J Am Acad Audiol 30:792–801 (2019)

Effect of Intensity Level and Speech Stimulus Type on the Vestibulo-Ocular Reflex

DOI: 10.3766/jaaa.18016

Mary Easterday*
Patrick N. Plyler*
James D. Lewis*
Steven M. Doettl*

Abstract

Background: Accurate vestibulo-ocular reflex (VOR) measurement requires control of extravestibular suppressive factors such as visual fixation. Although visual fixation is the dominant suppressor and has been extensively studied, the mechanisms underlying suppression from nonvisual factors of attention and auditory stimulation are less clear. It has been postulated that the nonvisual suppression of the VOR is the result of one of two mechanisms: (1) activation of auditory reception areas excites efferent pathways to the vestibular nuclei, thus inhibiting the VOR or (2) cortical modulation of the VOR results from directed attention, which implies a nonmodality-specific process.

Purpose: The purpose of this research was to determine if the VOR is affected by the intensity level and/or type of speech stimulus.

Research Design: A repeated measures design was used. The experiment was single-blinded.

Study Sample: Participants included 17 adults (14 females, three males) between the ages of 18–34 years who reported normal oculomotor, vestibular, neurological, and musculoskeletal function.

Data Collection and Analysis: Each participant underwent slow harmonic acceleration testing in a rotational chair. VOR gain was assessed at 0.02, 0.08, and 0.32 Hz in quiet (baseline). VOR gain was also assessed at each frequency while a forward running speech stimulus (attentional) or a backward running speech stimulus (nonattentional) was presented binaurally via insert earphones at 42, 62, and 82 dBA. The order of the conditions was randomized across participants. VOR difference gain was calculated as VOR gain in the auditory condition minus baseline VOR gain. To evaluate auditory efferent function, the medial olivocochlear reflex (MOCR) was assayed using transient-evoked otoacoustic emissions (right ear) measured in the presence and absence of broadband noise (left ear). Contralateral acoustic reflex thresholds were also assessed using a broadband noise elicitor. A three-way repeated measures analysis of variance was conducted to evaluate the effect of frequency, intensity level, and speech type on VOR difference gain. Correlations were conducted to determine if difference gain was related to the strength of the MOCR and/or to the acoustic reflex threshold.

Results: The analysis of variance indicated that VOR difference gain was not significantly affected by the intensity level or the type of speech stimulus. Correlations indicated VOR difference gain was not significantly related to the strength of the MOCR or the acoustic reflex threshold.

Conclusions: The results were in contrast to previous research examining the effect of auditory stimulation on VOR gain as auditory stimulation did not produce VOR suppression or enhancement for most of the participants. Methodological differences between the studies may explain the discrepant results. The removal of an acoustic target from space to attend to may have prevented suppression or enhancement of the VOR. Findings support the hypothesis that VOR gain may be affected by cortical modulation through directed attention rather than due to activation of efferent pathways to the vestibular nuclei.

*Department of Audiology and Speech Pathology, University of Tennessee Health Science Center, Knoxville, TN

Corresponding author: Mary Easterday, Department of Audiology and Speech Pathology, University of Tennessee Health Science Center, Knoxville, TN 37996-0740; Email: mlyttle@uthsc.edu

This research was supported by a student investigator award from the American Academy of Audiology Foundation.

Poster presentation at the 29th Annual American Academy of Audiology National Convention (Indianapolis, IN, April 7, 2017).

Key Words: assessment, MOCR, medial olivocochlear reflex, tasking, TEOAE, transient evoked otoacoustic emissions, vestibular, vestibulo-ocular reflex, VNG, VOR

Abbreviations: CD = critical difference; MOCR = medial olivocochlear reflex; RCT = rotary chair testing; RMS = root mean square; SHS = sinusoidal harmonic acceleration; SPV = slow-phase velocity; TEOAEs = transient-evoked otoacoustic emissions; VOR = vestibulo-ocular reflex

INTRODUCTION

The vestibular system is responsible for translating afferent biological signals associated with head rotation and gravitational forces into efferent biological signals for motor control of the musculoskeletal and ocular systems. The resulting motor responses are responsible for maintaining postural and ocular stability (i.e., balance and spatial orientation; Baloh et al, 2011). Thus, normal balance function and maintenance depend on the vestibular sensory periphery sending the proper afferent signals to the central vestibular system and the central vestibular system sending the proper efferent motor signals to the ocular muscles and/or spinal cord. There are three motor reflexes directly related to the vestibular peripheral input and central regulation: the vestibulo-ocular reflex (VOR), vestibulo-spinal reflex, and vestibulo-colic reflex. The VOR specifically is useful for evaluation of vestibular function because of more direct innervation of the peripheral vestibular projections into the oculomotor pathway (Bronstein et al, 2015).

The VOR is responsible for stabilizing stationary images on the fovea of the retina during active high intensity (<2 Hz or 80°/sec) head rotation. In other words, the VOR is necessary for clear and stable foveal vision during motion (Goebel et al, 2000). The stabilization of the image is achieved through the head rotation activating the vestibular end organs. The end organs then send the afferent signals to the vestibular nuclei, which routes the information to the ocular motor pathways to generate the efferent motor reflex. The VOR functions through a three-neuron arc system with sensory, inter, and motor neurons. The pathway runs via cranial nerves VIII (vestibulocochlear), III (oculomotor), and VI (abducens) (Purves et al, 2001; Bronstein et al, 2015). The vestibular nuclei operate as a pair between ears. The side that is being stimulated/excited extends the excitatory signals to the contralateral oculomotor nuclei while also sending inhibitory signals ipsilaterally, thus eliciting the motor reflex of the eye. Ideally, the reflex results in eye movement that is opposite in direction and equal in magnitude relative to the head movement, which fixates the eye on a visual target and maintains foveal vision (Goumans et al, 2010).

When the vestibular system is stimulated with rotational head or body movement as with rotary chair testing (RCT), the VOR presents as a nystagmic eye movement as it aims to maintain eye position relative to the placement of the head. Each nystagmic beat consists of a slow compensatory phase as eyes drift away from center as the head moves followed by a fast corrective phase as eyes quickly return to center. The relationship between the slow phase velocity of the nystagmus and the speed of the head/body movement is known as the VOR gain (VOR gain = slow phase velocity/velocity of rotation). A gain of 1 or 100% corresponds to nystagmus that is equal in magnitude but opposite in direction of the rotational head movement.

Although the VOR is responsive to vestibular input, it can be overridden (suppressed) by visual input, as at times the VOR is an inappropriate response to maintain proper vision. For example, in the case of visually tracking a moving target, such as tracking a ball that has been thrown, the VOR is suppressed by the visual input as it would be counterintuitive to initiate a movement in the opposite direction of the movement of the head when the goal is to keep the eyes moving with the head to track the flight of the ball (Halmagyi and Gresty, 1979). However, clinical vestibular evaluations require measurement of the VOR free of any amount of suppression to ensure accurate results. Therefore, visual suppression is prevented during vestibular assessment by conducting testing with the eyes closed or in total darkness. With visual suppression controlled, the VOR is induced through head/body movement and measured quantitatively. Whereas visual input suppresses the VOR, the effect of nonvisual input is less clear (Barr et al, 1976; Moller et al, 1990; Jacobson et al, 2012).

Barr et al (1976) assessed VOR gain using RCT in the following nonvisual conditions on adults: imaginary stationary target, mental arithmetic, and imaginary moving target. Results indicated the imaginary stationary target had the highest VOR gain (0.94–0.96), followed by mental arithmetic (0.65–0.97), and then the imaginary moving target (0.32–0.64). These findings indicated significant VOR gain reduction for mental arithmetic and imaginary moving targets, but minimal VOR gain change for the imaginary stationary target. The authors noted high interparticipant and intraparticipant variability, with some individuals going from significant suppression to full enhancement simply by changing their imagined frame of reference from a target that moved or rotated with the chair to a target that was fixed or stationary in space.

Moller et al (1990) assessed VOR gain using RCT in similar conditions as Barr et al (1976) but added acoustic and proprioceptive conditions. The test conditions were as follows: mental alerting in darkness, stationary

visual target, moving visual target, imaginary stationary target in darkness, imaginary moving target in darkness, proprioceptive moving targets, acoustic stationary speaker target, and acoustic moving speaker target. For each set, the mental alerting in the dark served as the baseline condition. Results revealed a significant increase in VOR gain (enhancement) for the stationary speaker targets; however, the moving speaker targets resulted in a significant reduction in VOR gain (suppression) when compared with the baseline condition. The use of the baseline VOR gain as a reference point demonstrated that VOR gain can be increased (enhanced) or reduced (suppressed) depending on the nonvisual stimulus type. Moreover, the acoustic stationary speaker target increased the VOR gain to approximately 1.0, indicating stationary acoustic stimuli could cause complete enhancement of the VOR.

More recently, Jacobson et al (2012) assessed the effects of visual and nonvisual stimuli on VOR gain and RCT. VOR gain was assessed in the following conditions: darkness with alerting, darkness without alerting, visual, auditory moving speaker target, somatosensory, imaginary moving target, and auditory + somatosensory moving speaker target. With darkness with alerting serving as baseline, results revealed that all conditions significantly reduced (suppressed) VOR gain. Results also revealed an 86% reduction in VOR gain when attending to a visual target and a 28% reduction in VOR gain when attending to an auditory speaker target. These results were in agreement with Moller et al (1990) who also indicated that VOR gain was reduced for moving auditory speaker targets.

Although prior research suggests auditory stimulation can influence the VOR, the mechanism responsible for such an auditory–vestibular interaction remains unclear. Jacobson et al (2012) postulated two possible explanations for the presence of VOR suppression stemming from nonvisual input. The first is activation of the auditory efferents in response to acoustic stimulation also activates vestibular efferents because of the close proximity of these pathways, thereby suppressing the VOR. Thus, one hypothesis holds that increasing auditory stimulation should increase excitation of efferent pathways to the vestibular nuclei and should further reduce VOR gain (increase suppression). In addition, VOR suppression may be related to measures of auditory efferent activity such as the medial olivocochlear bundle reflex (MOC) and/or the acoustic reflex. The second explanation is that cortical modulation of the VOR is a product of directed attention. Thus, a second hypothesis holds that the use of auditory stimuli that has an attentional component should reduce VOR gain (increase suppression). To gain insight into the underlying mechanisms of nonvisual VOR suppression, the present study addressed the following questions.

- Does increasing the intensity of a speech stimulus reduce VOR gain during RCT and are changes in VOR gain related to other measures of auditory efferent activity?
- Does the use of a speech stimulus with an attentional component reduce VOR gain during RCT?

METHODS

Participants

A power analysis using Jacobson et al (2012) values indicated 16 participants to be sufficient for this study. This investigation recruited 17 healthy participants (14 female and three male; ages 18–34 years) with no history of otologic or neurologic dysfunction or disorders. Each participant denied the use of medications or illicit drugs known to affect nystagmus. Participants were required to have clear ear canals, normal hearing, and normal middle ear immittance before testing. Otoscopy was initially performed to ensure ear canals were clear. The Interacoustics Titan was used for immittance testing to ensure normal middle ear function. Normative immittance values were set as follows: 0.9–2.0 cm^3 for ear canal volume, +100 to −200 daPa for middle ear pressure, and 0.2–2.0 mL for tympanic membrane peak admittance. Hearing was screened at 20 dB HL at 500, 1000, 2000, and 4000 Hz in a sound-treated booth using a GSI 61 audiometer with insert earphones (Grason-Stadler, Eden Prairie, MN). This study was approved by the Institutional Review Board at the University of Tennessee, and all participants signed an informed consent before participation in this study. This study was funded by the American Academy of Audiology Student Research Vestibular Grant and the participants received payment for their participation. Testing was performed at the University of Tennessee Audiology Clinic.

Speech Stimuli

The stimuli consisted of a speech sample from the Harvard IEEE sentence list (Rothauser et al, 1969) spoken by a female voice. Examples of sentences include: "Tea served from the brown jug is tasty" or "A dash of pepper spoils beef stew." The speech samples were digitized and time-reversed end to end (Hawley et al, 2004) as time-reversed speech has been shown to control for nonlanguage attentional functions (Brown et al, 2012). The time-reversed speech tokens were unintelligible although they shared the same temporal–spectral structure of the forward speech. The forward and backward samples contained 30 sentences spoken in succession and were 72 sec in duration. The speech samples repeated as needed until testing was completed.

Speech stimuli were loaded onto an xDuoo X3 HI-FI music player (Xduoo Technology, Bao'an District, Shen Zhen, China). The media device was attached to the rear of the rotary chair and speech stimuli were presented via ER-1 insert headphones at the following intensity levels: silent (baseline), 42, 62, and 82 dBA. Insert earphones were used to ensure that the intensity level of the speech stimuli was accurate during rotations. Intensity levels were chosen by replicating 62 dBA from Jacobson et al (2012) and expanding it ±20 dBA to represent soft and loud speech. Each speech sample was calibrated before experimental testing using a 2cc coupler.

Procedures

RCT

Participants were seated in a rotational chair (Interacoustics NyDiag 200; Interacoustics; Eden Prairie, MN) and wore video-oculography goggles in a darkened room. The chair contained a headrest pillow and the participants were instructed to keep their head still and against the pillow during the test session. The participants were monitored for any head movement during the procedures. Each participant underwent RCT for three sinusoidal harmonic acceleration (SHA) frequencies (0.02, 0.08, and 0.32 Hz) in silent to establish baseline. For each SHA frequency, the participants also underwent RCT for three intensity levels (42, 62, and 82 dBA) and two speech types (forward and backward) for a total of 21 trials. The SHA frequencies were chosen so that findings could be directly compared with previous VOR gain research (Jacobson et al, 2012). Moreover, although each participant completed seven conditions at each SHA frequency, each condition was not replicated as Jacobson et al (2012) reported that VOR gain at these SHA frequencies was not significantly affected by replication. For the silent condition, insert earphones remained in the ears, but the media device was turned off. For the auditory conditions, onset and cessation of the auditory stimulus coincided with the onset and cessation of the chair rotation. VOR gain was measured for each of the 21 trials for each participant. Each participant was instructed to keep eyes open during all testing. During the auditory conditions, the participants were informed they would hear a stimulus through the earphones. They were not instructed to directly attend to the speech stimulus. All conditions were randomized to control for possible order effects.

Efferent Assessment

Measures of the efferent auditory system included the medial olivocochlear reflex (MOCR), assayed using transient-evoked otoacoustic emissions (TEOAEs) and the acoustic reflex threshold. These measures were chosen as they assay efferent pathways of the auditory system (Warren and Liberman, 1989; Hill et al, 1997; Guinan, 2006).

A custom laboratory system was used to measure the MOCR. Stimulus presentation and data collection were controlled via MATLAB through the ARLas software (provided by Dr. Shawn S. Goodman at the University of Iowa, Iowa City, IA). Band-limited clicks were presented to the right ear to evoke otoacoustic emissionss. Concurrent with measurement of TEOAEs, broadband noise was alternately turned-on and turned-off in the left ear every 15 sec. The underlying assumption is that the MOC efferents are activated only during the noise-on condition. Interleaving the noise and quiet conditions provides a means to control for slow drifts in the middle ear impedance (Goodman et al, 2013). TEOAEs were measured for a total of 7 min (3.5 min in the quiet condition and 3.5 min in the noise condition).

The clicks and noise had a flat magnitude spectrum through 12 kHz. Clicks were presented using a linear paradigm at a level of 64 dB peak SPL (as generated in an IEC711 coupler) and a rate of 12.6 clicks/sec (interstimulus interval of 79.5 msec). The level of the contralateral noise was 50 dB SPL (root mean square [RMS]; as generated in an IEC711 coupler).

Ear canal sound pressure recordings were high-pass filtered (low frequency cutoff = 500 Hz; order = 512). An artifact rejection algorithm (based on both the RMS level and crest factor of the ear canal recording) identified and removed recordings contaminated by high-level noise. For each condition (noise and quiet), retained recordings were divided into odd- and even-numbered recordings. Each set of recordings was then synchronously averaged. The signal (which may contain emission energy) was estimated by adding the averaged odd- and even-numbered recordings. Physiologic and equipment noise was estimated by subtracting the averaged odd- and even-numbered recordings.

The signal measured during the quiet condition (corresponding to a time window extending from 3.5–20 msec relative to the peak amplitude of the evoked click) was analyzed for the presence/absence of the TEOAE energy in nine frequency bands (1/3 octave wide; center frequencies from 0.75 to 4.76 kHz). Within each frequency band, the RMS pressure levels of the signal and noise were calculated and compared. A TEOAE was classified as present if the signal-to-noise ratio within the frequency band was at least +6 dB.

MOCR strength was quantified as the percent difference between TEOAEs measured in the noise and quiet conditions. First, the RMS value of the complex difference between frequency domain representations of

Table 1. Mean VOR Gain Values for Each SHA Hz, Intensity Level, and Speech Type.

Intensity Level	Speech Type	0.02 Hz	0.08 Hz	0.32 Hz
Silent	Baseline (none)	42 (28)	50 (23)	43 (20)
42 dBA	Forward	39 (14)	48 (22)	48 (25)
	Backward	41 (15)	47 (15)	47 (22)
62 dBA	Forward	39 (15)	48 (22)	45 (28)
	Backward	40 (14)	52 (17)	48 (28)
82 dBA	Forward	38 (15)	48 (19)	50 (26)
	Backward	38 (16)	49 (23)	47 (21)

Note: Standard deviation shown in parentheses.

the TEOAE measured in quiet was calculated in each frequency band. As per this method, the difference metric describes the total MOCR-induced change to the emission, as it captures changes in both magnitude and phase. For each participant, bootstrapping (Goodman et al 2013) was used to determine whether the difference was statistically significant ($p \leq 0.01$). Significant values were then normalized by the RMS level of the TEOAE measured in quiet, and multiplied by 100 to yield the percent difference between the TEOAE measured in noise and quiet (i.e., MOCR%; Backus and Guinan, 2007; Marshall et al, 2014; Mishra and Dinger, 2016). The final estimate of the MOCR strength was calculated as the average MOCR% across frequency bands.

The Interacoustics Titan, calibrated to manufacturer recommendations, was used to measure the left contralateral, broadband noise acoustic reflex threshold. Threshold was defined as the level at which the reflex (a) was replicated across repeated measures, (b) was ≥0.02 mmhos, and (c) increased in magnitude when the activator was increased by 5 dB.

RESULTS

VOR gain values were averaged across participants for each SHA frequency for the baseline and auditory conditions (Table 1). Each baseline measurement was conducted in silence. The effect of auditory stimulation on VOR gain was calculated as the difference in VOR gain between the auditory and baseline conditions at each SHA frequency (VOR difference gain = auditory VOR gain − baseline VOR gain). A negative number indicated VOR gain was reduced or suppressed,

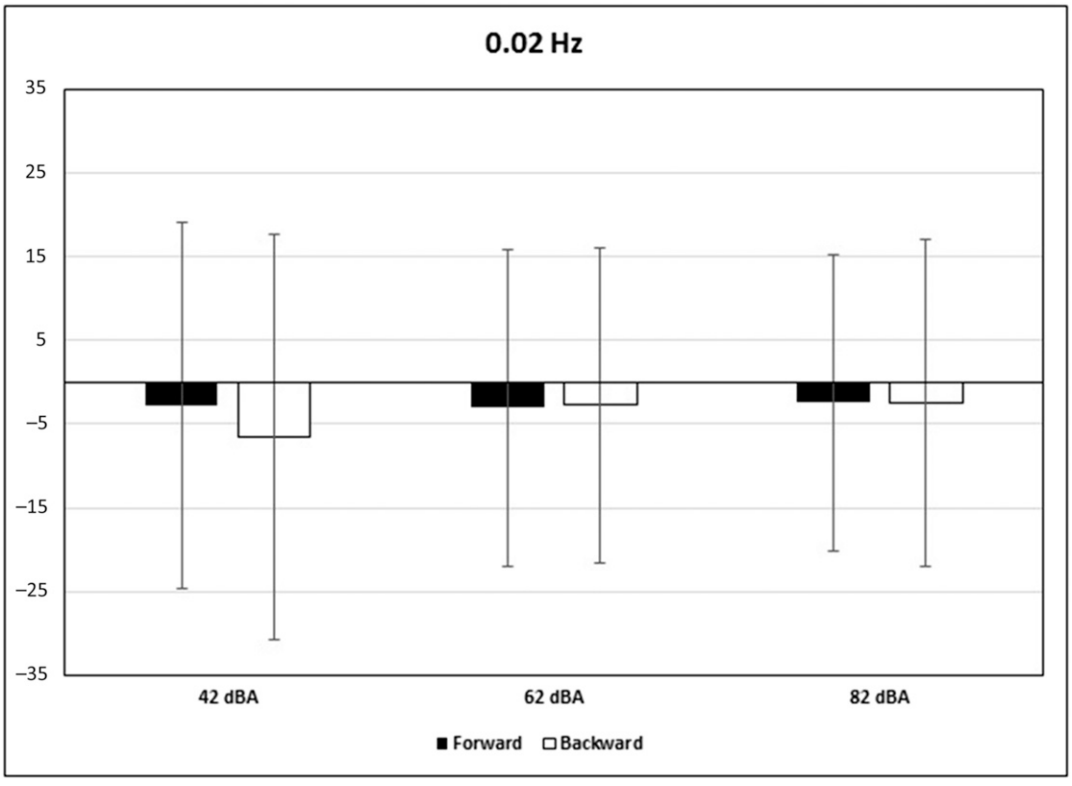

Figure 1. Mean VOR difference gain values for 0.02 Hz for each intensity level and speech type. Standard deviation bars are shown.

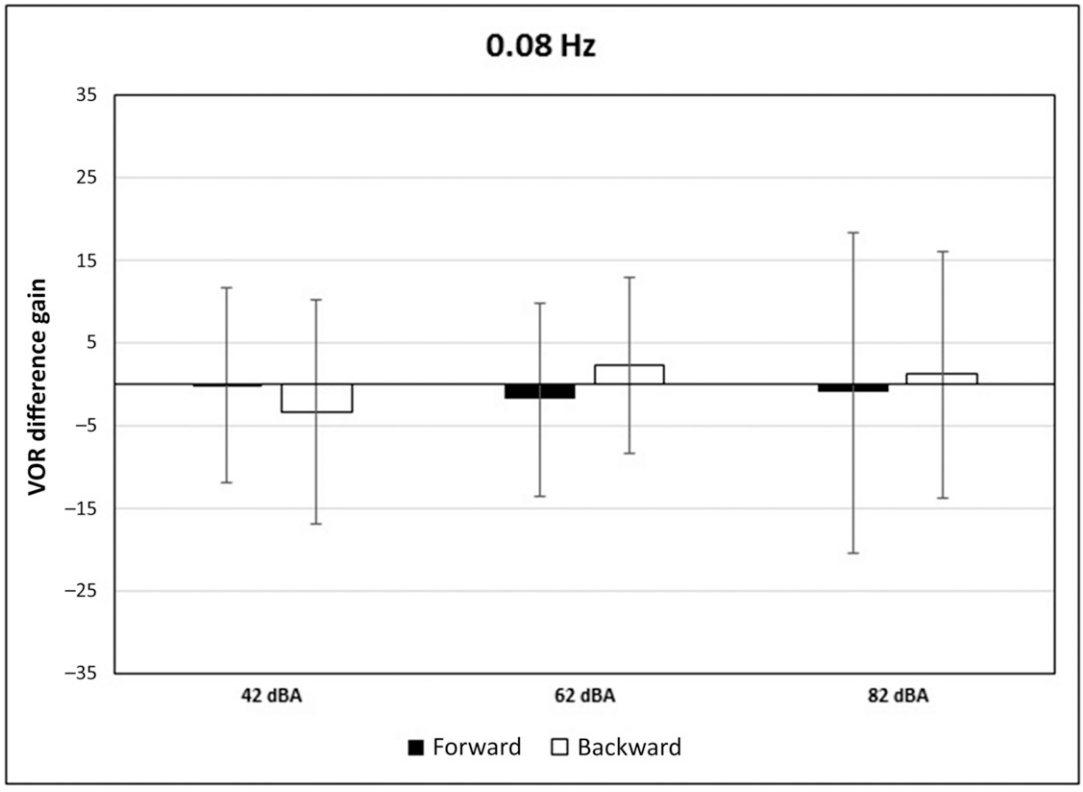

Figure 2. Mean VOR difference gain values for 0.08 Hz for each intensity level and speech type. Standard deviation bars are shown.

whereas a positive number indicated VOR gain was increased or enhanced with auditory stimulation. VOR difference gain values were averaged across participants for each SHA frequency, intensity level, and speech type (Figures 1–3).

A three-way repeated measures analysis of variance was conducted to evaluate the effect of SHA frequency, intensity level, and speech type on VOR difference gain. Independent variables were SHA frequency (0.02, 0.08, and 0.32 Hz), intensity level (42, 62, and 82 dBA), and speech type (forward and backward). The VOR difference gain served as the dependent variable. SHA frequency was included in the analysis to determine if any possible observed effects were related to frequency of rotary chair stimulation. The analysis of variance did not reveal a significant effect for SHA frequency, intensity level, or speech type (Table 2). VOR difference gain values were then averaged across SHA frequency, intensity level, and speech type for each participant.

Correlations were conducted to determine if VOR difference gain was related to baseline VOR gain, the strength of the MOCR, or to the acoustic reflex threshold. Results were not significant for the VOR baseline gain $r_{(16)} = -0.25$, $p > 0.05$; MOCR, $r_{(16)} = 0.05$, $p > 0.05$; or acoustic reflex threshold, $r_{(16)} = 0.03$, $p > 0.05$. These results indicated the VOR difference gain was not affected by the intensity level of the stimulus or by the type of speech stimulus used. Results further indicated VOR difference gain was not related to the VOR baseline gain, the strength of the MOC reflex, or the acoustic reflex threshold.

DISCUSSION

Previous research indicated that auditory stimulation can enhance or suppress the VOR during RCT (Moller et al, 1990; Jacobson et al, 2012); however, the mechanism responsible for such an auditory–vestibular interaction remained unclear. Jacobson et al (2012) postulated that VOR suppression may be due to activation of efferent pathways to the vestibular nuclei or to cortical modulation through directed attention. The purpose of this study was to test these hypotheses to determine if the VOR was affected by the intensity level and/or the type of speech stimulus. If VOR gain was related to efferent pathway activation, increasing the intensity level of the auditory stimuli should reduce VOR gain. If VOR gain was related to directed attention, the use of an auditory stimulus that contains an attentional component such as speech should reduce the VOR gain. Results indicated the VOR was not affected by the intensity level of the stimulus or by the type of speech stimulus used. Results further indicated that VOR was not related to the baseline gain,

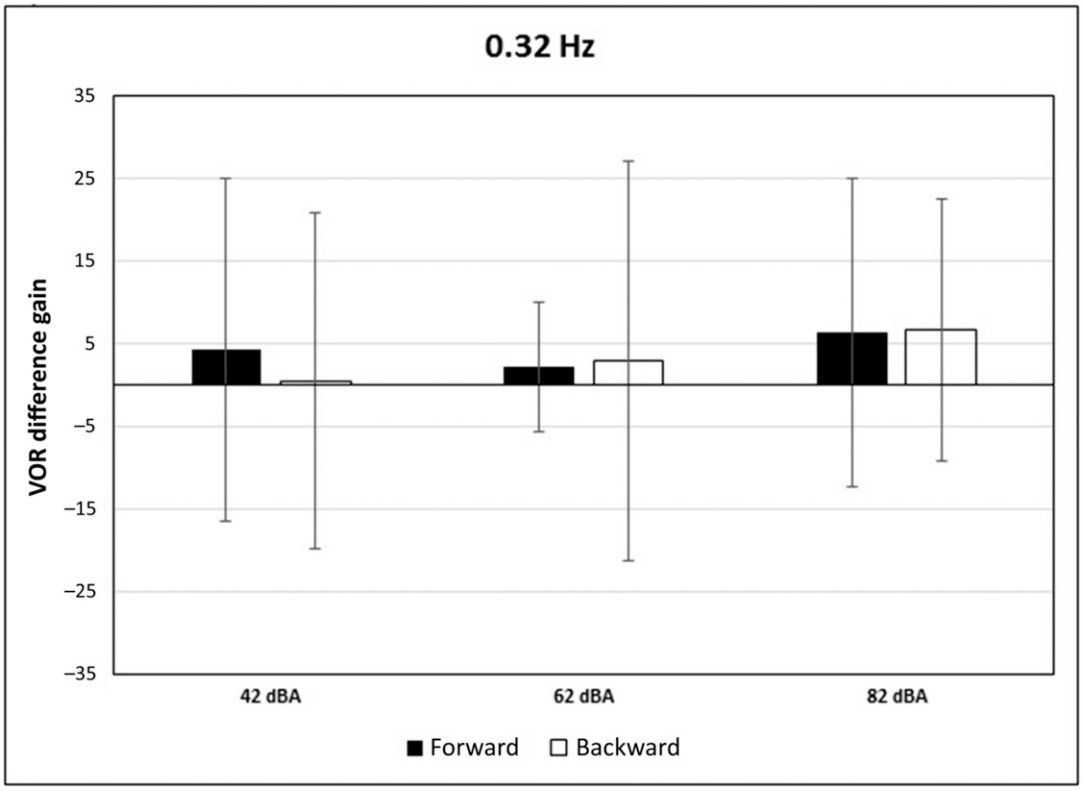

Figure 3. Mean VOR difference gain values for 0.32 Hz for each intensity level and speech type. Standard deviation bars are shown.

the strength of the MOC reflex, or the acoustic reflex threshold.

The results of the present study were in contrast to previous research examining the effect of auditory stimulation on VOR gain. For example, Jacobson et al (2012) reported significant reduction in VOR gain when using auditory stimulation, whereas Moller et al (1990) reported significant increase in VOR gain when using auditory stimulation. Therefore, it was possible that some participants in the present study exhibited a reduction in VOR gain when presented auditory stimuli, whereas other participants exhibited an increase in VOR gain when presented auditory stimuli. If true, the effect of auditory stimulation on VOR gain would have been nullified when averaged across participants.

In an attempt to further explore this possibility, individual data were analyzed and are presented in Table 3. VOR gain values were collapsed across SHA frequency in silent to establish the baseline value. VOR gain values were also collapsed across SHA frequency, intensity level, and speech type to establish the auditory value. To identify significant changes in a participant's VOR gain between baseline and auditory conditions, a 95% confidence interval of the three baseline SHA frequencies (i.e., 95% critical difference [CD]) was calculated for each participant. Differences between the baseline and auditory VOR gain values were considered significant if the auditory VOR gain exceeded the upper or lower baseline CD. VOR gain values exceeding the lower CD were considered evidence of VOR suppression, whereas values

Table 2. Analysis of Variance Results

	df	*F*	*p*-value	η^2	Ω
Speech type	1	0.001	0.973	0.000	0.050
Intensity level	2	0.939	0.402	0.059	0.197
Frequency	2	0.686	0.511	0.044	0.155
Speech type × intensity level	2	1.357	0.273	0.083	0.269
Speech type × frequency	2	0.257	0.775	0.017	0.087
Intensity level × frequency	4	0.360	0.836	0.023	0.126
Speech type × intensity level × frequency	4	0.080	0.988	0.005	0.065

Table 3. Individual Data Averaged across SHA Frequency, Intensity Level, and Speech Type

Participant	Baseline VOR Gain	CD	Auditory VOR Gain	Percentage Change
1	24 (8.5)	14.33–33.67	26.1 (10.7)	−9.03
2	27 (6)	20.12–33.88	31.1 (7.1)	−15.47
3	53.3 (25.4)	24.55–82.05	50.6 (15.5)	5.00
4	56.3 (8.5)	46.68–65.92	50.2 (6.6)	10.85
5	39.3 (7.5)	30.81–47.79	42 (18)	−6.78
6	96 (44.8)	45.25–146.75	79.6 (22.3)	17.07
7	42.3 (9.8)	31.19–53.41	52.5 (12.9)	−24.02
8	28.6 (4.9)	23.02–34.18	35.6 (9.5)	−24.42
9	23.6 (1.1)	**22.29–24.91**	**35.1 (10.8)**	**−48.36**
10	38.6 (3.5)	34.63–42.57	39 (8.3)	−1.01
11	63.6 (8)	54.45–72.75	61.2 (11.7)	3.84
12	64 (4.5)	**58.81–69.19**	**73.5 (18.8)**	−14.93
13	62.3 (19.3)	40.35–84.25	62.1 (19.2)	0.36
14	43 (15.1)	25.88–60.12	37.8 (10.9)	12.02
15	60.6 (4.1)	**55.89–65.31**	**38.1 (15.6)**	**37.18**
16	21.6 (3.2)	17.96–25.24	22.9 (7.2)	−5.90
17	18.3 (8.7)	**8.41–28.19**	**29 (8.5)**	**−58.48**

exceeding the upper CD were considered evidence of VOR enhancement. Examination of Table 3 indicates that only one participant exhibited significant VOR suppression (participant 15), three participants exhibited significant VOR enhancement (participants 9, 12, and 17), and 13 participants were within the CD (no change).

As previously mentioned, Jacobson et al (2012) reported an average VOR gain reduction of 28% when participants were instructed to direct their attention to a moving auditory target. The percentage of VOR gain change was calculated as [1 − (auditory VOR gain/baseline VOR gain) × 100]. For comparison, the percentage of change in VOR gain from baseline was also calculated using the individual data and the same equation (Table 3). A positive percentage indicated VOR gain was reduced or suppressed, whereas a negative percentage indicated VOR gain was increased or enhanced with auditory stimulation. Differences between the baseline and auditory VOR gain values were considered significant if the percentage of change exceeded the 28% value reported by Jacobson et al (2012). Thus, values exceeding 28% were considered evidence of VOR suppression, whereas values exceeding −28% considered evidence of VOR enhancement. Examination of Table 3 indicates that only one participant exhibited significant VOR suppression (participant 15), two participants exhibited significant VOR enhancement (participants 9 and 17), and 14 participants exhibited no significant change. Taken together, the analysis of the individual data indicates auditory stimulation did not produce VOR suppression or enhancement for most of the participants.

It is possible that methodological differences between the studies may explain the discrepant results. Jacobson et al (2012) and Moller et al (1990) reported a significant reduction in VOR gain (suppression) when participants used a speaker source that rotated with the participant. By contrast, Moller et al (1990) reported a significant increase in VOR gain (enhancement) when participants used a fixed, stationary speaker source. Thus, the location of the sound source during RCT may impact the ability to use spatial cues, thereby affecting VOR gain in different ways. Stated differently, the location of the auditory target in space may impact VOR gain more than the intensity level or type of auditory target used.

For example, when the acoustic target is fixed to the chair and rotates with the participant, participants may suppress the VOR to focus on the auditory target directly in front of them, similar to the example of tracking a moving target in space. Conversely, when the acoustic target is fixed to the wall while the participant rotates, participants may enhance the VOR to search for the reference target in the dark, similar to the example of fixating on a stationary target while moving in space. The present study used insert earphones which eliminated the ability to use spatial cues. In doing so, auditory images moved from localized points in space to within the head of the participant. Thus, the removal of an auditory target that either rotated with the participant or remained fixed in space may explain why participants in the present study were not affected by auditory stimulation. In addition, participants were informed they would hear a stimulus during the auditory conditions, but they were not instructed to directly attend to the speech stimulus. It is possible that

participants did not attend to either speech stimulus, regardless of the intensity level, thereby resulting in minimal effects. Future studies should examine the effect of direct versus indirect attention to the speech stimulus on VOR suppression.

The results of the present study do not support the hypothesis that VOR suppression may be due to activation of auditory efferents rather than activation of vestibular efferents. If VOR gain was related to efferent pathway activation, increasing the intensity level of the auditory stimuli should have reduced VOR gain regardless of how the stimuli were delivered. The use of insert earphones that removed spacial cues would not be expected to impact VOR gain if the efferent control hypothesis was correct. Similarly, VOR gain was not correlated with other measures of auditory efferent control such as the MOCR and the acoustic reflex. This conclusion is in agreement with Jacobson et al (2012) who suggested if auditory efferent pathways can suppress the VOR, the effect would be modest. Results of the present study do, however, appear to support they hypothesis that VOR suppression may be due to cortical modulation through directed attention to a moving or stationary auditory target. Although the use of an auditory stimulus than contained an attentional component did not impact the VOR gain, it appears that the removal of an acoustic target from space for one to attend to either consciously or subconsciously prevented suppression or enhancement of the VOR. This finding is in agreement with Jacobson et al (2012) who suggested that attention directed to a nonvisual target allowed participants to suppress the VOR.

Clinical Implication and Limitations

The assessment of the VOR using RCT is an important measure that aids in assessment, differential diagnosis, and subsequent rehabilitation planning of the vestibular patient. To ensure accurate VOR measurement, extravestibular suppressive factors such as visual fixation, auditory stimulation, and attention must be controlled. If the VOR is inaccurately captured, it could have significant clinical implications of misdiagnosis and/or improper treatment. Clinicians should exert caution as auditory stimuli in the examination room have the potential to serve as fixation targets which may result in suppression or enhancement of the VOR, thereby producing inaccurate test results. Consideration should be made for controlling the auditory stimulation in the test environment during RCT to ensure the measured VOR response is an accurate representation of vestibular function with little to no influence from nonvestibular factors.

Last, all participants in the present study were young with normal hearing and no history of vestibular disorders. It is possible the effects of auditory stimulation on the VOR could be different in an older population with a history of hearing loss and/or vestibular disorders. Future studies should investigate the effects of aging, hearing loss, and vestibular pathology on the ability to use auditory stimulation to affect the VOR. In addition, future research is needed to determine to role of spacial hearing on the VOR.

CONCLUSIONS

The purpose of this study was to determine if the VOR was affected by the intensity level and/or the type of speech stimulus. Results indicated that the VOR was not affected by the intensity level of the stimulus or by the type of speech stimulus used. Results further indicated that VOR was not related to the baseline gain, the strength of the MOC reflex, or the acoustic reflex threshold. Clinicians should be aware that the location of the sound source during RCT may impact the ability to use spatial cues, thereby affecting VOR gain in different ways. Findings support the hypothesis that VOR gain may be affected by cortical modulation through directed attention rather than due to activation of efferent pathways to the vestibular nuclei.

REFERENCES

Backus BC, Guinan JJ. (2007) Measurement of the distribution of medial olivocochlear acoustic reflex strengths across normal-hearing individuals via otoacoustic emissions. *J Assoc Res Otolaryngol* 8(4):484–496.

Baloh RW, Honrubia V, Kerber KA. (2011) *Baloh and Honrubia's Clinical Neurophysiology of the Vestibular System*. New York, NY: Oxford University Press.

Barr CC, Schultheis LW, Robinson DA. (1976) Voluntary, non-visual control of the human vestibulo-ocular reflex. *Acta Otolaryngol* 81: 365–375.

Bronstein AM, Patel M, Arshad Q. (2015) A brief review of the clinical anatomy of the vestibular-ocular connections—how much do we know? *Eye* 29:163–170.

Brown EC, Muzik O, Rothermel R, Matsuzaki N, Juhasz C, Shah AK, Atkinson MD, Fuerst D, Mittal S, Sood S, Diwadkar VA, Asano E. (2012) Evaluating reverse speech as a control task with language-related gamma activity on electrocorticography. *Neuroimage* 60(4):2335–2345.

Goebel JA, Isipradit P, Hanson JM. (2000) Manual rotational testing of the vestibulo- ocular reflex. *Laryngoscope* 110:517–535.

Goodman S, Mertes I, Lewis J, Weissbeck D. (2013) Medial olivocochlear-induced transient-evoked otoacoustic emission amplitude shifts in individual subjects. *J Assoc Res Otolaryngol* 14: 829–842.

Goumans J, Houban MM, Dits J, Van Der Steen J. (2010) Peaks and troughs of three- dimensional vestibulo-ocular reflex in humans. *J Assoc Res Otolaryngol* 11:383–393.

Guinan JJ, Jr. (2006) Olivocochlear efferents: anatomy, physiology, function, and the measurement of efferent effects in humans. *Ear Hear* 27:589–607.

Halmagyi GM, Gresty MA. (1979) Clinical signs of visual-vestibular interaction. *J Neurol Neurosurg Psychiatry* 42:934–939.

Hawley ML, Litovsky RY, Culling JG. (2004) The benefit of binaural hearing in a cocktail party: effect of location and type of interferer. *J Acoust Soc Am* 115:833–843.

Hill JC, Prasher DK, Luxon LM. (1997) Evidence efferent effects on auditory afferent activity and their functional relevance. *Clin Otolaryngol Allied Sci* 22:394–402.

Jacobson GP, Piker EG, Do C, McCaslin DL, Hood L. (2012) Suppression of the vestibulo-ocular reflex using visual and nonvisual stimuli. *Am J Audiol* 21:226–231.

Marshall L, Lapsley Miller JA, Guinan JJ, Shera CA, Reed CM, Perez ZD, Delhorne LA, Boege P. (2014) Otoacoustic-emission-based medial-olivocochlear reflex assays for humans. *J Acoust Soc Am* 136:2697–2713.

Mishra S, Dinger Z. (2016) Influence of medial olivocochlear efferents on the sharpness of cochlear tuning estimates in children. *J Acoust Soc Am* 14(2):1060.

Moller C, White V, Odkvist LM. (1990) Plasticity of compensatory eye movements in rotatory tests: II. The effect of voluntary, visual, imaginary, auditory, and proprioceptive mechanisms. *Acta Otolaryngol* 109:168–178.

Purves D, Augustine G, Fitzpatrick D, Katz L, LaMantia A, McNamara J, Williams M, eds. (2001) Central vestibular pathways: eye, head, and body reflexes. In *Neuroscience*. 2nd ed. Sunderland, MA: Sinauer Associates. https://www.ncbi.nlm.nih.gov/books/NBK10987/. Accessed November 17, 2016.

Rothauser EH, Chapman WD, Guttman N, Nordby KS, Silbigert HR, Urbanek GE, Weinstock M. (1969) IEEE recommended practice for speech quality measurements. *IEEE Trans Audio Electroacoust* 17: 225–246.

Warren E, Liberman M. (1989) Effects of contralateral sound on auditory-nerve responses. I. Contributiuons of cochlear efferents. *Hear Res* 37:89–104.