Frequency Following Response Study

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Abstract

Persistent rhythmic auditory stimuli neurologically manifest as a cortical frequency-following response (Oster 1973; Smith et al. 1975; Marsh et al. 1975; Smith et al. 1978; Hink et al. 1980). Both Oster (1973) and Hink et al. (1980) have demonstrated a frequency-following response (FFR) to binaural beating with an evoked-potential EEG protocol in the context of hearing-acuity research.

This study was designed to further the above-cited previous FFR work with respect to binaural beating using multiple-subject trials (N = 7) and an appropriate evoked-potential protocol. Results showed subjects exposed to specific binaural-beat stimuli evidenced increases in amplitude of time-domain averaged EEG in frequencies matching the binaural-beat stimuli when compared to the silence-baseline condition. Elevation in EEG amplitudes in comparison to the silence-baseline condition was also seen in reaction to alternative stimuli.

Statistical analysis revealed non-significant increases in 16 Hz time-domain averaged EEG amplitudes during the 16 Hz binaural-beat stimulus periods over the silence-baseline condition when the increases in EEG during the alternative stimuli were considered. There was, therefore, no evidence of a 16 Hz FFR. Significant ($p =< 0.05$) increases in 7 Hz EEG amplitudes were, however, demonstrated during the 7 Hz stimulus condition, which provided evidence of a 7 Hz FFR during the 7 Hz binaural-beat stimulus periods even when the increases in EEG during the alternative stimuli were considered.

Key Words: reticular, frequency-following response, sound, binaural beats, brain waves

Background

An auditory "frequency-following response" is defined as a brain-wave (EEG) frequency response that corresponds to the frequency of an auditory stimulus (Smith, Marsh, & Brown 1975). There have been several free-running EEG studies that suggest that a frequency-following response (FFR) to binaural beats may, somehow encourage alterations in overall brain waves and, therefore, arousal states (see Foster 1990; Sadigh 1990; Hiew 1995; Brady 1997). Even though an FFR to binaural beating has been

demonstrated (Oster 1973 & Hink et al. 1980), an FFR to binaural beats in frequency ranges associated with such state changes has not been previously demonstrated using apropos evoked-potential EEG protocols.

The sensation of auditory binaural beats occurs when two coherent sounds of nearly similar frequencies are presented, one to each ear, and the brain detects phase differences between these sounds. Within the olivary nuclei the brain integrates the two signals, producing a sensation of a third sound called the binaural beat. Perceived as a fluctuating rhythm at the frequency of the difference between the two (stereo left and right) auditory inputs, binaural beats originate within the brainstem's superior olivary nuclei, the sites of contralateral integration of auditory input (Oster 1973). This auditory sensation is neurologically routed to the reticular formation (Swann et al. 1982) and simultaneously volume conducted to the cortex where it can be objectively measured as an FFR (Oster 1973; Smith, Marsh, & Brown 1975; Marsh, Brown & Smith 1975; Smith et al. 1978; Hink et al. 1980).

Much speculation surrounds the possibility that the very-low amplitude auditory FFR somehow engenders psychophysiological state changes. Well-accepted studies demonstrate the presence of an EEG FFR to auditory stimuli recorded at the vertex of the human scalp (Oster 1973; Smith, Marsh, & Brown 1975; Marsh, Brown & Smith 1975; Smith et al. 1978; Hink et al. 1980). Is it possible that sufficient exposure (amplitude, duration, and frequency) to auditory stimuli may influence ongoing brain-wave activity? The perceptual experience of binaural beats could be seen as a psychologically "disruptive force" or "patterning force" required in the induction of a discrete altered state of consciousness (Tart 1975). Binaural beats also affect management functions of the reticular activating system through traditional neural pathways. The reticular formation alters the electrical potentials of the thalamus and cerebral cortex (measurable by EEG), arousing or quieting this so-called higher center of the brain (Swann et al. 1982). So, the reticular activating system plays an important role in understanding changes in brain waves and states of consciousness (Tice 1989 & Estes 1995), which may somehow be engendered by binaural-beat environments.
A critical point, however, is that FFRs to binaural beats (proof that the sensation of binaural beating has a neurological efficacy) in archetypal brain-wave frequency ranges associated with reported altered states of consciousness have not been objectively demonstrated using apropos evoked-potential EEG protocols. This then would appear to be a vital step in understanding the reported effectiveness of rhythmic sound stimuli, including binaural beating, and the possible neural underpinnings.

The Study

This replication study used multiple-subject trials (N = 7) designed to objectively verify an FFR to beta and theta binaural-beat stimuli through the use of an appropriate evoked-potential protocol. This study was designed to determine if a 16 Hz (beta) binaural beat would engender a 16 Hz FFR and if a 7 Hz (theta) binaural beat would engender a 7 Hz FFR. These frequencies were used because they were in keeping with arousal states

purportedly encouraged by listening to binaural beats (Atwater 1997 and referenced cited therein).

The hypothesis postulated that subjects exposed to binaural-beat stimuli would evidence increases in amplitude of time-domain averaged EEG in frequencies matching binaural-beat stimuli when compared to a silence-baseline condition. Elevation in EEG amplitude (an arousal response) could be expected in the case of a placebo stimulus as well as the alternative binaural-beat stimulus. Significant increases in 16 Hz- and 7 Hz-EEG amplitudes during comparable binaural-beat stimuli periods over the silence-baseline condition would imply the development of an FFR to the respective binaural beat.

To control for subject expectation, an eighteen-episode Latin-Square protocol provided for two seconds of a binaural-beat stimulus at 16 Hz, 7 Hz, and two seconds of a placebo tone (without a binaural beat). Between each two-second-stimulus interval were two seconds of silence. The onset pulses were reversed in the middle of the protocol so as to phase-cancel the gross brainstem response, the evoked potential of the tones themselves (vs. the binaural-beat component). The entire test sequence lasted about thirty-five minutes.

Subjects were volunteer adults, both male and female. None of the subjects had prior experience listening to binaural beats. All subjects reported normal hearing. None of the subjects reported a history of neurologic disorders. All subjects executed an Informed Consent Form explaining the nature and purpose of this research and the risks, if any, associated with his/her participation therein.

A computer presented the audio stimuli. A series of sound files (Microsoft's .wav format) provided the stimulus periods. Each sound file (22050 Hz / 8 Bit / Stereo) was automatically played in the Latin-Square sequence through a 16 Bit stereo sound card to subjects' Sony(tm) MDR E464 in-ear stereo headphones. All subjects were tested in an isolated, double-wall soundproofed, electrically shielded booth after EEG electrode placement and continuity testing. Subjects lay supine on a waterbed heated to 33° C (=/-.5°). To aid in the reduction of eye-movement artifact, a small (5 by 20 cm.) soft fabric bag filled with rice was placed over the closed eyes of the subjects.

The stimuli integrated into the Latin-Square were as follows:

16 Hz Binaural Beat Frequency Mix

Left-Ear Frequencies	Right-Ear Frequencies
292 Hz	308 Hz
192 Hz	208 Hz

7 Hz Binaural Beat Frequency Mix

Left-Ear Frequencies	Right-Ear Frequencies
296 Hz	303 Hz

196 Hz 203 Hz

Placebo A

Left-Ear Frequency	Right-Ear Frequency
300 Hz	300 Hz

Placebo B

Left-Ear Frequency	Right-Ear Frequency
200 Hz	200 Hz

EEG recordings were made during the entire Latin-Square protocol outlined above. The resulting EEG record provided 180 one-second 256 integer arrays of data for analysis from each two-second stimulus and silence condition. Subjects were connected to a 24-channel digitizing EEG computer (Neurosearch-24(tm), LEXICOR Medical Technology Inc., Boulder, Colorado) using V151 software. The entire standard 10/20 International System montage of electrodes was used (Electro-Cap(tm)). The reference was linked-ears balanced for impedance by metered calibration to less that 1k-ohm difference. The electrode at the midline vertex served as ground. The nineteen active EEG channels and reference electrode placements were tested to ensure contact resistance of 10K ohms or less and balanced closely for impedance level. Electro-Gel(tm) was used in the prescribed manner to provide for adequate electrical conductivity. All EEG data were recorded and saved on a 386-AT computer in raw form.

The Neurosearch-24 (NRS-24) sampling rate of 256 samples per second was used with the high pass filter set to off. The NRS-24 provided for an EEG frequency response of 1-64 Hz (less 60 Hz, due to a notch filter), a frequency resolution of 1 Hz, and a temporal resolution of one second. Each epoch was one second long, creating an integer array of 256 points per channel per epoch. Since each stimulus and each silence period was two seconds, each was represented by an integer array of 512 points.

The NRS-24 was calibrated for frequency response of each channel at each sampling rate using a swept frequency approach, according to the procedures outlined in the NRS-24 software manual. The purpose of this calibration was to compensate for the roll-off of the anti-aliasing analog filters. Frequency response corrections were applied to ensure flat spectrums across the frequencies of interest. The NRS-24 was also normalized for amplitude response at 10 Hz at each channel for every combination of sampling rate and gain setting. This procedure compensated for any differences in amplification across all channels for each sampling rate and gain setting. Normalization was used in conjunction with frequency-response calibration to ensure similar spectral amplitudes across channels. Both calibration and normalization verifications were periodically conducted thereafter. All parameters remained stable throughout the time of the testing and no adjustments were needed to recalibrate or renormalize.

Analysis

Preliminary EEG-signal analyses consisted of 1) the analog-to-digital conversion of the raw brain waves; 2) editing the digitized EEG containing obvious eye-movement artifact and high-frequency muscle activity; 3) extracting, ordering, and phase aligning one-second binaural-beat stimuli, placebo, and silence-baseline epochs, with software written by the author; 4) time-domain averaging 180 one-second (256 sample integer array) epochs of each stimulus condition, and 5) performing Fast-Fourier Transform (FFT), with SimpleFFT software from Copelyright Software, of the time-domain averaged data. Results of FFT computation yielded amplitude values in microvolts for the brain-wave frequencies of interest.

Statistical computation was accomplished with Kwikstat 4.6 Professional software from TexaSoft, Cedar Hill, Texas. Statistical analysis consisted of using averaged FFT values for the binaural-beat stimuli periods, placebo, and silence-baseline condition for each subject. Multiple comparisons following a one-way Analysis of Variance (Dennett's Test) equating the silence-baseline condition (as a control mean) with the respective binaural-beat stimuli conditions and the placebo condition were made using the averaged FFT values.

Results

As set forth in the hypothesis, subjects exposed to binaural-beat stimuli evidenced increases in amplitude of time-domain averaged EEG in frequencies matching binaural-beat stimuli when compared to the silence-baseline condition. As expected, some elevation in EEG amplitudes in comparison to the silence-baseline condition was also seen in reaction to both the placebo stimulus and the alternative binaural-beat stimulus. There were non-significant increases in 16 Hz time-domain averaged EEG amplitudes during the 16 Hz binaural-beat stimulus periods over the silence-baseline condition when the increases in EEG during the placebo and the 7 Hz binaural-beat stimuli were considered. There was, therefore, no evidence of a 16 Hz FFR.

Multiple Comparison/Dunnett's Test Summary Data for 16 Hz EEG Response

Group Means and Standard Deviations

Silence Baseline:	mean = .0331	s.d. = .0209	n = 7
16 Hz Stimulus:	mean = .065	s.d. = .0354	n = 7
7 Hz Stimulus:	mean = .0362	s.d. = .0168	n = 7
Placebo:	mean = .0553	s.d. = .0337	n = 7

Analysis of Variance Table

Source	S.S.	DF	MS	F	Approx p
Total	.02	27			

Treatment	.	3	.	2.11	0.1257
Error	.02	24	.		

Error term used for comparisons = . with 24 d. f.

Dunnett's Comp. (2-tailed)	Difference	P	Q	Critical q (.05)
Silence Baseline-16 Hz Stimulus	= 0.0319	4	2.141	2.51
Silence Baseline-Placebo	= 0.0222	3	1.49	2.35
Silence Baseline7 Hz Stimulus	= 0.0031	2	.208	2.06

Comparisons marked with an asterisk "*" are significantly different.

Significant (p =< 0.05) increases in 7 Hz EEG amplitudes were, however, demonstrated during the 7 Hz stimulus condition, which provided evidence of a 7 Hz FFR during the 7 Hz binaural-beat stimulus periods even when the increases in EEG during the placebo and 16 Hz binaural-beat stimuli were considered.

Multiple Comparison/Dunnett's Test Summary Data for 7 Hz EEG Response

Group Means and Standard Deviations

Silence Baseline:	mean = .0686	s.d. = .0397	n = 7
7 Hz Stimulus:	mean = .1706	s.d. = .0995	n = 7
16 Hz Stimulus:	mean = .105	s.d. = .0677	n = 7
Placebo:	mean = .1249	s.d. = .067	n = 7

Analysis of Variance Table

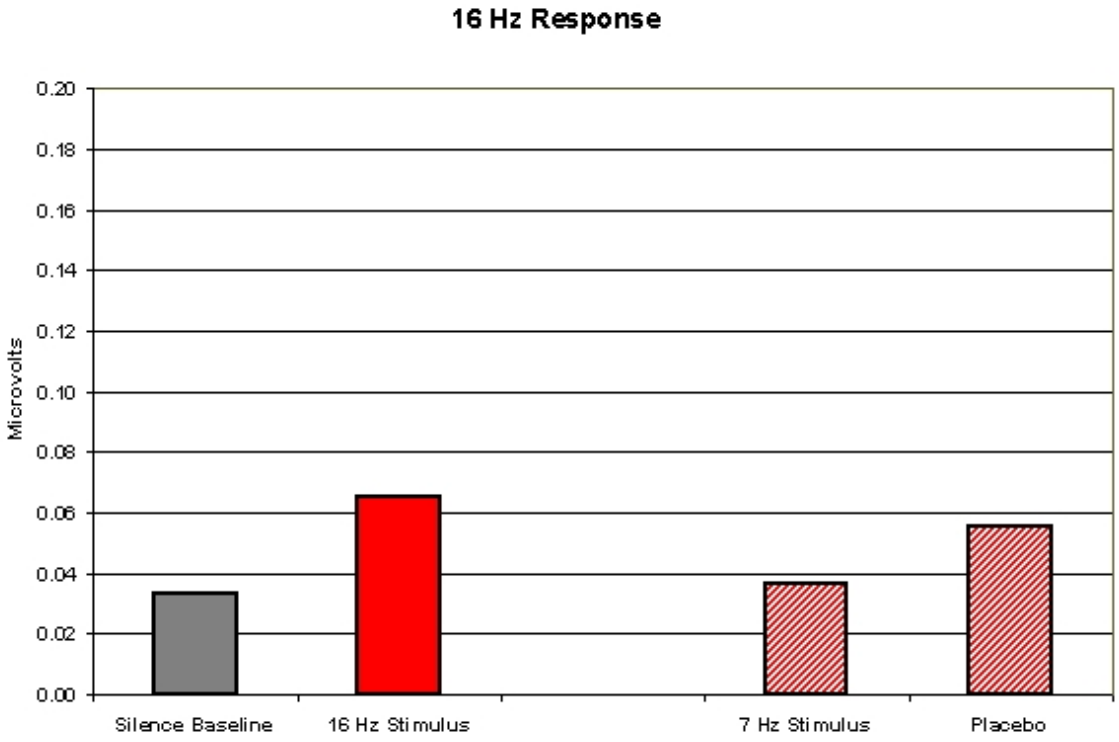
Source	S.S.	DF	MS	F	Appox p
Total	.16	27			
Treatment	.04	3	.01	2.46	0.087
Error	.12	24	.01		

Error term used for comparisons = .01 with 24 d. f.

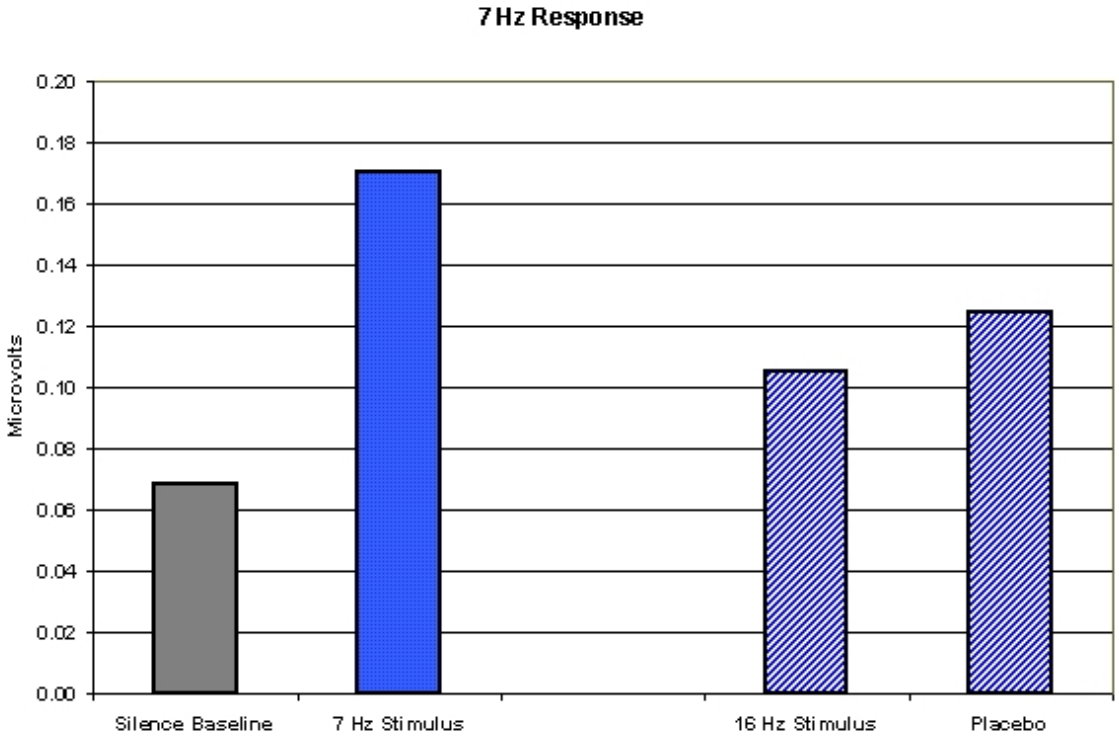
Dunnett's Comp. (2-tailed)	Difference	P	Q	Critical q (.05)
Silence Baseline-7 Hz Stimulus	= 0.102	4	2.662	2.51 *
Silence Baseline-Placebo	= 0.0563	3	1.47	2.35
Silence Baseline-16 Hz Stimulus	= 0.0364	2	.95	2.06

Comparisons marked with an asterisk "*" are significantly different.

The following graphs show the anticipated arousal response to the placebo stimulus and the alternative binaural-beat stimulus as well as substantial EEG amplitude increases in the appropriate binaural-beat stimuli periods over the silence-baseline condition.



The 16 Hz response to the 16 Hz stimulus, although elevated, was non-significant when
the 7 Hz and placebo conditions were included in the multiple comparison procedure.



The 7 Hz response to the 7 Hz stimulus was significant (p =< 0.05), even when the 16 Hz condition and the placebo condition were included in the multiple comparison.

Comments

Even though very small, this study furthered previous work and objectively demonstrated an FFR to 7 Hz binaural beating. This study showed at least some neurological evidence of an FFR in brain-wave frequencies typically associated with theta levels of arousal. Additionally, there was a promising increase in the beta response level during the beta stimulus compared to the silence-baseline condition. Perhaps in a larger study with additional numbers of subjects, this trend would continue to the point of statistical significance indicating a 16 Hz FFR. There have been several EEG studies suggesting that an FFR to binaural beats may somehow encourage alterations in overall brain waves and, therefore, arousal states.

Much speculation still surrounds the possible mechanism by which rhythmic sound patterns (binaural beats, in this case) appear to engender changes in cortical arousal (ongoing brain-wave activity vs. the FFR). As the reticular is responsible for regulating cortical arousal (Swann et al. 1982; Empson 1986; Newman & Baars 1993; Newman 1997a,b; Petty 1998), it is possible that the reticular formation serves as the mechanism of change in arousal levels engendered by externally initiated (e.g., music, rhythmic drumming, or binaural beats) coherent oscillations within the superior olivary nuclei and further the cholinergic neurons within the nucleus reticularis.

Additionally, four decades of investigation have shown that exposure to such stimuli under appropriate circumstances can provide access to expanded states of consciousness (Atwater 1997). Several free-running EEG studies (Foster 1990; Sadigh 1990; Hiew 1995; Brady 1997, among others) suggest that binaural beats induce alterations in cortical arousal states. These cited studies, by default, also document measurable changes in the extended reticular-thalamic activating system during exposure to binaural beats because the reticular formation neurochemically regulates cortical arousal (Swann et al. 1982; Empson 1986; Newman & Baars 1993; Newman 1997a,b and Petty 1998).

It would appear that the rhythmic frequencies of an auditory stimulus (when objectively demonstrated by an FFR) affect cholinergic neurons within the nucleus reticularis. Such an intercourse seemingly modifies the membrane transport and production of acetylcholine and consequently results in changes in arousal states. These suppositions are compatible with current knowledge of the reticular formation and suggest a neural mechanism, an instrument for the regulation of cortical levels of arousal using rhythmic audio stimuli.

The implications in the enhancement of human performance as it relates to the control of generalized arousal levels such as the basic rest/activity cycle, sleep cycles, mood and motivational states, orienting and vigilance, etc., via the use of rhythmic sound and binaural-beat stimuli are intriguing. The voluntary regulation of arousal levels through the use of persistent rhythmic sound stimuli such as binaural beating is the subject of another paper.

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