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Habituation and the Orienting Response in the Auditory Cortical Evoked Potential

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ABSTRACT

Habituation of the auditory cortical evoked potential, the GSR, and heart rate was measured in 100 male subjects. Stimuli were 31 tones of 1 sec duration with an ISI of 33 sec. All stimuli were sinusoidal, at a frequency of 1000 Hz, and at an intensity of 95 dB (re 20 N/cm^2). The EEG was measured from bipolar electrode placement to the C_z and T_3 scalp locations. Evoked potentials were averaged over 3 successive blocks of 10 stimuli. The GSR habituation scores were the regression coefficients over trials of the response amplitude in square root conductance. Significant habituation of all evoked potential amplitude components was found. This was a rapid process occurring between the first and the second 10 stimuli. The habituation score for the most significant evoked potential variable (P200) was found to correlate significantly with the GSR and heart rate habituation scores. These results were interpreted as suggesting that the amplitude of the evoked potential was enhanced by the orienting reaction.

DESCRIPTORS: Evoked potential, GSR, Heart rate, Habituation, Orienting response.

Ritter, Vaughan, and Costa (1968), using a novel experimental design, identified several types of habituation phenomena within the auditory cortical evoked potential. Stimuli were presented in successive blocks of 30 with short interstimulus intervals (ISIs) of 2 sec within blocks and with long ISIs (5 min) between blocks. With this design they were able to average the stimuli both within a block and over the same stimulus positions of the different blocks. They found that the major habituation effects took place in the amplitude of the P200 component. Within the blocks, habituation of this component was initially very rapid for the first one or two stimuli, but was nonsignificant thereafter. Ritter et al. suggested that this habituation was an artifact produced by the short ISI within blocks. The effects of ISI on the evoked potential are well known (Davis, Mast, Yoshie, & Zerlin, 1966) and within this type of design the first stimulus is necessarily preceded by a long interval.

Habituation of this same P200 component across blocks was much slower. Ritter et al. argued that this slow habituation effect was similar to that found in the visual evoked potential by Bogacz, Vanzulli, Handler, and Garcia-Austt (1960), Walter (1964), and Perry and Copenhaver (1965). They further argued that, although its determinants were obscure, it could not represent orienting response habituation since it took too long to develop.

In other experiments, Ritter et al. interpolated unexpected stimuli into the stimulus sequence and observed a new positive component in the evoked potential at 300 msec. They argued that this P300 component was an aspect of the orienting response. They observed that there was no change in the P200 component in response to unexpected stimuli and therefore concluded that this component was independent of orienting behavior.

One way of investigating orienting components in the evoked potential would be to compare them with indices of orienting from other physiological response systems. The GSR response is probably the most widely used and validated measure of orienting. One problem, however, with relating the evoked potential to the GSR is that whereas averaging of many responses is necessary to produce reliable measures in the evoked potential, the GSR produces only a limited number of responses and more transient effects. It also needs longer interstimulus intervals to allow responses from previous stimuli to recover. For these reasons, a simple replication of the Ritter design would not work for concomitant measurement of GSR and evoked potential. In the present experiment, subjects were presented with auditory stimuli of fairly strong intensity at half min intervals. The strong intensity

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enabled fairly reliable evoked potentials to be obtained with averages of only 10 stimuli, at the same time producing enough GSR responses to calculate habituation scores in all subjects. Heart rate responses were also measured.

Method

Subjects

One hundred male subjects were tested. Of these, 84 were taken from a twin register compiled at the Institute of Psychiatry. Their mean age was 24.2 yrs, ranging between 17 and 44 yrs. The remaining 16 subjects were Institute staff. Genetic analysis of the twin data is reported elsewhere (Rust, 1975; Rust, Note 1).

Apparatus

The EEG was measured using a Mingograph EEG polygraph. Skin resistance was measured with an apparatus built in the department and described elsewhere (Venables & Martin, 1967, model b1). Electrodes were Ag/AgCl with a diameter of 9.04 mm. A saline lubricating jelly (Johnson and Johnson Ltd.) was used as the electrolyte. The EKG was measured from the right forearm to the left mastoid. EEG and skin resistance and EKG were all recorded on a tape recorder for subsequent computer analysis. Tonal stimuli were generated by an Audio Oscillator (SG65A Advance) and were presented binaurally through stereophonic headphones.

Procedure

The subject was seated in a soundproof dark room, and was asked to keep his eyes closed during testing. The subject was informed before the experiment about what to expect. He was told that he would be hearing about 30 tones, each of 1 sec duration through the headphones with an interval of about half a min between each. He then received 31 stimuli, each of 1 sec duration, at a regular ISI of 33 sec. All stimuli were sinusoidal, at a frequency of 1000 Hz, and at an intensity of 95 dB (re 20 N/cm^2). One channel of EEG was measured from bipolar electrode placement to the C_z and T_3 scalp locations. A time constant of 0.3 sec and a frequency filter of 70 Hz were used. The skin resistance was measured from the first and second fingers of the left hand.

Analysis of Psychophysiological Analog Data

For the EEG a Linc-8 computer was used to average the evoked potentials for the 500 msec following each stimulus. For each subject, averaging was carried out over 3 successive blocks of 10 stimuli (data from the response to the first stimulus was not included). There were thus 3 averages per subject.

The skin resistance was also scored on the Linc-8 computer using a system of programs developed by L. Law in the department (Martin, Levy, & Slubicka, 1975). A response to a stimulus was defined where a response onset was detected within 5 sec of stimulus onset. The amplitude of this response was then the difference between the resistance at the peak of this response and at its onset. This score for each stimulus was then transformed into square root conductance units (in μmhos) and a habituation score was calculated from the responses to the first 21 stimuli. This was the linear regression coefficient of response amplitude on trials. Failure to respond was treated as a response of amplitude zero.

The Linc-8 computer was used to transform the EKG data to a heart rate histogram. The largest and the smallest interbeat intervals (IBIs) in the 9 sec following stimulus onset were then found. Heart rate maximum and heart rate minimum responses were then calculated as the difference between these IBIs and the average IBI of the 5 sec before and the 20 sec after each stimulus. These scores were then transformed into bpm. The regression slopes of these scores over trials were then calculated for the first 21 stimuli to give measures of heart rate maximum response habituation and heart rate minimum response habituation.

The first evoked potential response was not included in the averaging since this response can easily be contaminated by muscle artifact. Some subjects tend to jump a little on hearing the first stimulus. There is no reason however to exclude this first response from the heart rate or GSR records since these are not contaminated in this way. In many ways the first GSR response is the most informative, and while some have argued that it is not appropriate to include it in the calculation of GSR habituation, attempts to adjust for it inevitably lead to contamination of the GSR habituation scores by other sources (Martin & Rust, 1976).

Results

With the stimulus paradigm used there was very little evidence for the existence of a separate P300 component. Between one-quarter and one-fifth of the subjects showed some evidence of two positive peaks between 150 and 350 msec after stimulus onset. Only four components were clearly present for all subjects. These included a large negative deflection at about 100 msec (N100) and a large positive deflection at about 200 msec (P200). The other two components were defined as P50, the largest positive deflection prior to N100; and N400, the largest negative deflection following P200. Latency and amplitude scores for these four components were measured. Amplitude scores were then transformed into absolute differences between successive positive and negative components. There were thus seven evoked potential variables for each average, these being P50, N100, P200 and N400 latencies and N100, P200 and N400 amplitudes. Latencies were measured in msec and amplitudes in μV.

For these seven evoked potential variables analysis of variance was carried out on the 84 twin subjects to test the significance of any habituation effects over the 3 successive averages. The results of this, together with the means, are given in Table 1. The first F-test shows the significance of the difference between the first average and the mean of the following two averages. The second test com-

TABLE 1

Habituation of the auditory cortical evoked potential

Variable	Means[a]			*F*-ratios (*df*=1/83)	
	Averages				
	a	b	c	a vs (b and c)	b vs c
P50 Latency	61.16	58.99	59.47	3.57	0.25
N100 Latency	113.60	112.40	113.18	0.66	0.90
P200 Latency	226.52	228.67	230.29	2.76	1.39
N400 Latency	407.61	406.59	409.41	0.04	2.72
N100 Amplitude	25.00	21.01	20.61	73.27*	0.90
P200 Amplitude	37.08	32.72	31.96	77.79*	2.96
N400 Amplitude	27.32	25.55	25.68	5.81*	0.11

[a]Latencies in msec, Amplitudes in μV.
*$p<.05$.

pares the second average with the third. It can be seen that the latencies show no significant change during the experiment. The amplitude scores on the other hand show very marked habituation, but this occurs entirely between the first and the second average. This amplitude habituation is significant for all three components, but is largest for the N100 and P200. Since these scores have the absolute N100 measurement in common it seems likely that it is this component which is primarily involved.

The mean amplitude of the GSR response was .724 square root μmhos with a standard deviation of .437. The mean habituation score was $-.0346$ (SD=.0216). Subjects gave responses on average to 17.8 of the 21 stimuli (SD=3.52). The habituation score for the most significant evoked potential component (P200) was then correlated with the GSR habituation score. For this analysis all 100 subjects were included. Evoked potential habituation was calculated as the difference between the second and first averages, which gives the best estimate of the regression slope given that responses have had to be averaged. The third average was not included so that both GSR and evoked potential scores were from the responses to the same stimuli (except that the first stimulus was included in the GSR score). The correlation was reliable, $r(98)=.22$, thus providing some evidence for a relationship between GSR amplitude and evoked potential P200 amplitude habituation.

Heart rate was measured only on the 84 twin subjects. The mean heart rate maximum habituation and heart rate minimum habituation scores were .044 and $-.043$ with standard deviations of .13 and .15 respectively. Both these habituation rates were significant and they had an intercorrelation of .74. The correlations of these two heart rate habituation scores with GSR habituation were only .09 and .03. However their correlations with the habituation score for P200 evoked potential amplitude were both reliable, $r(82)=.24$. Here again therefore we have evidence of a relationship of P200 evoked potential amplitude habituation with habituation in another psychophysiological system.

Discussion

It thus seems that rapid habituation is a characteristic of all the amplitude components of the evoked potential, the effect being strongest for the change in amplitude between the N100 and P200 components. The reason that these effects have not generally been found in the literature is probably that the large number of responses which are usually included in averaging swamps the effect. Ritter et al. (1968) also found this rapid habituation for the P200 component but concluded that it was an artifact of their short ISIs between stimuli within blocks. However, the results of the present experiment clearly show that the effect exists for ISIs as long as 33 sec and it is therefore unlikely that the effect is entirely due to short ISIs, although we would certainly expect these to contribute to the effect where ISIs are short. A closer look at the data presented by Ritter et al. suggests that the issue is not as clear cut as they imply even in their experiment. They have claimed that the P200 component showed a continuous slow 'habituation' over blocks, yet in their first experiment they found that the significant decrement in this component took place only between their first and their subsequent three blocks. There was no change after the first block.

One disadvantage of using more intense stimuli to overcome the problem of stability with averages of a few stimuli is that we cannot be sure whether the stimuli are producing orienting, defensive, or startle responses. The 95 dB stimulus intensity used in the present experiment is on the borderline between orienting and defensive responses, but closer to the orienting. The fact that the responses to the first stimulus were left out of the evoked potential scores makes it unlikely that startle responses were involved. The significant correlation of evoked potential habituation with GSR and heart rate habituation could mean that the former reflects either an orienting or a defensive response, or both. However it may be that this division of the possible responses into two types is rather arbitrary. At the stimulus intensity used the distinction is a theoretical rather than an empirical one. An interpretation in terms of orienting is in fact supported by the data from the first experiment of Ritter et al. where weaker stimuli were used.

It could be argued that the scoring method used

for the evoked potential components was such as to confound P200 with P300 where the latter occurred, and consequently that the results claimed for P200 were in fact due to the occasional occurrence of P300. Inspection of the data suggests that this is an unlikely cause of the effect. If it were so we would expect habituation to be shown for the latency as well as for the amplitude components, which is clearly not the case. We would also expect a correlation between P200 amplitude habituation and P200 latency habituation which again was not found (the correlation was only .05). Further it is notable that the habituation for N100 amplitude is larger than that for N400 amplitude, though only the latter could possibly be contaminated by a P300.

It is clear that in contrast to the claim of Ritter et al. (1968), the earlier amplitude components of the evoked potential as well as the later ones show an initial rapid habituation effect. There is also some evidence that there is some small but significant relationship between this habituation and that which occurs in the GSR and heart rate response systems. It seems reasonable to conclude from this that all aspects of the evoked potential amplitude, and not just P300, are affected by the orienting reaction.

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REFERENCE NOTE

1. Rust, J. Genetic sources of variation in electrodermal measures: A twin study. Manuscript submitted for publication, September 1975.

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