Bone Conduction Improvement After Surgery for Conductive Hearing Loss

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Abstract The objective is to evaluate change in post-operative bone conduction in patients who underwent surgery for conductive/mixed hearing loss due to various reasons. The study design is of retrospective case review and tertiary referral center setting. Five-hundred patients with unilateral conductive/mixed hearing loss were divided into five equal groups (each representing different causes for pre-operative hearing loss), who underwent appropriate surgical correction and had a follow-up audiogram available. The intervention comprises surgery (like myringoplasty and ossiculoplasty with closed or open cavity mastoidectomy for chronic otitis media, stapes mobilization for tympanosclerosis and stapedotomy for otosclerosis) for conductive/mixed hearing loss. Significant improvement or worsening in bone conduction was defined as 15 dB or more improvement or worsening of bone conduction threshold at least in two frequencies between 500 and 4000 Hz. All the other groups also showed a consistent pre-operative bone conduction reduction with an equally consistent improvement in post-operative bone conduction improvement to a varying degree with otosclerosis group having maximum percentage of patients with post-operative bone conduction improvement (60%). The measurement of bone conduction is not necessarily a true reflection of the function of the inner ear. Middle ear makes a contribution to bone conduction and correction of a middle ear conductive lesion causes an apparent improvement in inner ear function. The apparent inner ear hearing loss caused in this way may be reversible to some extent.

Keywords Bone conduction · Carhart's effect · Pseudoperceptive hearing loss

Introduction

There have been three primary components of bone conduction hearing as suggested by Tonndorf [1]. These include distortional, inertial-ossicular and external canal-osseotympanic components. Each of them contributes to the eventual bone conduction sensation level. The clinical importance of these components in bone conduction measurement has been described [2]. Distortional component includes distortion and stimulation of the otic capsule caused by vibrational sound force. When a vibrating tuning fork or bone conduction measurement probe rests on the mastoid process, these vibrations causes distortion of the membranous cochlea and thus hearing sensation. Inertial-ossicular component involves out of phase movements of ossicular chain (due to inertia of the ossicles that are suspended in a middle ear cavity by ligaments that separate them from rest of the temporal bone) when a vibrational sound force is applied on the skull bone along the axis of ossicles causing movement of the stapes in the oval window. This component is greatest for frequencies below 800 Hz and requires intact ossicular chain. The final component for bone conduction is contributed by external auditory canal. Because the skull bones are interconnected, when a bone vibrator is placed on the skull, it causes vibration of bony external auditory canal creating sound. Though most of the sound escapes from the unoccluded ear, a part of it strikes the tympanic membrane, which along with the inertial-ossicular component stimulates the cochlea. This component increases substantially when the external canal is blocked. It also underlines the

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importance of intact tympanic membrane, though minor, for bone conduction threshold especially involving low frequency. Therefore when a sound is applied to skull, it is not only transmitted via bone but equally via the external auditory canal and ossicular chain.

Thus the bone conduction we measure pre-operatively in cases of conductive hearing loss due to external or middle ear pathology is not entirely a measure of inner ear function. Huizing [3] proved this variation in bone conduction threshold experimentally and termed this phenomenon as middle ear bone conduction loss or pseudoperceptive deafness. It is also known as the “Carhart Effect” as Carhart [4] was the first to describe the improvement in bone conduction in otosclerosis after stapes surgery. This effect has also been well studied in various other disorders that affect the conductive mechanism [5, 6]. There have been efforts to mathematically predict the Carhart effect to estimate the effect of external and middle components at each frequency [8].

An improvement of bone conduction has been observed after various types of surgery for conductive hearing loss. So this study was carried out to evaluate and compare the amount of bone conduction improvement in early post-operative period (3–6 months) in various conditions.

Materials and Methods

Following middle ear conditions were included in the evaluation of change in bone conduction and were divided into five groups.

- Group I: Tubotympanic chronic otitis media (CSOM) with perforation of tympanic membrane with intact ossicular chain undergoing cortical mastoidectomy with Type I tympanoplasty.
- Group II: Tubotympanic CSOM with perforation of tympanic membrane with ossicular discontinuity undergoing intact canal wall mastoidectomy with short or long columella ossiculoplasty.
- Group III: Atticoantral CSOM with cholesteatoma undergoing canal wall down mastoidectomy with short or long columella ossiculoplasty.
- Group IV: Tympanosclerosis related ossicular fixation with perforation of tympanic membrane undergoing intact canal wall mastoidectomy with intact ossicular chain or appropriate ossiculoplasty.
- Group V: Otosclerosis with stapes surgery.

Random selection was made consisting of 100 patients in each group who underwent appropriate surgical correction and had a follow up audiogram available. Patients with bilateral middle ear pathology or patients having

sensorineural hearing loss in opposite clinically normal middle ear were not included in study to rule out other causes for sensorineural hearing loss like noise induced hearing loss, ototoxicity and presbycusis.

Audiological evaluation included retrospective analysis of bone conduction in pre-operative and early post-operative period for frequency of 500, 1000, 2000 and 4000 Hz. Comparison was made for change in mean bone conduction between pre-operative and post-operative period and statistical significance determined. Comparison was also made between the percentages of patients having significant worsening of bone conduction in pre-operative period and subsequent improvement of bone conduction in various groups to assess the effect of that particular condition on bone conduction. Pre-operatively 15 dB or more depression of bone conduction threshold at least in two frequencies between 500 and 4000 Hz was considered to be significant. Significant improvement or worsening in bone conduction was defined as 15 dB or more improvement or worsening of bone conduction threshold at least in two frequencies between 500 and 4000 Hz as suggested by Mustafa Tuz et al. [9].

Results

The age of patients in each group did not vary from each other significantly and were comparable. The mean pre-operative bone conduction of each group did not differ significantly from each other and were statistically comparable. Similarly the mean post-operative bone conduction did not differ significantly among different groups and were statistically comparable. However in each group there was a significant improvement in bone conduction in post-operative period in all the groups with the maximum gain in group V (otosclerosis group) with an average gain in bone conduction of 11.1 dB and minimum gain in group III (cholesteatoma group) with an average gain in bone conduction of 7.7 dB (Table 1). Group V (otosclerosis group) had maximum percentage of patients with pre-operative bone conduction reduction (89%) having 15 dB or more depression of bone conduction threshold at least in two frequencies between 500 and 4000 Hz (Table 2). Also, Group V (otosclerosis group) had maximum percentage of patients with post-operative bone conduction improvement (60%) having 15 dB or more improvement of bone conduction threshold at least in two frequencies between 500 and 4000 Hz. All the other groups also showed a consistent pre-operative bone conduction reduction with an equally consistent improvement in post-operative bone conduction improvement to a varying degree. A total of 13 patients out of 500 (2.6%) showed post-operative worsening of bone conduction having 15 dB or more depression of bone

Table 1 Significant improvement in bone conduction in post-operative period is seen in all the groups

Middle ear condition	Pre-operative bone conduction mean (SD)	Post-operative bone conduction mean (SD)	Change in bone conduction mean (SD)	<i>P</i> value
Group I	16.1 (10.3)	6.4 (8.6)	−9.7 (7)	<0.001
Group II	18 (8.8)	9.2 (8.3)	−8.8 (7.3)	<0.001
Group III	17.4 (8.1)	9.7 (7.3)	−7.7 (7.1)	<0.001
Group IV	17.5 (8.9)	8.8 (7.4)	−8.7 (7.4)	<0.001
Group V	22.4 (9.4)	11.1 (7.6)	−11.3 (7.8)	<0.001

Table 2 A consistent pre-operative bone conduction reduction with an equally consistent improvement in post-operative bone conduction improvement to a varying degree is seen in all the groups

Middle ear condition	Pre-operative bone conduction reduction (15 dB or more loss in at least two frequencies between 500 and 4000 Hz)	Post-operative bone conduction improvement (15 dB or more improvement in at least two frequencies between 500 and 4000 Hz)	Post-operative bone conduction worsening (15 dB or more loss in at least two frequencies between 500 and 4000 Hz)
Group I			
Number of patients	65	45	0
Group II			
Number of patients	74	43	2
Group III			
Number of patients	77	31	5
Group IV			
Number of patients	70	31	4
Group V			
Number of patients	89	60	2

conduction threshold at least in two frequencies between 500 and 4000 Hz with two each in group II and V, five in group III and four in group IV.

Discussion

While direct involvement of cochlea [10] and enzymatic actions [11] have been considered as a cause for sensorineural hearing loss in otosclerosis and tympanosclerosis, chemical labyrinthitis (due to bacterial toxins and inflammatory intermediates) and infective labyrinthitis are considered the cause for depressed bone conduction in chronic otitis media with or without cholesteatoma [12]. Chronic otitis media can definitely cause labyrinthitis and can cause sensorineural hearing loss but it does not explain the reason behind the depressed bone conduction in so many patients without any symptoms of inner ear involvement. Similarly cochlear otosclerosis may be a definite entity but the belief that it can cause pure sensorineural hearing loss have been strongly criticized by histopathological examination of temporal bones [13, 14]. Audiometric studies by Glorig et al. [15] concluded that otosclerosis does not increase the

sensorineural hearing loss above that to be expected in the general population. We believe that direct involvement of cochlea and enzymatic actions may be causing a part of sensorineural hearing loss in advanced cases of otosclerosis, but they too have a mechanical basis behind the depressed bone conduction as seen by frequent over closure of air bone gap in such cases. Mechanical factors (contributed by middle ear and external ear) do play an important role in bone conduction reduction in majority of the cases of chronic otitis media and otosclerosis [3–9]. Bone conduction can be depressed in middle ear pathologies due to both mechanical and chemical factors and surgery can not only close air bone gap but also improve bone conduction.

Mechanical occlusion of round window with granulations, stiffening of ossicular chain due to granulations or cholesteatoma around them, ossicular discontinuity and perforation are the principle mechanical factors affecting bone conduction in chronic otitis media [16, 17]. Our study supports the mechanical theory with a consistent pre-operative bone conduction reduction with an improvement in post-operative bone conduction to a varying degree (between 31 and 45%) in perforation of tympanic membrane with or

without ossicular discontinuity as well as in cholesteatoma. Mustafa Tuz et al. [9] observed improvement in post-operative bone conduction in half of the patients who were affected pre-operatively. Hence, one should not hesitate in operating a patient with abovementioned findings with impaired bone conduction fearing a poor functional outcome. It must be kept in mind that both air and bone conduction thresholds may improve after surgery particularly in cases with cholesteatoma and extensive middle ear disease.

While Mustafa et al. [9] did not find stiffening of ossicular chain commonly seen in tympanosclerosis to adversely affect pre-operative bone conduction, Vartiainen et al. [16] found it so. Our results are similar to Vartiainen et al. with 70% of the patients having depressed pre-operative bone conduction. We feel the difference might be because we took only those patients with middle ear and mastoid involvement with tympanosclerosis while not including those patients with simple myringosclerosis that the former group could have included.

Depressed pre-operative bone conduction with subsequent improvement in post-operative bone conduction with over closure of air bone gap is almost universal in cases of otosclerosis and in our study these patients had the maximum improvement among all the groups.

Conclusions

Although the bone conduction thresholds are frequently elevated in chronic otitis media, tympanosclerosis and otosclerosis in pre-operative period, it may not be due to direct inner ear involvement in all the cases. In most of the cases it may be due to mechanical factors (so called Carhart effect or pseudoperceptive deafness). Successful results can be achieved in most of these patients regardless of the deteriorated bone conduction thresholds.

References

1. Tonndorf J (1968) A new concept of bone conduction. *Arch Otolaryngol Head Neck Surg* 87:595–600
2. Richard WH (2002) Pure tone evaluation. In: Katz J, Burkard R, Medwetsky L (eds) *Handbook of clinical audiology*, 5th edn. Lippincott Williams & Wilkins, Baltimore, pp 76–78
3. Huizing EH (1964) Bone conduction loss due to middle ear pathology—pseudoperceptive deafness. *Int Audiol* 3:89–98
4. Carhart R (1950) Clinical application of bone conduction. *Arch Otolaryngol Head Neck Surg* 51:798–807
5. Huizing EH (1960) Bone conduction—the influence of middle ear. *Acta Otolaryngol (Stockh) Suppl.* 155:1–99
6. Allen GW, Fernandez C (1960) The mechanism of bone conduction. *Ann Otol Rhinol Laryngol* 69:5–28
7. Dirks D, Malmquist C (1969) Comparison of frontal and mastoid bone conduction thresholds in various conductive lesions. *J Speech Hear Res* 12:725–746
8. Gatehouse S, Browning GG (1982) A re-examination of the Carhart effect. *Br J Audiol* 16:215–220
9. Tuz M, Dogru H, Uygur K, Gedikli O (2000) Improvement in bone conduction threshold after tympanoplasty. *Otolaryngol Head Neck Surg* 123:775–778
10. Ruedi L (1965) Histopathological confirmation of labyrinthine otosclerosis. *Laryngoscope* 75:1582–1609
11. Causse J, Chevance LG, Bretlau P, Jorgensen MB, Uriel J, Berges J (1977) Enzymatic concept of otospongiosis and cochlear otospongiosis. *Clin Otolaryngol* 2:23–32
12. Paparella MM, Morizono T, Le CT et al (1984) Sensorineural hearing loss in chronic otitis media. *Ann Otol Rhinol Laryngol* 93:623–629
13. Shuknecht HF (1983) Cochlear otosclerosis: an intractable absurdity. *J Laryngol Otol Suppl* 8:81–83
14. Huer MM, Goycolea MV, Paparella MM, Oliveira JA (1991) Otosclerosis: The University of Minnesota temporal bone collection. *Otolaryngol Head Neck Surg* 105:396–405
15. Glorig A, Gallo R (1962) Comments on sensorineural loss in otosclerosis. In: Shuknecht HF (ed) *Otosclerosis*, Little Brown & Co, Boston, pp 63–78
16. Vartiainen E, Karjalainen S (1987) Factors influencing sensorineural hearing loss in chronic otitis media. *Am J Otolaryngol* 8:13–15
17. Levine BA, Shelton C, Berliner K, Sheehy JI (1989) Sensorineural loss in chronic otitis media: is it clinically significant. *Arch Otolaryngol Head Neck Surg* 115:814–816