Pharmacology Biochemistry and Behavior, Vol. 3, pp. 195–200. ANKHO International Inc., 1975. Printed in the U.S.A.

Marijuana Influenced Changes in GSR Activation Peaking During Paired-Associate Learning[1]

MICHAEL J. COHEN[2] AND WILLIAM H. RICKLES

Department of Psychiatry, UCLA School of Medicine and Veterans Administration Hospital, Sepulveda, California

AND

BRUCE D. NALIBOFF[3]

Department of Psychiatry, UCLA School of Medicine

(Received 8 July 1974)

COHEN, M. J., W. H. RICKLES AND B. D. NALIBOFF. *Marijuana influenced changes in GSR activation peaking during paired-associate learning.* PHARMAC. BIOCHEM. BEHAV. 3(2) 195–200, 1975. – Activation Peaking (AP) refers to a patterned physiological response occurring during learning. Marijuana has been found to interfere with both paired-associate learning and phasic GSR activity. Therefore, a study was performed to assess the effects of marijuana intoxication on paired-associate learning and concomitant GSR AP. Two marijuana usage categories were employed – light and heavy usage Ss. Within each category four groups were run in a design to test state-dependent effects. Each S was seen twice with a seven-day inter-session interval. The groups were P-P, P-M, M-M and M-P with P = placebo and M = 14 mg Δ^9 THC. At each session S learned a nine-word paired-associate list to a criterion of one correct recitation, and then received 100 percent overlearning. No usage or group differences were found in level of basal conductance, except lights showed habituation over sessions and heavies did not. Magnitude of phasic GSR activation, aligned for AP, was significantly reduced for both heavy usage and marijuana intoxicated Ss. Also, only on placebo days was an AP effect evident. The results were discussed in terms of marijuana's effects on learning and physiology with emphasis on possible mechanisms of action.

Marijuana Paired-associate learning GSR Skin conductance Activation-peaking

THE effect of marijuana intoxication on neurophysiologic systems associated with arousal and attention appears to be a complex interaction involving the task, set and setting, among other variables. A simple depressant or stimulant effect cannot account for the various results generated by studies of marijuana's influence on arousal and attention. Under the condition of moderate marijuana intoxication, researchers found fluctuating levels of vigilance [21,32]. Subjectively, experiences of heightened sensual sensitivity and increased meaning have been reported [28]. Several tonic central nervous system (CNS) measures suggested increased arousal associated with marijuana intoxication. For example, spectral analysis of the EEG revealed that intoxicated Ss had energy displacement toward higher frequencies: also an S's characteristic frequency of the alpha rhythm was speeded by 1.0 Hz [9,10]. The contingent negative variation (CNV) is augmented by marijuana, while comparable doses of alcohol depress the magnitude of the CNV [16]. Marijuana's effect on the evoked potential is, as yet, unclear, with both augmentation and attenuation reported. [17, Roth, Galanter, Weingartner, Vaughan and Wyatt, unpublished.] Marijuana appears to leave the motor

[1]This study was supported by NIMH Grant 5 ROI MH 19488-02. Computing assistance was obtained from the Health Science Computing Facility, UCLA, sponsored by NIH Special Resources Grant RR-3.

[2]Send reprint request to Michael J. Cohen, Ph.D., Clinical Psychophysiology Laboratory, Veterans Administration Hospital, Sepulveda, CA 91343. Appreciation is expressed to Keren McIntyre and Charlotte Whitaker for assistance in data gathering and analysis.

[3]Now at Department of Psychology, Bowling Green University, Bowling Green, Ohio 43403

system relatively unaltered in humans [22]. Aside from the well-documented tachycardia, reddening and congestion of the ocular conjunctiva and hypothermia [5, 11, 17], marijuana does not have a major influence on autonomic nervous system (ANS) basal levels.

Psychophysiological studies of attention and arousal have concentrated on the orienting response, basal activity, and conditioned responding. Germana [6] pointed out that activational responding associated with the formation of a conditioned response has had wide replicability, but little recognition among interested scientists. Germana termed the above phenomenon activation peaking (AP). As a stimulus takes on the properties of a conditioned stimulus the magnitude of the galvanic skin response (GSR) increases systematically to some learning point. Additional exposure to the learning situation result in successive decrements in the activation measures [2, 4, 7, 12]. The AP phenomenon probably can be seen in other autonomic nervous system innervated organs, as well [1,6].

Perusal of the literature relating marijuana intoxication to behavioral activity indicated that intoxication interfered with learning as measured by several performance variables [25, 33, 34]. These studies also suggested that past usage history with marijuana was a significant factor in accounting for the behavioral effects associated with the drug. Apparently, performance decrements were less severe as usage increased [3,34]. The study reported below assessed the effects of marijuana intoxication and past usage history of marijuana on a paired-associate learning task. Measures of GSR AP were concomitantly recorded to see if performance changes induced through marijuana intoxication were accompanied by correlated changes in AP.

METHOD

Subjects

All Ss were male, paid volunteers, ranging between 18 and 27 years of age. Initially, Ss were screened with the Minnesota Multiphasic Inventory (MMPI) and the Wechsler Adult Intelligence Scale (WAIS). Current norms for college students for the MMPI and scores of 110 to 140 on the verbal scale of the WAIS were used in subject selection. Aside from hashish, no other drugs were used with regularity. Thirty-two Ss from our light usage category were selected. Light usage was operationally defined as smoking not more than 3 times per week and not less than once per month. These Ss smoked primarily on weekends and in the company of others. We considered this group to be social users. Thirty Ss of heavy usage were also selected. Heavy usage was defined as smoking 4 or more times per week. These Ss reported going to school and/or work while intoxicated and also smoked marijuana while alone. Subjects were assigned to groups on a random schedule with the stipulation that, within each usage group, all cells contained n Ss before any cell contained n + 1 Ss. This stipulation insured the equal distribution of Ss able to learn the task as well as protected the integrity of the double blind.

Design

All Ss were seen on two occasions, seven days apart. The basic design was a 2 X 4 X 2 X 6 split-plot with two repeated measures, ([15], p. 311). The between-groups treatment conditions were usage, (either light or heavy) and drug groups. The four drug groups were placebo–placebo (P–P), placebo–marijuana (P–M), marijuana–marijuana (M–M) and marijuana–placebo (M–P). These four groups represent the conditions needed to test for state-dependency of learned materials. One repeated measures treatment was the two days of the study. The other repeated measure represented six data points aligned and analyzed for activation peaking. The data points are explained below under the heading of dependent variables.

Apparatus and Physiological Measures

Upon ariving at the laboratory, the subject was instrumented for recording GSR and EKG on a Beckman Type R polygraph. The EKG was analyzed only for the well-documented increases in heart rate associated with marijuana intoxication. The GSR was recorded monopolar with a 2 cm Ag/AgCl electrode placed in the center of the right palm. The volar surface of the right forearm was skin drilled and served as the inactive site [23]. Current density was 9 $\mu A/cm^2$ of skin area and 0.1 N NaCl electrode gel formed the interface between the skin and electrode. (The electrode jelly was provided by Mr. Jack Day of the Manned Spacecraft Center, NASA, Clearwater, Texas.)

Procedure

For one week prior to testing, and during the inter-session interval, S was encouraged to remain drug-free. Following electrode attachment, 5 min of baseline activity was obtained. Then S smoked two "marijuana" cigarettes. On placebo days each cigarette contained 1000 mg of exhaustively alcohol-extracted marijuana. On marijuana days, 500 mg of marijuana was placed in the center of the cigarette and 250 mg of placebo was flanked on each end. The marijuana contained 1.4% Δ^9 THC; therefore, 14 mg of Δ^9 THC was available to the S. Smoking was by deep inhalation and hold and S was given about 20 min to complete smoking. Approximately 50 percent of available THC is burned in the cigarette, or left in the roach, so an average S inhaled about 7.0 mg THC [13, 14, 30]. Double blind conditions were maintained throughout the study.

After smoking, five additional minutes of baseline physiology were recorded, followed by an orienting sequence of ten 500 Hz, 75 Db tones, each presented for 1.0 sec. At the conclusion of the orienting sequence, instructions were given for learning a nine-item paired-associate list. Each item consisted of a nonsense syllable (CVC trigram) and a common English word. The stimuli were transmitted via a Kodak 850 Carousel projector, located in an equipment room, through one-way glass into the separate S's room. The nonsense syllable was presented for 1.0 sec, followed by 0.5 sec of blank screen time, while the projector changed slides, and the word was then available for 1.0 sec. Thus, the inter-pair interval was 1.5 sec. With each slide change the 1.0 sec orienting tone was sounded. This was done both to mask the noise of the projector changing slides and to alert S to be attentive to the new slide pair. Thusly, the initial orienting sequence was conducted to stabilize S's physiology to the alerting tone. The word pairs were programmed on a fixed inter-trial interval of 20 sec. The list was presented in three random rotations until attainment of criterion learning. Criterion learning was defined as correctly anticipating all nine words within one complete rotation and within the

1.5 sec inter-pair interval. Once criterion was reached it was immediately followed by 100 percent over-learning, i.e., the total trials given to a S was twice the number needed to attain criterion learning.

On Day 2, immediately preceding the learning phase, S was presented the nine, Day 1 nonsense syllables for one trial and asked to recall the words that had been associated with them. The nonsense syllable was presented for 1 sec, followed by 20 sec of blank screening and S was given the full amount of time to respond. After this phase, S was asked to learn a second list of words, consisting of both new nonsense syllables and new words, equated to List 1 in difficulty of pronunciation, frequency of occurrence in daily usage, and meaningfulness [29,31]. If, on either day, S failed to reach criterion within 15 trials, he was dropped from the study. This was done to assure learning occurred under the acute intoxication of marijuana.

Dependent Variables

Measures of basal skin resistance were obtained during the following phases of the experiment: 5 min presmoking period, 5 min postsmoking period, the first trial of learning, and for 5 min following the over-learning. Phasic GSR activity was measured for each presentation of the nonsense syllables. The minimum acceptable response was a change of 500 ohms initiated within the range of 0.5 to 2.0 sec from onset of the nonsense syllable. A 1.5 sec range for initiating a GSR was utilized in hopes of assuring that these GSRs were generated to the nonsense syllable and were not response produced, i.e., caused by S verbally saying the word. However, it is possible that the magnitude of some GSRs were produced by the confounding of the perception of the nonsense syllable and the verbal response to it. For both basal and phasic activity, resistance scores were converted to conductance and square root transforms of the conductance data were used in analyses. Hereafter, phasic GSRs will be referred to as skin conductance responses (SCRs). Skin conductance activity was aligned for analysis of AP, (see [6]). Six peaking points were selected: Point (1) the average of the nine SCRs to the first presentation of the nonsense syllables; (2) the SCRs prior to the first correct response, but not including responses assigned to Point (1), averaged over all nine words; (3) the average of first correct responses, i.e., the average of the SCRs occurring on the trials that each word was correctly anticipated for the first time; (4) the average SCRs occurring on the last error +1 trial. Last error +1 trial was defined as the trial at which four successive correct responses were initiated, again this was the average for all nine words; this point contains some of the points in (3) whereby the subject made no errors on a word after the first correct response. Point (4) defines a learning criterion for words which are correctly anticipated and later missed; (5) the average of the SCRs to all trials after (4) but exclusive of the last trial; (6) average of the nine SCRs to the last presentation of the nonsense syllables. It is important to remember that AP alignment is done for individual Ss so that physiological responding for all the words are aligned at some common definition of learning.

RESULTS

On Day 1, 12 Ss failed to reach criterion learning. Eleven of these Ss were intoxicated with marijuana. Of these 11 Ss, 7 were light users and 4 were heavy users. On Day 2, 2 light and 1 heavy user did not attain criterion learning. All were marijuana intoxicated. For various reasons (see [3,25] for details), 2 more light users and 5 additional heavy users were dropped from the study. About half of these Ss were placebo intoxicated. The Ss successfully completely the task were divided equally among the four drug conditions and the two usage categories, so that there were 5 Ss in each of the 8 cells.

Details of the behavioral data can be found in [3] and [25]. Briefly, light usage marijuana intoxicated Ss required significantly more trials to reach criterion than light usage placebo Ss on both days. Also, recall of Day 1 learning was found to be state-dependent. Those Ss changing drug state from learning to recall remembered about 30 percent fewer words than Ss re-entering the same state. Heavy usage Ss showed no difference in trials to cirterion, between drug states, and accuracy of recall was not found to be state-dependent. Regardless of drug state, the heavy usage Ss' trials to criterion data looked more like the data for the light marijuana intoxicated Ss, compared to the light placebo Ss.

Basal Conductance

No main effects were found on the basal conductance data for usage, groups or days. There was a significant effect over phases of the experiment, $F(3,96) = 19.50$, $p<0.001$. Also of interest was a significant Usage X Days interaction, $F(1,32) = 4.54$, $p<0.05$ and a Usage X Phases interaction, $F(3,96) = 5.40$, $p<0.002$.

Post hoc tests for simple main effects on the above interactions yielded the following information: On Day 1, lights were significantly higher on basal conductance than heavies with no difference between usage categories on Day 2. Also, light usage Ss showed significantly higher basal activity on Day 1 compared with their Day 2 levels, while for the heavies there was no difference between days. Light usage Ss were significantly higher than the heavies on basal conductance during the postsmoking, learning, and post-learning phases, but both usage category Ss showed activation from presmoking levels to all other phases.

Activation Peaking

Analysis of the AP alignment indicated a significant main effect of usage on the magnitude of the SCRs, $F(1,32) = 21.67$, $p<0.001$. Heavy Ss had smaller magnitude SCRs than the lights. (See Fig. 1) There was also a significant main effect of groups, $F(3,32) = 4.51$, $p<0.01$. Post hoc analysis showed that the P–P group had a significantly higher magnitude of responding than the M–M group. There were no other reliable differences among the groups. The main effect of peaking points was also significant, $F(5,160) = 14.22$, $p<0.001$. Post hoc trend analysis indicated significant linear and quadratic components. The only significant interaction effect was Days X Groups X Peaking Points, $F(15,160) = 1.76$, $p<0.05$. Tests of simple-simple main effects were performed. It was found that significant AP levels occurred only on placebo days. (See Fig. 2) That is, significant F tests were found only for the data generated by the P–P group on Days 1 and 2, the P–M group on Day 1 and the M–P group on Day 2.

Recall

Analysis of SCRs to the nonsense syllables, presented for

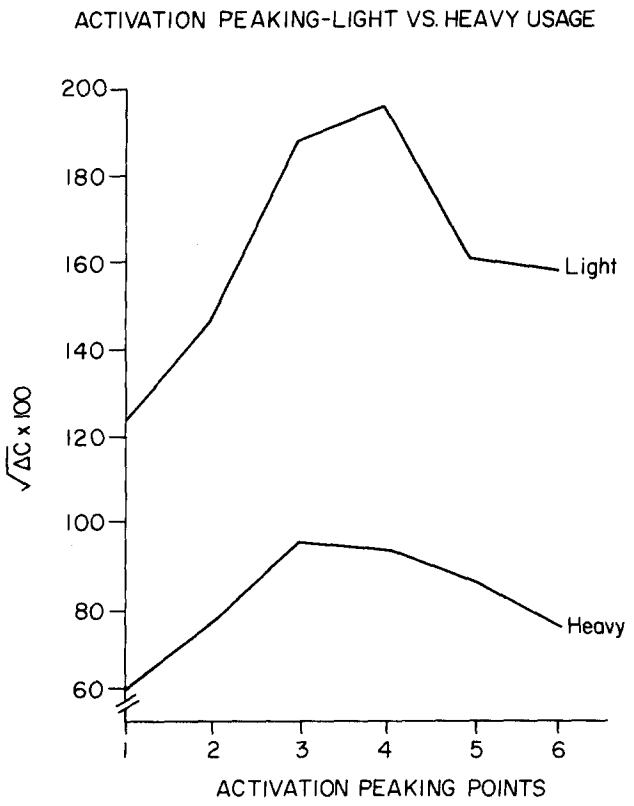

FIG. 1. Magnitude of SCR Activation Peaking for subjects of light and heavy past marijuana usage.

recall of Day 1 learning, yielded no significant effects of state change on the magnitude of responding. In essence, physiological responding, as represented by the SCR, showed no state-dependent effects.

DISCUSSION

Both behaviorally and physiologically, chronic marijuana users, whether intoxicated or not, looked more like their intoxicated social counterparts than like the nonintoxicated social users. For a discussion of the behavioral data see [3].

Evaluating tonic SCR activity indicated no acute effects of marijuana on basal conductance but several differences between the social and chronic usage populations were evident. The social marijuana users were, initially, more aroused on Day 1 compared to the chronic users, and the social users showed habituation on Day 2. The chronic users were, initially, at lower levels of arousal and had no significant change in basal conductance levels from Day 1 to Day 2. In addition, looking at the separate phases of the experimental session, one sees that social users' arousal levels were increased above initial baseline for all other phases, while chronic usage Ss had a much smaller effect. The basal conductance data are in agreement with West [35], who suggested that heavy usage of marijuana led to a rather complacent individual. West termed this phenomenon the amotivational syndrome. The chronic users in our study did not have the physiologic indicants of anxiety that are usually seen with Ss placed in a new and rather intimidating situation. With history of drug use being an ex-post-facto variable, it could not be determined if marijuana led to the development of an amotivational syndrome, or some underlying personality variable accounts for both the syndrome and marijuana usage. The patterning of basal activity is suggestive of Roessler's [26] work showing that people with high ego strength are initially more anxious, explore the new situation and subsequently become less anxious (social users?). Low ego strength individuals start out either high or low anxious and remain so (heavies?).

The analysis of the AP data yielded two basic results of interest. First, chronic usage Ss had diminished SCR magnitude during the learning task, compared to social users. Second, regardless of past history, acute marijuana intoxication also resulted in depressed levels of SCR activity. As was the situation with the basal data, the usage effect on SCRs during learning is difficult to assess. Was the severe diminution in responding indeed related to long-term use of marijuana, or was this physiologic phenomenon a correlate of some underlying personality variable? Cohen and Rickles [3] previously suggested that the chronic users seemed to be less task-oriented, as assessed within the experimental situation. SCR magnitude correlates with speed of learning [18,19]. Our chronic users, compared to the social users, had depressed SCRs and the nonintoxicated chronic Ss required more trials to attain criteria than their social user counterparts. A methodological consideration needs to be brought out. Of the 15 Ss unable to learn to criterion, 14 were marijuana intoxicated. It is possible that the obtained results, especially that marijuana reduced SCR magnitude, could be an artifact of S selection. That is, anxious Ss may have had more difficulty with the task and with the added burden of marijuana intoxication, failed to learn. This might mean that successful marijuana intoxicated Ss were less anxious than their placebo counterparts.

However, the marijuana-induced reduction in SCR AP is consistent with earlier investigations concerned with the relationship between marijuana and the SCR ([17], Rickles, Cohen & Klitzner, Unpublished). Rickles, Cohen and Klitzner examined the magnitude of the SCR to orienting stimuli during the dynamic processes of habituation, dishabituation and rehabituation. Marijuana consistently reduced the overall response level but did not appear to effect the dynamics. Although the intoxicated Ss' SCRs were severely reduced, compared to nonintoxicated Ss, when the phasic SCRs were converted to individual ratio scores, habituation to repeated presentations of the orienting tone occurred; the magnitude increased when the tone was given signal qualities; and the SCR again showed habituation when the signal qualities were removed for a group of chronic usage Ss. Low, Klonoff and Marcus [17] studied the influence of high (9.1 mg THC), low (4.8 mg THC) and placebo doses of marijuana on several CNS and ANS measures. They reported that SCR reactivity decreased for both high and low dosage groups compared to the placebo group. However, there seemed to be no differences in SCR magnitude between the high and low dosage groups. With the present study the same phenomenon obtains. Examination of Fig. 2 showed that while on marijuana days responding was smaller, the basic quadratic function remained intact. The curves for marijuana and for placebo days are generally of the same shape, but with a substantial difference in magnitude.

What inferences may be drawn regarding the mechanisms of action on the SCR as an index of arousal in a learning situation? The depression of SCR responding without similar change in response dynamics suggested that

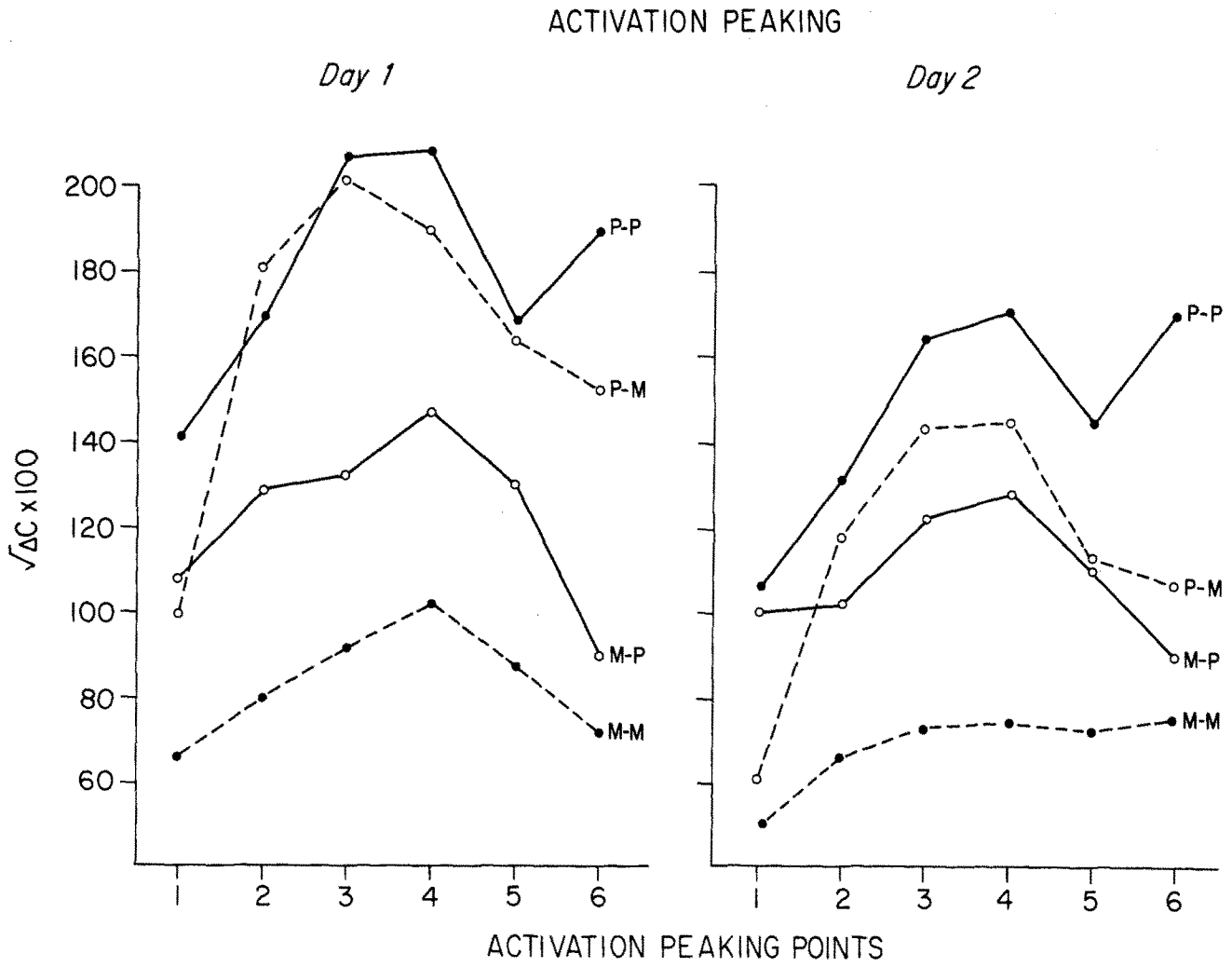

FIG. 2. SCR Activation Peaking partitioned by groups and days.

marijuana was acting peripherally to block sympathetic output to the target membranes in the skin and ecrine sweat glands. This interpretation is consistent with the known anticholinergic properties of marijuana [8], and the presence of a sympathetic-cholinergic transmittal situation in SCR innervation [24]. Mitigating this view was the lack of alteration in basal skin conductance under marijuana intoxication. Basal conductance levels are maintained by tonic neural output [24]. Therefore, a peripheral blocking action, similar to curare or atropine, would be expected to depress basal conductance. Basal conductance levels were unaffected by marijuana; therefore it seems more probable that CNS cholinergic systems concerned with memory and motivation, but not ANS basal levels of arousal, are implicated. It is, certainly, well established that cholinergic CNS systems are concerned with memory and learning.

These cholinergic mediated neural systems are largely located within the limbic system, an area already implicated both inferentially and neurochemically as sites of marijuana activity [17,20]. Drawing on animal data, we would reject the hippocampus and hippocampal gyrus as major sites of marijuana action because ablation of these structures eliminated habituation of the orienting response and interfered with establishment of memory traces – neither of which are much affected by marijuana ([3,25], Rickles, Cohen and Klitzner, Unpublished). A more likely structure is the amygdaloid nuclei. Stimulation or ablation of the amygdala does not alter basal ANS levels, but phasic modulation is affected [24]. This structure has recently been shown to mediate motivational aspects of learning. Thus, a hypothesis which implicates the amygdala as the major CNS structure altered by marijuana is consistent with attenuation of the magnitude of SCR orienting, diminished motivation, no alteration in basal skin conductance, normal habituation of the orienting response and altered but not abolished ability to lay down memory traces while S is marijuana intoxicated.

A further question may be raised – how does attenuation of SCR orienting affect learning? The magnitude of SCR orienting has been positively correlated with speed of learning [18,19]. Perhaps one parameter of learning paired-associates required that some threshold of cumulative activation associated with a particular nonsense syllable word pair must be exceeded before a stable learning trace is

laid down. If this accumulation of associated activation was necessary, more learning trials would be required to reach the cumulative threshhold during marijuana intoxication. Further, the paradoxical increase in selective attention but diminished vigilance found in marijuana intoxication may be related to an inability of the individual to shift attention easily because the orienting response is too small to cause orienting distractions. The same mechanism may underlie the fluctuating levels of vigilance. If orienting-arousal functions as an error signal, in the servo-mechanical sense, bringing attention into sharper focus and, therefore, maintaining vigilance, a drastic diminution in the orienting response and error signal would result in an organism which could not maintain vigilance well, (not enough error signal to cause re-focus). The organism would not be easily distracted because the orienting response to external stimuli was not great enough to distract the focus of attention.

Clearly, there are many neurophysiological systems which underlie learning, and, doubtlessly, others are also affected by marijuana. Skinner [27] has shown that marijuana extract prevents the normal functioning of the non-specific thalamo-cortical system; a system he believes to control selective perception and attention in the cat. It remains for future research to elaborate these effects and the interaction with those hypothesized herein.

REFERENCES

1. Blatt, S. J. Patterns of cardiac arousal during complex mental activity. *J. abnorm. Psychol.* **63:** 272–282, 1961.
2. Brown, C. H. The relation of magnitude of galvanic skin responses and resistance levels to the rate of learning. *J. exp. Psychol.* **20:** 262–278, 1937.
3. Cohen, M. J. and W. H. Rickles Jr. Performance on a verbal learning task by subjects of heavy past marijuana usage. *Psychopharmacologia* **37:** 323–330, 1974.
4. Finesmith, S. Systematic changes in GSR activity as a physiological correlate of paired-associate learning. Paper presented at the meeting of the Midwestern Psychological Association, Chicago, 1959.
5. Galanter, M., R. Wyatt, L. Lemberger, H. Weingartner, T. Vaughan and W. T. Roth. Effects on humans of Δ^9 THC administered by smoking. *Science* **176:** 934–936, 1972.
6. Germana, J. Psychophysiological correlates of conditioned response formation. *Psychol. Bull.* **70:** 105–114, 1968.
7. Germana, J. and W. B. Pavlik. Autonomic correlates of acquisition and extinction. *Psychonom. Sci.* **1:** 109–110, 1964.
8. Goodman, L. S. and A. Gilman. *The Pharmacological Basis of Therapeutics.* New York: McMillan, 1970, pp. 276–313.
9. Heath, R. G. Marijuana effects on deep and surface electroencephalography of man. *Archs gen. Psychiatry, Chicago* **26:** 577–587, 1972.
10. Hollister, L. E., S. Sherwood and A. Cavasino. Marijuana and the human electroencephalogram. *Pharmac. Res. Communs* **2:** 305–309, 1970.
11. Holtzman, D., R. Lovell, J. Jaffe and D. Freedman. 1-Δ^9 tetrahydrocannabinol: neurochemical and behavioral effects in the mouse. *Science* **163:** 1464–1467, 1969.
12. Kintsch, W. Habituation of the GSR component of the orienting reflex during paired-associate learning before and after learning has taken place. *J. Math. Psychol.* **2:** 330–340, 1965.
13. Kiplinger, G. R. and J. Manno. Dose response relationships to cannabis in human subjects. *Pharmac. Rev.* **23:** 339–347, 1971.
14. Kiplinger, G. R., J. Manno, B. Rodda and R. B. Forney. Dose response analysis of the effects of tetrahydrocannabinol in marijuana. *Clin. Pharmac. Ther.* **12:** 650–657, 1971.
15. Kirk, R. E. *Experimental Design Procedures for the Behavioral Sciences.* Belmont, California: Brooks-Cole, 1968, p. 311.
16. Kopell, B. S., J. R. Tinklenberg and L. E. Hollister. Contingent negative variation amplitude: marijuana and alcohol. *Archs gen. Psychiat.* **27:** 801–811, 1972.
17. Low, M. D., H. Klonoff and A. Marcus. The neurophysiological basis of the marijuana experience. *Can. Med. Ass. J.* **108:** 157–165, 1973.
18. Maltzman, I. and M. P. Mandell. The orienting reflex as a predictor of learning and performance. *J. exp. Res. Personality* **3:** 99–106, 1968.
19. Maltzman, I. and D. C. Raskin. Effects of individual differences in the orienting reflex on conditioning and complex processes. *J. exp. Res. Personality* **1:** 1–6, 1965.
20. McIsaac, W. M., G. E. Fritchie, J. E. Idönpään-Heikkila, B. T. Ho and L. E. Englert. Distribution of marijuana in monkey brain and concomitant behavioral effects. *Science* **230:** 593–594, 1971.
21. Meyer, R. E., R. C. Pillard, L. M. Shapiro and S. Miren. Administration of marijuana to heavy and casual marijuana users. *Am. J. Psychiat.* **128:** 198–204, 1971.
22. National Commission on Marijuana and Drug Abuse. Marijuana, a signal of misunderstanding. Washington: U.S. Government Printing Office, 1972.
23. Rickles, W. H. Jr. Engineering considerations in GSR research. *Aeromed. Rev.* **4:** 1–20, 1964.
24. Rickles, W. H. Jr. Central nervous system substrates of some psychophysiological variables. In: *Handbook of Psychophysiology*, edited by N. S. Greenfield and R. A. Sternbach. New York: Holt, Rinehart and Winston, 1972, pp. 93–121.
25. Rickles, W. H. Jr., M. J. Cohen, C. A. Whitaker and K. E. McIntyre. Marijuana induced state-dependent verbal learning. *Psychopharmacologia* **30:** 349–354, 1973.
26. Roessler, R. Personality, psychophysiology and performance. *Psychophysiology* **10:** 215–227, 1973.
27. Skinner, J. E. Abolition of a conditioned, surface-negative, cortical potential during cryogenic blockade of the non-specific thalamo-cortical system. *Electroenceph. clin. Neurophysiol.* **31:** 197–209, 1971.
28. Tart, C. T. *On Being Stoned, A Psychological Study of Marijuana Intoxication.* Palo Alto: Science and Behavior Books, 1971.
29. Thorndike, E. L. and I. Lorge. *The Teacher's Word Book of 30,000 Words.* New York: Columbia University Press, 1944.
30. Truitt, E. B. Jr. Biologic disposition of THC. *Pharmac. Rev.* **23:** 273–278, 1971.
31. Underwood, B. J. and R. W. Schultz. *Meaningfulness and Verbal Learning.* Philadelphia: Lippincott, 1960.
32. Volavka, J., R. I. Dornbush, S. Feldstein, G. Clare, A. Zaks, M. Fink and A. M. Freedman. Marijuana, EEG and behavior. *Ann. N. Y. Acad. Sci.* **191:** 206–215, 1971.
33. Weil, A. T. and N. E. Zenberg. Acute effects of marijuana on speech. *Nature* **222:** 434–437, 1969.
34. Weil, A. T., N. E. Zenberg and J. Nelson. Clinical and psychological effects of marijuana in man. *Science* **162:** 1234–1247, 1968.
35. West, L. J. On the marijuana problem. In: *Psychotomimetic Drugs,* edited by D. Efron. New York: Raven Press, 1970, p. 327.