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CHANGES IN AUDITORY EVOKED RESPONSE INDUCED BY ALCOHOL

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INTRODUCTION

There has been a long-standing interest in and concern about the effects of alcohol on perception. This is related to the problem of hazards resulting from alcohol intake, in addition to an interest in the mechanisms of alcoholism and its complications. Anecdotally, it is not unusual for people who suffer from some hearing deficit to describe a subjective experience of further reduction of hearing after alcohol intake. To our knowledge, there have been no studies on the effects of alcohol on auditory perceptual activity in man. The averaged auditory evoked response appears to be a useful means of studying this problem. By the use of computer techniques it is possible to obtain a profile of the neurophysiologic concomitants of the auditory perceptual activity, utilizing as an intermediate step electroencephalographic recordings obtained at the scalp. These response profiles have been demonstrated to be meaningfully related in the waking state in man to the perceptual threshold, stimulus intensity, (4, 9) and levels of attention and vigilance (3, 6). In addition, the growing interest in the auditory evoked response and the effects of various conditions upon it leads naturally to a concern about the effect of alcohol. Nakai (8) demonstrated that in cats alcohol reduced the auditory evoked response obtained at the auditory cortex.

METHOD

Ten white, male college students with a mean age of 18.9 were studied. They were seated in an acoustic enclosure (Industrial Acoustics Co. №1203A) directly beneath a KLH №19 speaker, approximately 30 inches below the face of the speaker. The speaker was driven by a rectangular pulse of 0.2 millisecond duration, via a Bogen amplifier (MV 30A) whose gain was fixed to yield a sound pressure of 90 db re 0.0002 dynes/cm^2 30 inches below the speaker in response to a 1000 cycle sinewave the peak-to-peak amplitude of which was equal to that of the rectangular stimulus. Monopolar determinations were obtained utilizing as the active lead an electrode placed 5 cm to the left of the midline along an imaginary line connecting the two external auditory meati. The combination of the two ear lobes formed the reference electrode. Consequently, the data obtained has polarity opposite to that obtained with bipolar recording. Resistances were kept below 5000 ohms.

The EEG data were recorded by means of the Grass Model 7 polygraph, and the model 7 wide band A.C. EEG amplifier high frequency cut-off filter was set at 75 cycles/sec, with the 60-cycle filter on. This effectively limited all electrical potential to below 60 cycles/sec and consequently acted as an equivalent to the "muscle filter" switch appearing on earlier Grass model polygraphs. The gain of the preamplifier was set to clip surges of activity greater than 100 μv referred to the input.

Computation of the average evoked potentials was accomplished by means of the Magnetic Drum Average Response Computer (MDARC) (11). The frequency response of the MDARC is approximately that between 3 and 50 cycles/sec. Hence the system response is effectively limited by the computer.

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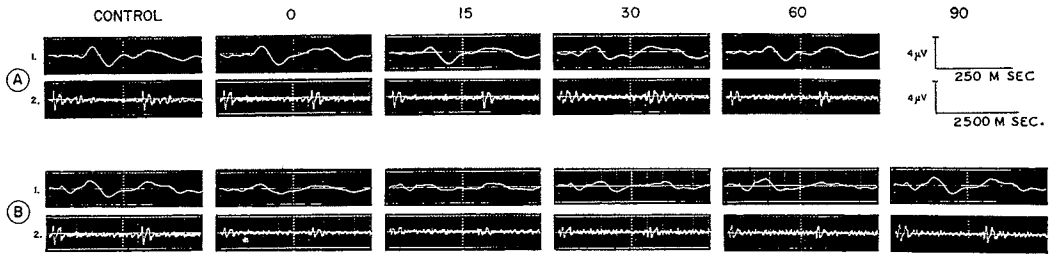

FIG. 1. Comparison of the effects of water and alcohol on the auditory evoked response for the entire subect group reveals a greater reduction with alcohol. It should be emphasized that time 0 is actually the response obtained following the five-minute ingestion time and the 6.8 minute data sampling period.

The averaged evoked response (AER) to 270 clicks was obtained on each of two consecutive days while the subjects were reading. Subsequently all subjects were asked to drink 100 cc of water with ice, or 100 cc of 90-proof whiskey with ice. One-half the group drank alcohol on the first day and water on the second; for the other half the conditions were reversed. The time for the ingestion of the water and the whiskey was kept at five minutes. Immediately after intake an evoked response to 270 clicks was obtained; this procedure was repeated 15, 30 and 60 minutes following ingestion.

Little has been written of the explicit criteria for the measurement of the characteristics of the AER. Peak-to-peak, or peak-to-trough measurements, have been described by several authors for determining amplitudes of the AER; the latency of peaks and troughs has also been described by these authors for determining the temporal characteristics (2–4, 9).

Because of the complexity of the wave forms, the above criteria are often insufficient. For example, there is the problem of the configuration of those components which have negative peaks at approximately 60 and 160 msec. Most often their configuration is relatively simple and resembles what Giblin (5) alludes to in describing somatosensory responses as a V form; in others the configuration resembles what he has described as W form. An additional problem in measurement arises from the fact that occasionally there is a plateau instead of a peak.

On the basis of our experience we have evolved the following criteria: when a W configuration is present, the highest of the

TABLE 1

Amplitude Characteristics of the Auditory Evoked Response Obtained with Alcohol and Water

Amplitude	Agent	Run 1 Control	Run 2	Run 3	Run 4	Run 5	Run 6
			Time				
			0	15 min	30 min	60 min	90 min
A	Alcohol						
	Mean	8.8	7.3	5.3	4.9	6.5	7.0
	S.D.	3.4	3.4	1.7	2.3	2.2	2.2
	Water						
	Mean	8.8	8.3	8.1	8.3	8.4	—
	S.D.	1.9	2.4	2.6	2.8	2.4	
B	Alcohol						
	Mean	10.9	7.7	6.0	6.6	7.0	8.1
	S.D.	2.3	2.1	2.2	2.3	2.3	1.6
	Water						
	Mean	11.5	10.9	11.1	9.6	10.0	—
	S.D.	2.2	2.6	2.4	2.5	2.0	
C	Alcohol						
	Mean	9.6	7.2	5.6	6.5	6.5	8.0
	S.D.	2.4	2.1	2.5	1.8	1.8	2.2
	Water						
	Mean	10.4	9.4	9.9	8.5	8.7	—
	S.D.	3.3	2.2	2.7	1.6	1.9	
D	Alcohol						
	Mean	7.9	6.5	5.6	5.4	5.6	6.9
	S.D.	3.4	2.8	2.3	2.1	2.1	2.4
	Water						
	Mean	8.7	7.5	7.8	7.9	7.7	—
	S.D.	3.3	2.4	2.2	1.9	2.6	

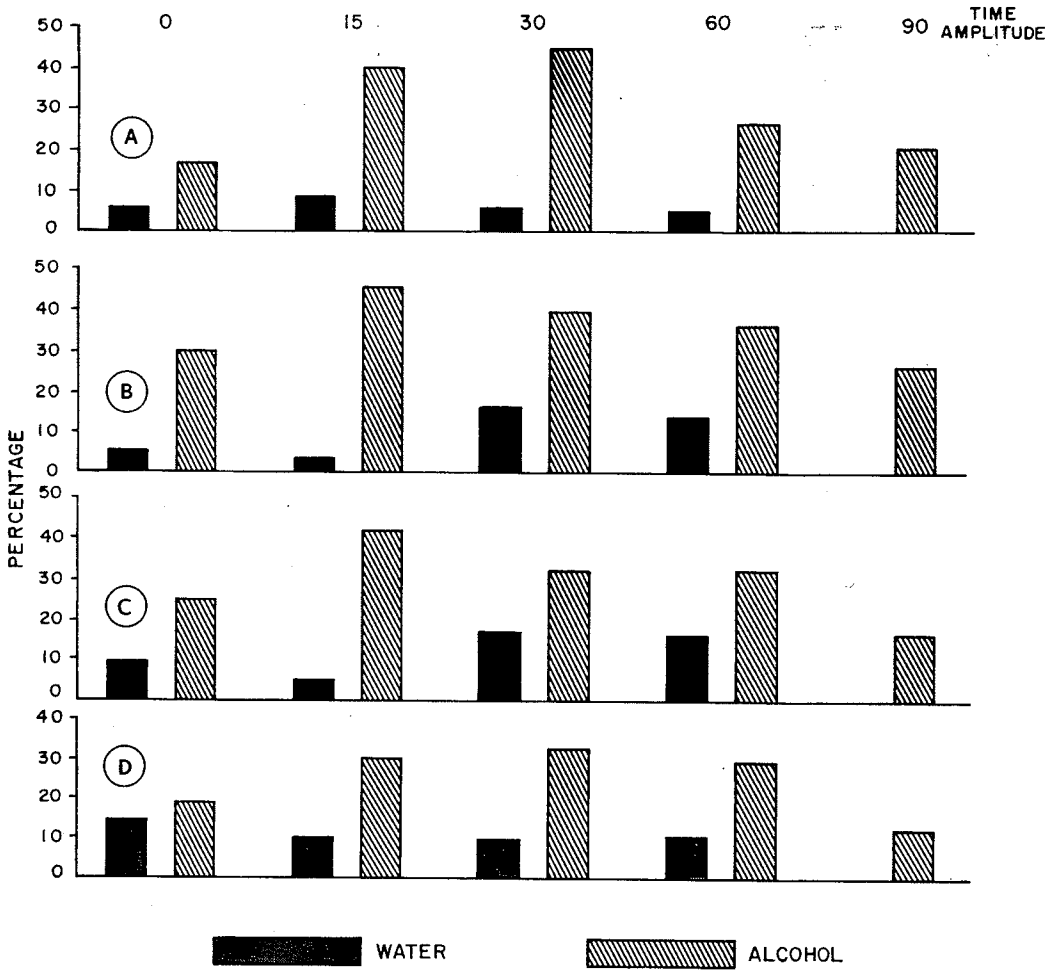

FIG. 2. Data collected from a subject demonstrating the striking difference between water and alcohol effect. (The upper series, demonstrating water effect, shows throughout the apparent absence of the 30 msec downward positive deflection which we have observed infrequently.)

two peaks is chosen for the amplitude and latency determinations of that component. This method has also been used to measure the evoked response to light (7). Where a plateau is present, the midpoint is chosen for latency determination.

With the above exceptions, the AER obtained is multiphasic and regularly consists of five components: a positive peak at 30 msec, a negative peak at 60 msec, a positive peak at 100 msec, a negative peak at 160 msec, and a positive peak at 220 msec. The times are approximate. (See Figure 1.) This yields four successive peak-to-peak amplitudes, measured in terms of the perpendicular distance between the successive peaks (designated A–D), a total duration of the response which corresponds to the interval from time zero to the positive peak at 220 msec, and four successive latencies measured from time zero (designated 1–4). (Bickford [1] has described a myogenic response, which on the basis of his findings accounts for the early peaks prior to 25 msec. Consequently, this activity is not measured in our studies. He has also de-

scribed contamination of the activity involved in the 30 msec positive peak. It is our impression that the utilization of a high-frequency cut-off minimizes this problem.)

RESULTS

The 24-hr. test retest reliability for the baseline AER was significant for all amplitudes, latencies and duration. The amplitude coefficients were: A = .66, B = .75, C = .70, D = .79. A, B, and C were significant at better than the .05 level; D was significant at $p < .01$. The coefficients for latencies were: 1 = .86, 2 = .79, 3 = .76, 4 = .83. All were significant at $p <$ 01. The coefficient for duration was .81, also significant at the .01 level.

The amplitude characteristics of the evoked response obtained with alcohol and water are summarized in Table 1. Analysis of variance, comparing the effects of both agents, was performed for each amplitude and yielded the following results. All amplitudes were significantly lower for alcohol than for water. Amplitude A, F = 6.45, $p < .05$; Amplitude B, F = 15.85, $p <$ .01; Amplitude C, F = 12.21, $p < .01$; Amplitude D, F = 4.64, $p < .05$. (The run means averaged over the two agents did not differ significantly.) The performance curves for the two agents differed significantly for amplitudes B and C at $p < .05$ and were not significant for amplitudes A and D. (See Figures 1 and 2.)

Individual mean amplitude comparisons were performed between control and runs following the ingestion of both agents. After alcohol ingestion: Amplitude A was significantly lower at 15 and 30 minutes; Amplitude B was significantly lower for all runs; Amplitude C was significantly lower at 0, 15, 30 and 60 minutes; Amplitude D was not significantly lower for any of the time intervals. There was no significant change of latencies or duration as regards means and standard deviations (Table 2).

Following water ingestion, the evoked response was not significantly different from the control at any time for all characteristics with the single exception of the variance of duration at 30 minutes.

TABLE 2

Latency Characteristics of the Auditory Evoked Response Obtained with Alcohol and Water

Latency	Agent	Run 1 Control	Run 2	Run 3	Run 4	Run 5	Run 6
			Time				
			0	15 min	30 min	60 min	90 min
1)	Alcohol						
	Mean	36.9	38.9	37.5	38.1	41.4	31.7
	S.D.	11.0	11.9	10.1	14.1	10.3	8.8
	Water						
	Mean	38.1	37.8	38.6	37.5	38.1	
	S.D.	8.4	9.9	11.7	8.4	10.6	
2)	Alcohol						
	Mean	66.7	66.4	63.3	70.8	69.4	69.7
	S.D.	12.2	11.7	10.8	9.4	10.1	10.7
	Water						
	Mean	69.4	71.8	69.7	71.4	67.5	
	S.D.	11.3	10.3	10.7	9.1	10.1	
3)	Alcohol						
	Mean	110	106.9	104.7	115.5	105.6	108.9
	S.D.	11.8	10.3	10.2	12.8	13.1	11.5
	Water						
	Mean	107.2	107.8	107.2	108.7	104.2	
	S.D.	10.2	7.9	9.8	8.4	8.0	
4)	Alcohol						
	Mean	178.4	172.2	175.6	180	173.9	173.1
	S.D.	15.9	16.5	11.4	9.9	16.4	9.7
	Water						
	Mean	174.7	178.3	176.1	177.8	175.6	
	S.D.	15.0	13.8	10.1	12.8	15.2	
Duration	Alcohol						
	Mean	228.6	224.7	229.2	231.1	225.3	225.3
	S.D.	10.9	14.1	12.1	18.3	13.1	24.5
	Water						
	Mean	228.3	226.9	235.8	236.7	231.9	
	S.D.	15.4	14.6	15.4	9.4	18.5	

DISCUSSION

The findings establish that alcohol significantly reduces the AER in man. The data do not provide information as to the site of action. Findings regarding the nature of the visual evoked response indicate that the early activity is related to the primary sensory pathways and the later activity is related to conduction via the

reticular formation (10). Studies by Ciganek suggest that the visual and auditory evoked responses are analogous in configuration; he formulated the hypothesis that the early and late activities of the auditory evoked response have origins analogous to those observed in the visual evoked response (2). If this proves to be the case, then the alcohol effect on the auditory evoked response will involve reduction of activity along both pathways. Whether this has a separate effect on each, or a primary effect on the end organ, or on an integration mechanism, remains to be determined.

Additional studies will be necessary to determine the behavioral significance of the alcohol effect on the AER. The significant amplitude reductions induced by alcohol ingestion which are consistent with those observed in the reduction of stimulus intensity and in the reduction of levels of attention probably reflect, insofar as a psychic event is concerned, a reduction of input of auditory information into the perceptual and cognitive systems. This is in keeping with daily occurrences in which we become aware of external stimuli being experienced as fluctuating in levels of intensity, depending on the degree to which we attend to them. The fact that it is not experienced as change in the actual external stimulus is probably the result of a "corrective cognitive function."

SUMMARY

Alcohol affects the auditory evoked response in man. All amplitudes are significantly reduced with maximal effect observed at 15–30 minutes after ingestion. In the dosage used there was no significant change in the latencies.

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