Frequency transposition around dead regions simulated with a noiseband vocoder

Deniz Başkent[a] and Robert V. Shannon
Department of Biomedical Engineering, University of Southern California, Los Angeles, California 90089 and Department of Auditory Implants, House Ear Institute, 2100 West Third Street, Los Angeles, California 90057

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In sensorineural hearing loss, damage to inner hair cells or the auditory nerve may result in dead regions in the cochlea, where the information transmission is disrupted. In cochlear implants, similar dead regions might appear if the spiral ganglia do not function. Shannon *et al.* [J. Assoc. Res. Otolaryngol. **3**, 185–199 (2002)] simulated dead regions of varying size and location using a noiseband vocoder. Phoneme recognition by normal-hearing subjects was measured under two frequency-place mapping conditions: the frequency range corresponding to the dead region was (1) removed or (2) reassigned to bands adjacent to the dead region to simulate the off-frequency stimulation of neurons at the edge of a dead region. The present study extends the results of Shannon *et al.* by including a frequency transposition mapping condition, where the overall acoustic input frequency range was distributed over the entire remaining nondead region. The frequency transposed map provided more acoustic information when compared to the map with the frequency range corresponding to the dead region removed. However, speech perception did not improve for many simulated dead region conditions, possibly due to the spectral distortions in the frequency-place mapping. © *2006 Acoustical Society of America.* [DOI: 10.1121/1.2151825]

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I. INTRODUCTION

Sensorineural hearing loss is often caused by damage in hair cells or the auditory nerve. When the damage is in the outer hair cells, the active amplification mechanism in the cochlea diminishes and the auditory filters become broader (Glasberg and Moore, 1986). Such reduced frequency selectivity is believed to be one of the main factors leading to poor speech recognition in noise by hearing-impaired listeners (Stelmachowicz *et al.*, 1985). When the damage is in the inner hair cells or in the auditory nerve, the efficiency of the information transmission to higher stages of the auditory pathway is reduced (Moore and Glasberg, 1997). Listeners with this type of pathology usually demonstrate poor speech recognition even in quiet listening conditions (Pauler *et al.*, 1986). In cochlear implants, the hair cells are bypassed and the sound information is delivered by direct electrical stimulation of the auditory nerve. However, the survival rate or the functionality of the spiral ganglia, or abnormalities in more central auditory nerves, might affect speech recognition abilities of cochlear implant users (Gomaa *et al.*, 2003). Histopathological studies on temporal bones taken post mortem from hearing-impaired (Schuknecht and Gacek, 1993) or implant subjects (Khan *et al.*, 2005) showed that the cochlear pathology may differ for each patient. In hearing impairment, if the inner hair cells or the auditory nerve are severely damaged, the transduction from mechanical vibrations of basilar membrane to action potentials might be disrupted for that particular region, resulting in a dead region (Moore and Glasberg, 1997). In implants, if the spiral ganglia did not survive, the transmission of the electrical signal to the nerve might similarly be hindered.

Shannon *et al.* (2002) simulated dead regions with cochlear implant listeners, by selectively turning off electrodes, and in normal-hearing listeners, by using a noiseband vocoder. The analysis bands of the vocoder represented the acoustic input, and the carrier bands represented the cochlear stimulation range. The dead regions were simulated by dropping carrier bands of the vocoder. In the first frequency-place mapping condition, the acoustic information falling in the dead region was also dropped, preserving the matched spectral mapping for the remaining regions. Speech recognition was measured as a function of the size and location of the dead region. The simulated cochlear locations of the dead regions were apical, middle, and basal, with the logarithmic center frequencies of around 1, 2.4, and 5 kHz, respectively. Once normalized for the baseline condition where there was no dead region, performances by implant users and normal-hearing subjects were similar. Speech recognition by all subjects decreased as the size of the dead region increased. The effect was more pronounced for dead regions simulated in the apical region, where frequencies around 1 kHz were missing.

Because there is no or minimal information transmission in a dead region, the audiometric threshold of a tone falling in the dead region would theoretically be infinite. At loud levels, however, the neighboring healthy regions can be stimulated by the spread of basilar membrane vibrations, resulting in an off-place detection of the tone (Florentine and Houtsma, 1983). In this case, even though the correct place for the tone frequency was not stimulated, the audiogram

[a]Electronic mail: deniz_baskent@starkey.com

would show a finite but high threshold for the tone frequency. Numerous studies (Rankovic, 1991; Hogan and Turner, 1998; Vickers *et al.*, 2001) have indicated that amplification in dead regions might be more harmful than beneficial, probably due to such distortions in the frequency-place excitation patterns. Similar spectral distortions may arise in electrical hearing if an electrode is located in a region where the spiral ganglia do not function or are mostly vanished. The current levels, then, might have to be set to high values to stimulate a sufficient number of spiral ganglia to produce a sensation. Irregularities in the stimulation levels, measured for electrodes located at different cochlear locations, have been shown for stimulation modes that produce a narrow spread of activation, such as bipolar (Pfingst and Xu, 2004; Pfingst *et al.*, 2004) or tripolar (Bierer, 2005) configurations. The strong current fields produced due to high stimulation levels might lead to off-place stimulation of the ganglia on the edges of the dead region. In the second condition, Shannon *et al.* (2002) simulated such off-place stimulation as a local distortion in the frequency-place mapping. They reassigned the envelopes from the analysis bands in the dead region to the carrier bands immediately neighboring the dead region. The results with the reassigned condition did not differ from the results of the first condition, where the bands were simply dropped.

As a feasible alternative to amplification in the dead regions, where there was no benefit for some hearing-impaired subjects, frequency transposition was suggested. The idea was to shift the frequency information from the dead region into an area of residual hearing. Most studies showed no significant improvement in speech recognition with frequency transposition (e.g., McDermott and Dean, 2000). However, Turner and Hurtig (1999) did show a benefit for some hearing-impaired subjects, when they kept the relative distance of formant peaks proportionate in the transposition of the high frequencies.

The dead regions with hearing-impaired listeners limit the stimulation range of the cochlea that can be used for transmission of the acoustic information. A similar problem occurs with cochlear implant users when the stimulation range of the electrode array is limited. For example, if the array is inserted shallower than a full insertion, the apical cochlear regions, which would normally respond to low frequencies, will not be activated by electrical stimulation. Başkent and Shannon (2005) simulated shallow insertions with Med-El 40+ implant users and applied frequency transposition by compressing the entire speech spectrum to the limited stimulation range. An improvement in phoneme recognition was observed when the input frequency range was moderately compressed, but the performance dropped as the compression rate increased.

The present study is an extension of the Shannon *et al.* (2002) study and explores the feasibility of frequency transposition as a method to increase speech information transmission in the presence of dead regions, simulated with a noiseband vocoder. The first mapping condition in Shannon *et al.* (2002), where the acoustic information in the dead region was discarded, was repeated as the baseline condition in the present study. Dead regions of varying size and cochlear location were simulated with a noiseband vocoder and phoneme recognition by normal-hearing listeners was measured. In a second mapping condition, frequency transposition, the entire input spectrum was distributed over the remaining carrier bands that represented the nondead cochlear region. This second map is different from the locally distorted mapping condition simulated by Shannon *et al.*, where the analysis band envelopes from the dead region were assigned only to the carrier bands at the edges of the simulated dead region. In the frequency transposition map used in the present study, the distortion is distributed evenly across the entire nondead region (Fig. 1).

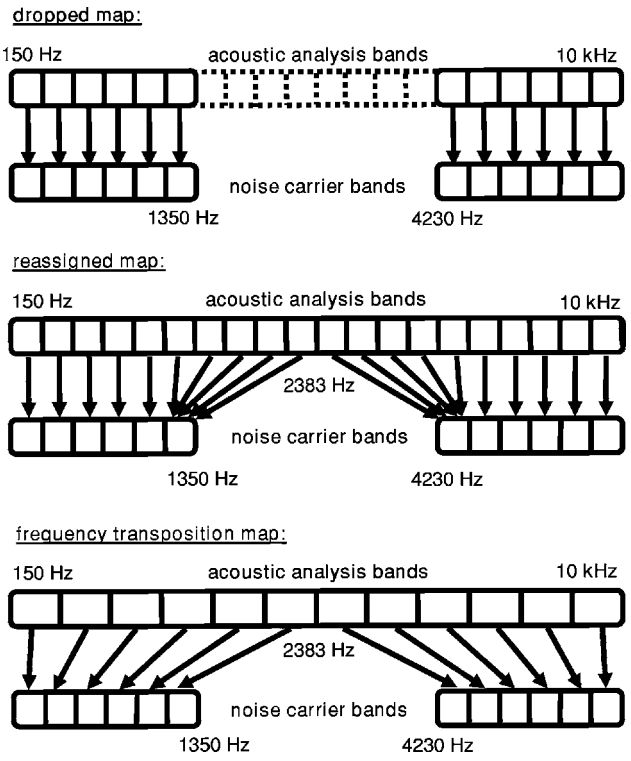

FIG. 1. Three frequency-place mapping conditions with a simulated dead region in the middle cochlear location. In the dropped condition, shown at the top, the acoustic information was removed from the analysis band range, and the remaining frequency range was assigned to matching carrier bands. This map was the first condition in the Shannon *et al.* study and the baseline condition of the present study. In the reassigned condition, shown in the middle, acoustic information was reassigned to the bands neighboring the dead region, simulating the off-place stimulation of auditory nerves. This map was the second condition in the Shannon *et al.* study and was not used in the present study. In the frequency transposition condition, shown at the bottom, the entire input acoustic spectrum was distributed over the remaining carrier bands. This map was used in the present study only.

II. METHOD

A. Subjects

Seven normal-hearing listeners, aged 22 to 36, participated in the experiment. All subjects were native speakers of American English and had hearing thresholds better than 20 dB HL at audiometric frequencies between 125 and 8000 Hz.

The performance of new subjects may increase during the first few testing sessions with noiseband vocoder experiments. The subjects who participated in the present study had

TABLE I. Summary of the simulated dead regions. In columns from left to right, the size of the dead region increases, as shown by the number of the carrier bands dropped. In rows from top to bottom, the simulated cochlear location of the dead region changes from base to apex. The top line in every entry shows which carrier bands were eliminated, numbering starting from the basal end, consistent with the Nucleus electrode array. The corresponding frequency ranges are presented in the bottom lines.

	Size			
Location	2 bands	4 bands	6 bands	8 bands
Basal	5–6	4–7	3–8	2–9
	4230–5635 Hz	3665–6504 Hz	3175–7506 Hz	2571–8664 Hz
Middle	10–11	9–12	8–13	7–14
	2065–2571 Hz	1789–3175 Hz	1550–3665 Hz	1350–4230 Hz
Apical	15–16	14–17	13–18	12–19
	950–1350 Hz	750–1550 Hz	550–1789 Hz	350–2065 Hz

extensive experience with vocoder processing from previous experiments, and they were also familiar with the stimuli. Therefore no training was provided and the data collection started from the first session.

B. Stimuli

The speech recognition tasks consisted of medial vowel and consonant discrimination. Vowel stimuli were taken from materials recorded by Hillenbrand *et al.* (1995). Listeners heard ten presentations, spoken by five male and five female talkers, of 12 medial vowels (ten monophthongs and two diphthongs) presented in a /h/-vowel-/d/ context (heed, hid, head, had, hod, hawed, hood, who'd, hud, heard, hayed, hoed). Chance level on this test was 8.33% correct. Consonant stimuli were taken from materials recorded by Turner *et al.* (1992) and Fu *et al.* (1998). Listeners heard six presentations, spoken by three male and three female talkers, of each of 14 medial consonants /b d f g k m n p s ʃ t θ v z/, presented in an /a/-consonant-/a/ context. Chance level on this test was 7.14% correct.

Subjects were tested once with all 84 consonant presentations and 120 vowel presentations for each condition. All stimuli were presented via loudspeaker in a sound field at 70 dB on an A-weighted scale, without lip-reading. Tokens were presented in random order by custom software (Robert, 1998). Subjects were asked to select the phoneme they heard from a menu displayed on the screen. No feedback was provided. On average, each session lasted from 2 to 4 h, and subjects were able to finish the experiment in three to four sessions.

C. Experimental procedure

A summary of the experimental conditions is given in Table I. From left to right, the size of the simulated dead region increases, and from top to bottom, the location of the simulated dead region moves from base to apex. The top line in every entry shows the number of the bands eliminated from the 20 carrier bands, with the numbering starting from the basal end, consistent with the Nucleus cochlear implant. The corresponding frequency ranges are presented in the bottom lines of each entry.

A noiseband vocoder (Shannon *et al.*, 1995) was implemented in Matlab to simulate the dead regions. To maintain consistency with Shannon *et al.* (2002), the SPEAK Table 9 of Nucleus cochlear implant (Cochlear Corporation, 1995) was used as the basis for the cutoff frequencies of spectral bands. The phonemes were band-pass filtered into 20 bands with sixth-order Butterworth filters, with the overall spectral range of 150 Hz to 10 kHz. The cutoff frequencies of the filters were partitioned linearly for frequencies up to 1550 Hz, and logarithmically for higher frequencies, as shown in the second column of Table II. The speech envelope was extracted from each analysis band by half-wave rectification and low-pass filtering using a third-order Butterworth filter with a cutoff frequency of 160 Hz. The carrier bands were produced by band-pass filtering white noise with sixth-order Butterworth filters. The speech envelopes were used to modulate the noise carrier bands. Eventually, the processed speech stimuli were produced by combining the modulated noise bands.

Dead regions were simulated by eliminating carrier bands in the synthesis stage of the vocoder processing. In the dropped map, analysis bands corresponding to the dead region were discarded, and the remaining analysis bands matched the remaining carrier bands. In the frequency transposition map, the entire analysis frequency range was distributed over the remaining carrier bands. The partitioning of the new analysis bands was similar to the partitioning of the carrier bands. Figure 1 shows an example of a dead region simulated in the middle cochlear location and with different mapping conditions applied. The cutoff frequencies of individual bands are shown in Tables II–IV for all simulated dead region conditions. The second column in each table shows the frequency partitioning of the analysis and carrier bands when there was no dead region. The subsequent columns show the cutoff frequencies of the analysis bands with frequency transposition and dropped maps, as the size of the dead region increases, from left to right. For all dead region conditions, the cutoff frequencies used for the carrier bands with both dropped and frequency transposition maps were the same as the cutoff frequencies used for the analysis bands with the dropped map.

TABLE II. Cutoff frequencies for individual vocoder bands for the dead region simulated in the basal cochlear location. The first column shows the numbering of the bands. The second column shows the partitioning of analysis and carrier bands when there was no dead region. The subsequent columns show cutoff frequencies of analysis bands with the frequency transposition and dropped maps, for the dead regions of 2, 4, 6, and 8 bands. The carrier bands for each dead region condition were the same as the analysis bands with the dropped map.

	None	2 bands, basal		4 bands, basal		6 bands, basal		8 bands, basal	
	analysis and carrier bands (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)
apical end	150	150	150	150	150	150	150	150	150
19-20	350	363	350	379	350	396	350	416	350
18-19	550	581	550	618	550	660	550	710	550
17-18	750	803	750	864	750	937	750	1025	750
16-17	950	1027	950	1117	950	1226	950	1358	950
15-16	1150	1253	1150	1375	1150	1524	1150	1706	1150
14-15	1350	1480	1350	1638	1350	1829	1350	2067	1350
13-14	1550	1710	1550	1904	1550	2142	1550	2440	1550
12-13	1789	1986	1789	2226	1789	2524	1789	2900	1789
11-12	2065	2307	2065	2604	2065	2976	2065	3449	2065
10-11	2383	2679	2383	3046	2383	3509	2383	4102	2383
9-10	2751	3113	2751	3564	2751	4138	2751	4882	2751
8-9	3175	3616	3175	4171	3175	4882	3175	⋯	⋯
7-8	3665	4201	3665	4882	3665	⋯	⋯	⋯	⋯
6-7	4230	4882	4230	⋯	⋯	⋯	⋯	⋯	⋯
5-6	4882	⋯	⋯	⋯	⋯	⋯	⋯	⋯	⋯
4-5	5635	4882	5635	⋯	⋯	⋯	⋯	⋯	⋯
3-4	6504	5842	6504	4882	6504	⋯	⋯	⋯	⋯
2-3	7506	6989	7506	6204	7506	4882	7506	⋯	⋯
1-2	8664	8361	8664	7878	8664	6993	8664	4882	8664
basal end	10 000	10 000	10 000	10 000	10 000	10 000	10 000	10000	10 000

III. RESULTS

The percent correct scores, averaged across subjects, are presented in Figs. 2 and 3 for vowels and consonants, respectively, as a function of the simulated dead region size. The scores were corrected for chance, using the chance levels of 8.33% for vowels and 7.14% for consonants:

$$PC_{\text{corrected for chance}} = (PC_{raw} - \text{Chance level})/(100 - \text{Chance level}).$$

With the adjustment, a raw percent correct score at chance level equals 0% when corrected for chance. In each figure, the panels from left to right present results with the conditions of dead regions simulated in cochlear locations of basal, middle, and apical, respectively. The filled symbols show the scores with the dropped map, and the open symbols show the scores with the frequency transposition map. The error bars show one standard deviation. The dots under the scores indicate the dead region conditions where the performances with the two maps differed significantly, determined by *posthoc* Tukey multiple comparisons. Three dots show a significance level of $p<0.001$, two dots show a significance level of $p<0.01$, and one dot shows a significance level of $p<0.05$.

A two-way repeated measures ANOVA showed that the identification of vowels and consonants decreased significantly with both maps as the size of the dead region increased, however the rate of decrease was different for each map and for each simulated cochlear location (Table V). Similar to the study of Shannon *et al.*, speech recognition was poorer for dead regions simulated in middle and apical locations compared to the basal location. This finding is consistent with the speech intelligibility index (SII) which predicts more contribution from the low- and middle-frequency ranges to overall speech understanding (ANSI S3.5, 1997). The performances observed with the dropped and frequency transposition maps were significantly different, except for the consonant recognition with the conditions of dead regions simulated in middle cochlear locations.

In the dropped map, acoustic information that would fall into the dead region was discarded. The frequency transposition map provided the full input acoustic information, but the mapping of the spectral information to cochlear place was distorted. Frequency transposition was advantageous for consonant recognition with large dead regions (six and eight bands) simulated in middle and apical locations and for vowel recognition with a large dead region simulated in the apical location. However, it produced poorer vowel and consonant recognition with basal dead regions, and poorer vowel

TABLE III. Similar to Table II, except for a dead region simulated in the middle cochlear location.

	None	2 bands, middle		4 bands, middle		6 bands, middle		8 bands, middle	
	analysis and carrier bands (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)	frequency transposition (Hz)	dropped map (Hz)
apical end	150	150	150	150	150	150	150	150	150
19-20	350	367	350	388	350	412	350	440	350
18-19	550	591	550	640	550	700	550	772	550
17-18	750	819	750	903	750	1009	750	1137	750
16-17	950	1050	950	1175	950	1333	950	1529	950
15-16	1150	1285	1150	1454	1150	1671	1150	1946	1150
14-15	1350	1521	1350	1739	1350	2022	1350	2383	1350
13-14	1550	1760	1550	2030	1550	2383	1550	⋯	⋯
12-13	1789	2048	1789	2383	1789	⋯	⋯	⋯	⋯
11-12	2065	2383	2065	⋯	⋯	⋯	⋯	⋯	⋯
10-11	2383	⋯	⋯	⋯	⋯	⋯	⋯	⋯	⋯
9-10	2751	2383	2751	⋯	⋯	⋯	⋯	⋯	⋯
8-9	3175	2797	3175	2383	3175	⋯	⋯	⋯	⋯
7-8	3665	3281	3665	2855	3665	2383	3665	⋯	⋯
6-7	4230	3849	4230	3418	4230	2931	4230	2383	4230
5-6	4882	4514	4882	4091	4882	3602	4882	3036	4882
4-5	5635	5293	5635	4894	5635	4422	5635	3860	5635
3-4	6504	6206	6504	5853	6504	5426	6504	4903	6504
2-3	7506	7276	7506	6998	7506	6654	7506	6221	7506
1-2	8664	8530	8664	8366	8664	8158	8664	7889	8664
basal end	10 000	10 000	10 000	10 000	10 000	10 000	10 000	10 000	10 000

recognition with smaller size dead regions in middle and apical locations, compared to the dropped map.

IV. DISCUSSION

The dropped map of the present study was similar to the first condition presented in the Shannon *et al.* (2002) study. The effects of the dropped map on speech recognition observed in the present study were similar to results observed by Shannon *et al.*; there was a decrease in the performance as the size of the simulated dead region increased. The effects were more pronounced for the simulated middle and apical cochlear locations, with the corresponding frequency ranges of 1–2 kHz (Tables III and IV). This finding is consistent with the SII which shows that these frequencies are most important for speech recognition (ANSI S3.5, 1997). Pauler *et al.* (1986) also demonstrated a correlation between poor speech recognition by hearing-impaired subjects and the nerve damage in these cochlear regions. As a result, the decrease in the performance with the dropped map seems to be a direct consequence of missing acoustic information.

In the frequency transposition map, the overall acoustic input range was distributed over the nondead cochlear region. This condition was motivated by the idea of frequency transposition that was previously suggested for hearing aid users with steeply sloping hearing loss (Turner and Hurtig, 1999; McDermott and Dean, 2000) and for cochlear implant users with partial electrode array insertion (Başkent and Shannon, 2005). The frequency transposed condition provides more acoustic information, yet at the expense of introducing distortions in the frequency-place stimulation patterns in the cochlea. Results of the present study showed that the frequency transposition did not necessarily improve speech recognition compared to the dropped map for many simulated dead region conditions; only for six- to eight-band apical dead regions some benefit was observed. This finding cannot be explained by SII, which would predict no drop in performance as the frequency transposition map provides the entire acoustic information for all dead region conditions. The drop in the performance, therefore, is more likely to be caused by the spectral distortions in the mapping of this acoustic information.

Note that the effects observed in the present study are instantaneous effects obtained with normal-hearing subjects. It is possible that listeners might learn over time how to make use of the additional acoustic information, even if presented in a distorted map. Many studies showed a significant improvement in speech recognition by implant users over the course of a few months following the surgery (e.g., Tyler *et al.*, 1997). However, contrary to the high degree of the plasticity shown with pediatric implant users (Sharma *et al.*, 2002), adaptation by adult implant users might be limited. Kral *et al.* (2002), for example, showed a sensitive period of plasticity in the cat auditory system. Başkent and Shannon (2004) measured speech recognition by implant subjects as a

TABLE IV. Similar to Table II, except for a dead region simulated in the apical cochlear location.

	None	2 bands, apical		4 bands, apical		6 bands, apical		8 bands, apical	
	analysis and carrier bands (Hz)	frequency transposition (Hz)	dropped (Hz)	frequency transposition (Hz)	dropped (Hz)	frequency transposition (Hz)	dropped (Hz)	frequency transposition (Hz)	dropped (Hz)
apical end	150	150	150	150	150	150	150	150	150
19-20	350	384	350	443	350	576	350	1150	350
18-19	550	631	550	779	550	1150	550	⋯	⋯
17-18	750	887	750	1150	750	⋯	⋯	⋯	⋯
16-17	950	1150	950	⋯	⋯	⋯	⋯	⋯	⋯
15-16	1150	⋯	⋯	⋯	⋯	⋯	⋯	⋯	⋯
14-15	1350	1150	1350	⋯	⋯	⋯	⋯	⋯	⋯
13-14	1550	1337	1550	1150	1550	⋯	⋯	⋯	⋯
12-13	1789	1563	1789	1362	1789	1150	1789	⋯	⋯
11-12	2065	1826	2065	1612	2065	1383	2065	1150	2065
10-11	2383	2133	2383	1906	2383	1661	2383	1408	2383
9-10	2751	2491	2751	2253	2751	1993	2751	1721	2751
8-9	3175	2908	3175	2662	3175	2389	3175	2099	3175
7-8	3665	3395	3665	3143	3665	2861	3665	2557	3665
6-7	4230	3962	4230	3710	4230	3424	4230	3112	4230
5-6	4882	4624	4882	4378	4882	4096	4882	3785	4882
4-5	5635	5396	5635	5166	5635	4899	5635	4600	5635
3-4	6504	6296	6504	6094	6504	5857	6504	5588	6504
2-3	7506	7356	7506	7189	7506	7002	7506	6786	7506
1-2	8664	8571	8664	8479	8664	8368	8664	8238	8664
basal end	10 000	10 000	10 000	10 000	10 000	10 000	10 000	10 000	10 000

function of the spectral distortions in the frequency-place mapping and analyzed the results for pre- and postlingually deafened subjects. Speech recognition by the prelingually deafened subjects was the best when the frequency-place map of the experimental processor was most similar to the map used in their own implant processor. Performance by the postlingually deafened subjects, however, was best when the experimental map was closest to the normal map of the healthy auditory system. Okazawa *et al.* (1996) similarly observed that postlingually deafened implant users had similar cortical activity to normal-hearing control group, while the PET images of the prelingually deafened subjects were significantly different from both the normal-hearing and the postlingually deafened subjects. These studies suggest that the auditory system of a postlingually deafened subject might be hardwired during the sensitive developmental pe-

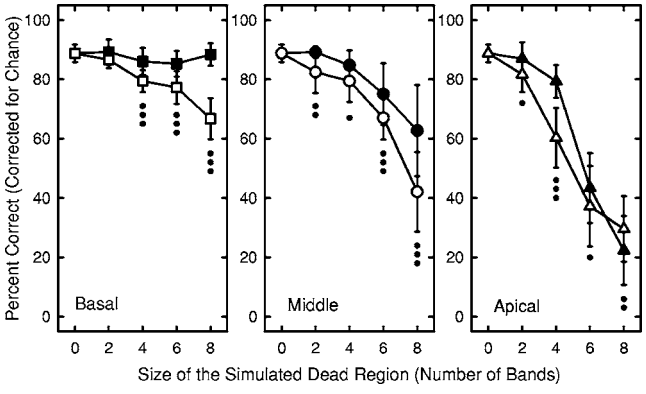

FIG. 2. Percent correct scores, averaged across subjects, for vowel recognition. The scores are presented as a function of the size of the simulated dead region, expressed by the number of carrier bands dropped. The simulated cochlear location of the dead region changes from basal to apical, as shown in the panels from left to right. The filled symbols represent the scores with the dropped map, where the spectrum in the dead region was eliminated from the analysis bands. The open symbols represent the scores with the frequency transposition map, where the entire acoustic input spectrum was distributed over the remaining carrier bands. The error bars show one standard deviation. The dots under the scores show the performances with the two maps that differed significantly, determined by a *posthoc* Tukey test; three dots for $p<0.001$, two dots for $p<0.01$, and one dot for $p<0.05$.

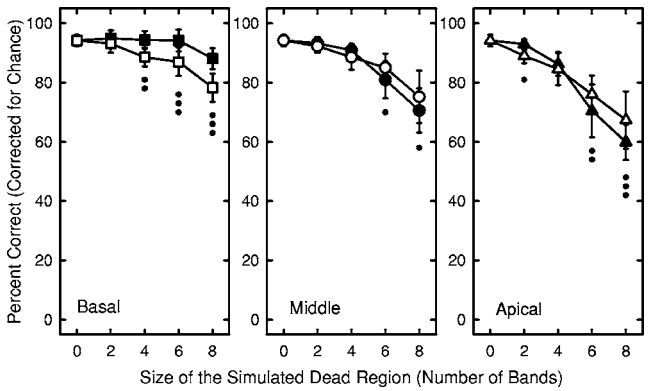

FIG. 3. Similar to Fig. 2, except the speech stimuli were consonants.

TABLE V. F and p values determined by a two-way repeated measures ANOVA. The columns show the values for the dead regions simulated in basal, middle, and apical cochlear locations. The rows show the significance of the effects of the size of the dead region and the mapping condition, and the interaction between the dead region size and the mapping condition, for vowel and consonant recognition.

		Basal	Middle	Apical
Vowel recognition	Size of the dead region	$F(4,24)=35.92$, $p<0.001$	$F(4,24)=47.74$, $p<0.001$	$F(4,24)=143.09$, $p<0.001$
	Mapping condition	$F(1,6)=89.26$, $p<0.001$	$F(1,6)=86.64$, $p<0.001$	$F(1,6)=27.04$, $p<0.01$
	Interaction between the dead region size and the mapping condition	$F(4,24)=23.63$, $p<0.001$	$F(4,24)=12.60$, $p<0.001$	$F(4,24)=13.62$, $p<0.001$
Consonant recognition	Size of the dead region	$F(4,24)=53.15$, $p<0.001$	$F(4,24)=44.51$, $p<0.001$	$F(4,24)=70.16$, $p<0.001$
	Mapping condition	$F(1,6)=34.52$, $p<0.001$	$F(1,6)=1.85$, $p=0.22$	$F(1,6)=8.04$, $p<0.05$
	Interaction between the dead region size and the mapping condition	$F(4,24)=7.22$, $p<0.001$.	$F(4,24)=6.34$, $p<0.01$	$F(4,24)=7.08$, $p<0.001$

riod with normal acoustic patterns, and the adaptation by adult implant users might be limited. Fu *et al.* (2002), for example, introduced a spectral shift in the frequency-place maps of implant users from the normal settings (by 2–4 mm expressed in distance on the cochlea). At the end of the experimental period of 3 months, where the subject had used the shifted map exclusively, speech recognition scores were still lower than the baseline performance obtained with normal settings. Svirsky *et al.* (2004) and Fu *et al.* (2005) showed that adaptation due to simple exposure to a map might be a lengthy process. Fu *et al.* (2005) also showed that a more targeted training might be necessary to facilitate adaptation. Results from the present study can be used as guidelines in fitting implant users. If a matched frequency-place map is more beneficial for the patient in the beginning of the adaptation process, changing the map from the matched map to the frequency transposed map gradually might ease the progress Svirsky *et al.* (2003). In addition, as Başkent and Shannon (2005) showed with simulations of partial electrode array insertions with implant users, there might be an optimal map between the two maps used in the present study, where the frequency transposition would be applied only moderately.

With any simulation study, one should be careful translating the results to real applications with implant users. There are many factors in electrical stimulation that would affect speech perception, such as electrode configuration, stimulus mode, channel interactions, nerve survival pattern, etc. The vocoder processing is only a rough imitation of implant signal processing and does not capture the effects of any of the factors mentioned above. In addition, there are aspects of auditory perception other than speech recognition that might be crucial for implant users, such as the quality of the perceived sounds, which was not covered in the present study.

There is an inherent trade-off in the noiseband vocoder processing; it is usually not possible to preserve the overall acoustic input range, the number of channels, and the bandwidth of individual vocoder bands all at the same time. In the present study, the bandwidth of individual carrier bands was kept the same for all conditions. Therefore, as the size of the simulated dead region increased, the number of channels used in the vocoder decreased. We also had intended to use the entire acoustic input range in the frequency transposition map. When the entire analysis band range was partitioned into a number of channels, the bandwidth of individual analysis bands was larger than the bandwidth of the analysis bands used in the dropped mapping condition. The varying number of channels or bandwidth might have affected the results in addition to the spectral distortions. Follow-up experiments can be designed to complement the present study and to understand the effects of such additional factors. In one experiment the number of channels could be the same for all conditions at the expense of varying the bandwidth of carrier bands and, in another, the bandwidth of all bands could be the same at the expense of losing some acoustic information.

V. CONCLUSION

The results of the present simulation study imply that simply providing more acoustic information to cochlear implant or hearing aid users with dead regions might not be sufficient to improve their speech recognition. If not implemented carefully, spectral distortions introduced by transposing frequencies might cause degradation in speech recognition, especially for postlingually deafened patients. However, there is a possibility that the patients can adapt to the frequency transposition map, especially if training is also provided. Normal-hearing subjects, after all, are used to the redundancy in the speech signal, and the instantaneous effects of spectral distortions might be more detrimental for these listeners compared to real patients, who would have to learn to efficiently use all speech cues available to them. Starting

the patient with a map that is more beneficial for speech recognition and then introducing the frequency transposition in small steps gradually might make such adaptation easier for the patient.

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American National Standards Institute (**1997**). "Methods for Calculation of the Speech Intelligibility Index," S35.

Başkent, D., and Shannon, R. V. (**2005**). "Interactions between cochlear implant electrode insertion depth and frequency-place mapping," J. Acoust. Soc. Am. **117**, 1405–1416.

Başkent, D., and Shannon, R. V. (**2004**). "Frequency-place compression and expansion in cochlear implant listeners," J. Acoust. Soc. Am. **116**, 3130–3140.

Bierer, J. A. (2005). "The tripolar electrode configuration and measures of channel interactions in cochlear implant subjects," 28th Midwinter Meeting, Assoc. Res. Otolaryn., New Orleans, LA.

Cochlear Corporation (**1995**). Technical Reference Manual, Englewood, CO.

Florentine, M., and Houtsma, A. J. (**1983**). "Tuning curves and pitch matches in a listener with a unilateral, low-frequency hearing loss," J. Acoust. Soc. Am. **73**, 961–965.

Fu, Q.-J., Nogaki, G., and Galvin, III, J. J. (**2005**). "Auditory training with spectrally shifted speech: Implications for cochlear implant patient auditory rehabilitation," J. Assoc. Res. Otolaryngol. **6**, 180–189.

Fu, Q.-J., Shannon, R. V., and Galvin, III, J. J. (**2002**). "Perceptual learning following changes in the frequency-to-electrode assignment with the Nucleus-22 cochlear implant," J. Acoust. Soc. Am. **112**, 1664–1674.

Fu, Q.-J, Shannon, R. V., and Wang, X. (**1998**). "Effects of noise and spectral resolution on vowel and consonant recognition: Acoustic and electric hearing," J. Acoust. Soc. Am. **104**, 3586–3596.

Glasberg, B. R., and Moore, B. C. J. (**1986**). "Auditory filter shapes in subjects with unilateral and bilateral cochlear impairments," J. Acoust. Soc. Am. **79**, 1020–1033.

Gomaa, N. A., Rubinstein, J. T., Lowder, M. W., Tyler, R. S., and Gantz, B. J. (**2003**). "Residual speech perception and cochlear implant performance in postlingually deafened adults," Ear Hear. **24**, 539–544.

Hillenbrand, J., Getty, L. A., Clark, M. J., and Wheeler, K. (**1995**). "Acoustic characteristics of American English vowels," J. Acoust. Soc. Am. **97**, 3099–3111.

Hogan, C. A., and Turner, C. W. (**1998**). "High-frequency audibility: Benefits for hearing-impaired listeners," J. Acoust. Soc. Am. **104**, 432–441.

Khan, A. M., Whiten, D. M., Nadol, Jr., J. B., and Eddington, D. K. (**2005**). "Histopathology of human cochlear implants: Correlation of psychophysical and anatomical measures," Hear. Res. **205**, 89–93.

Kral, A., Hartmann, R., Tillein, J., Heid, S., and Klinke, R. (**2002**). "Hearing after congenital deafness: Central auditory plasticity and sensory deprivation," Cereb. Cortex **12**, 797–807.

McDermott, H. J., and Dean, M. R. (**2000**). "Speech perception with steeply sloping hearing loss: Effects of frequency transposition," Br. J. Audiol. **34**, 353–361.

Moore, B. C. J., and Glasberg, B. R. (**1997**). "A model of loudness perception applied to cochlear hearing loss," Aud. Neurosci. **3**, 289–311.

Okazawa, H., Naito, Y., Yonekura, Y., Sadato, N., Hirano, S., Nishizawa, S., Magata, Y., Ishizu, K., Tamaki, N., Honjo, I., and Konishi, J. (**1996**). "Cochlear implant efficiency in pre- and postlingually deaf subjects: A study with H2(15)0 and PET," Brain **119**, 1297–1306.

Pauler, M., Schuknecht, H. F., and Thornton, A. R. (**1986**). "Correlative studies of cochlear neuronal loss with speech discrimination and pure-tone thresholds." Arch. Oto-Rhino-Laryngol. **243**, 200–206.

Pfingst, B. E., and Xu, L. (**2004**). "Across-site variation in detection thresholds and maximum comfortable loudness levels for cochlear implants," J. Assoc. Res. Otolaryngol. **5**, 11–24.

Pfingst, B. E., Xu, L., and Thompson, C. S. (**2004**). "Across-site threshold variation in cochlear implants: Relation to speech recognition," Audiol. Neuro-Otol. **9**, 341–352.

Rankovic, C. M. (**1991**). "An application of the articulation index to hearing aid fitting," J. Speech Hear. Res. **34**, 391–402.

Robert, M. E. (**1998**). CONDOR: Documentation for Identification Test Program, House Ear Institute, Los Angeles, CA

Schuknecht, H. F., and Gacek, M. R. (**1993**). "Cochlear pathology in presbycusis," Ann. Otol. Rhinol. Laryngol. **102**, 1–16.

Shannon, R. V., Galvin, III, J. J., and Başkent, D. (**2002**). "Holes in hearing," J. Assoc. Res. Otolaryngol. **3**, 185–199.

Shannon, R. V., Zeng, F.-G., Kamath, V., Wygonski, J., and Ekelid, M. (**1995**). "Speech recognition with primarily temporal cues," Science **270**, 303–304.

Sharma, A., Dorman, M. F., and Spahr, A. J. (**2002**). "Rapid development of cortical auditory evoked potentials after early cochlear implantation," NeuroReport **13**, 1365–1368.

Stelmachowicz, P. G., Jesteadt, W., Gorga, M. P., and Mott, J. (**1985**). "Speech perception ability and psychophysical tuning curves in hearing impaired listeners," J. Acoust. Soc. Am. **77**, 620–627.

Svirsky, M. A., Silveira, A., Neuberger, H., Teoh, S. W., and Suarez, H. (**2004**). "Long-term auditory adaptation to a modified peripheral frequency map," Acta Oto-Laryngol. **12**, 381–386.

Svirsky, M. A., Sinha, S., Neuberger, H. S., and Talavage, T. M. (**2003**). "Gradual adaptation to shifts in the peripheral acoustic frequency map," 26th Midwinter Meeting, Asssoc. Res. Otolaryn. Daytona Beach, FL.

Turner, C. W., and Hurtig, R. R. (**1999**). "Proportional frequency compression of speech for listeners with sensorineural hearing loss," J. Acoust. Soc. Am. **106**, 877–886.

Turner, C. W., Fabry, D. A., Barrett, S., and Horwitz, A. R. (**1992**). "Detection and recognition of stop consonants by normal-hearing and hearing-impaired listeners," J. Speech Hear. Res. **35**, 942–949.

Tyler, R. S., Parkinson, A. J., Woodworth, G. G., Lowder, M. W., and Gantz, B. J. (**1997**). "Performance over time of adult patients using the Ineraid or Nucleus cochlear implant," J. Acoust. Soc. Am. **102**, 508–522.

Vickers, D. A., Moore, B. C. J., and Baer, T. (**2001**). "Effects of low-pass filtering on the intelligibility of speech in quiet with and without dead regions at high frequencies," J. Acoust. Soc. Am. **110**, 1164–1175.