J Am Acad Audiol 13: 50–58 (2002)

Temporally Jittered Speech Produces Performance Intensity, Phonetically Balanced Rollover in Young Normal-Hearing Listeners

Terence T. Miranda*
M. Kathleen Pichora-Fuller*†

Abstract

This study investigates whether temporally jittered stimuli will produce performance-intensity, phonetically balanced (PI-PB) rollover in young adults with normal hearing. Although not yet explicitly stated in the literature, there is clinical and theoretical evidence to suggest that PI-PB rollover, such as that found in cases of acoustic neuroma, is caused by neural dyssynchrony in the auditory system. Sixteen participants were tested with intact and temporally jittered word lists in quiet at 40, 55, 65, and uncomfortable listening level −5 dB HL. The results show significant rollover in the jittered but not the intact conditions. The results are consistent with the existing evidence that suggests that neural PI-PB rollover is caused by decreased neural synchrony and support the claim that temporal jitter simulates neural dyssynchrony. Furthermore, these results are consistent with the hypothesis that synchrony coding plays an important role in the perception of high-level speech.

Key Words: Neural dyssynchrony, performance intensity, phonetically balanced rollover, temporal jitter, temporal processing, word recognition

Abbreviations: ABR = auditory brainstem response, ALSR = average localized synchronized rate, ANF = auditory nerve fiber, ANOVA = analysis of variance, FFT = fast Fourier transform, MCL = most comfortable listening level, NU-6 = Northwestern University Auditory Test No. 6, PI-PB = performance intensity, phonetically balanced, SNK = Student-Newman-Keuls, SPIN = Speech Perception in Noise test, UCL = uncomfortable listening level

Sumario

Este estudio investiga si los estímulos con variancias (*jitter*) temporales producen regresión fonémica (*rollover*) en estudios de rendimiento/intensidad con material fonéticamente balanceado (PI-PB) en adultos jóvenes con audición normal. Aunque no ha sido explícitamente aclarado en la literatura, existe evidencia clínica y teórica que sugiere que la regresión fonémica en PI-PB (PI-PB *rollover*), como la encontrada en casos de neurinoma del acústico, es debida a una dis-sincronía en el sistema auditivo. Se evaluaron dieciséis participantes utilizando listas de palabras intactas y con variancias (*jitter*) temporales, en silencio a 40, 55 y 65 dB HL, así como a un nivel de molestia en la audición (UCL) de −5 dB HL. Los resultados muestran una regresión fonémica significativa en las condiciones con variancia, pero no con el material intacto. Estos resultados son consistentes con la evidencia existente que sugiere que la regresión fonémica neural en PI-PB es debida a una disminución en la sincronía neural, y apoya la sugerencia de que la variancia temporal en la señal estimula la dis-sincronía neural. Más aún, estos resultados son consistentes con la hipótesis de que la codificación de la sincronía (*synchrony coding*) juega un papel importante en la percepción del lenguaje de alto nivel.

Palabras Clave: Dis-sincronía neural, rendimiento/intensidad, balance fonético, regresión fonémica, variancia (*jitter*) temporal, procesamiento temporal, reconocimiento de palabras

Abreviaturas: ABR = respuestas auditivas del tallo cerebral, ALSR = función de tasa sincronizada de localización promedio, ANF = fibra auditiva neural, ANOVA = análisis de variancia, FFT = transformación rápida de Fourier, MCL = nivel confortable de audición, PI-PB = rendimiento/Intensidad con balance fonémico, SNK = Student-Newman-Keuls, SPIN = prueba de Percepción del Lenguaje en Ruido, UCL = nivel molesto de audición, UN-6 = Prueba Auditiva de la Universidad Northwestern No. 6

*University of British Columbia, School of Audiology and Speech Services, †Institute for Hearing Accessibility Research, Vancouver, British Columbia

Reprint requests: Terence T. Miranda or M. Kathleen Pichora-Fuller, University of British Columbia, 5804 Fairview Avenue, Vancouver, BC V6T 1Z3

Performance-intensity, phonetically balanced (PI-PB) rollover has been accepted for many years as a clinical indicator of retrocochlear pathology (e.g., Martin, 1997; Stach, 1998). Rollover is seen when word recognition scores decrease with increases in presentation level. The mechanism by which retrocochlear pathology leads to PI-PB rollover has not yet been well explained in the literature.

There are lines of evidence, both clinical and theoretical, which point to decreased neural synchrony as the cause of PI-PB rollover in clients with retrocochlear disorder. Neural synchrony, or periodicity coding, refers to the ability of auditory nerve fibers (ANFs) to phase-lock or discharge to one phase of an incoming stimulus (Rose et al, 1971).

Clinical evidence comes from two different populations: acoustic neuroma sufferers and the elderly. Both are reported to have a higher incidence of pronounced PI-PB rollover than the general clinical population. Furthermore, for both populations, there is reason to believe that there is decreased neural synchrony. The results of a number of studies suggest a connection between the clinical measure of PI-PB rollover and neural dyssynchrony in these two populations.

Neural dyssynchrony can be characterized by a number of physiologic measures. Clinically, the auditory brainstem response (ABR) is a widely used electrophysiologic measure that relies on neural synchrony. Mechanisms of damage to the auditory nerve that result in an abnormal ABR include compression, ischemia, hemorrhage, and demyelination/degeneration; any or all of these conditions can coexist (Jacobson et al, 1994). What all of these mechanisms have in common is the disruption of the myelin sheath that coats the auditory nerve. When the myelin sheath becomes damaged, leakage of current could ensue, resulting in conduction failure or a slowing of conduction and a corresponding temporal dispersion of electrical activity, thereby causing ABR waveform components to be depressed, delayed, or abolished (Jacobson et al, 1994). Importantly, the ABR is not a test of whether afferent information passes through the auditory nerve and brain stem but rather it is a test of whether the information passes through in a synchronized manner. Although neural synchronization is a necessary condition for a normal ABR trace, it is not the only condition required for recording activity at a remote electrode (Jacobson, 1994). Nonetheless, if audiometric thresholds are taken into account and if the ABR is abnormal, there is a very high likelihood of retrocochlear disorder (Stach, 1998).

In cases of acoustic neuroma, neural transmission is disrupted, resulting in decreased neural synchrony that, in turn, yields an abnormal ABR (Selters and Brachmann, 1977). Other clinical studies provide substantial evidence associating surgically or radiographically confirmed acoustic neuroma with pronounced PI-PB rollover (e.g., Jerger and Jerger, 1971; Dirks et al, 1977; Bess et al, 1979; Meyer and Mishler, 1985). Because decreased neural synchrony is identified as the primary mechanism by which acoustic neuroma leads to abnormal ABR, and because acoustic neuroma is associated with PI-PB rollover, it follows that a cause of PI-PB rollover may be decreased neural synchrony.

Further evidence pointing to neural dyssynchrony as a cause of PI-PB rollover can be found in studies of hearing in the elderly. Although hearing loss in the elderly is commonly attributed to cochlear pathology, it can also be attributed to retrocochlear pathology. Insight into the nature of age-related retrocochlear pathology is provided by animal studies. In particular, studies of aging mice show a degeneration of ganglion cells including demyelination with loosening and unraveling of myelin sheaths (Willott, 1991). Furthermore, physiologic changes in measures such as the compound action potential (Hellstrom and Schmiedt, 1990) and ABR (Boettcher et al, 1993) in quiet-reared gerbils support the suggestion that neural dyssynchrony is a characteristic of auditory aging (Schneider, 1997a). Neural dyssynchrony has been offered as a possible explanation for a number of age-related changes in psychoacoustic and speech measures in humans, including increased frequency difference limens, reduced binaural masking-level differences, and poorer speech perception in noise (Schneider and Pichora-Fuller, 2001). Clinically, several authors have found that pronounced PI-PB rollover, though rare in the general clinical population, is more common among aged listeners (e.g., Jerger and Jerger, 1971; Gang, 1976; Dirks et al, 1977; Shirinian and Arnst, 1980). Because there is substantial evidence to suggest that neural dyssynchrony is more common in the older adult population (for reviews, see Schneider, 1997a; Miranda, 2000) and because pronounced PI-PB rollover is also more common in older adults, support is given to the proposed association between rollover and neural dyssynchrony.

Although neural dyssynchrony is a likely cause of PI-PB rollover, findings of significant amounts of PI-PB rollover have also been attributed to mechanical factors (i.e., absent acoustic reflexes). Wormald et al (1995) argued that because contraction of the stapedius muscle attenuates the lower frequencies much more than the higher frequencies, in cases where the stapedius muscle is paralyzed, there is decreased attenuation of the lower frequencies. Consequently, at higher sound levels, this would result in an upward spread of masking (i.e., masking of the higher frequencies), and because more of the key information necessary for understanding speech is in the higher frequencies (above 1 kHz), this upward spread of masking may lead to decreased speech discrimination. Clinical evidence from cases of unilateral stapedectomy and Bell's palsy (McCandless and Goering, 1974) and idiopathic facial paralysis (McCandless and Schumacher, 1979) support the mechanical basis for PI-PB rollover.

A neural-mechanical interaction has also been proposed as a basis for PI-PB rollover such that speech abnormalities attributable to a neural basis are exacerbated by failure of the normal mechanical function of the acoustic reflex (Hannley and Jerger, 1981). This proposal was based on a study of patients with confirmed acoustic tumors in which those with absent reflexes had significantly greater PI-PB rollover than those whose reflexes were elicited at one or more frequencies. However, because the stage of the acoustic tumors was not documented, it is possible that the patients with absent acoustic reflexes also had tumors that were more advanced than did the patients with present or only partially absent reflexes. Therefore, the possibility remains that the high degree of rollover in the retrocochlear patients with absent reflexes may not be strictly indicative of mechanical effects but rather neural effects caused by increased pressure on the auditory nerve, increased demyelination, and a corresponding increased disruption of neural synchrony.

Taken together, the findings in cases of acoustic neuroma and the elderly provide clinical and experimental evidence supporting an association between neural dyssynchrony and PI-PB rollover. In addition, a theoretical link between PI-PB rollover and neural dyssynchrony can be established using the average localized synchronized rate (ALSR) computational model of Young and Sachs (1979).

The ALSR model of Young and Sachs (1979) attempts to explain how speech can be coded by the auditory system at high presentation levels where neural firing rate saturates. At low stimulus levels, the population response (the average rate response of a large number of ANFs) to a complex sound (e.g., the speech sound [e]) well represents the spectrum of that sound insofar as firing rate is higher for fibers whose characteristic frequencies correspond to formant peaks. However, when the stimulus level is high, the firing rates of the neurons saturate and the formant information is lost since the firing rate is high for all neurons. The ALSR, a measure that combines the rate at which a neuron discharges (amount of firing) and its synchrony (timing of firing), enables the transmission of information regarding formant frequencies to be recovered, even at high intensities. For example, at high stimulus levels, formant energy peaks will activate a large number of neurons, corresponding to the spread of activation along the basilar membrane, but the timing, or phase-locking, of firing will be linked to the dominant formant frequencies. By using a combined measure of firing rate and temporal correlation with a predefined frequency band, the ALSR computation estimates the magnitude of neural response in given frequency channels, effectively extracting formant peaks. The ALSR is a theoretical construct; there is as yet no physiologic evidence to support a central mechanism that combines rate and synchrony information appropriate to the characteristic frequencies of the projecting fibers (Greenberg, 1996).

The ALSR is not the only explanation for how the auditory system encodes spectrally complex stimuli (e.g., vowels) at high intensities. There is evidence to suggest that the low spontaneous rate neurons (comprising 15% of ANFs) may encode the spectral envelope on the basis of rate-place information, even at the highest stimulus levels, because of an extended neuronal dynamic range (for reviews, see Greenberg, 1996; Geisler, 1998). However, the majority (85%) of ANF activity becomes saturated at high intensities, and it is not clear how the brain would be able to give priority to the low spontaneous rate neurons over the more numerous high and medium spontaneous rate neurons.

The fact remains that speech intelligibility typically remains good at high presentation levels. Whereas the physiologic basis for speech intelligibility at lower presentation levels is well understood, the physiologic explanation for speech intelligibility at high presentation levels is not yet fully understood. Taking into account the ALSR theory and clinical findings for the acoustic neuroma and elderly populations, it seems reasonable to hypothesize that neural synchrony may be more important when the speech signal is presented at higher intensity levels. Therefore, disruptions in synchrony coding may be at the root of PI-PB rollover.

Recent experiments in our laboratory have used temporally jittered stimuli in an attempt to simulate internal, neural dyssynchrony. Jittering was accomplished by introducing time-varying delay values in the speech soundfile according to a low-pass noise model. The purpose of the present study was to investigate whether temporally jittered stimuli would simulate, in young normal-hearing participants, the type of speech perception deficits observed in patients with retrocochlear disorder. We wanted to determine if external temporal jitter would produce significant PI-PB rollover in young listeners with normal hearing.

METHOD

Participants

Sixteen listeners participated in this experiment, 11 females and 5 males. All participants spoke English as their first language, were between the ages of 22 and 34 years (mean = 27.3 years, SD = 3.5 years), and had 16 to 25 years of education. None of the participants had a history of hearing problems. All participants had bilateral speech reception thresholds and pure-tone air-conduction thresholds at 0.25, 0.5, 1, 2, 4, and 8 kHz $\leq$ 20 dB HL. Otoscopic screening revealed no abnormalities, tympanograms were all type A, and ipsilateral acoustic reflexes were within normal limits (70–100 dB HL) in the test (left) ear at 0.5, 1, and 2 kHz for all participants (Stach, 1998). None of the participants were trained in the field of communication disorders. Each participant was required to give informed consent and received remuneration of $10 following completion of each experimental session.

Design

Each participant attended two sessions of approximately 1 hour each. The sessions were separated by at least 1 week to reduce the effects of practice. Uncomfortable listening level (UCL) for speech was determined for each participant based on a number of suggested procedures (Mueller and Bright, 1994; Martin, 1997; Stach, 1998). After obtaining the most comfortable listening level (MCL) in the left ear, each participant was given the following instructions:

> I want to find the level that is uncomfortably loud for you. This is a level that you would be able to tolerate for only 1 or 2 minutes. If you look at the chart posted in front of you, the uncomfortable level is somewhere between "Annoying" and "Extremely Uncomfortable." I'm going to start talking and increasing the volume. I want you to say "Stop" when the level becomes uncomfortable. Are you ready?

The experimenter then began reading a passage (see Miranda, 2000) at a moderate pace, talking at the participant's MCL and increasing the presentation level, in steps of 5 dB, every 1 to 2 seconds (at specific locations in the passage) until the participant said "Stop." This level was recorded, and the participant was instructed that the procedure would be repeated two more times. During the second estimation, the beginning level was set at MCL + 10 dB HL, and in the third estimation, the initial level was set at MCL – 10 dB HL. The average final presentation level of the three trials, rounded to the nearest 5 dB, was taken as the UCL.

PI-PB functions were created for each participant by measuring word recognition scores at 40, 55, 65, and UCL−5 dB HL in three conditions: one intact and two different jittered conditions. Participants were randomly assigned to one of four groups, with four participants per group. Two groups heard intact word lists first, one group with lists presented in ascending intensities and the other group with lists presented in descending intensities. The other two groups heard word lists in one type of jitter first, one with lists presented in ascending and the other with lists presented in descending intensities. All groups heard word lists in the second type of jitter last, with half of the participants being tested with ascending intensities and the other half being tested with descending intensities. Word recognition was tested in the intact and first jittered condition using 50-word lists (1A, 1B, 2A, 2B, 3A, 3B, 4A, and 4B) of Northwestern University Auditory Test No. 6 (NU-6; Tillman and Carhart, 1966). The second jittered condition was tested using 50-word Central Institute for the Deaf W-22 lists (Hirsh et al, 1952). To counterbalance the effects of any potential list differences, the order of list presentation was changed for each participant within a group such that for each group, each of the word lists was presented an equal number of times at each intensity level in the condition being tested.

Procedures

Digitized compact disc recordings of all stimuli were fed from a JVC XL-Z232 compact disc player into a Grason-Stadler GSI-16 audiometer and then into TDH-50P headphones (left ear only). To prevent crossover, speech noise was delivered to the right ear at 5, 20, 30, or UCL−40 dB effective masking level, depending on the speech presentation level. All equipment was calibrated to ANSI 3.6 1969 standards. The experimental sessions took place with the participant seated in a double-walled, sound-attenuating Industrial Acoustics Corporation booth.

Participants were instructed to say and write the word that they heard even if they were not sure or if the words seemed unclear. To familiarize the participant with the task and the talker's voice, three intact items selected from the W-22 corpus of words were presented to each participant at his or her MCL for speech. The experimenter recorded each spoken response as correct or incorrect during testing and noted errors. The responses recorded by the experimenter were compared with the participant's written responses, with written responses taking precedence on the few occasions when discrepancies occurred.

Stimuli

The intact NU-6 and W-22 word lists were purchased on a commercial compact disc recording (Auditec, St. Louis, MO). Each target word and the accompanying carrier phrase were extracted from the original CD using commercial software (Goldwave; Craig, 2000), redigitized at a sampling rate of 20 kHz, and saved as a *.SND file on a PC hard drive. These sound files were used to produce a jittered version of the word discrimination lists.

Since synchrony coding (phase-locking) occurs for low- but not high-frequency inputs (Pickles, 1988), only the frequency components of the speech signal below 1.2 kHz were jittered. A fast Fourier transform (FFT) was used to separate the incoming signal into its component frequencies. For the first jittered condition, the speech (NU-6 lists) was then divided into two bands, one above and the other below 1.2 kHz, and then converted back to the time domain using an inverse FFT. For the second jittered condition, the speech (W-22 lists) was divided into four bands, one above 1.2 kHz and three below 1.2 kHz (0–0.4, 0.4–0.8, and 0.8–1.2 kHz). For both jittered conditions, only the components below 1.2 kHz were jittered. The two jittered conditions differed in terms of the number of different bands below 1.2 kHz that were jittered independently.

The low-frequency band of the stimuli was jittered using in-house software (Schneider, 1997b; Jaeger, 2000). A digital soundfile recorded with a sampling rate of 20 kHz contains sequential values corresponding to the amplitude of the speech time waveform, with one sample value taken every 0.05 msec. When a soundfile is temporally jittered, the sequence of the amplitude values is altered. The amount of jitter will vary according to how the sequence is altered. Using our method, the sequence of amplitude values in the soundfile is altered by shifting them by delay values. The delay value applied to each sample is determined using a low-pass, band-limited white noise model. Such a noise has amplitude values ranging between a maximum and a minimum (e.g., +20 and −20 dB SPL), with a mean of 0 and a standard deviation. The higher the upper cutoff frequency of the noise, the more rapid are the changes in its amplitude. Bandwidth represents the upper cutoff frequency of the low-pass band-limited noise. The higher the bandwidth value, the more rapid the changes in the amplitude of the noise and, since delay values are dependent on the amplitude of the noise, the more rapid the changes in delay values. For each data point in the digitized sound file, the program selects a delay value by referring to a noise generated with an experimenter-specified standard deviaton and bandwidth, determining the amplitude value of the noise at the corresponding point in time, and then converting this amplitude into a delay value. The delay value determines the position (in time) of the sample in the original file whose amplitude value is to be substituted for the data point under consideration. The larger the standard deviation, the greater the range of the delay values that can be used in jittering the signal. Note that, when the program selects an amplitude value from the low-pass band-limited white noise, the delay value will be positive or negative depending on whether the amplitude value is positive or negative. If the value is positive, then the substituted value is taken from a point later in the file, whereas if the value is negative, then the substituted value is taken from a point earlier in the file. If the selected amplitude value is zero, then the delay value is zero and the original amplitude value is retained.

Table 1 Mean Percent Correct Scores for Intact and Jittered NU-6 and W-22 Lists at Each of Four Presentation Levels

Jitter Condition	*Presentation Level (dB HL)*	*Mean % Score*	*Standard Error*
NU-6, intact	40	98.4	0.46
NU-6, intact	55	99.0	0.32
NU-6, intact	65	98.3	0.57
NU-6, intact	(UCL − 5)	95.8	1.03
NU-6, jitter 1-band	40	74.1	1.59
NU-6, jitter 1-band	55	79.8	1.36
NU-6, jitter 1-band	65	76.3	2.29
NU-6, jitter 1-band	(UCL − 5)	66.3	2.74
W-22, jitter 3-band	40	76.5	1.20
W-22, jitter 3-band	55	80.5	1.28
W-22, jitter 3-band	65	79.0	1.68
W-22, jitter 3-band	(UCL − 5)	66.6	2.14

For both jittered conditions, the specified values for jittering were SD = 0.50 msec and bandwidth = 0.5 kHz. In the first jittered condition, all signal components below 1.2 kHz were jittered using one noise exemplar. In the second jittered condition, each of the three bands below 1.2 kHz was jittered using a unique noise exemplar; therefore, at any given sample point, the delay value applied to one band was independent of the delay value applied to the other two bands. After jittering, the jittered low-frequency band(s) and the intact high-frequency band were recombined, resampled at 44.1 kHz, saved as *.WAV files, and written back to compact disc, with a different disc for each of the three conditions. The 1.0-kHz calibration tone was saved as the first track of each compact disc and was used for calibration.

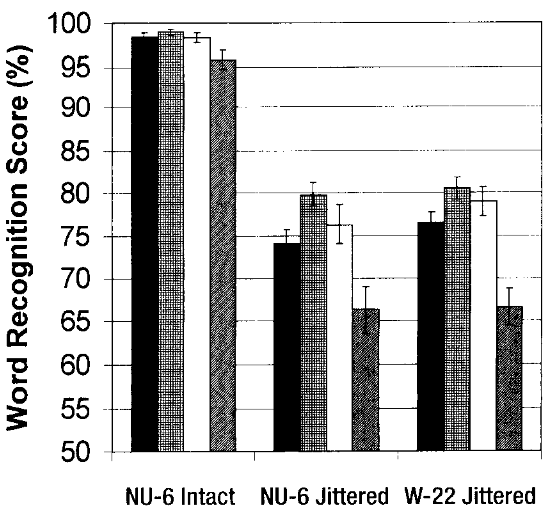

Figure 1 Mean percent correct score at each of four presentation levels (40, 55, 65, and UCL−5 dB HL) for intact NU-6 and jittered NU-6 and W-22 word lists.

RESULTS

Word Recognition Scores

Word recognition scores were measured for each of the 16 participants using intact NU-6 and jittered NU-6 and W-22 lists presented at four levels, 40, 55, 65, and UCL −5 dB HL. The average UCL measured was 90.6 dB HL (max. = 105, min. = 80; SD = 7.5 dB HL). Mean percent correct scores in each condition appear in Table 1 and Figure 1. The maximum scores are higher for the intact lists than for either of the jittered lists. Whereas the scores for the intact lists remain high as presentation level increases, scores for the jittered lists decline as presentation level increases. This description was confirmed by an analysis of variance (ANOVA) with group as a between-subjects factor and jitter condition and level as within-subjects factors. There was no significant main effect of group on percent correct scores ($F = 0.84$, $df = 3, 12$, $p = .50$), but there were significant main effects of jitter condition ($F = 237.7$, $df = 2, 24$, $p < .001$) and presentation level ($F = 30.7$, $df = 3, 36$, $p < .001$), as well as a significant interaction effect of jitter condition × presentation level ($F = 6.0$, $df = 6, 72$, $p < .001$). A Student-Newman-Keuls (SNK) test of multiple comparisons confirmed that the scores were significantly ($p < .01$) higher in the intact condition than in the jittered conditions. The SNK showed that the scores in the jittered conditions did not differ significantly from each other. Another SNK test confirmed that scores in the intact condition did not differ

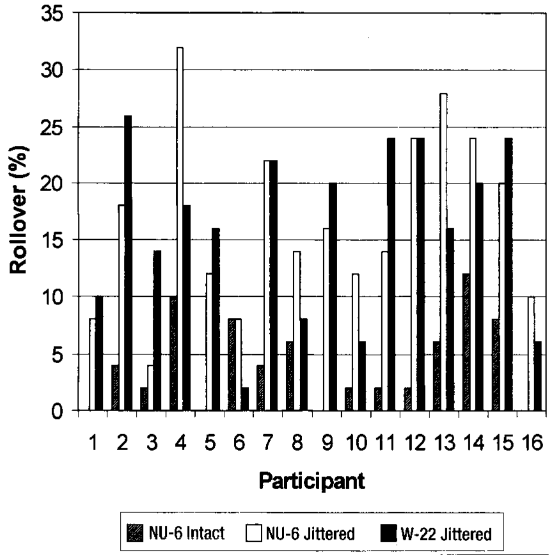

Figure 2 Rollover (%) for each of the 16 individual participants for intact NU-6 and jittered NU-6 and W-22 word lists.

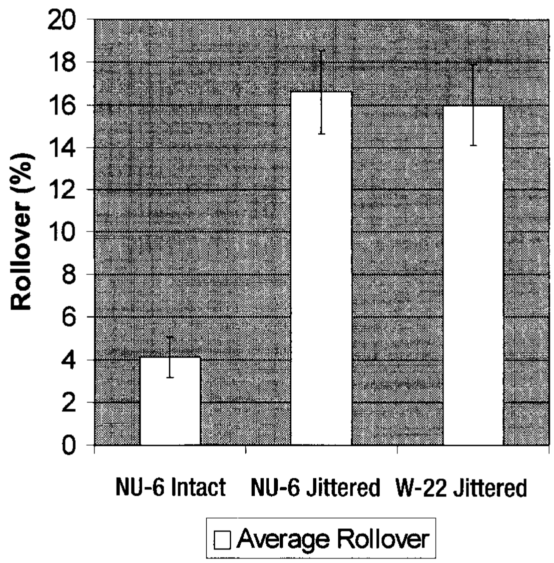

Figure 3 Mean rollover (%) for intact NU-6 and jittered NU-6 and W-22 word lists.

significantly as presentation level increased. It also showed that scores in the two jittered conditions did not differ significantly from each other at the lowest three presentation levels, where they were always lower than scores in the intact condition. The SNK also confirmed that, in the two jittered conditions, scores at presentation level 4 were significantly lower than at the lowest three presentation levels. The scores at the highest presentation level, for the jittered NU-6 and W-22 conditions, did not differ significantly from each other.

PBmax

PBmax, the maximum score of the PI-PB function, is an important component of the rollover calculation. Three PBmax scores were recorded for each of the 16 participants (one for the intact condition and each of the two jitter conditions). PBmax scores were greater in the intact condition than in either of the two jitter conditions. This description was confirmed by an ANOVA with group as a between-subjects factor and jitter condition as a within-subjects factor. There was no significant main effect of group on PBmax scores ($F = 0.95$, $df = 3, 12$, $p = .45$), but there was a significant main effect of jitter condition ($F = 117.4$, $df = 2, 24$, $p < .001$). An SNK test confirmed that PBmax in the intact condition was significantly ($p < .01$) greater than in the jitter conditions, which did not differ significantly from each other.

Rollover

For each PI-PB function (1 per condition, 3 per participant, 48 in total), a rollover calculation (rollover = PBmax − PBmin, where PBmin is the lowest percent score obtained at an intensity greater than that used to obtain PBmax) was made. Figure 2 shows the rollover scores obtained for each participant in each condition and Figure 3 shows the mean rollover scores. It is clear that the amount of rollover obtained in the jittered conditions is substantially greater than the rollover in the intact condition. This description was confirmed by an ANOVA with group as a between-subjects factor and jitter condition as a within-subjects factor. There was no significant main effect of group on rollover ($F = 0.415$, $df = 3, 12$, $p = .75$), but there was a significant main effect of jitter condition ($F = 27.9$, $df = 2, 24$, $p < .001$). An SNK test confirmed that rollover in the intact condition was significantly ($p < .001$) less than in the jitter conditions, which did not differ significantly from each other.

Correlations

PBmax and rollover scores were tested for their degree of correlation to the sex, age, years of education, left and right pure-tone averages (0.5, 1, and 2 kHz), speech reception threshold (SRT) (left ear), MCL (left ear), UCL (left ear), left ipsilateral acoustic reflex thresholds (0.5, 1, and 2 kHz), and handedness of each participant.

In general, the correlation analysis showed that rollover was sometimes, but not always, significantly ($p < .05$) positively correlated to UCL and significantly ($p < .05$) negatively correlated to PBmax. These results are not surprising since the higher the UCL, the higher was the maximum presentation level, and the lower the PBmax, the less opportunity there was to observe a decline in score. PBmax was also sometimes significantly ($p < .05$) negatively correlated with SRT. Neither rollover nor PBmax was significantly ($p < .05$) correlated to such participant variables as sex, age, years of education, pure-tone average, MCL, acoustic reflex threshold, or handedness.

DISCUSSION

In summary, word recognition scores were high and did not exhibit rollover when the young normal-hearing listeners heard intact speech. Importantly, for both jitter conditions, PBmax was not as high as in the intact condition, and significant rollover was observed when speech was presented at higher intensity levels.

Because the participants in the present study had ipsilateral acoustic reflexes within the normal range and did not exhibit rollover when intact speech was presented, natural mechanical and neural bases for the rollover are ruled out. Thus, the rollover that was observed must be attributed to the simulation of neural dyssynchrony that involved externally jittering the signal. Importantly, the simulated dyssynchrony disproportionately disrupted word recognition at high presentation levels, thereby ruling out the possibility that the jittering simply degraded the signal in a level-independent fashion. In contrast, other kinds of signal degradation, such as low-pass filtering, would be expected to yield better scores at high presentation levels than at lower levels. Consistent with the clinical and theoretical considerations presented in the introduction, the results of the present study support the hypothesis that PI-PB rollover can be caused by disruptions of synchrony coding.

The lack of difference between the two jitter conditions suggests that various types of dyssynchrony could produce PI-PB rollover, and further research will be required to determine how to characterize the exact nature of the dyssynchrony found in particular pathologies or individual cases.

Acknowledgment. This research was funded by the National Science and Engineering Research Council of Canada and the Canadian Institutes for Health Research. We wish to thank Carol Jaeger for developing the software to jitter the stimuli and Trudy Adam for conducting statistical analyses. We also wish to thank Bruce Schneider and Dennis Phillips for their advice. This article is based on the master's thesis of the first author.

A student poster submission of this material was displayed at the American Academy of Audiology's 2001 annual convention held in San Diego, CA, April 18–21, 2001.

REFERENCES

American National Standards Institute. (1969). *Specifications for Audiometers.* (ANSI S3.6 1969). New York: ANSI.

Bess FH, Josey AF, Humes LE. (1979). Performance intensity functions in cochlear and eighth nerve disorders. *Am J Otolaryngol* 1(1):27–31.

Boettcher FA, Mills JH, Norton BL. (1993). Age-related changes in auditory evoked potentials of gerbils: I. Response amplitudes. *Hear Res* 71:137–145.

Craig S. (2000). *Goldwave Version 4.12* [computer program]. St. John's, NF: Goldwave.

Dirks DD, Kamm C, Bower D, Betsworth A. (1977). Use of performance-intensity functions for diagnosis. *J Speech Hear Disord* 42:408–415.

Gang RP. (1976). The effects of age on the diagnostic utility of the rollover phenomenon. *J Speech Hear Disord* 31:63–69.

Geisler CD. (1998). *From Sound to Synapse.* New York: Oxford University Press.

Greenberg S. (1996). Auditory processing of speech. In: Lass NJ, ed. *Principles of Experimental Phonetics.* St. Louis: Mosby, 362–407.

Hannley M, Jerger J. (1981). PB rollover and the acoustic reflex. *Audiology* 20:251–258.

Hellstrom LI, Schmiedt RA. (1990). Compound action potential input/output functions in young and quiet-aged gerbils. *Hear Res* 50:163–174.

Hirsh I, Silverman S, Reynolds E, et al. (1952). Development of materials for speech audiometry. *J Speech Hear Disord* 17:321–327.

Jacobson JT. (1994). Prelude to auditory evoked potentials. In: Jacobson JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Needham Heights, MA: Allyn and Bacon, 3–19.

Jacobson GP, Jacobson JT, Ramadan N, Hyde M. (1994). Auditory brainstem response measures in acoustic nerve and brainstem disease. In: Jacobson JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Needham Heights, MA: Allyn and Bacon, 387–426.

Jaeger CP. (2000). *Jitter14.exe* [computer program]. Vancouver: CJA Consulting.

Jerger J, Jerger S. (1971). Diagnostic significance of PB word functions. *Arch Otolaryngol* 93:573–580.

Martin FN. (1997). *Introduction to Audiology*, 6th Ed. Boston: Allyn and Bacon.

McCandless GA, Goering DM. (1974). Changes in loudness after stapedectomy. *Arch Otolaryngol* 100:344–350.

McCandless GA, Schumacher MH. (1979). Auditory dysfunction with facial paralysis. *Arch Otolaryngol* 105:271–274.

Meyer DH, Mishler ET. (1985). Rollover measurements with Auditec NU-6 word lists. *J Speech Hear Disord* 50:351–356.

Miranda TT. (2000). *Temporal Jitter Produces PI-PB Rollover.* Master's thesis, Vancouver, The University of British Columbia.

Mueller HG, Bright KE. (1994). Selection and verification of maximum output. In: Valentine M, ed. *Strategies for Selecting and Verifying Hearing Aid Fittings*. New York: Thieme Medical, 38–63.

Pickles J. (1988). *An Introduction to the Physiology of Hearing,* 2nd Ed. San Diego: Academic.

Rose JE, Hind JE, Anderson DJ, Brugge JF. (1971). Some effects of stimulus intensity on response of auditory nerve fibers in the squirrel monkey. *J Neurophysiol* 34:685–699.

Schneider BA. (1997a). Psychoacoustics and aging: implications for everyday listening. *J Speech Lang Pathol Audiol* 21:111–124.

Schneider BA. (1997b). *Jitspin2.exe* [computer program]. Toronto: University of Toronto.

Schneider BA, Pichora-Fuller MK. (2001). Implications of perceptual deterioration for cognitive aging research. In: Craik FIM, Salthouse TA, eds. *Handbook of Aging and Cognition*, 2nd Ed. New York: Lawrence Erlbaum, 155–219.

Selters WA, Brackmann DE. (1977). Acoustic tumor detection with brain stem electric response audiometry. *Arch Otolaryngol* 103:181–187.

Shirinian MJ, Arnst DJ. (1980). PI-PB rollover in a group of aged listeners. *Ear Hear* 1:50–53.

Stach BA. (1998). *Clinical Audiology: An Introduction*. San Diego: Singular.

Tillman TW, Carhart R. (1966). *An Expanded Test for Speech Discrimination Utilizing CNC Monosyllabic Words: Northwestern University Auditory Test No. 6. Technical Report No. SAM-TR-66-55.* Brooks Air Force Base, TX: USAF School of Aerospace Medicine.

Willott JF. (1991). *Aging and the Auditory System: Anatomy, Physiology and Psychophysics.* San Diego: Singular.

Wormald PJ, Rogers C, Gatehouse S. (1995). Speech discrimination in patients with Bell's palsy and a paralysed stapedius muscle. *Clin Otolaryngol* 20:59–62.

Young ED, Sachs MB. (1979). Representation of steady-state vowels in the temporal aspects of the discharge patterns of auditory-nerve fibers. *J Acoust Soc Am* 66:1381–1403.