Facial Nerve Stimulation From Cochlear Implants

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Objective: To evaluate the incidence of facial nerve stimulation from cochlear implants and to better define the segment of nerve being stimulated and the causes of stimulation.

Study Design: Retrospective patient case review and a temporal bone dissection study.

Setting: A tertiary care setting.

Patients: All patients given a cochlear implant at the Hospital of the University of Pennsylvania. This encompassed only adult patients.

Intervention: All patients had surgical insertion of either a 3M single channel, Nucleus 22-channel, or CLARION multichannel cochlear implant.

Main Outcome Measures: Demonstration of facial nerve stimulation with a cochlear implant and determination of affected electrodes; measurement of electrode location and distances between the labyrinthine segment of the facial nerve and the cochlea in temporal bone dissections; and determination of the relationship between the labyrinthine facial nerve and the cochlea using computed tomography evaluation.

Results: The overall incidence of facial nerve stimulation using all three devices was 14% (8 of 58). Otosclerosis and otosphylls appear to be predisposing conditions to stimulation. The mid-cochlear electrodes, located near the labyrinthine facial nerve, appear to cause stimulation of the VIIth nerve most commonly. Computed tomographic evaluation of the bone between the labyrinthine fallopian canal and the cochlea may provide some indication of potential facial nerve problems.

Conclusion: Facial nerve stimulation from the use of cochlear implants is more prevalent in patients with otosclerosis and otosphylls. The labyrinthine segment of the facial nerve is the most likely area being stimulated in most patients. Preoperative computed tomographic evaluation may be beneficial in determining the possibility of this problem. Key Words: Cochlear implant—Facial nerve stimulation.


Facial nerve stimulation from the use of activated cochlear implants is a well-known side effect in a small but significant number of patients. When initially reported by Cohen and Hoffman (1,2) the incidence was quite low. With multiple subsequent reports, however, the incidence appears to be higher (3–8), up to 14.6% in adult patients (3). Facial nerve stimulation in the pediatric implant population has been reported infrequently (8,9). This problem has been described with the use of the 3M/House single-channel device (3M, St. Paul, MN, U.S.A.) and with the multichannel Nucleus (Cochlear Corp., Englewood, CO, U.S.A.) and Ineraid devices. Currently, there are no published studies of which we are aware that report facial stimulation with the CLARION (Advanced Bionics Corp., Sylmar, CA, U.S.A.) implant. It has been documented that facial nerve stimulation is often delayed and progresses with time (3,4,7,8). The specific mechanisms causing the stimulation and the reasons for its progression are unknown; however, several pathways of current spread have been proposed (3–8,10–12). This problem has been noted to occur more frequently with otosclerosis. It has been suggested that the labyrinthine segment of the facial nerve is the most likely area of stimulation in most patients (2,7,13).

MATERIALS AND METHODS

Fifty-four patients have undergone a total of 58 cochlear implant placements since 1980 at the University of Pennsylvania Medical Center. Five 3M/House single-channel devices, 42 Nucleus 22-channel devices, and 10 CLARION multichannel devices have been implanted and activated. One Nucleus device was explanted secondary to infection before activation. The records from all patients receiving cochlear implants from 1980 to 1997 were reviewed regarding facial nerve stimulation, timing and progression of stimulation, and for the method of stimulus elimination. The preoperative computed tomography (CT) scans of five of the seven patients that stimulated were available and were retrospectively and blindly reviewed. One of the five patients also had a postoperative CT scan, which was reviewed.

Eight temporal bones were dissected. The distance from the labyrinthine segment of the facial nerve to the scala tympani and scala vestibuli was then measured. These bones were then

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implanted with a Nucleus 22-channel practice electrode into the scala tympani. The electrode closest to the labyrinthine portion of the facial nerve was recorded.

RESULTS

3M single-channel device

Of the five patients who were given the 3M single-channel device, one had facial nerve stimulation, which occurred 8 years after implantation (patient no. 1, Table 1). The facial nerve stimulation was eliminated by reducing the clipping level. At 12 years, the problem recurred and again was eliminated by reducing the clipping level further.

CLARION device

Ten patients have undergone implantation and activation with the CLARION multichannel device. One experienced facial nerve stimulation (patient no. 1, Table 1). This patient had previously been given the 3M single-channel device and was the same patient in the single-channel series (described previously) who experienced facial nerve stimulation. The single-channel device was explanted secondary to failure of the implant. The CLARION device was placed in that same ear because of previous infectious problems in the contralateral ear. On activation of the device, facial stimulation occurred from channel 4 and progressed to involve channels 2–8 within 2 months. Facial stimulation was eliminated without affecting the hearing by setting the M levels below the level of facial stimulation. To our knowledge, this is the first reported case of facial nerve stimulation with the CLARION cochlear implant.

Nucleus 22-channel device

Forty-two patients have undergone implantation and activation with the Nucleus 22-channel device. Six of these patients experienced facial nerve stimulation for an incidence of 14.3% (Table 1). Three of the four patients with otosclerosis in our patient group experienced facial nerve stimulation. Two of these same patients also had a diagnosis of Meniere’s disease. The patient with only otosclerosis (patient no. 2) had a single-channel implant in the contralateral ear. No facial stimulation was observed with this device. Evidence of severe cochlear otosclerosis was evident on the preoperative CT scan. A Nucleus implant was inserted into the scala vestibuli secondary to partial ossification of the scala tympani just beyond the first segment of the basilar turn.

Two of our three patients with otitic syphilis also had facial nerve stimulation. The other patient had idiopathic, possibly noise-induced, hearing loss. All patients with stimulation had a full insertion of the active electrodes and insertion of 3–10 supporting rings.

Two patients had stimulation of the facial nerve on the first day of activation and the remainder were delayed from 2 days to 8 months. All patients had progression of the facial nerve stimulation, which initially involved only one or two electrodes and eventually involved four to seven electrodes (Table 1). This progression continued from 3 weeks to 4 years.

Radiologic evaluation

The preoperative CT scans of five of the seven patients who had facial nerve stimulation were available and reviewed. This was done to evaluate the relationship of the labyrinthine segment of the facial nerve to the cochlea. One scan did not cover the area of concern adequately, leaving only four scans for comparison.

In the patient with idiopathic hearing loss (patient no. 7, Table 1), there appeared to be minimal bone to actual dehiscence of bone between the cochlea and labyrinthine segment of the facial nerve bilaterally (Fig. 1). This patient had two electrodes that immediately caused facial stimulation on activation of the device, which eventually required elimination of seven electrodes. A postoperative scan obtained on this patient demonstrated that the implant was abutting the labyrinthine portion of the facial nerve (Fig. 2).

The patient with acquired luetic hearing loss (patient no. 6, Table 1) had significant radiographic changes in the temporal bone bilaterally, consistent with otosyphilis (Fig. 3). The patient with congenital syphilis as the cause of her hearing loss demonstrated minimal radiographic demineralization on CT. There appeared, however, to be a connection between the cochlea and labyrinthine segment of the nerve on the implanted side but not on the nonoperated side.

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age of implant</th>
<th>Sex</th>
<th>Hearing loss etiology</th>
<th>CT type</th>
<th>Side</th>
<th># of electrodes inserted</th>
<th>Onset of VII stimulation from activation</th>
<th>Time until stabilization</th>
<th>Stimulating electrodes Initial</th>
<th>Total</th>
<th>Hearing affected</th>
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<tbody>
<tr>
<td>1</td>
<td>67</td>
<td>M</td>
<td>Idiopathic</td>
<td>3-M</td>
<td>R</td>
<td>1</td>
<td>8 years</td>
<td>4 years</td>
<td>–</td>
<td>–</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>80</td>
<td></td>
<td></td>
<td>Clarion</td>
<td>R</td>
<td>16</td>
<td>Immediate</td>
<td>2 months</td>
<td>ch-4</td>
<td>ch2-8</td>
<td>No</td>
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<tr>
<td>3</td>
<td>71</td>
<td>M</td>
<td>Otosclerosis</td>
<td>L</td>
<td>22 + 10</td>
<td>2 weeks</td>
<td>3 months</td>
<td>12</td>
<td>12,14,15,17</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>M</td>
<td>Otosclerosis/Meniere's</td>
<td>L</td>
<td>22 + 7</td>
<td>2 days</td>
<td>2 months</td>
<td>13,15</td>
<td>12,13,15–17</td>
<td>No</td>
<td></td>
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<tr>
<td>5</td>
<td>80</td>
<td>F</td>
<td>Otosclerosis/Meniere's</td>
<td>L</td>
<td>22 + 3</td>
<td>8 months</td>
<td>1 year</td>
<td>18</td>
<td>17–20</td>
<td>Yes</td>
<td></td>
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<td>6</td>
<td>68</td>
<td>F</td>
<td>Late congenital syphilis</td>
<td>L</td>
<td>22 + 10</td>
<td>Immediate</td>
<td>3 months</td>
<td>10</td>
<td>7–11</td>
<td>No</td>
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<tr>
<td>7</td>
<td>48</td>
<td>P</td>
<td>Acquired syphilis</td>
<td>L</td>
<td>22 + 4</td>
<td>1 month</td>
<td>9 months</td>
<td>10,12</td>
<td>12–16</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>59</td>
<td>M</td>
<td>Idiopathic-pass noise induced</td>
<td>L</td>
<td>22 + 6</td>
<td>Immediate</td>
<td>4 years</td>
<td>15,17</td>
<td>14–20</td>
<td>Yes</td>
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TABLE 2. Temporal bone dissection

<table>
<thead>
<tr>
<th>Bone #</th>
<th>Distance of labyrinthine segment of facial nerve to scala tympani</th>
<th>Distance of labyrinthine segment of facial nerve to scala vestibuli</th>
<th>Nucleus 22-channel electrode closest to labyrinthine segment of facial nerve</th>
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<tr>
<td>1</td>
<td>0.1mm</td>
<td>0.5mm</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>0.4mm</td>
<td>0.8mm</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>0.4mm</td>
<td>0.6mm</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>0.1mm</td>
<td>0.4mm</td>
<td>13</td>
</tr>
<tr>
<td>5</td>
<td>0.2mm</td>
<td>0.7mm</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>0.4mm</td>
<td>0.8mm</td>
<td>8</td>
</tr>
<tr>
<td>7</td>
<td>0.4mm</td>
<td>0.6mm</td>
<td>8</td>
</tr>
<tr>
<td>8</td>
<td>0.3mm</td>
<td>0.7mm</td>
<td>9</td>
</tr>
<tr>
<td>Average</td>
<td>0.29mm</td>
<td>0.64</td>
<td>10</td>
</tr>
</tbody>
</table>

The fourth patient whose scan was evaluated had a history of otosclerosis and Meniere's-like symptoms. There appeared to be good bone between the cochlea and the facial nerve in this patient. Her stimulation was delayed 8 months from the time of activation.

Temporal bone dissection

Eight temporal bones were dissected and implanted with a Nucleus 22-channel practice electrode array. The distance between the labyrinthine segment of the facial nerve and the scala tympani and scala vestibuli were measured. The electrode closest to this point was identified (Table 2). These measures demonstrated that the distance between the labyrinthine segment of the facial nerve and cochlea was small but that there was also variation among specimens. It was also apparent that the scala tympani was uniformly closer to the nerve than the scala vestibuli. The electrode closest to the nerve varied from numbers 8–13. The electrodes that were involved in facial nerve stimulation in our patients ranged from numbers 10–18 initially and eventually involved electrodes 7–20 (Fig.4).

DISCUSSION

Facial nerve stimulation has been a known complication of cochlear implantation for many years. Nevertheless, its incidence remains unclear. Earlier studies indicated that it was a relatively rare complication affecting ≈1% of patients (1,2,14). More recent studies have demonstrated a variable reported incidence of facial nerve stimulation (Table 3) (3–9). Cochlear implants, when activated, generate electrical fields that allow some current to spread outside the cochlea (3,4,10–12,15,16). Facial nerve stimulation may thus be regarded as an electrical shunt between the cochlear implant and the facial nerve (7). The mechanism of this stimulation is unknown, however, but has been postulated to occur through multiple different routes by various authors. In certain cases, it is apparent that the tympanic segment of the nerve is the portion being stimulated. Graham et al. found with a single-channel device that intraoperative placement at the round window or promontory resulted in facial nerve stimulation, and placement of the device deeper into the cochlea eliminated this (5). Niparko et al. had two patients using the Nucleus device who received stimulation from electrodes that were adjacent to the round window, suggesting spread of current through the cochleostomy to the tympanic or vertical segments of the nerve (3). Bredberg and Lindstrom have also noted stimulation from electrodes in the basal segment of the cochlea near the round window (6). It has been suggested

FIG. 1. A: Coronal CT scan with bone algorithm demonstrates diminution in the amount of bone between the labyrinthine segment of the facial nerve (arrow) and the superior aspect of the cochlea on the right side. The changes are present to a lesser degree on the left side. B: Coronal magnified view of the right side of the temporal bone shows no significant bone between the facial nerve and the cochlea.
that a low impedance shunt exists at the base of the modiolus of the cochlea (10–12) and that this could also account for stimulation with basal electrodes (3).

Most cases of facial nerve stimulation occur from the electrodes deep within the cochlea (3,6). With the Nucleus device, a bipolar stimulating configuration is usually used. The current required to produce auditory stimulation with a bipolar configuration is significantly higher than with monopolar stimulation. In addition, all the electrodes are located within the cochlea, and it has been demonstrated that most of the current passes directly through the perilymph (12). It has been suggested that current is shunted to the labyrinthine segment of the facial nerve, which causes stimulation (2,7,13). The superior most aspect of the basal turn of the cochlea is known to be close to this portion of the facial nerve. If the bone separating the superior basal turn from the labyrinthine segment of the facial nerve is thin or dehiscent or if through some process the impedance of bone is lowered, it is likely that current spread through the perilymph could stimulate the facial nerve in this area.

Certain causes of hearing loss may lower the impedance of bone and predispose patients using a cochlear implant to facial nerve stimulation. It has been suggested that otosclerosis can be a predisposing condition (4,7,13). Otosclerosis is a disorder of bone metabolism that creates

![FIG. 2. Coronal CT scan with bone algorithm shows the cochlear implant (larger arrow) adjacent to the facial nerve (small arrow) on the left side. No bone separates the implant from the nerve.](image1)

![FIG. 3. Coronal CT scan with bone windowing demonstrating demineralization around both cochleae, comparable with either otospongiosis or otosphyllis. On the left side, the facial nerve and the spongiotic bone are contiguous.](image2)
immature spongy bone that is often softer than normal labyrinthine bone (4,7). These otospongiotic changes in the cochlear capsule may reduce the bone impedance, allowing more current than usual to reach the facial nerve (6,13). In the most serious cases, massive bone resorption can occur, allowing for the possibility of close contact between the electrode array and the facial nerve (4). Three of our four patients with otosclerosis experienced facial nerve stimulation.

Two of the seven patients who experienced stimulation suffered from Meniere’s disease. These two patients also had otosclerosis and are included in that group. It is possible that endolympathic hydrops may contribute via some unknown mechanism to facial nerve stimulation after cochlear implants, and Shea and Domico reported that three of their eight patients with facial nerve stimulation had hydrops (8). It is more likely, however, that otosclerosis is the predisposing factor in our two patients.

Two of our cases had luetic hearing loss. One patient had late-onset congenital syphilis, known to cause hearing loss in up to 38% of patients, usually in adulthood (17). The other patient suffered from acquired syphilis, which can have otitic involvement most often in its tertiary form (18). The facial nerve stimulation that occurred after cochlear implant activation in these patients is likely a result of bony changes that occur in late syphilis. These changes include a productive periostitis with diffuse endosteal inflammation and a gummatous periostitis/osteomyelitis (17). Although these changes most commonly affect the semicircular canals and labyrinthine capsule, the cochlear canal can also be involved (17). It is likely that the bony changes reduce the impedance of the cochlear capsule in a manner similar to that of otosclerosis, allowing current from the cochlear implant to reach the neighboring facial nerve more easily. The bone between the facial nerve and cochleae could also have been thinned in this process. In the temporal bone, the radiographic changes of otosyphilis can resemble otospongiosis (Fig. 3).

From our temporal bone observations, the amount of bone in the superior most basal turn of the cochlea sepa-

rating the scala tympani from the labyrinthine segment of the facial nerve can be variable (Table 2). In some instances the bone has been found to be extremely thin. This could theoretically predispose the facial nerve to unwanted stimulation from current spreading through the perilymph. It was also seen, from Table 2, that the distance from the scala tympani to the facial nerve is consistently less than the distance from the scala vestibuli to the facial nerve. From this observation, one could propose that implantation into the scala vestibuli may reduce the incidence of stimulation of the labyrinthine segment of the facial nerve. A scala vestibuli insertion was required in three of our patients given the Nucleus implant; however, one of these patients (patient no. 2, Table 1) still experienced stimulation.

After the temporal bone specimens were implanted with the Nucleus 22 electrode array, it was noted that the electrode adjacent to the labyrinthine segment of the facial nerve can vary even when the insertion is consistently a full 25 mm from the round window. This includes all the active electrodes and the 10 supporting rings. The electrodes recorded to be closest to the labyrinthine segment of the facial nerve in the temporal bone specimens ranged from numbers 8–13 with a full insertion (Table 2). This may be due to some small variation in cochlear length, slight variation of the position of the nerve to the cochlea, or more likely to positioning differences and some bending of the electrode array that can occur with insertion. These observations could account for some of the variation seen among patients when examining the specific electrodes that caused stimulation. When the numbering of electrodes causing stimulation on our patients was adjusted for a less than full 25-mm insertion length, the electrodes causing stimulation ranged from numbers 6–17 (Fig. 5). This would correspond well with our temporal bone work. In comparing a similar calculation to the seven patients in Bredberg’s (6) series who did not have basal stimulation, their stimulating electrodes would have ranged from numbers 8–17, which also correlates well with our patients and the temporal bone observations.
These findings prompted us to reexamine the temporal bone CT scans of the patients in our series who experienced stimulation to determine the proximity of the labyrinthine portion of the fallopian canal to the cochlea. This relationship can easily be seen if fine-cut bone window coronal images of the temporal bone are obtained. Five of the seven patients had preoperative CT scans available for review and one had a postoperative CT. In four of these, the scans were detailed enough to evaluate the relationship of the cochlea and labyrinthine segment of the facial nerve. Three of these four scans demonstrated abnormalities of the bone between the labyrinthine facial nerve and the cochlea. These abnormalities were bilateral in two patients and only on the implanted side in one. They ranged from dehiscence of bone to demineralization and thinning. One patient appeared to have adequate bone in this area. Her facial nerve, however, did not stimulate until 8 months after activation, shortly after she suffered some mild closed head trauma (patient no. 4, Table 1). The patient with bilateral dehiscence of bone separating the cochlea from the labyrinthine segment of the facial nerve had a postoperative scan demonstrating the electrode array directly adjacent to the nerve (Fig. 2). He experienced immediate facial nerve stimulation on activation and eventually experienced stimulation from seven electrodes, which was the largest number in our series. It is suggested from this examination that the bone separating the labyrinthine segment of the facial nerve from the cochlea should be evaluated in the preoperative CT. With further study, this may prove helpful for determining the risk of postoperative facial nerve stimulation or at least determining the side of implantation when other criteria are symmetric.

We have observed that, in all of our cases of stimulation with multichannel implants, the extent of electrode involvement does not remain constant with time. Rather, it appears that asymptomatic electrodes adjacent to the initial offending ones eventually cause facial nerve stimulation. This expansion is usually self-limiting, as we have not seen a case involving more than seven electrodes; however, stimulation in patient no. 7 (Table 1) continued to spread for 4 years. Others have reported more extensive involvement requiring explantation (1,2,8).

It is unknown why the facial nerve stimulation is often delayed and migrates to adjacent electrodes. It is known that with electrical stimulation changes in bone can occur. If this occurs with the current of a cochlear implant, it is possible that the bone separating the cochlea and the facial nerve may thin with time, allowing facial nerve stimulation to begin or spread. The effects of electrical fields, however, appear to enhance osteogenesis exclusively without altering bone resorption in the long bones (19). Marsh et al. have also found evidence for bone formation in the cochlea of a patient who died 5 months after implantation (13). It is unknown if the electrical stimulation from the implant influenced this bone formation, but given these observations it would seem unlikely that electrical current would cause any further thinning of bone in the cochlea.

It has been well documented that there can be considerable trauma to the cochlea on insertion of the electrode array (13,20). It is possible that this could contribute to an increased risk of facial nerve stimulation, but the severity of this trauma does not appear to fracture or thin the outer wall of the cochlear canal where the facial nerve is located.

It is possible, however, that the physical pressure exerted on the outer wall of the cochlea by the Nucleus 22-channel implant from the spring-like effect of its initially straight, but now curled, electrode array may with time cause erosion of the thin bone separating the labyrinthine facial nerve from the cochlea. An electrode adjacent to the facial nerve in this instance could cause direct stimulation. The length and number of electrodes involved in this process, however, goes against direct contact of the nerve by a specific electrode or two in most cases. The length of the electrode array causing facial stimulation in our patients ranged from ~3 mm to nearly 5.5 mm. The portion of the cochlea directly adjacent to the labyrinthine segment of the facial nerve is only 1 or 2 mm.
suggesting that the current is indeed spread through the perilymph.

Treatment of facial nerve stimulation after cochlear implants is often simple and effective but can be quite challenging in some instances. The offending electrodes may be reprogrammed out of the MAP, usually without reducing the patient's hearing capacity once they have adjusted to the new program. In cases in which the patient's hearing is adversely affected, the current for the offending electrodes can often be set below the stimulus level inducing the facial nerve symptoms while still reaching auditory stimulation. In some patients the hearing is significantly affected by the elimination of electrodes, reducing the usefulness of the implant dramatically, as occurred in patient nos. 4 and 7. Botulinum toxin injections have been successfully used in severe cases when reprogramming is not effective (16).

It is thought that steering and focusing of the current is improved if the electrodes in the array are placed near the cells of the auditory nerve (21). If the electrodes are placed closer to the modiolus, as is done with the CLARION implant, it may be possible to reduce the flow of current to the facial nerve that is adjacent to the outer rim of the cochlea and therefore farther away from the stimulus. Some current spread through the perilymph, however, would still be expected. In the Nucleus implant, the electrodes are bands. This characteristic would therefore place them physically closer to the nerve. Even with the CLARION device, 1 patient in our 10 experienced stimulation, so it remains to be seen whether this electrode design will reduce the overall incidence of facial nerve stimulation.

CONCLUSION

Facial nerve stimulation from the use of cochlear implants is not uncommon and has occurred in patients using the 3M single-channel, the Nucleus 22-channel, the Ineraid, and the CLARION multichannel devices. Our incidence is ~14%. Otosclerosis and otitic syphilis appear to be predisposing conditions. The labyrinthine segment of the facial nerve is the most likely area of stimulation in most patients. Preoperative evaluation of a coronal bone window CT scan to examine the bone separating the cochlea from this portion of the VIIth nerve may be useful.

REFERENCES


