human temporal bones from the Otopathology Laboratory, part of the Department of Otolaryngology at the University of Minnesota. Immediately after removal, the temporal bones were placed in 10% buffered formalin. They were then decalcified, embedded in celloidin, and sectioned at a thickness of 20 μ m. Every 10th section was stained with hematoxylin and eosin for histologic study under light microscope at various magnifications.

We selected 8 temporal bones from 7 deceased donors with cholesteatoma, 4 bones from males and 4 from females, the mean age of 62.5 ± 29.56 years (range, 7 to 86 years). We also selected 70 temporal bones from 47 donors with chronic otitis media without cholesteatoma (COM), 50 bones from males and 20 from females, the mean age of 52 ± 31.75 years (range, 6 months to 93 years). COM group is defined as temporal bones with any sign of chronic inflammatory changes in the middle ear cleft, such as granulation tissue, cholesterol granuloma, fibrosis, or fibrocystic structures. For our control group, we included 27 temporal bones from 19 donors with no history of chronic ear disease, 17 bones from males, and 10 from females, mean age of 47 ± 26.54 years (range, 1 to 88 years).

We excluded temporal bones with a history of otologic surgery (such as mastoidectomy), congenital malformation of the middle ear, temporal bone fractures, Paget disease, otosclerosis, congenital cholesteatoma, irradiation of the head and neck, or leukemia; because all of those conditions could cause changes of the bone covering the ICA or FN in the middle ear. We reviewed all clinical records for any history of past ear disease (such as acute otitis media).

For all 3 groups (cholesteatoma, COM, and controls), we excluded any temporal bones with removal and fixation artifacts, and neoplastic tissue infiltration. In addition, we also scrutinized the contralateral ears from deceased donors with cholesteatoma or COM.

Analysis of Dehiscence

Carotid Artery Canal and Fallopian Canal—Using light microscopy, we analyzed temporal bones for any dehiscence or microdehiscence in the bony wall covering of the ICA

canal. We analyzed every stained slide from the tensor tympani tendon until the lowest section available. We defined dehiscence as a lack of continuity in the bony wall; we defined microdehiscence as a small gap in the bony covering filled with connective tissue³. We considered the size of the dehiscence of the carotid artery canal as clinically relevant when it was larger than 3 mm, because most endoscopes used for middle ear surgery have a diameter of this size¹⁷, however, we highlight that serious complications can also occur, due to the proximity of the ICA to the tympanic membrane, during any middle ear procedure. We documented the location of dehiscence in the bony covering of the ICA, the presence of any inflammatory cells, and remodeling of bone by osteoblasts or osteoclasts (bony erosion). We defined dehiscence of the Fallopian canal as a lack of its bony covering along the entire tympanic segment of the FN and noted dehiscence of the bone of any size.

We measured the thickness of the bone that covers the carotid artery canal, and the size of dehiscence using a Nikon Eclipse E400 system equipped with measurement software (NIS-Elements BR 4.30.01 64-bit, Nikon, Japan). The measurement point we used was the distance of the bone covering the ICA canal, just below the tensor tympani muscle canal. This point was obtained from an acute angle formed by a straight line crossing the external auditory canal and touching the anterior annulus of the tympanic membrane, and then ending at the carotid artery canal. We considered this measurement to best mimic the placement of the endoscope (Fig. 2).

Statistical Analysis

We calculated and compared the relative risk among the 3 groups for dehiscence of both the ICA and Fallopian canals. In addition, to calculate and compare the narrowest bony coverage of the carotid artery canal, and the length of dehiscence, we used the Mann-Whitney U test. For these analyses, we used SPSS 23.0 software for Windows (SPSS Inc., Chicago, IL). Findings were considered statistically significant when *P* values were less than 0.05.

RESULTS

Our final groups comprised of 8 temporal bones from 7 donors with cholesteatoma and 70 temporal bones from 48 donors with COM. Our control group included 27 temporal bones from 19 donors with no history of chronic ear disease.

In 7 (87.5%) of the 8 temporal bones with cholesteatoma, we observed dehiscence of the ICA canal. All 8 bones had signs of acute inflammation and erosion close to the dehiscent bone covering the carotid artery canal. All 8 bones also showed signs of chronic inflammatory changes and bony erosion at the anterior portion of the middle ear. The location of dehiscence was close to the promontory in 3 (42.85%) of the bones, close to the Eustachian tube in 4 (57.15%), and 5 (62.5%) of them had dehiscence in the Fallopian canal.

In 51 (72.85%) of the 70 temporal bones with COM, we observed dehiscence of the ICA canal. However, 52 of the bones in the COM group showed signs of inflammation, and 50 had bony erosion. In almost 50% of the bones, dehiscence was close to the promontory. Fallopian canal dehiscence was found in 40 (57.14%) out of 70 temporal bones.

In 14 (51.85%) of the 27 temporal bones in the control group, we observed dehiscence of the ICA canal in the middle ear; 6 of them had signs of erosion of the ear's bony coverage. Yet, only 3 (11.11%) out of the 27 temporal bones in the control group had dehiscence over the Fallopian canal. None of the 19 deceased donors in the control group had signs of effusion in the middle ear (Fig. 3). In reviewing their clinical histories, we found that 11 had no history of ear infections, 3 had a history of diabetes, and 4 had a history of renal disease.

The relative risk of dehiscence of the carotid artery canal in the cholesteatoma group was 1.6875 times higher than in the control group (95% confidence interval [CI], 1.0781 to 2.6413, $P=0.0221$). The relative risk of dehiscence in the COM group was 1.4051 times higher than in the control group (CI, 0.9508 to 2.0765, $P=0.0879$). The relative risk of the Fallopian canal dehiscence in the cholesteatoma group was 5.6250 times higher than in the control group (CI, 1.7039 to 18.5697, $P=0.0046$). The relative risk of Fallopian canal dehiscence in the COM group was 5.1429 times higher than in the control group (CI, 1.7360 to 15.2353, $P=0.0031$).

We found dehiscence of the ICA canal larger than 3 mm in the middle ear in 1 (12.5%) of the 8 temporal bones in the cholesteatoma group, 6 (8.57%) of the 70 bones in the COM group, and 2 (7.41%) of the 27 bones in the control group. The presence and size of dehiscence of the carotid artery and Fallopian canals are summarized in Table 1.

Table 2 highlights the incidence of dehiscence among the 3 groups by age (i.e., donors who were younger than 18 years vs. those who were at least 18 years old). Although we observed a difference in the incidence of dehiscence in the ICA canal between age groups, this difference was not statistically significant.

We also analyzed 6 contralateral ears from deceased donors with cholesteatoma (1 bone) or COM (5 bones). All of the contralateral ears showed no signs of middle ear disease but only 1 (16.67%) of them had dehiscence in the ICA canal. None of them had any signs of erosion in the carotid artery canal, and only 1 ear had erosion in the Fallopian canal.

DISCUSSION

Synopsis of new findings

In our study, we found an increased incidence and size of dehiscence in the ICA canal in temporal bones with chronic middle ear disease (as compared with nondiseased ears). Also, we found a significant increase in dehiscence of the Fallopian canal. Both presence and duration of inflammation in the middle ear cleft appear to play a major role in the increased risk of the dehiscence in the ICA and Fallopian canals.

Strengths of the study

COM and cholesteatoma are both associated with a higher incidence of dehiscence in the ICA and Fallopian canals. Many studies have shown this association radiologically or by surgical observations; nevertheless, to our knowledge, this is the first histopathologic study to report these findings. In addition, the previous studies published in this matter reported the incidence of dehiscence of these structures in normal temporal bones, without including

patients with middle ear disease. It is important to mention that the number of temporal bones with cholesteatoma is low, therefore our analysis could be naturally inflated.

Comparisons with other studies

Endoscopic techniques allow enhanced exposure of the operative field (reducing the need for drilling) and adequate passage of light (enhancing intraoperative visualization). But a few safety concerns must be kept in mind. The tip of the endoscope can become excessively hot, especially when a xenon light source is used, and accidental movement of the tip of the endoscope can result in secondary trauma¹⁷. Even with high-magnification, angled, and high-definition views, it is critical to know the presence and the most frequent location of dehiscence in the carotid artery and Fallopian canals.

Both groups with chronic middle ear disease showed an increased incidence of ICA canal dehiscence larger than 3 mm and an increased incidence of erosion in the canal. Its bony coverage, when present, was also thinner compared to the control group. These findings are important when one considers that the diameter of the tip of the endoscope is 3 mm. Furthermore, any dehiscence of the ICA canal is vulnerable to surgical damage considering the shape and size of the instruments used in otologic surgeries.

In patients with cholesteatoma and concomitant infections, several inflammatory factors are found in the ME. For example, IL-6 affects fibroblast proliferation and osteoblast development, leading to osteofibrosis, bony erosion, tympanosclerotic degeneration, and chronic disease. We found a significant difference in the incidence of Fallopian canal dehiscence between the diseased groups and non-diseased control group. These findings suggest inflammatory changes may be involved in the erosion of the bone covering these structures.

We found an increased incidence of ICA canal dehiscence in adults (18 years or older) compared to children in both the diseased and control groups. Wang et al.² studied high-resolution CT scans of the carotid artery canal from 208 adults who were 50 years or older. They found the incidence of dehiscence in the ICA canal was higher in the group over 50 years of age. The incidence of Fallopian canal dehiscence was also increased in adults compared to children in all 3 groups. Interestingly, we found a significantly higher incidence of dehiscence in the disease groups compared to the control group when considering adults only. These findings suggest the duration of middle ear inflammation can be an important factor in the development of dehiscence. Choi et al.¹⁸ studied CT scans from 254 patients with middle ear cholesteatoma, and 54% of them revealed Fallopian canal dehiscence with dehiscence larger in size among patients with a history of a long-term inflammatory conditions of the middle ear.

Clinical applicability

The introduction of the endoscope as a tool for middle ear surgery has brought several new possibilities to the surgeon, including the preservation of health tissue and better removal of the pathologic matter, especially in spaces otherwise inaccessible in the temporal bone¹⁹. Additionally, the use of the endoscope has the advantage of decreasing the surgical trauma of the procedures by providing the possibility of more focused approaches²⁰.

Moreano et al.^{3,10} reported the incidence of dehiscence in the ICA and Fallopian canals in temporal bones with no middle ear disease. Compared to this study, our data demonstrate a higher incidence of dehiscence in these structures among patients with cholesteatoma and COM. Furthermore, we also observed that the most common location of dehiscence in the ICA and Fallopian canals are in the mesotympanum. Thus, our findings demonstrate the vulnerability of these structures to injuries during transcanal procedures, such as myringotomy, or endoscopic ear surgeries. In endoscopic procedures, the 2-dimension view provided by the endoscope could also increase the chances of such complications.

CONCLUSION

Our study indicates that the dehiscence in the carotid artery and Fallopian canals is more common in temporal bones from adults with chronic middle ear disease, suggesting the importance of a scrupulous evaluation of the dehiscence in the ICA and Fallopian canals prior to endoscopic surgery.

Acknowledgments

Funding

This project was funded by NIH NIDCD U24 DC011968, International Hearing Foundation, Starkey Hearing Foundation, and Lions 5M International.

References

1. Lund AD, Palacios SD. Carotid artery-cochlear dehiscence: a review. *Laryngoscope*. 2011; 121:2658–2660. [PubMed: 22109767]
2. Wang CH, Shi ZP, Liu DW, et al. High computed tomographic correlations between carotid canal dehiscence and high jugular bulb in the middle ear. *Audiol Neurotol*. 2011; 16:106–112. [PubMed: 20606423]
3. Moreano EH, Paparella MM, Zeltermann D, et al. Prevalence of carotid canal dehiscence in the human middle ear: a report of 1000 temporal bones. *Laryngoscope*. 1994; 104:612–618. [PubMed: 8189992]
4. Hasebe S, Sando I, Orita Y. Proximity of carotid canal wall to tympanic membrane: a human temporal bone study. *Laryngoscope*. 2003; 113:802–807. [PubMed: 12792314]
5. Penido NO, Borin A, Fukuda Y, et al. Microscopic anatomy of the carotid canal and its relations with cochlea and middle ear. *Braz J Otorhinolaryngol*. 2005; 71:410–414. [PubMed: 16446952]
6. Gil JLM, Palau EM, Jurado FJA, et al. Abnormal internal carotid artery in the middle ear and severe otorrhagia after myringotomy. *Acta Otorrinolaringol Esp*. 2010; 59:142–144.
7. Leuin SC, Handwerker J, Rabinov JD, et al. Carotid laceration during myringotomy. *Otolaryngol Head Neck Surg*. 2009; 140:946–947. [PubMed: 19467423]
8. Shinnabe A, Hara M, Matsuzawa S, et al. A case of multiple abnormalities with eustachian tube obstruction by a protruded internal carotid artery. *Int J Ped Otorhinolaryngol*. 2011; 75:441–443.
9. Cömert E, Cömert A, Tunçel Ü, et al. Surgical anatomy of facial nerve for revision transmastoid surgery. *J Craniofac Surg*. 2014; 25:619–622. [PubMed: 24514887]
10. Moreano EH, Paparella MM, Zeltermann D, et al. Prevalence of facial canal dehiscence and of persistent stapedial artery in the human middle ear: a report of 1000 temporal bones. *Laryngoscope*. 1994; 104:309–320. [PubMed: 8127188]
11. Baxter A. Dehiscence of the Fallopian canal. *J Laryngol Otol*. 1971; 85:587–594. [PubMed: 5581361]

12. Lien CF, Weng HH, Liu CF, et al. Risk factors for internal carotid artery injury in adults during simple nasopharyngeal surgeries. *Eur Arch Otorhinolaryngol.* 2014; 271:1693–1699. [PubMed: 23982669]
13. Silvola J, Kivekäs I, Poe DS. Balloon dilation of the cartilaginous portion of the eustachian tube. *Otolaryngol Head Neck Surg.* 2014; 151:125–130. [PubMed: 24705223]
14. Inal M, Muluk NB, Dag E, et al. The Pitfalls and Important Distances in Temporal Bone HRCT of the Subjects with High Jugular Bulbs – Preliminary Report. *Adv Clin Exp Med.* 2015; 24:315–324. [PubMed: 25931366]
15. Zielnik-Jurkiewicz B, Stankiewicz-Szymczak W. Pro-inflammatory interleukins in middle ear effusions from atopic and non-atopic children with chronic otitis media with effusion. *Eur Arch Otorhinolaryngol.* 2016; 273:1369–1378. [PubMed: 26078091]
16. Choi JW, Park YH. Facial Nerve Paralysis in Patients With Chronic Ear Infections: Surgical Outcomes and Radiologic Analysis. *Clin Exp Otorhinolaryngol.* 2015; 8:218–223. [PubMed: 26330915]
17. Tarabichi M. Endoscopic transcanal middle ear surgery. *Indian J Otolaryngol Head Neck Surg.* 2010; 62:6–24. [PubMed: 23120674]
18. Choi JW, Park YH. Facial nerve paralysis in patients with chronic ear infections: surgical outcomes and radiologic analysis. *Clin Exp Otolaryngol.* 2015; 8:218–223.
19. Presutti L, Nogueira JF, Alicandri-Ciufelli M, et al. Beyond the middle ear. Endoscopic surgical anatomy and approaches to the inner ear and lateral skull base. *Otolaryngol Clin N Am.* 2013; 46:189–200.
20. Strauß G, Bahrami N, Hofer M, et al. The HD-panoramic visualization system: a new visualization system for ENT surgery. *Eur Arch Otorhinolaryngol.* 2009; 266:1475–1487. [PubMed: 19099317]

Figure 1.

A: Anatomic view of the right internal carotid artery and Fallopian canals and their relationship with the structures of the middle ear. B: Interpretation of an endoscopic view of the left middle ear, with the tympanic membrane removed.

1 = internal carotid artery; 2 = inferior tympanic artery; 3 = caroticotympanic artery; 4 = round window niche; 5 = stapes; 6 = facial nerve running in the Fallopian canal; 7 = incus; 8 = malleus; 9 = tensor tympani muscle; 10 = chorda tympani; 11 = stapedial tendon; 12 = cochleariform process; 13 = hypotympanic air cell; 14 = posterior malleolar ligament; 15 = Eustachian tube

Figure 2.

Histologic section of a healthy left ear stained with hematoxylin and eosin, 1× magnification. A: Anatomic relations of the ICA with the middle ear structures, as well as its proximity to the tympanic membrane. B: Graphic representation of the endoscope to the measurement point of the bone covering over the internal carotid artery (arrow).

C = cochlea; EAC = external auditory canal; ET = Eustachian tube; FN = facial nerve; I = incus; ICA = internal carotid artery; M = malleus; ME = middle ear; S = stapes; TM = tympanic membrane; TTM = tensor tympani muscle; V = vestibule

Figure 3. Histologic section of a diseased left ear stained with hematoxylin and eosin, 1 × magnification. A: Dehiscence (star), with middle ear effusion, inflammation, and thickening of the mucosa over the promontory. B: A thickened tympanic membrane, with effusion in the middle ear, and erosion and dehiscence of the internal carotid artery and Fallopian canals (stars).

EAC = external auditory canal; FN = facial nerve; RWN = round window niche

Table 1

Characteristics of dehiscence of carotid artery and Fallopian canals in diseased and non-diseased groups.

	Cholesteatoma	Chronic Otitis Media	Control
Carotid artery canal dehiscence length	7 (87.5%)[*] (<i>P</i> = 0.0221)	51 (72.85%) (<i>P</i> = 0.0879)	14 (51.85%)
< 3 mm	6 (75%)	45 (64.28%)	12 (44.44%)
3 mm	1 (12.5%)	6 (8.57%)	2 (7.41%)
Erosion in the internal carotid artery canal	8 (100%)[*] (<i>P</i> < 0.0001)	50 (71.42%)[*] (<i>P</i> = 0.0015)	6 (22.22%)
Dehiscence position			
ME	3 (42.85%)	34 (66.67%)	8 (57.14%)
ET	4 (57.14%)	17 (33.33%)	6 (42.85%)
Dehiscence length (µm)	709.31 (<i>P</i> = 0.2143)	390.87 (<i>P</i> = 0.3456)	117.58
Bony coverage thickness (µm)	306.11 (<i>P</i> = 0.8366)	336.49 (<i>P</i> = 0.8066)	406.72
Dehiscence in the Fallopian canal	5 (62.5%)[*] (<i>P</i> = 0.0046)	40 (57.14%)[*] (<i>P</i> = 0.0031)	3 (11.11%)
Erosion in the Fallopian canal	6 (75%)[*] (<i>P</i> = 0.0013)	54 (77.14%)[*] (<i>P</i> = 0.0004)	4 (14.81%)
Active inflammation	7 (87.5%)	52 (74.28%)	0
Bilateral disease	1 (12.5%)	23 (52.85%)	NA

ET = Eustachian tube; ME = middle ear; NA = not applicable

^{*} Statistically significant (as compared with the control group)

Table 2

Incidence of dehiscence of carotid artery and Fallopian canals in diseased and non-diseased groups, according to the age. The number in parentheses represents the percentage of the temporal bones that presents dehiscence in the evaluated structures, as compared to the total number of bones presented in each group.

<u>Carotid Artery Canal Dehiscence</u>			
	Cholesteatoma	Chronic Otitis Media	Control
< 18 years	2 (25%) ($P=0.3206$)	19 (27.14%) ($P=0.6835$)	2 (7.41%)
18 years	5 (62.5%) ($P=0.0621$)	32 (45.71%) ($P=0.1443$)	12 (44.44%)

<u>Fallopian Canal Dehiscence</u>			
	Cholesteatoma	Chronic Otitis Media	Control
< 18 years	1 (12.5%) ($P=0.7074$)	13 (18.57%) ($P=0.5623$)	1 (3.7%)
18 years	4 (50%)* ($P=0.0047$)	27 (38.57%)* ($P=0.0046$)	2 (7.41%)

* Statistically significant (as compared with the control group)