Superior Canal Dehiscence Size: Multivariate Assessment of Clinical Impact

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Objective: To examine the association between dehiscence length in patients with superior semicircular canal dehiscence syndrome and their clinical findings, including objective audiometric and vestibular testing results.

Study Design: Retrospective study.

Setting: Tertiary referral center.

Patients: Patients included in this study were diagnosed with superior semicircular canal dehiscence syndrome and underwent surgical repair of the dehiscence through middle fossa craniotomy. The dehiscence length was measured intraoperatively in all cases.

Main Outcome Measures: Correlation between dehiscence length with pure-tone average (PTA), average bone-conduction threshold, maximal air-bone gap, cervical vestibular evoked myogenic potential thresholds, and presenting signs and symptoms.

Results: The correlation between dehiscence length and maximal air-bone gap was statistically significant on both univariate and multivariate regression analyses. The correlations between dehiscence length and PTA, average bone-conduction threshold, cervical vestibular evoked myogenic potential threshold, and presenting signs and symptoms were not statistically significant.

Conclusion: The dehiscence length correlated positively with the maximal air-bone gap in patients with superior semicircular canal dehiscence. The correlation was statistically significant. The dehiscence length did not correlate with the other variables examined in this study. Key Words: Conductive hearing loss—Hyperacusis—Oscillopsia—Superior semicircular canal dehiscence—Tullio phenomenon—Third window—Vertigo.

database (from March 1996 to December 2010), 117 of whom have had surgery for SCD repair. Of these patients, 106 patients had intraoperative measurement of dehiscence length. Twenty patients were excluded from the study because of previous otologic surgery (stapedectomy, perilymphatic fistula repair, previous SCD repair). These patients were excluded because the audiometric or vestibular findings could be confounded by the previous surgery and, therefore, might not be solely caused by the dehiscence. One patient was excluded because of a history of sudden profound sensorineural hearing loss in the same ear as SCD before presenting to our clinic for evaluation. The remaining 85 patients were included in this study. The average age was 46 years (range, 29–66 yr). Forty-three were men, and 42 were women. Forty-one dehiscences involved the right superior semicircular canal, and 44 involved the left.

The diagnosis of SCD was based on clinical testing and physical examination findings. Clinical findings were retrieved from the database. Symptoms considered included chronic dis-equilibrium, autophony, sound- and pressure-induced vertigo, pulsatile tinnitus, and positional vertigo. Clinical signs evaluated included nystagmus induced by tones with an audiometer, Val-salva maneuver, or external auditory canal pressure. Head tilt induced by tones with an audiometer also was examined. These 10 signs and symptoms were evaluated, and the total number of positive findings in each patient was recorded (from a minimum of zero to a maximum of 10 signs and symptoms). Extended bone-conduction audiometry (using an audiometer specially calibrated to detect bone conduction thresholds as low as −20 dB nHL) was used to measure conductive hyperacusis.

All patients had audiograms before surgery. Preoperative cervical vestibular evoked myogenic potential (cVEMP) responses were measured in 73 patients. Computed tomography of the temporal bones with reconstructions in the plane of the superior semicircular canal as well as orthogonal to that plane was performed in every patient. In all cases, the computed tomographic (CT) scans were consistent with SCD on the side for surgery.

All patients in this study underwent SCD repair via a middle fossa craniotomy as previously described (9). The location of the SCD in the middle fossa was found with the aid of image navigation. The size of the dehiscence was measured using a small scale several millimeters long laid next to the dehiscence and viewed under high magnification. The length of the dehiscence was measured under the operating microscope with an estimated error of 0.2 mm.

The dehiscence was plugged by packing small pieces of previously harvested temporalis fascia into the lumen of the bony superior canal on both ends of the dehiscence. Bone chips and bone pate were used to reinforce the plugs. After surgery, patients remained under observation in a neurosurgical intensive care unit overnight. Patients were discharged from the hospital on the second or third postoperative day. Dexamethasone 6 to 8 mg was given 3 to 4 times daily for the first 24 hours. If there was no evidence of sensorineural hearing loss or pan-labyrinthine hypofunction on bedside testing, then the steroids were tapered over 5 days. Otherwise, the taper was prolonged for 10 to 14 days. Bedside testing consisted of the Weber and Rinne tuning fork tests and assessment of the vestibuloocular reflexes with rapid rotary head impulses (head thrusts).

Statistical analyses for this study were performed using STATA (StataCorp LP, College Station, TX, USA) and Excel (Microsoft, Seattle, WA, USA). Simple linear and Poisson regression analyses were used to examine the association of dehiscence length and clinical findings, including audiometric and vestibular test results. Statistical significance was defined as \( p < 0.05 \). Sample size calculation showed that for \( \alpha = 0.05 \) and \( \beta = 0.8, 85 \), patients were needed to detect a correlation coefficient of 0.3.

RESULTS

Pure-Tone Average

Patients with SCD often present with hearing loss. Pure-tone average (PTA) is a commonly used measure of the degree of hearing loss in patients. It is calculated by taking the average of air-conduction thresholds at 500, 1,000, and 2,000 Hz. Figure 1 shows the scatter plot of PTA versus dehiscence length. The linear regression line showed a positive association between PTA and dehiscence length. Simple linear regression analysis showed the Pearson’s correlation coefficient \( r \) to be 0.16 (\( p = 0.15 \)). Because age and sex are both important predictors of hearing loss (10), it is important to control for them when examining the association between PTA and dehiscence length. Multivariate regression analysis of PTA and dehiscence length, controlling for age and sex (Table 1), showed the regression coefficient \( \beta_1 \) to be 1.36 (\( p = 0.08 \)).

FIG. 1. Simple linear regression of SCD length and pure-tone average (PTA). The linear regression line shows a positive correlation between SCD length and PTA (slope = 1.17). The Pearson’s correlation coefficient is not statistically significant (\( p = 0.15 \)).

Bone-Conduction Thresholds

Hyperacusis for bone-conducted sounds often is seen in SCD. This is indicated by bone-conduction thresholds below 0 dB nHL. We examined the correlation between the average low-frequency bone-conduction threshold (the average of bone-conduction thresholds at 250, 500, and 1,000 Hz) and the dehiscence length. Figure 2 shows the scatter plot of average bone-conduction threshold versus dehiscence length. The linear regression line showed a slightly positive association between average bone conduction threshold and dehiscence length. Simple linear regression analysis showed the Pearson’s correlation coefficient to be 0.05 (\( p = 0.67 \)). Multivariate regression analysis...
analysis of average bone-conduction threshold and dehiscence length, controlling for age and sex (Table 1), showed the regression coefficient to be 0.31 (p = 0.63). The R^2 for the multivariate model was 0.11. Thus, there was no significant correlation of average bone-conduction threshold with dehiscence length.

**Maximal Air-Bone Gap**

The combination of bone conduction hyperacusis and elevation in air-conduction thresholds results in an air-bone gap. We examined the correlation between maximum air-bone gap and the dehiscence length. Figure 3 shows the scatter plot of maximal air-bone gap versus dehiscence length. The linear regression line shows a positive association between maximal air-bone gap and dehiscence length. Simple linear regression analysis showed the Pearson’s correlation coefficient to be 0.22 (p = 0.046). Multivariate regression analysis of maximal air-bone gap and dehiscence length, controlling for age and sex (Table 1), showed the regression coefficient to be 2.45, p = 0.021. The R^2 for the multivariate model was 0.11. Thus, there was a significant correlation between maximal air-bone gap and dehiscence length, but only 11% of the variance in air-bone gap data can be accounted for by the combination of age, sex, and dehiscence length.

**Vestibular Evoked Myogenic Potentials**

Patients with SCD often have a decreased cervical VEMP threshold. Cervical VEMP threshold was documented in 73 of 85 patients in our study. Figure 4 shows the scatter plot of cVEMP threshold versus dehiscence length. The linear regression line shows a negative association between cVEMP threshold and dehiscence length. Simple linear regression analysis showed that cVEMP threshold was not significantly associated with the dehiscence length (Pearson’s correlation coefficient, r = 0.13, p = 0.26). Because age and sex are both predictors of VEMP threshold (11,12), it is important to control for them when examining the association between cVEMP

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\text{Ave BC indicates average bone-conduction threshold; cVEMP, cVEMP threshold; Max ABG, maximal air-bone gap; MLR, multivariate linear regression coefficient p value; PTA, pure-tone average; R^2, coefficient of determination for the multivariate model; SD, standard deviation; SLR, simple linear regression coefficient p value.}
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**TABLE 1.**

<table>
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<th></th>
<th>Mean</th>
<th>Range</th>
<th>SD</th>
<th>SLR</th>
<th>MLR</th>
<th>R^2</th>
</tr>
</thead>
<tbody>
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<td>SCD length (mm)</td>
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<td>1–7</td>
<td>1.29</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>PTA (dB HL)</td>
<td>17.6</td>
<td>0–45</td>
<td>9.52</td>
<td>0.15</td>
<td>0.08</td>
<td>0.15</td>
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<tr>
<td>Ave BC (dB HL)</td>
<td>1.00</td>
<td>–10 to 35</td>
<td>7.83</td>
<td>0.67</td>
<td>0.63</td>
<td>0.11</td>
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<tr>
<td>Max ABG (dB HL)</td>
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<td>0–55</td>
<td>12.7</td>
<td>0.046</td>
<td>0.02</td>
<td>0.11</td>
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<tr>
<td>cVEMP (dB)</td>
<td>73.3</td>
<td>45–106</td>
<td>10.2</td>
<td>0.26</td>
<td>0.23</td>
<td>0.025</td>
</tr>
</tbody>
</table>

FIG. 2. Simple linear regression of SCD length and average bone-conduction thresholds. The linear regression line shows a slightly positive correlation between SCD length and average bone-conduction thresholds (slope = 0.29). The Pearson’s correlation coefficient is not statistically significant (p = 0.67).

FIG. 3. Simple linear regression of SCD length and maximal air-bone gap. The linear regression line shows a positive correlation between SCD length and maximal air-bone gap (slope = 2.14). The Pearson’s correlation coefficient is statistically significant (p = 0.046).

FIG. 4. Simple linear regression of SCD length and cVEMP threshold. The linear regression line shows a negative correlation between SCD length and cVEMP threshold (slope = −1.11). The Pearson’s correlation coefficient is not statistically significant (p = 0.26).
determination (analysis of cVEMP threshold and dehiscence length, threshold and dehiscence length. Multivariate regression analysis of cVEMP threshold and dehiscence length, controlling for age and sex (Table 1), showed the regression coefficient to be $-1.22 (p = 0.23)$. Coefficient of determination ($R^2$) for the multivariate model was 0.025. Thus, although cVEMP thresholds may be reduced in SCD syndrome compared with controls, there was no significant correlation between cVEMP threshold and dehiscence length.

**SCD Signs and Symptoms**

We examined whether dehiscence length was associated with the total number of vestibular and auditory signs and symptoms in SCD patients. Each SCD patient in this study was assessed for the presence of 7 vestibular and 3 auditory signs and symptoms (listed in the Methods). The median number of signs and symptoms in our patient cohort was 6 (range, 1–10). One patient had only vestibular complaints, whereas 2 had only auditory complaints. The remaining 82 patients experienced both auditory and vestibular signs and symptoms. Poisson regression, a correlation technique used for count data, was used to analyze the association between the number of presenting signs and symptoms and dehiscence length. The Poisson regression coefficient was 0.03 and was not statistically significant ($p = 0.39$).

**DISCUSSION**

Superior canal dehiscence syndrome was first described by Minor et al. (1) in 1998 after examining a group of patients presenting with vertigo and oscillopsia. High-resolution temporal bone CT scan of these patients confirmed a dehiscence of bone covering the superior semicircular canal. It was later found that some of these patients also can present predominantly with auditory signs and symptoms, especially low-frequency conductive hearing loss and hyperacusis to bone-conducted sounds (5,6). The mechanism in which SCD causes its clinical manifestations can be explained by the third-window hypothesis. Normally, the inner ear has 2 mobile windows: the oval and the round windows. In the presence of SCD, a third-mobile window is created in the inner ear, which acts as a shunt pathway for sound energy to travel through the labyrinth instead of the cochlea. This can lead to both auditory and vestibular signs and symptoms (1,2,13,14).

The reason why SCD causes predominantly vestibular findings in some patients, auditory complaints in others, and a combination of both vestibular and auditory abnormalities in yet another group of patients is unclear. Some have postulated that dehiscence size and/or location may determine the clinical presentation in SCD. It may seem logical to assume that larger dehiscences would cause an increase in the number and severity of auditory and vestibular signs and symptoms because the shunt pathway created by a larger dehiscence might be expected to cause more significant effects on inner ear mechanics. However, a mechano-acoustic model analysis of inner ear mechanics suggests otherwise.

Songer and Rosowski (15) developed a mechano-acoustic model of SCD to examine the effect of SCD length on hearing. In this model, a two-port network was used, and the transmission matrix parameters were derived using measurements in chinchilla ears. A lumped-element model of SCD was created based on anatomic parameters calculated from a histologic reconstruction of the chinchilla inner ear. According to this model, when the dehiscence is very small, there should be very little effect on auditory sensitivity (as defined by cochlear potential normalized by sound pressure). As the dehiscence size approaches the cross-sectional area of the superior canal lumen, the auditory sensitivity decreases. However, once the dehiscence size exceeds the cross-sectional area of the superior canal lumen, there is little further effect on auditory sensitivity.

In this study, we examined the association between dehiscence length and clinical manifestations in SCD patients. Our analysis showed that only maximal air-bone gap was statistically significantly associated with dehiscence length. All other variables that we examined, including PTA, average bone-conduction threshold, cVEMP threshold, and the total number of auditory and vestibular signs and symptoms, did not correlate significantly with SCD length. Because this study only focused on patients who underwent surgical repair of SCD, and because only those who were affected more severely would seek surgical treatment for SCD, it is possible that the dehiscence length of our study cohort may be too large to show any effect of dehiscence length on most of the studied variables. Specifically, although the smallest dehiscence length we measured was approximately 1 mm, most of the dehiscences were longer than 2 mm in our study cohort. Igarashi (16) found that the diameter of the human semicircular canal was 1.44 mm. Thus, the dehiscence lengths in this cohort of patients who underwent surgery may have largely exceeded a value below which graded effects on auditory or vestibular function might be seen. However, the fact that there was a statistically significant correlation between maximal air-bone gap and dehiscence length suggests that, although the effect of dehiscence length on air conduction and bone conduction thresholds individually may be small, when the dehiscence was large, their combined effects are still detectable.

A recent study by Pfammatter et al. (7) examined the correlation between dehiscence length and patient symptoms in 27 patients with SCD. The dehiscence length was estimated using temporal bone CT scan alone in their study. They found that patients with larger dehiscences ($\geq 2.5$ mm) presented predominantly with both vestibular and auditory abnormalities, whereas patients with smaller dehiscences ($< 2.5$ mm) presented with either auditory or vestibular dysfunction. They also found that patients with SCD length of 2.5 mm or greater were more likely to have a cVEMP threshold of 80 dB or lower, whereas those with SCD length less than 2.5 mm were more likely to have a cVEMP threshold of greater than 80 dB. In another...
in our case versus tical techniques used for data analysis (Poisson regression).

The dehiscence lengths were not stated specifically in that study for comparison. Another possible source of disparity is the different statistical techniques used for data analysis (Poisson regression in our case versus t tests in the study by Pfammatter et al.).

Our study found a statistically significant association between maximal air-bone gap and dehiscence length. This finding is similar to the study by Yuen et al., although average air-bone gap was used in their study instead. It is important to point out that our data were much more scattered than those of the study by Yuen et al., as evidenced by the small $R^2$ in our multivariate regression analysis (0.11) compared with theirs (0.78). The $R^2$ of our regression model also did not improve when fitted quadratically (data not shown). One possible source of difference was that patients who had previous SCD repair were included in the Yuen study. In fact, in 1 case, the patient was included twice (both preoperatively and postoperatively). Because previous otologic surgery is a known risk factor for postoperative hearing (17), inclusion of patients with previous otologic surgery may lead to biased results.

In considering studies correlating dimensions of SCD with signs and symptoms, caution must be exercised when interpreting data obtained from CT measurements but not confirmed at the time of surgery. It is our experience that high-resolution computed tomography commonly overestimates the size of the dehiscence that is actually found at the time of surgery. In the present study, using measurements actually obtained at surgery, we found that the only significant correlation of SCD size with auditory and vestibular signs and symptoms was for the maximal air-bone gap.

One aspect of vestibular function was not investigated in this study: the correlation between dehiscence length and the rotational sensitivity of the dehiscent superior canal. Cremer et al. (3) showed that when dehiscence length exceeded 5 mm, there was a significantly lower gain of the affected canal’s angular vestibuloocular reflex (AVOR) compared with that of normal canals or canals with SCD lengths shorter than 5 mm. This effect has been attributed to “autoplugging” of the canal, that is, the presumed herniation of the dura into the lumen of the dehiscent canal to a sufficient extent to compress the membranous duct and dampen endolymph flow under normal circumstances even before surgery. Demonstrating quantitative AVOR gains requires magnetic search coil or high-speed video recordings that are beyond the scope of this study of a large cohort of patients. The present study does not negate the “autoplugging” effect of large dehiscences on AVOR gain, but it points out that effects of dehiscence length on auditory and vestibular signs and symptoms are otherwise so small or variable that they cannot be detected in a cohort of this size or using these common clinical measures.

CONCLUSION

In this study, we examined the correlation between SCD length and objective auditory and vestibular measurements in SCD patients. We also examined the correlation between SCD length and the presenting signs and symptoms in these patients. We found that there was a statistically significant correlation between SCD length and maximal air-bone gap. There was no statistically significant correlation between SCD length and PTA, average bone-conduction thresholds, cVEMP thresholds, and the total number of auditory and vestibular signs and symptoms.

REFERENCES


