applied gallamine was to eliminate outward sodium currents almost completely, suggesting that the open sodium channels were occluded by the positively charged gallamine molecules. Occasionally, the combination of slowed inactivation and occlusion could be seen as a rapid initial decline in inward current, followed by a later decay that was slower than Na^+ inactivation in the control records. This effect was particularly noticeable at potentials near the Na^+ equilibrium potential. In general, gallamine did not change the time course of sodium activation or maximum sodium permeability. The nodal action potential following internal application of gallamine was prolonged by two to three times, as might be expected from the voltage-clamp results.

In addition to gallamine, we examined the effects of internally and externally applied acetylcholine, carbamylcholine, and *d*-tubocurarine. No effects on sodium permeability were seen at concentrations up to 10 m*M*. At this high concentration, acetylcholine and carbamylcholine (but not *d*-tubocurarine) slightly reduced the potassium conductance (10 to 20 percent), presumably because of their structural resemblance to TEA^+. Since gallamine is effective at much lower concentrations, its action is likely to be reasonably specific.

The actions of gallamine on the potassium channel resembled those produced by TEA^+ (*7–9*), except that gallamine was approximately five times more potent than TEA^+. In most respects, the effects of internal gallamine on the sodium channel mimicked those of another neuromuscular blocking agent, pancuronium bromide (*2*), although gallamine did not decrease maximum sodium permeability. Although gallamine did not affect the sodium channel acutely, this may not remain true with long-term exposure to the drug.

The high affinity of gallamine for the potassium channels of nerve membranes may have significant consequences in gallamine-paralyzed preparations. Particularly in neurophysiological studies, total paralysis is often required. The minimum paralyzing concentration (calculated on the assumption that the extracellular fluid is approximately 20 percent of body weight) of gallamine in human, cat, and rat are approximately 19 μM, 11 μM to 25 μM, and 69 μM, respectively (*10–12*), and concentrations up to ten times these levels are occasionally employed. The voltage-clamp data show that even 10 μM gallamine produces a detectable effect on g_K. Indeed, at concentrations lower than those required for neuromuscular block, gallamine has been shown to effect repetitive firing in both mammalian muscle and amphibian nerve fibers (*13–15*). The entry of gallamine to the central nervous system in intact preparations is impaired by the blood-brain barrier; but, particularly in experimental studies, this barrier is often breached by surgical or pathological processes. Consequently, it is important to note that while the myelinated axons of the mammalian central and peripheral nervous systems have few exposed potassium channels (*16*), their central synaptic endings are significantly affected by potassium-blocking drugs (*17*). Thus, one would expect gallamine, upon gaining access to the central nervous system, to have an excitatory effect on central nerve activity commensurate with its potassium-blocking property, and such an effect has been reported (*18*).

In view of the potent effects of gallamine on neural potassium and sodium channels in mammalian and amphibian nerve fibers, its continued use as a specific neuromuscular blocking agent should be reexamined, especially in circumstances where the blood-brain barrier may be disrupted by surgical or pathological processes.

K. J. SMITH
C. L. SCHAUF

Department of Physiology,
Rush University,
Chicago, Illinois 60612

References and Notes

1. D. Bovet, F. DePierre, Y. deLestrange, *C. R. Acad. Sci.* **224**, 496 (1947).
2. J. Z. Yeh and T. Narahashi, *J. Gen. Physiol.* **69**, 293 (1977).
3. B. Hille, *ibid.* **58**, 599 (1971).
4. T. L. Pencek, C. L. Schauf, F. A. Davis, *J. Pharmacol. Exp. Ther.* **204**, 400 (1978).
5. T. Brismar, *J. Physiol. (London)* **298**, 171 (1980).
6. S. Y. Chiu and J. M. Ritchie, *Nature (London)* **284**, 170 (1980).
7. B. W. Payton and D. G. Shand, *Br. J. Pharmacol. Chemother.* **28**, 23 (1966).
8. A. D. Grinnell, *J. Physiol. (London)* **210**, 17 (1970).
9. C. M. Armstrong, *Q. Rev. Biophys.* **2**, 179 (1975).
10. F. H. M. Derkx, I. L. Bonta, A. Lagendijk, *Eur. J. Pharmacol.* **16**, 105 (1971).
11. R. W. Gardier, M-F. Ganansia, A. L. Delaunois, W. Hamelberg, *Anesthesiology* **40**, 494 (1974).
12. J. V. Donlon, J. J. Savarese, H. H. Ali, *Anesth. Analg. (Cleveland)* **58**, 377 (1979).
13. W. F. Riker and W. C. Wescoe, *Ann. N.Y. Acad. Sci.* **54**, 373 (1951).
14. J. J. Jones and J. L. H. Laity, *Br. J. Pharmacol.* **24**, 360 (1965).
15. M. D. Sokoll, K. L. Dretchen, S. D. Gergis, J. P. Long, *Anaesthesiology* **38**, 157 (1973).
16. M. Horakova, W. Nonner, R. Stämpfli, *Proceedings of the XXIV International Congress on Physiological Science* (1968), p. 198; T. Brismar, *Acta Physiol. Scand.* **102**, 67 (1978); J. D. Kocsis and S. G. Waxman, *Nature (London)* **287**, 348 (1980).
17. M. Lemeigan, *Neurophysiology* **11**, 551 (1972); *ibid.* **12**, 641 (1973).
18. S. Salama and S. Wright, *Br. J. Pharmacol.* **7**, 1 (1952); G. C. Salmoiraghi and F. A. Steiner, *J. Neurophysiol.* **26**, 581 (1963); P. Andersen and D. R. Curtis, *Acta Physiol. Scand.* **61**, 100 (1964); D. R. Curtis and R. W. Ryall, *Exp. Brain Res.* **2**, 66 (1966); _____, J. C. Watkins, *ibid.*, p. 97; L. M. Halpern and R. G. Black, *Science* **155**, 1685 (1967); A. Galindo, K. Krnjevic, S. Schwartz, *Exp. Brain Res.* **5**, 87 (1968).
19. Supported by the Morris Multiple Sclerosis Research Fund, USPHS research grant RO1 NS15741, and National Multiple Sclerosis Society grant RG 1313A2 to C.L.S. K.J.S. is a postdoctoral fellow (FG527A) of the National Multiple Sclerosis Society.

9 February 1981

A Novel Illusion of Bars Made from Triangles

Abstract. *A repetitive pattern of a triangular luminance profile may be perceived as a triangular-wave grating or as a square-wave grating. This illusion may reflect the operation of cortical phase-selective mechanisms that are biased toward particular phase relations and favor abrupt luminance gradients.*

Visual illusions can provide insights about the processing of information by the visual system. Presumably these perceptual errors highlight neural operations that normally escape notice during everyday visual experience. Recently we happened to discover a novel illusion produced by viewing a one-dimensional grating pattern with a triangular spatial luminance profile (Fig. 1). Rather than maintain its initial appearance, the display soon became multistable—the percept alternated between a true triangular-wave grating and what appeared to resemble more closely a square-wave grating (Fig. 2) of the same period. We then inquired about the conditions necessary for experiencing this illusory square-wave grating and considered potential reasons for its occurrence.

The triangular-wave grating in Fig. 1 was generated electronically on a cathode-ray tube (CRT) display (*1*). Once perceived, the illusory square-wave grating remains present for several seconds before reverting to the genuine triangular wave. Typically, the alternations of these two distinct percepts resemble the reversals of a Necker cube. As a rule, each luminance peak and trough in the triangular-wave grating becomes the edge of an illusory bar. The illusion itself is bistable in that the light bars may appear either to the left or to the right of the luminance peak (Fig. 2, C and D); as a result, the illusory square-wave grating may appear to shift in phase (*2*). Some observers have spontaneously reported that the bars of the illusory square-wave grating often appear to lie in different depth planes, such that the set of dark bars appears behind the set of light bars. The illusion, when it is seen, encompasses the entire display rather than being spatially local. By varying spatial frequency on the CRT display and recording the percentage of the total viewing time the illusion was experienced (its predominance), we found the optimum value to be around 1 cycle/deg. The illusion may be experienced at any orientation.

The illusion is not experienced until the contrast amplitude of the triangular wave reaches a level sufficient to push at least two of the frequency components above their own threshold. On the basis of the characteristic shape of the human contrast sensitivity function, we predicted that less contrast above detection threshold would be required to first create the illusion at a low spatial frequency than at a high spatial frequency. This prediction was confirmed: at 0.5 cycle/deg, contrast had to be raised only 8 to 10 dB above absolute detection threshold in order for the triangular-wave grating to be seen as an illusory square-wave grating some of the time; at 2 cycle/deg, although the minimum detection threshold was actually lower than at 0.5 cycle/deg, almost a full log unit of additional contrast (20 dB) was needed for the subject to begin experiencing the illusion. Further increases in contrast enhanced the predominance of the illusion at the lower, but not at the higher, spatial frequency.

We considered three possible explanations for the illusion. First, the illusion could be characterized as a perceptual inference (*3*). If an array of bars were situated in depth relative to a background plane and were illuminated from an appropriate angle from either side, the resulting luminance profile (and hence retinal image) would resemble a triangle wave. Consistent with this interpretation is the perception of depth that sometimes accompanies the illusion and that can itself assume either of two spatial organizations depending on whether light bars are seen to the left or to the right of a luminance peak. We have been unsuccessful, however, in biasing the illusion toward one particular depth organization by adding potent information, in the form of real shadows, about the direction of the source of illumination (*4*).

A second explanation is that the illusion arises from the superimposition, by eye movements, of the proximal stimulus upon an afterimage (*5*). If the proximal stimulus is superimposed on an afterimage with a phase difference of $\pm 90°$ (and multiples of π rad), summation of these two wave forms could yield a wave form resembling the illusion. To test this hypothesis, we presented experienced observers with the triangular-wave grating for short durations (approximately 100 msec) to eliminate the role of eye movements. Under these conditions, observers often reported that the flashed grating resembled the illusory square-wave grating (*6*). In a second condition, observers were instructed to fixate a point on a continuously presented triangular-wave grating of 0.5 cycle/deg. This instruction, which should have minimized superimposition by eye movements, did not systematically influence the predominance of the illusion compared with instructions to scan the stimulus freely. In a third condition, we produced an afterimage of a triangular-wave grating by transilluminating a photographic negative of the grating. Despite this effective stabilization of the pattern on the retina, observers still experienced the illusion. These converging lines of evidence militate against explanations of the illusion based solely on the superimposition of an afterimage and proximal stimulus.

A third potential explanation of the illusion is suggested by the fact that the triangular-wave grating and a real square-wave grating share identical Fourier components that differ in terms of their phase relations (*7*). A square-wave percept could result from a triangular-wave grating if the analysis of the phase information of the frequency components were performed by an unstable process or one that tended to be biased toward particular phase relationships. If the illusory square-wave grating results from instability or bias among phase-selective mechanisms, then decreasing (by adaptation) the relative sensitivity of the phase mechanism signaling "square-wave" phase should temporarily bias the percept in favor of the triangular-wave grating. To test this hypothesis we had observers track the appearance and disappearance of the illusion in each of three conditions: without prior adaptation (baseline), with direct adaptation to a real square-wave grating, and with interocular adaptation to a real square-wave grating. In the two adaptation conditions the observer was exposed to a 1 cycle/deg vertical square-wave grating for 2 minutes, during which the observer continuously scanned the display irregularly to minimize differential light adaptation of the retina. After this initial period of adaptation, the genuine square-wave grating was replaced by a triangle-wave grating of the same orientation and spatial frequency, and the observer reported the fluctuations in the appearance of the grating for a 30-second period. The

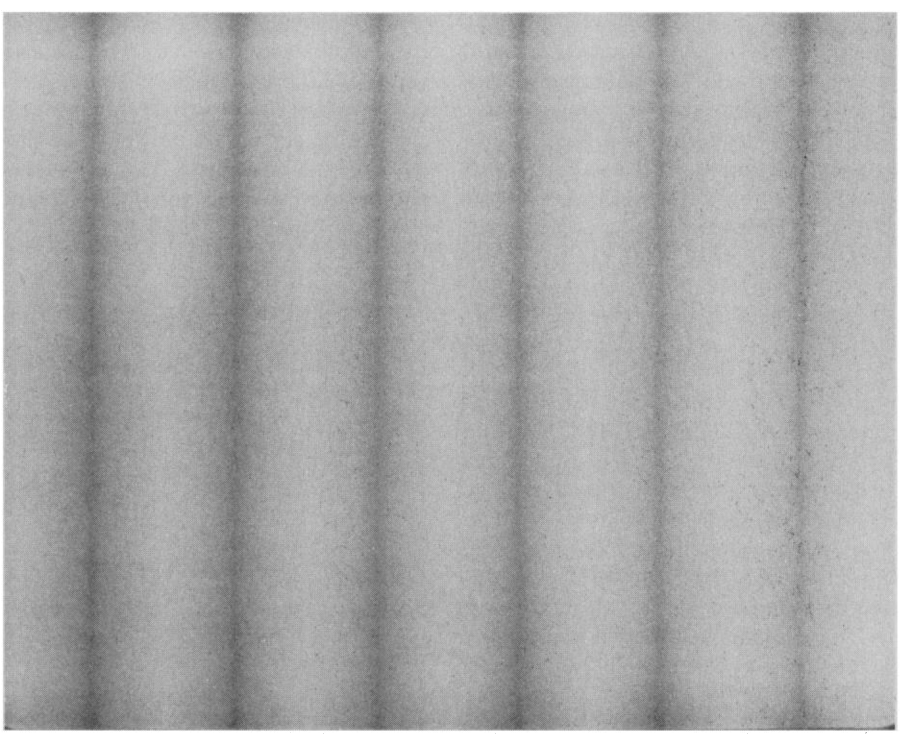

Fig. 1. Grating with a triangular spatial luminance profile. The points of peak luminance appear as thin, bright, vertical stripes; the luminance falls off gradually and symmetrically on both sides of these peaks. After a few moments of viewing this display, however, the resulting percept belies the actual luminance distribution in that it more closely resembles a set of bars with abrupt edges. Initially, the illusion may take some time to appear, after which it appears and disappears. Shaking the figure horizontally may facilitate the appearance of the illusion.

observer was then readapted to the genuine square-wave grating for 20 seconds, after which another 30-second test period followed. This adapt-and-test sequence was continued until completion of seven test periods. Three observers were tested (Fig. 3).

Without prior adaptation to a real square-wave grating (U), the illusion was experienced for about half of the observation period, with individual durations averaging 3 seconds. When the same eye was used during adaptation and test (D), predominance fell by about 50 percent, so that the test display was seen more frequently as a triangular-wave grating (*8*). Moreover, predominance was reduced significantly even when one eye was adapted to the genuine square-wave grating and the other used during the test (I) (the unstimulated eye, during both the adaptation and test, always viewed a second CRT display of the same luminance and dimensions). For both conditions of adaptation—direct and interocular—recovery from adaptation (as evidenced by a return to the predominance before adaptation) occurred within about 4 minutes. This interocular transfer of adaptation implicates a cortical or central mechanism.

In two corollary experiments we found that adaptation of a 1 cycle/deg sinusoidal grating of the same orientation, contrast, and spatial frequency as the triangular-wave grating had no effect on the predominance of the illusion. This result indicates that the reduction in predominance of the illusion with square-wave adaptation was not due to spatial frequency adaptation. We also found that adapting to a horizontal square-wave grating but testing with a vertical triangular-wave grating yielded much smaller reductions in the predominance of the illusion, thus demonstrating that the underlying process is orientation-selective.

In a final experiment we used a computer to generate on the CRT compound gratings consisting of the first three frequency components shared by the square-wave and triangular-wave gratings. These components were placed in either peaks-add (triangular) or peaks-subtract (square) phase, and their relative amplitudes were set to correspond to those of a true triangular-wave grating or those of a true square-wave grating. When both phase and amplitude of the components corresponded to those of a triangular-wave grating, the illusion was robustly experienced; this result demonstrates that three components are sufficient to trigger the process responsible for the illusion. When the components were in square-wave phase the pattern never looked triangular, regardless of the amplitude of the components. The illusion was vivid when the components were of square-wave amplitude but in triangular-wave phase. This last observation specifically implicates phase as the essential ingredient for the illusion.

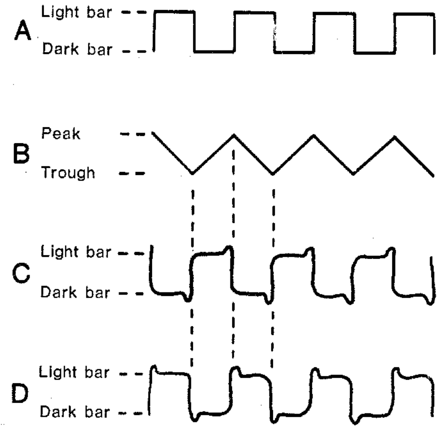

Fig. 2. Spatial luminance profiles of (A) a square-wave grating and (B) a triangular-wave grating. (C and D) Perceived brightness profile of the illusion. The illusion may assume one of two states. As denoted by the dashed lines, a luminance ramp of the triangular-wave grating may become either a light bar or dark bar of the illusion.

The results from all these experiments support the hypothesis that the illusion stems from instability among phase-selective mechanisms. Although this hypothesis is consistent with other evidence for the existence of phase-selective mechanisms in human vision (*9*), it does not explain why viewing a triangular-wave grating leads to the illusory square wave whereas viewing a genuine square-wave grating never generates an illusory triangular wave. This lack of symmetry implies that certain phase relationships are inherently more stable and that a phase anisotropy could lead to a bias on the part of the visual system for abrupt edges rather than gradual luminance gradients.

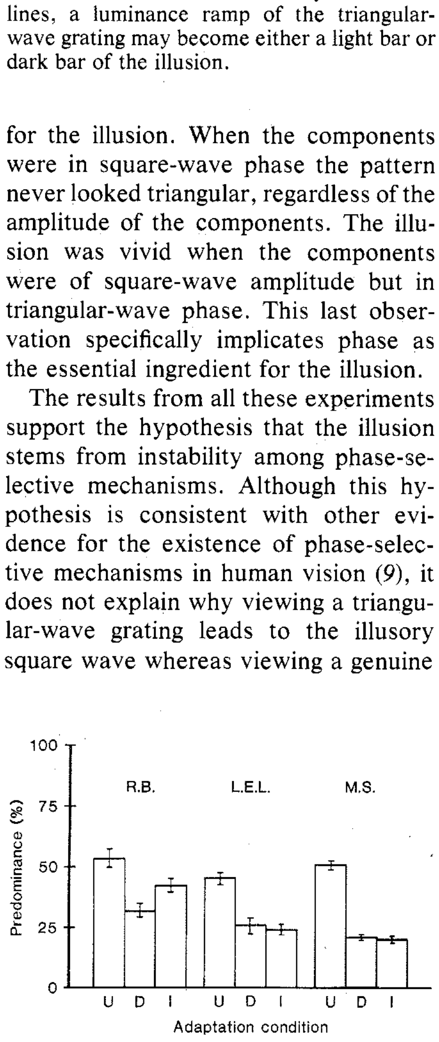

Fig. 3. Percentage of total viewing time (predominance) during which the observer experienced the square-wave illusion after different conditions of adaptation. For the unadapted (*U*) condition, the observer viewed the test stimulus—a 1 cycle/deg vertical triangular-wave grating of 30 percent contrast. During direct (*D*) and interocular (*I*) adaptation, a 2-minute monocular adaptation period to a 1 cycle/deg vertical square-wave grating of higher contrast preceded the viewing of the test display.

L. E. LEGUIRE
R. BLAKE
M. E. SLOANE

Cresap Neuroscience Laboratory, Northwestern University, Evanston, Illinois 60201

References and Notes

1. Observers operated a two-position switch to track the appearance and disappearance of the illusion during a prolonged observation period; successive durations were recorded to the nearest 10 msec. The observers viewed a CRT display from a distance of 114 cm; the display subtended a visual pattern of 7° by 5° with mean luminance of 7 cd/m^2.
2. The illusion is not simply a variant of other conventional brightness illusions such as the Craik-O'Brian-Cornsweet illusion [T. Cornsweet, *Visual Perception* (Academic Press, New York, 1970)]. With Mach-band illusions, certain luminance gradients are immediately and continuously misperceived as abrupt edges; observers never experience those edge illusions as transitory. The square-wave illusion, however, is unstable, and when it is experienced the polarity of the illusion—the phase of the apparent square wave—can assume one of two states. Moreover, the square-wave illusion, unlike Mach-band phenomena, requires some initial viewing time before it is first experienced; subsequently, the illusion is readily perceived. In that respect it behaves much like global stereopsis [B. Julesz, *Foundations of Cyclopean Perception* (Univ. of Chicago Press, Chicago, 1971)].
3. J. E. Hochberg, *Perception* (Prentice-Hall, Englewood Cliffs, N.J., 1978).
4. We attached a real three-dimensional cube (3 by 3 by 3 cm) to the center of the face of the CRT and illuminated the display obliquely from the left with a 75-W incandescent bulb. The real cube cast a prominent shadow, which clearly evidenced the direction of illumination. Despite this potent cue, however, the illusion remained multistable in that, over time, the dark (or receding) bars were seen on either side of the peak of the triangle wave.
5. An explanation based upon superimposition of an aftereffect on a proximal stimulus has been suggested by C. S. Furchner and A. P. Ginsburg [*Vision Res.* **18**, 1641 (1978)] to explain "monocular rivalry" of a compound wave form. The compound wave form consisted of a fundamental and a third harmonic.
6. The homogeneous raster of the CRT was changed to a 1 cycle/deg triangular-wave grating of the same mean luminance for 100 msec every 10 seconds for ten consecutive trials. When the screen became homogenous again, the observer reported whether the flashed grating appeared triangular or square. Observers reported that the flashed grating appeared square 20 to 80 percent of the trials, depending on the observer. Reversals were not experienced, as the exposure duration was too brief.
7. A square-wave grating can be represented by the Fourier series

$$\left\{\sin x + \frac{\sin 3x}{3} + \frac{\sin 5x}{5} + \ldots\right\}$$

and a triangular-wave grating by

$$\left\{\cos x + \frac{\cos 3x}{3^2} + \frac{\cos 5x}{5^2} + \ldots\right\}$$

In addition to a phase difference of the frequency components, the amplitude of the frequency components for the triangular-wave grating declined as a square of the reciprocal of the frequency from the fundamental.
8. Mean strength of the illusion was significantly reduced for all three subjects in both the direct

[R.B.: $t(84) = 4.01$, $P < .001$; L.E.L.: $t(78) = 3.10$, $P < .001$; M.S.: $t(124) = 9.32$, $P < .001$] and interocular [R.B.: $t(87) = 2.28$, $P < .025$; L.E.L.: $t(86) = 3.76$, $P < .001$; M.S.: $t(127) = 9.68$, $P < .001$] adaptation conditions. Subject R.B. showed a significant difference in the strength of the illusion in the interocular and direct adaptation conditions [R.B.: $t(55) = 2.47$, $P < .02$].
9. D. C. Burr, *Vision Res.* **20**, 391 (1980); K. K. DeValois, *ibid.* **17**, 209 (1977); L. E. Arend and R. V. Lange, *ibid.* **19**, 1089 (1979); C. F. Stromeyer, A. F. Lange, L. Ganz, *ibid.* **13**, 2345 (1973); L. Maffei and A. Fiorentini, *ibid.* **13**, 1255 (1973).
10. This research was supported by NIH grant EY01596, NSF grant BNS7817948, a career development award to R.B., and NIH postdoctoral fellowship EY05436 to L.E.L. We thank J. Zacks, A. Arditi, and W. Martens for helpful comments and assistance and M. Banks for earlier discussion.

6 November 1980; revised 19 January 1981

Electroencephalographic Tolerance and Abstinence Phenomena During Repeated Alcohol Ingestion by Nonalcoholics

Abstract. *Certain measures of the auditory average evoked response are sensitive to alcohol and provide evidence for abstinence and tolerance during and after 10 days of alcohol consumption by nonalcoholics. Electroencephalographic techniques provide a single sensitive measure for the study of the etiology of tolerance and abstinence with particular reference to a new area of investigation with nonaddicted humans.*

Mendelson (*1*) proposed that the pharmacological criteria of alcoholism most amenable to systematic investigation are tolerance and physical dependence. The study of these phenomena and the relationship between them has been extensive, but largely limited to infrahuman species or to humans already suffering from a long history of chronic alcoholism. A further restriction on such investigations has been the relative absence of techniques whereby both tolerance and physical dependence are assessed by comparable response indices. This led Begleiter and Porjesz to conclude that "to choose one technique to measure tolerance and another to assess physical dependence will continue to produce incomprehensible findings" (*2*, p. 356). They proposed the use of electroencephalography (EEG) as a remedy, since the same objective and quantifiable response measurements can be used to assess the effects on central nervous system excitability of both exposure to ethanol (*3*) and its removal (*4*). Our investigation is, to our knowledge, the first attempt to use quantitative EEG measures in a study of both tolerance and withdrawal in nonalcoholic humans.

Eight male volunteers (*5*) were informed of the purpose and risks of the study before consenting to participate. Subjects remained on our clinical research unit for the 16 days of the study. A battery of electrophysiological and psychological tests, administered twice daily, included spontaneous occipital EEG; vertex auditory average evoked potentials (AEP's) following 0-, 20-, 50-, and 90-dB tone stimuli; heart rate; hand tremor; postural sway; smooth pendular pursuit eye movements; verbal recall; and intoxication self-rating in that order. The order of testing remained constant throughout the study. The 16 days of the study were divided into three phases; the first 3 days were a preliminary baseline (baseline 1), followed by a 10-day alcohol phase, and a 3-day final baseline (baseline 2). On each day of the investigation, the test battery was administered twice, once before and once 20 minutes after the subject drank a beverage. During the baseline periods, the beverage was 500 ml of orange juice, and during the alcohol phase, alcohol (1 g per kilogram of body weight) mixed to a 10 percent solution with an orange juice vehicle. During the alcohol phase, breath alcohol was tested by a standard breath analysis machine immediately after the measurement of the auditory evoked responses. For six subjects, the beverage was consumed at 1400 hours, and for two, at 1000 hours. During the alcohol phase, after completing the second test battery, subjects consumed additional alcohol (0.9 g/kg) over 2 to 3 hours to keep blood alcohol concentrations elevated over a significant portion of each day. For these days, before-beverage testing occurred approximately 19½ hours after the completion of the previous day's alcohol ingestion.

From the spontaneous EEG, power spectral analysis techniques were used to derive estimates of the percentage of alpha, beta, and theta powers in addition

Table 1. Auditory evoked response means and standard deviations associated with 90- and 50-dB stimulus levels for the three phases of the experiment. Significance levels are for comparisons of before- and after-beverage scores (analysis of variance).

Measure	Baseline 1 (days 1 to 3)	Alcohol (days 4 to 13)	Baseline 2 (days 14 to 16)
		90-dB stimulus	
Variance (μv)^2			
Before	1.13 ± 0.52	1.41 ± 0.49****	1.17 ± 0.43
After	1.08 ± 0.63	0.98 ± 0.40	1.39 ± 0.44
ANR (dB)			
Before	−14.75 ± 2.34	−13.97 ± 2.30***	−14.54 ± 2.50
After	−15.63 ± 3.43	−15.97 ± 2.56	−14.02 ± 2.12
N_1 latency (msec)			
Before	111.25 ± 13.38	104.53 ± 8.79***	103.58 ± 11.23
After	107.63 ± 9.68	109.17 ± 10.10	105.00 ± 13.26
P_2 latency (msec)			
Before	182.54 ± 16.11	176.53 ± 15.29*	173.91 ± 14.35
After	175.08 ± 11.17	172.33 ± 14.31	175.67 ± 13.74
N_1P_2 amplitude (μV)			
Before	9.06 ± 2.63	10.20 ± 2.46***	9.13 ± 2.74
After	8.71 ± 2.82	7.72 ± 4.34	9.87 ± 2.88
		50-dB stimulus	
Variance			
Before	0.62 ± 0.32	0.71 ± 0.32***	0.64 ± 0.26
After	0.67 ± 0.33	0.53 ± 0.27	0.69 ± 0.29
ANR			
Before	−17.38 ± 2.46	−17.22 ± 2.30***	−17.37 ± 1.98
After	−17.82 ± 2.36	−18.76 ± 2.19	−17.18 ± 1.92
N_1 latency			
Before	120.58 ± 16.55	120.75 ± 14.32	118.75 ± 14.23
After	121.46 ± 11.91	124.81 ± 17.72	115.43 ± 13.37
P_2 latency			
Before	195.88 ± 16.04	191.69 ± 15.98	186.42 ± 16.05
After	190.71 ± 20.81	192.79 ± 21.19	188.87 ± 22.60
N_1P_2			
Before	5.59 ± 1.95	6.34 ± 1.87**	6.31 ± 1.54
After	5.63 ± 2.02	5.01 ± 1.89	6.51 ± 1.68

*$P < .05$. **$P < .01$. ***$P < .005$. ****$P < .001$.

0036-8075/81/0605-1175$00.50/0