b-wave for all of the infants tested was 96 msec with the maximum white stimulus. It increased with decrease of stimulus luminance, a relationship significant beyond the .001 level of probability according to the results of a Spearman rank-order correlation. Mean latency of the infant *x*-wave was 57 msec. It did not change with stimulus luminance. These values are somewhat longer than those seen in adults.

The ERG of the newborn is highly differentiated. The components are similar to those of the adult ERG, but they are attenuated and modified in form. In both adult and infant the response depends not only on the intensity of the stimulus, but also on the wavelength. The electroretinogram was small in the infant; peak latencies of *a*-, *x*- and *b*-waves were slightly longer for the same stimulus luminance and the response seemed to be more rippled. But despite these differences, the wave pattern of the response is remarkably similar to that of adults (*14*). The first positive wave elicited in the infant by stimulation with orange light appears to be the same as the photopic or *x*-wave of the adult ERG because of its form, latency, and relationship to stimulation with red light.

The finding of the *x*-wave in the ERG of infants stimulated with light of long wavelength supports the assumption that photopic vision is functional in the newborn human. The finding of a *b*-wave suggests that the scotopic system also is active. Hence, both systems develop together at the retinal level. Our results may provide some electrophysiological support for behavioral studies of the development of responses to color and form such as those cited earlier.

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Auditory Habituation: A Test of a Centrifugal and a Peripheral Theory

Abstract. *Evoked potentials recorded at the cochlear nucleus of unanesthetized cats show that auditory habituation is a process with a rapid onset and a slower rate of dissipation, and that the habituation is a direct function of the rate of stimulation. Neither the middle ear muscles nor the reticular formation appear to participate in this process. It is suggested that auditory habituation might be a function of the interaction of the olivocochlear bundle and some intrinsic property of higher synaptic areas.*

A considerable body of evidence (*1*) indicates that some forms of afferent input are under centrifugal control. Studies of habituation of the evoked potentials recorded in the auditory pathway of unanesthetized animals also provide experimental support for this concept by indicating that habituation is under centrifugal control (see *2, 3*).

Another interpretation of auditory habituation which has been proposed is that known as the peripheral theory, according to which auditory habituation is mediated by the activity of the middle ear muscles. Guzmán-Flores (*4*) reports that the cutting of the middle ear muscles prevents habituation and the administration of curare abolishes habituation. Since the effective latency of these muscles (*5*) is far greater than the duration of stimuli usually employed, it is argued that habituation is produced by temporal conditioning of the muscles. The auditory stimulus acts as an unconditioned stimulus, and the interval between stimuli as a conditioned stimulus for the contraction of the muscles. An important implication of this theory is that a constant interval between stimuli is a necessary condition for habituation to occur. Another implication is that any change in this constant interval after habituation has developed should lead to dishabituation or a return to evoked potentials with amplitudes similar to those found before habituation. However, in one study (*6*) it is reported that habituation occurs in the auditory cortex despite bilateral cutting of the middle ear muscles. This observation, while not crucial, is not in strict agreement with the peripheral theory.

According to centrifugal theories, habituation is mediated by direct inhibition from a center in the brain. In one theory this role is allocated to the reticular formation, since barbiturate anesthetics will abolish habituation and extensive lesions in the mesencephalic reticular structures will prevent it (*2*). Habituation is also distinguished from adaptation or some fatigue process, since a change in the habituating stimulus pattern will produce an immediate dishabituation. This effect is regarded as a change in the animal's "attention," which is mediated by the reticular formation.

In another centrifugal theory it is argued that the direct inhibition comes from a cortical locus, but the experimental support for this concept is meager (*3*). According to this theory, a change in the stimulus pattern will produce dishabituation and an associated change in "attention."

The original evidence for the reticular theory was obtained from studies of habituation at the cochlear nucleus, but other workers (*7, 8*) were unable to find significant habituation in this region. However, more recently (*9*), habituation was reported to occur at the cochlear nucleus, but the decrements were considerably smaller than those of the original observations. In view of the conflict in the experimental data relating to both theoretical positions, the experiments reported here were designed to study habituation at the cochlear nucleus and to provide a test of both theories.

Fourteen cats were used. Bipolar electrodes were permanently implanted in the cochlear nucleus at Horsley-Clarke co-ordinates P 7.5, R 8.0, H −5.5 by a method previously reported

Table 1. A summary of the orthogonal trend analysis of the data on the rate of stimulation.

Source	Degrees of freedom	Mean square	Variance ratio (*F*)
Overall trend	10	0.0335	9.05 ($p < .01$)
Between treatment means	4	.5679	6.04 ($p < .01$)
Between treatment trends	40	.0079	1.98 ($p < .01$)
Linear trend	(4)	.0093	7.28 ($p < .01$)
Between subject means	4	.1615	
Subject by treatment means	16	.0940	
Between subject trends	40	.0037	
Subject by treatment trends	160	.0040	
Total	274		

(*10*). All placements were verified histologically after the experiment. After recovery from the operation, the animals were tested in a sound-proofed box (*9*). The habituating stimulus was a 20-msec burst of pulses (1000 per second) delivered by two 12.7-cm loudspeakers set in opposite walls of the test box. The pulse bursts were generated by wave-form and pulse generators and were of 85-db sound-pressure-level intensity (*11*). The "rise time" (*12*) of the stimulus was 10 μsec, which ensured a synchronous evoked response at the periphery (*13*). The variation in intensity within the range of movement of an animal in the box was ±1.5 db. The evoked responses were recorded by a pen recorder (*14*), and the peak-to-peak amplitude was measured. The first minute and then every alternate minute of recording was measured. A mean microvolt value was obtained for each of these minutes. This was then expressed as a percentage of a control value based on the mean microvolt figure for six stimuli given at random over a 5-minute period before testing. Each test session lasted 20 minutes, since previous work (*9*) had indicated that most of the habituation decrement occurred in this period.

Extraneous influences due to variation in the acoustic field (*15*) or to gross movements (*16*) were controlled by setting rigid criteria for the animal's position throughout a test session. The floor of the test box was schematically divided into four quadrants. A record was accepted only if the animal remained relaxed in one quadrant with its head 10 to 15 cm above the floor. The behavior of the animal was monitored continuously through one-way glass and any part of the record influenced by movement was discarded. Two cats anesthetized with sodium pentobarbital (60 mg/kg) and each with a monopolar electrode placed near the round window were used as a microphone to study the variation in the acoustic field. Oscillographic observations made with these animals in a range of positions similar to those of the awake animals revealed variations in the N_1 and N_2 components and in the cochlear microphonic of the round-window recording which reflect the physical variation of ±1.50 db (*17*).

In the first experiment we studied habituation in five cats as a function of five rates of stimulation: one stimulus every 20 seconds (1/20 seconds), one stimulus every 10 seconds (1/10 seconds), one stimulus every 5 seconds (1/5 seconds), one stimulus every second (1/1 second), and then 10 stimuli per second (10/1 second). All cats were tested at each of these rates of stimulation given in random order to each cat. The data were scaled (90 percent = 0.90), punched onto cards, and statistically analyzed by a modified form of Grant's orthogonal trend analysis (*18*) on a 1620 IBM computer. The results show that the degree of habituation is a direct function of rate of stimulation (Fig. 1*a*), since a significant difference was revealed between amplitude means and between linear trends of the treatments (Table 1). Three rates of stimulation (1/20 seconds, 1/10 seconds, 1/5 seconds) did not show significant habituation since they do not differ from zero trend according to Scheffe's criterion (*19*). The trends were similar in all the animals studied. The evoked responses were often monitored on an oscilloscope and a number of responses were photographed and measured. These data confirmed the trends apparent in the pen records. The decrements were not a function of an orienting response to stimulation, since this was seldom observed. This response occurred sometimes when the faster rates were introduced, but such orientation or "attention" toward the loudspeakers did not influence the rate of growth of the decrements. It can be concluded that "attention" to the stimulus is not a necessary condition for the rate of growth of habituation.

The rapid onset of this process is in agreement with our earlier observations (*9*) and with the general conclusion of Harris (*20*) that all forms of habituation show a negatively accelerated growth rate when plotted as a function of

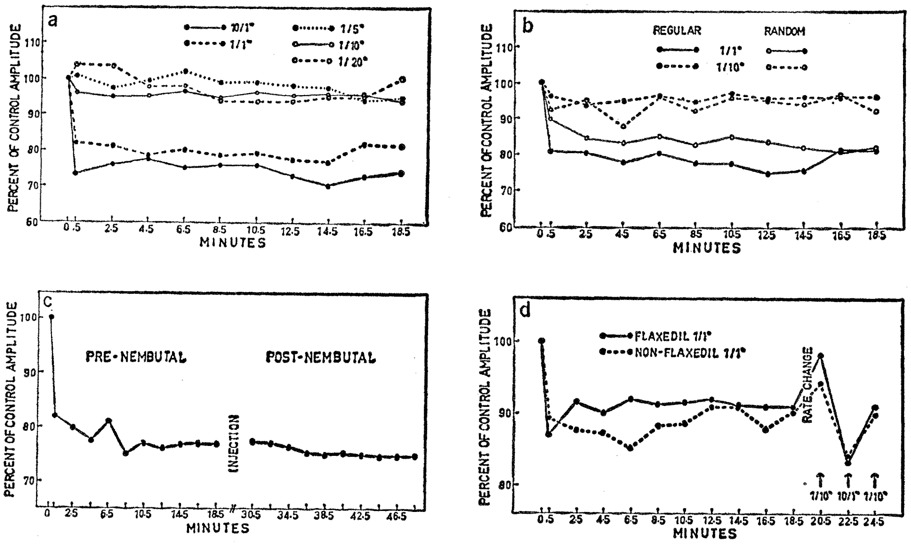

Fig. 1. The effects of repetitive stimulation on the amplitude of evoked potentials recorded in the cochlear nucleus of cats. (*a*) The relationship between the rate of stimulation and degree of habituation. Each point represents a mean figure for five animals. (*b*) The relationship between regular and random stimulation and degree of habituation. Each point represents a mean figure for four animals. (*c*) Effect of sodium pentobarbital on degree of habituation. Each point represents a mean figure for five animals. (*d*) Effects of Flaxedil and change of rate of stimulation on degree of habituation. Each point represents a mean figure for three animals. The intensity of all stimuli in (*a*) to (*d*) is 85 db relative to 0.0002 dyne/cm².

time, although the rapidity of growth depends on the system being studied. Previous studies of auditory habituation could not have revealed this rapid onset since they either did not record continuously or their units of time were too coarse (*1–3*, *8*).

Four cats were used for testing the role of a constant interval between stimuli. Two types of intervals were generated. One was the constant or regular interval already employed, and the other consisted of a set of stimuli with random intervals between them. Two restrictions were placed on randomization, in that each random set had an average rate equal to a regular stimulus rate of either 1/1 second or 1/10 seconds, and that no random interval was less than 280 msec. No significant difference was found between the degree of habituation produced by random and regular stimulus intervals (Fig. 1*b*). Thus a regular rate of stimulation is not a necessary condition for habituation.

The third experiment was a test of the reticular theory. Five cats were habituated for 20 minutes with a regular stimulus rate of 1/1 second. At the end of this period, each animal was injected intraperitoneally with a dose (60 mg/kg) of sodium pentobarbital. All reflexes were abolished 12 minutes after injection and the records were again read from this point. In contrast to previous reports, the administration of the drug did not abolish habituation (Fig. 1*c*). This result can be regarded as also bearing on the peripheral theory, since Carmel and Starr (*16*) have shown that similar doses of barbiturates block the reflex action of the middle ear muscles. Thus middle ear muscles appear unlikely to produce habituation by a tonic contraction.

A fourth experiment was designed to rule out the role of tonic contractions in the middle ear muscles and to determine whether any relation existed between rate of stimulation and dishabituation. Three cats were habituated for 20 minutes at a stimulus rate of 1/1 second. Each cat was tested twice, once under normal conditions and a second time 2 weeks later while immobilized with "Flaxedil" (*21*) and given artificial respiration. At the end of each 20-minute session a number of changes were made in the rate of stimulation. The rate (Fig. 1*d*) was decreased to 1/10 seconds and there was a consequent slow increase in response amplitude and partial dishabituation. The rate was increased to 10/1 second and the response amplitude immediately fell below the habituated level. The rate was then changed back to 1/10 seconds and the response amplitude again rose slowly. But it only rose to an intermediate level. This suggests that the 10/1 second rate had produced an effect which had a slower rate of dissipation than the effect due to the 1/1-second rate. Two other cats were tested without "Flaxedil" for a 20-minute period at the rate of 1/5 seconds. A very slight decrement occurred (2 to 4 percent), and then the rate was changed to 1/1 second with a marked consequent response decrement. The rate was changed back to 1/5 seconds but the response did not return immediately to the original amplitude. These results indicate that habituation is not produced by tonic contractions of the middle ear muscles and that "dishabituation" under these conditions is a function of the direction of change in stimulation rather than a function of change in itself. The animal's state of "attention" was not correlated with the changes observed. Furthermore, the results obtained with "Flaxedil" indicate that the degree of habituation is not an artefact of some change in position in the acoustic field.

From these data, it can be reasonably concluded that auditory habituation at the cochlear nucleus is a process with a rapid onset and a slower rate of dissipation and that the habituation is a direct function of rate of stimulation. Under the conditions employed, it does not appear to be dependent upon either temporal conditioning or tonic contractions of the middle ear muscles. The reticular formation does not seem to participate either directly or indirectly (*22*), since barbiturates fail to abolish habituation. Dishabituation appears to be a function of direction of change in the stimulus pattern, an observation which is difficult for all theories of auditory habituation.

While our observations on dishabituation would not be predicted by the cortical centrifugal theory, our data do not provide a crucial test of this theory. Furthermore, a report (*23*) of habituation in the N_1, N_2 responses to stimuli presented at the rate of 1/3 seconds provides some support for this theory, since the olivocochlear bundle is regarded as being part of this inhibitory circuit. However, it is also possible that the olivocochlear bundle is a local inhibitory circuit which acts independently of the cortex. Our earlier observations (*9*) that the degree of habituation increases as a direct function of a recording site being further along the auditory pathway suggests that as well as the olivocochlear bundle, other synaptic processes could be involved (*24*). The precise relationships comprising such an interaction remain to be determined, but we feel that the present results give some support to the contention of Sharpless that "it is difficult to believe that some highly specialized apparatus in the highest centres of the brain is necessary for habituation. The process is too pervasive" (*25*).

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