THE EFFECT OF STIMULANT DRUGS ON THE NEGATIVE AFTER-IMAGE THRESHOLD

by A. Eugene Palchanis

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CURRICULUM STUDIORUM

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INTRODUCTION

The challenge of an acceptable integration and explanation of the interaction of psychological and neurophysiological parameters of human behavior offers scientifically perplexing research possibilities. In the development of learning theories, the investigation of the phenomenon of reactive cortical inhibition has yet to yield a definitive explanation and clarification. Of paramount importance in the investigation of this phenomenon has been the precise role of the central nervous system, the structure and functions of which, have provided an entity which could be scientifically approached and explored.

Among the methods used to demonstrate the possible role of the central nervous system in reactive cortical inhibition has been the negative after-image phenomenon. Apropos to this method of investigation is the appearance of a new apparatus designed to produce and assess the negative after-image with considerable refinement. The potentials offered by experimentation with this apparatus have enhanced the interest of this writer in the reactive inhibition phenomenon.

The following pages report on a study which was undertaken to inquire into the role of the central nervous system as a generator of reactive cortical inhibition. The

involvement of the neurophysiological substratum was accomplished through the action of two sympathomimetic drugs.

The text of the report will be divided into three chapters followed by a summary and conclusions. Chapter One presents the research in this area in retrospect, an evaluation of this research and a statement of the problem directly studied. Chapter Two presents an outline of the experiment as it was conducted, and includes a description of the population used in the experiment and the type of statistical evaluation applied to the results obtained. Chapter Three presents the results of the experiment with an appropriate evaluation and discussion rendering an interpretation of these. The summary and conclusions will briefly give the implications of the results as well as suggestions for future research possibilities.

CHAPTER I

REVIEW OF THE LITERATURE

The research undertaken to explain and quantify reactive cortical inhibition has, at times, resulted in incomplete, inconsistent and contradictory findings. Recently, Barry[1] and Kovatch[2] have lucidly and comprehensively presented in their works the evolutional process leading to the various contemporary theories and hypotheses. Consequently, a similar presentation here would be redundant and, undoubtedly, replication.

However, pertinent to the present research are a number of studies having implications either directly or indirectly with regard to the method of investigation used in this study. In order to substantiate the relatedness of this study to previous work, these studies will be reviewed in this chapter from the points of view of methodology and contribution to the general understanding of the phenomenon under investigation. In order to ensure clarity of presentation the chapter will be divided into sections.

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1 William F. Barry, Introversion-Extraversion and the Negative After-Image Threshold, unpublished doctoral dissertation, University of Ottawa, April, 1961, vii-80 p.

2 Joseph D. Kovatch, Intra Cranial Pathology and the Negative After-Image Threshold, unpublished M.A. thesis, University of Ottawa, April, 1961, vii-44 p.

Section One will consider the studies which have provided a functional brain model offering research possibilities as well as those studies which have utilized an approach to the problem similar to that employed in this study. Section Two presents an evaluation of the studies and a statement of the problem to be directly investigated in the form of an experimental hypothesis.

1. Related Studies.

Köhler and Wallach,[3] in offering an explanation of reactive inhibition, have attempted a description of cortical conductivity in the occipital cortex utilizing figural after-effects. Their view of the visuopsychic cortex and nervous transmission therein, based on tissue polarization, has been opposed by Osgood and Heyer[4] as being untenable in view of the present neuroanatomical model of the cortex.

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3 Wolfgang Köhler and Hans Wallach, "Figural After-Effects", in Proceedings of the American Philosophical Society, 88, 1944, p. 269-357, quoted by John Armuskopf, "The Magnitude of Figural After-Effects as a Function of Duration of Test Period", in the American Journal of Psychology, Vol. 67, No. 4, December, 1954, p. 684-690.

4 C.E. Osgood and A.W. Heyer, "A New Interpretation of Figural After-Effects", Psychological Review, Vol. 59, No. 2, March, 1952, p. 98-118.

Eysenck[5] has employed the Köhler-Wallach theory in part to support his postulations with regard to individual differences in terms of reactive cortical inhibition patterns. Here, Eysenck maintains that certain personality dimensions would be identifiable on the basis of these patterns. Furthermore, he states that in assessing the action of stimulant drugs and in particular, amphetamine, which he uses as a prototype, a decrease in reactive cortical inhibition would be manifested in lowered negative after-image thresholds.

Such a position would be difficult to defend in view of the findings of Marrazzi and Hart[6] who point out that chemically, amphetamine produces inhibitory effects on the cerebral cortex and brainstem nervous transmission. Their findings favor a trophotropic blocking activity in these areas and subsequent increase in cortical inhibition.

Although Eysenck speaks of a cortical influence on the negative after-image, the interpretation strays more toward a peripheral sensory inhibition occurring at the retinal level of functioning. The use of amphetamine does not demonstrate retinal involvement in the negative after-image

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5 H.J. Eysenck, "Drugs and Personality, I. Theory and Methodology", Journal of Mental Science, Vol. 103, 1957, p. 119-131.

6 A.Marrazzi and E. Hart, "An Electrophysiological Analysis of Drugs Useful in Psychiatric States", Fed. Proc., Vol. 10, 1951, p. 322.

phenomenon inasmuch as research thus far has failed to identify conclusively any pharmacological action of the drug peculiar to the retinal photochemical process.

Eysenck and Aiba,[7] using a modified Lehman Device,[8] supported their postulations of the effects of ten milligrams of dextro-amphetamine sulfate on the suppression of the primary visual stimulus. Here, the interpretation of the findings was based on the pre-excitatory inhibition of the primary visual stimulus at the retinal level. This interpretation has been challenged by Barry[9] as being directly opposed to the commonly accepted interpretation of the negative after-image phenomenon and the vascillation of the authors in adhering to either a cortical or retinal inhibitory mechanism has been cited by Kovatch.[10]

Aiba,[11] studying the effects of ten milligrams of dextro-amphetamine sulfate on the after-effects produced by

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7 H.J. Eysenck and S. Aiba, "Drugs and Personality, IV. The Effects of Stimulant and Depressant Drugs on the Suppression of the Primary Visual Stimulus", Journal of Mental Science, Vol. 103, 1957, p. 661-665.

8 H. Lehman, "Preliminary Report on a Device for the Objective Measurement of the Negative After-Image", Science, Vol. 112, No. 2903, August, 1950, p. 199-201.

9 Barry, Op. Cit., p. 66.

10 Kovatch, Op. Cit., p. 11.

11 Satoru Aiba, "The Effects of Stimulant and Depressant Drugs on the Bidwell Phenomenon", British Journal of Psychology, Vol. 51, No. 4, 1960, p. 311-318.

the Bidwell phenomenon indicates that those subjects receiving the drug had lowered thresholds and contends that the drug enhances pre-excitatory inhibition by altering the response of the retina to a light stimulus. This explanation is inconsistent with the pharmacology of the drug used since the only action of the amphetamines on the eye itself is that of a mydriatic when introduced directly into the conjunctival sac. The above interpretation would seem to be that of an improvement of visual acuity which cannot be, ipso facto, a decrease in cortical inhibition.

Eysenck, Holland and Trouton[12] assessed the effects of ten milligrams of dextro-amphetamine sulfate on the after-effects produced by the Archimedes Spiral Apparatus. The results failed to support their postulations of the effect of the drug on the negative after-image threshold. An explanation for the findings was given in terms of the ineffectiveness of the stimulant drug.

Kaplan,[13] using a device similar to the one employed in this study, investigated the effects of three sympathomimetic drugs on the after-image threshold. His conclusions

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12 H.J. Eysenck, H. Holland and D.S. Trouton, "Drugs and Personality, III. The Effects of Stimulant and Depressant Drugs on Visual After-Effects", Journal of Mental Science, Vol. 103, p. 650-655.

13 Solomon D. Kaplan, "Autonomic Visual Regulation", reprinted from Psychiatric Research Reports, American Psychiatric Association, January, 1960, p. 104-114.

are not in agreement with Eysenck and his co-workers regarding the effect of the drugs on the threshold. In his study Kaplan found an increase in the threshold values. The interpretation of these results is in concurrence with the notion of a diencephalic link as a possible regulator of cortical inhibition as suggested by Barry.[14] The results reported by the author indicate that two of the drugs, twenty-five milligrams of ephedrine sulfate and 100 milligrams of sympatol produced significant effects while the third, five milligrams of amphetamine sulfate did not. The author does not offer any explanation for the failure of this drug to produce significant results, but a plausible assumption may be that the dosage level was too low.

In addition to a valuable explanation of cortical inhibition of visual functioning conceived as being the resultant of a possible differentiation in central nervous excitation as the result of the regulatory effects of the autonomic nervous system, the author also provides evidence of the effects of coffee, tea and tobacco on the negative after-image threshold; thus indicating the necessity of controlling these influences in similar experimentation.

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14 Barry, Op. Cit., p. 26.

Klein and Krech,[15] in localizing reactive cortical inhibition in the cerebral cortex, have contributed a theoretical dynamic model of reactive cortical inhibition conceived in terms of the neurophysiological substratum when they state that:

> (...) any neural activity induces heightened resistance in the area stimulated (...) the current flow initiated by stimulation of a defined cortical area results in heightened resistance within that area to further electrical activity. Should further stimulation occur, the resulting pattern of electrical activity would, as a consequence of this increased resistance, be "dampened", distorted or rerouted. In this event we can then speak of reactive or a temporary condition of decreased cortical conductivity: i.e., in one specific area and for a finite time cortical conductivity is reduced.[16]

This state of decreased cortical conductivity was seen among the organic subjects in the study of Kovatch as an increase in reactive cortical inhibition.[17]

2. Evaluation.

The studies which have been reviewed in the preceding section have attempted to demonstrate cortical mediation in reactive cortical inhibition, albeit, not always successfully. Some of the inconclusive results may be due to unreliable instruments -- a plausible assumption from the

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16 Klein and Krech, Op. Cit., p. 120-121.

17 Kovatch, Op. Cit.

fact that most of the authors have failed to report any evidence in this regard. Also, the reporting of some of the experiments implies a looseness of experimental method and little control of relevant, and possible contaminating variables. From these facts alone the results are justly the object of serious question.

In his work Barry has pointed to the inconsistencies of interpretation of the negative after-image phenomenon when he states that "(...) cortical inhibition is a central phenomenon. It may be the cause or effect of many sums, products and neurological quotients at the peripheral sensory level".[18] This statement brings to attention the fact that at times, the lack of agreement among authors and the rash postulations in the face of insufficient supportive evidence tend to complicate rather than explicate the nature and process of reactive cortical inhibition.

The study to be described in the following chapter was conducted in an effort to demonstrate the central nervous system involvement in reactive cortical inhibition via transitory modifications in cortical functioning. The essential tool used in the investigation was an apparatus of established validity and reliability. The problem investigated is stated in the form of a null hypothesis: There

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18 Barry, Op. Cit., p. 66.

will be no significant differences between the pre-drug and post-drug inhibition sums of groups of subjects after receiving either a stimulant drug or a placebo.

CHAPTER III

EXPERIMENTAL DESIGN

The experimental hypothesis advanced in the preceding chapter was investigated within the framework to be discussed below. The chapter will be divided into five sections, each discussing an aspect of the total design. Section One mentions the tool used in the experiment to evoke the negative after-image and establish the threshold values. Section Two presents the basis for the selection of the pharmacological agents and a description of these. Section Three deals with the method of selection and description of the population involved. These will be followed by Sections Four and Five treating respectively, the experimental method followed and the statistical evaluation applied to the results.

1. The Psychophysiological Tool.

The principal tool of the experiment was the NAIT apparatus designed and used by Barry,[1] and subsequently used

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1 William F. Barry, Introversion-Extraversion and the Negative After-Image Threshold, unpublished doctoral dissertation, University of Ottawa, April, 1961, vii-80 p.

by Kovatch[2] and Montville.[3] The complete description of the design and mechanics of the apparatus may be found in the report of the designer, and since no innovations were introduced by this writer, further elaboration at this point would be repetitious.

2. The Pharmacological Agents.

Two sympathomimetic drugs were used to introduce changes in the central nervous system functioning; an inert placebo was included for certain comparisons. The selection of the drugs was based largely on the available literature which offered descriptive information since it was not the object of this study to investigate the action of a particular drug. Specifically, the research was concerned with a class of drugs regarded as possessing stimulant properties.

In view of the body of information available pertaining to the amphetamines and their derivatives, amphetamine sulfate and dextro-amphetamine sulfate were selected.[4] Amphetamine is structurally related to epinephrine and is

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2 Joseph D. Kovatch, Intra Cranial Pathology and the Negative After-Image Threshold, unpublished M.A. Thesis, University of Ottawa, April, 1961, vii-44 p.

3 Paul G. Montville, study now in progress at the University of Ottawa, School of Psychology and Education.

4 Henceforth the drugs will be designated as amphetamine and d-amphetamine.

considered as being a sympathomimetic drug which stimulates the higher nerve centers, especially the cortex. The action of d-amphetamine is the same, but generally considered to be more rapid and without the side-effects of amphetamine, especially at higher dosage levels.

The dosage levels of ten milligrams of amphetamine and five milligrams of d-amphetamine were established for two reasons: (1) the dextro-rotary isomer of amphetamine is considered to produce twice the amount of central nervous stimulation as the parent drug amphetamine, thereby necessitating a proportion between the drugs in order to justify comparisons; and (2) because of the possible side effects occurring with higher dosage levels it would be imperative that a physician be in attendance at all times during the experiment -- a plan which did not prove feasible. The drugs and placebo were prepared in identically-sized capsules varying in shade of color from 'skin' color to light pink.

3. The Population.

The sample used in the study was engaged on a volunteer basis. The volunteers were fifty-one students of the School of Psychology and Education of the University of Ottawa in full-time attendance, and twenty-eight graduate nurses enrolled in the Introductory Psychology course at the School of Psychology and Education. Because of the nature

of the experiment certain requirements of eligibility were imposed upon the volunteers.

First, those persons under the age of twenty-one were excluded because of the legality involved in administering drugs. Secondly, the group was screened on the basis of medical reports obtained from the pre-registration physical examination required by the University. The criteria for exclusion from participation in the study were as follows: (1) elevated blood pressure which was assumed to be reflecting possible hypertension; (2) familial history of hypertension which would indicate caution because of the nature of the experimental conditions; (3) possible neurological disorder which might influence the general area under investigation; (4) history of inordinate or continued drug usage which might result in drug tolerance; (5) visual defects, particularly color blindness, which would directly affect performance on the experimental task; and (6) other contraindications which might derive from the acumen of the physician.[5]

Thirdly, and concommitant with the above medical evaluation, a pilot study was conducted by the experimenter in which all of the volunteers were requested to take the NAIT test. The purpose of the pilot study was twofold. Primarily,

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5 Evaluation of the medical reports was made by Gaetan Valois, M.D., Assistant Director of the Student Health Center of the University of Ottawa.

it was undertaken to serve as a check on the medical screening, particularly with regard to color blindness. Color blindness was established on the basis of the person's inability to correctly identify the color of the stimulus. Secondarily, it was intended to familiarize the experimenter with the operations of the NAIT apparatus, and thereby facilitate the actual experimental procedure which will be discussed in the next section.

4. The Experimental Method.

In reporting the experimental method followed by the researcher, the word subject will be used, and will refer to those volunteers who were 'qualified' to participate in the experiment on the basis of the screening procedure described in Section Three.

Subjects scheduled to report for the experiment were requested to abstain from coffee, tea and tobacco for a period of at least one hour prior to reporting because of their general stimulating properties as shown by Kaplan.[6] When testing immediately followed mealtime, subjects were requested to eat only a 'light' meal in order to permit maximum absorption rate of the drugs.

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6 Solomon D. Kaplan, "Autonomic Visual Regulation", reprinted from Psychiatric Research Reports, American Psychiatric Association, January, 1960, p. 110.

Integral to the general experimental procedure was the recording of (1) the time and content of the last meal; (2) the number of cups of tea or coffee and the amount of tobacco taken immediately before the experiment; and, (3) the hours of sleep during the previous night. This information was collected in order to assess any unwarranted reactions which might occur to either the drugs or placebo. Also, each subject was required to sign a consent slip freeing the researcher and the University from responsibility in the event of any drug-induced undesirable after-effects.

Two subjects reported to the testing situation at a time. This procedure allowed maximum observation of the subjects' overt behavioral reactions after having received either a drug or placebo. These general observations were also recorded.

Before receiving a capsule, the pre-drug blood pressure and heart rate were established and recorded to be studied later as indices of arousal due to the pharmacological agents.[7] Also, the pre-drug negative after-image threshold was established according to the procedure followed by Barry[8] and Kovatch.[9] Testing was done in a darkened room

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7 Blood pressure and heart rate were determined by a Registered Nurse.

8 Barry, Op. Cit.

9 Kovatch, Op. Cit.

and the subject was seated twelve inches from the NAIT apparatus (as measured by a string attached to the front panel). A three-minute dark-adaptation period was allowed during which time the directions were read to the subject. Each subject was given two sets of six trials, interrupted by a three-minute rest period to allow for the dissipation of reactive inhibition build-up. Each set of six trials constituted a session.

Following this pre-drug testing the subject received his capsule (capsules were administered randomly according to the double-blind method by the nurse). Each subject received his capsule when alone with the nurse to prevent awareness of slight color differences; the vials containing the capsules were concealed from view. After receiving the capsule, the subject was requested to be seated -- reading material was provided. Conversation was allowed but subjects were instructed that they should not discuss with each other how they 'felt' and should comment in this regard only when directly asked by the nurse. It was intended that this restriction would reduce suggestion effects since isolation of the subjects was virtually impossible. All comments made to the nurse were also recorded for future evaluation.

Each subject was then retested at forty-five and seventy-minute intervals after receiving either a drug or placebo. At these intervals, however, the directions were

given in an abbreviated form. These points of retesting were established on the basis of Davidoff's report[10] in which the peak of effectiveness for each drug is indicated. It was intended that retesting at these points covered the general range during the peak of effectiveness of both drugs. At each of these intervals the blood pressure and heart rate were again recorded. Between the forty-five and seventy-minute testings, the subjects were instructed to report their awareness of how they 'felt' to the nurse. This information was similarily recorded.

5. The Statistical Analysis.

This section offers a description of the statistical techniques applied to the results of the NAIT testings and also the measures of inferred physiological change accompanying the drug and placebo conditions. In order to maintain clarity of presentation this section will be further subdivided into two subsections. Subsection A will include the treatment of the raw NAIT readings originally expressed in microamperes and also the procedure followed in establishing the reliability of the inhibition sums obtained on both sessions. Subsection B will convey a brief presentation

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10 Eugene Davidoff, "A Comparison of the Stimulating Effect of Amphetamine, Dextroamphetamine and Dextro-N-Methyl Amphetamine (Dextro-Desoxyephedrine)", reprinted from Medical Records, Vol. 156, July, 1943, p. 422-424.

of the analysis applied to the descriptive data of the population. It will also include the method of analysis applied to the accessory and supportive data which was derived from the measures of physiological change.

A. Conversion of the Raw NAIT Readings and the Reliability of the Inhibition Sum.

Following the precedence of Barry, the microampere readings taken directly from the NAIT apparatus were converted to foot-candle measurements according to the scale provided in his report.[11]

Since it was not the intention of this researcher to further investigate the validity and reliability of the NAIT apparatus, and in view of the results already obtained in previous experimentation with the apparatus, the algebraically determined inhibition sum constituted the expression of reactive cortical inhibition. The inhibition sum was the difference between the threshold values obtained on trials one and six, for both sessions. It was assumed also, from previous research, that the inhibition sums obtained on Sessions I and II, if not significantly different, were reflecting the inter-trial reliability of the instrument.

Since two sets of six trials were given at each testing, each set yielding an independent inhibition sum, it

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11 Barry, Op. Cit., p. 45.

would be expeditious to combine these values and then use the mean for subsequent computations. In order to accomplish this reduction of data the consistency of the inhibition sums for Sessions I and II for the three testings was established to prevent unwarranted simplification of the results. This was accomplished according to the formula:[12]

$$r = \sqrt{\frac{\left[N\Sigma XY - (\Sigma X)(\Sigma Y)\right]^2}{\left[N\Sigma X^2 - (\Sigma X^2)\right]\left[N\Sigma Y^2 - (\Sigma Y^2)\right]}}$$

From the obtained results of this technique, a t test of the significance of the correlations was carried out according to the formula:

$$t = r\sqrt{\frac{N - 2}{1 - r^2}}$$

On the basis of the results of the above techniques, the pre-drug and post-drug inhibition sums were compared for each group. These comparisons were made using the mean inhibition sum for each group for both pre-drug and post-drug testings using a t test of the means from correlated data employing the formula:

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12 J.P. Guilford, Fundamental Statistics in Psychology and Education, New York, McGraw-Hill, 1956, p. 140.

$$t = \frac{D}{\sigma d}$$

where $\sigma d = \sqrt{\sigma M_1^2 + \sigma M_2^2 - 2 r \sigma M_1 \sigma M_2}$

and $\sigma M = \frac{\sigma}{\sqrt{N - 1}}$

B. Supplemental Data.

Independently of the treatment of the MAIT data, the similarity of the groups was examined and the effects of the drugs were evaluated according to the measures of physiological change. The comparability of the three groups was established on the basis of two factors: mean age and mean weight, according to the formula:

$$t = \frac{D}{\sigma d}$$

where $\sigma d = \sqrt{\sigma M_1 - \sigma M_2}$

and $\sigma M = \frac{\sigma}{\sqrt{N - 1}}$

The fluctuations of the obtained blood pressures and heart rates for each group during both the pre-drug and post-drug conditions were also examined according to the formula above in order to assess the action of the drugs in terms of

autonomic nervous system arousal. The final chapter will present the results of the total analytical process and an appropriate discussion in the light of implications toward current concepts of reactive cortical inhibition.

CHAPTER III

RESULTS AND DISCUSSION

The presentation of this chapter will again be facilitated by division into sections. Section One includes a discussion of the combined screening procedure and the resulting groups on the basis of the drug administration. Section Two presents the results of the correlation technique applied to the inhibition sums and the subsequent resulting comparisons. Section Three discusses the physiological changes which were manifested following the administration of the pharmacological agents, and the final section proposes a synthesis of the results.

1. The Results of the Screening and Drug Administration.

The combined screening procedure described in Chapter Two resulted in the elimination of sixteen volunteers from the original sample population. Two persons were excluded because of falling below the age limit, ten persons were automatically excluded because no medical records were available for them, two persons were excluded because of hypertension, one person because of color blindness and one person because of a past history of acute anxiety attacks. The experimental population then, on the basis of the combined screening criteria, was sixty volunteers.

Following the administration of the drugs and placebo the total group was differentiated into three groups of nineteen subjects on the basis of what agent each had received.[1] The descriptive data pertaining to the three groups appears in Tables I and II on pages twenty-four and twenty-five.[2]

2. The Inhibition Sum.

As was stated previously, the algebraically determined inhibition sum constituted the measure of reactive cortical inhibition. If it could be established that the group mean differences between Sessions I and II for all testings were not statistically significant, then these values could be combined in some manner with justification. Following the application of the statistical techniques to obtain first, a correlation between the two values and second, the significance of the differences between the correlated means, the necessary conditions were not met.

A significant mean difference for the placebo group was found on the seventy-minute testing. For both the amphetamine and d-amphetamine groups significant mean differences were ascertained on the forty-five minute and seventy-minute

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1 Three subjects failed to report for the experiment.

2 Coding of the groups will appear in the tables throughout this chapter as follows: I-Placebo, II-Amphetamine, and III-d-Amphetamine.

Table I.-

Data for the t Tests of Significance of the Mean Ages[a] of the Three Groups.

Groups (N=19)	Means	σ	σ_m	Diff.	σ_d	t	Sig.
I	25.10	5.22	1.277	0.13	1.506	.0863	--
II	24.97	3.10	0.800				
I	25.10	5.22	1.277	1.06	1.877	.5647	--
III	26.16	5.70	1.376				
II	24.97	3.10	0.800	1.19	1.592	.7475	--
III	26.16	5.70	1.376				

a Expressed in years.

Table II.-

Data for the t Tests of Significance of the Mean Body Weights of the Three Groups

Groups (N=19)	Means	σ	σ_m	Diff.	σ_d	t	Sig.
I	145.58	21.95	5.136	2.83	7.308	0.3872	--
II	142.75	23.70	5.199				
I	145.58	21.95	5.136	12.68	5.831	2.1743	.05
III	132.90	13.02	2.773				
II	142.75	23.70	5.199	9.85	5.892	1.6717	--
III	132.90	13.02	2.773				

testings. The data for these comparisons appear in summary in Table III on the following page.

Because of the significant mean differences which were found, it was necessary to compare the pre-drug and post-drug inhibition sums independently for Session I and Session II on all three occasions. From the data presented in Tables IV and V, pages twenty-eight and twenty-nine, it may be seen that there were no significant changes in the inhibition sums from the pre-drug level of any group. This relative constancy of the inhibition sum throughout the experimental conditions will be clarified in view of the data summarizing the arousal effects of the drugs which is discussed in the next section.

3. The Systemic Changes.

The fluctuations in blood pressure and heart rate following the ingestion of sympathomimetic drugs are understood to be reflecting the degree of arousal due to the action of the drugs. Because of the stimulating action of the drugs used, it would be expected that the above indices would manifest a state of increased activity of the autonomic nervous system. Contrary to expectations, the trend observed was toward a state of decreased activity. The significant drop in systiolic blood pressure measurements which occurred with the placebo group at the seventy-minute testing, and with the d-amphetamine group at both the forty-five and seventy-minute

Table III.-

Data for the t Tests of Significance of the Correlated Mean Inhibition Sums of the Three Groups.

Groups (N=19)	Testing	Mean I	σ	Mean II	σ	r_{12}	t	Sig.
I	Pre-Drug	4.37	8.58	3.47	9.28	.804	5.586	.01
	45-minute	6.10	20.79	2.47	4.39	.689	3.919	.01
	70-minute	4.63	0.07	1.74	0.49	.251	1.069	--
II	Pre-Drug	7.36	32.99	4.26	13.16	.902	8.601	.01
	45-minute	8.74	16.39	2.47	7.82	.112	0.490	--
	70-minute	7.79	15.82	1.26	7.89	.053	0.219	--
III	Pre-Drug	1.53	5.52	1.21	5.20	.719	4.263	.01
	45-minute	1.11	3.28	-.47	4.34	.340	1.491	--
	70-minute	3.53	4.32	1.53	3.05	.145	0.620	--

Table IV.-

Data for the t Tests of Significance of the Mean Inhibition Sums of the Three Groups on Session I.

Groups (N-19)	Testings	Mean	σ	σ_m	Diff.	σ_d	t	Sig.
I	Pre-Drug	4.37	8.58	2.023	1.73	5.301	0.3264	--
	45-minute	6.10	20.79	4.900				
	Pre-Drug	4.37	8.58	2.023	0.26	2.023	0.1285	--
	70-minute	4.63	0.07	0.017				
II	Pre-Drug	7.36	32.99	7.778	1.68	8.864	0.1895	--
	45-minute	8.74	16.39	3.864				
	Pre-Drug	7.36	32.99	7.778	0.43	8.643	0.0498	--
	70-minute	7.79	15.82	3.729				
III	Pre-Drug	1.53	5.52	1.301	0.42	1.512	0.2778	--
	45-minute	1.11	3.28	0.772				
	Pre-Drug	1.53	5.52	1.301	2.00	1.653	1.2099	--
	70-minute	3.53	4.32	1.019				

Table V.-

Data for the t Tests of Significance of the Mean Inhibition Sums of the Three Groups on Session II.

Groups (N=19)	Testings	Mean	σ	σ_m	Diff.	σ_d	t	Sig.
I	Pre-Drug	3.47	9.28	2.187	1.00	2.419	0.4134	--
	45-minute	2.47	4.39	1.035				
	Pre-Drug	3.47	9.28	2.187	1.73	2.191	0.7896	--
	70-minute	1.74	0.49	0.115				
II	Pre-Drug	4.26	13.16	3.102	1.79	2.414	0.7415	--
	45-minute	2.47	7.82	1.844				
	Pre-Drug	4.26	13.16	3.102	3.00	3.617	0.8294	--
	70-minute	1.26	7.89	1.861				
III	Pre-Drug	1.21	5.20	1.225	1.68	1.596	1.0526	--
	45-minute	-.47	4.34	1.024				
	Pre-Drug	1.21	5.20	1.225	0.32	1.420	0.2254	--
	70-minute	1.53	3.05	0.719				

testings, is not completely supported by the fluctuations of diastolic blood pressure and heart rate. Briefly stated, the drugs were ineffective in producing arousal and an unexplainable reaction occurred in the placebo group. The summary data for the systemic changes appears in Tables VI, VII, and VIII on the following pages. Appropriately, however, other considerations will be discussed in the following section.

4. Synthesis of the Results.

A complete explanation of the obtained results must also take into account factors of paramount importance -- the validity and reliability of the NAIT apparatus as a tool for the assessment of reactive cortical inhibition. In view of the studies of Barry[3] and Kovatch,[4] this writer is prepared to accept the inhibition sum obtained from the NAIT apparatus as a measure of reactive cortical inhibition. Furthermore, the reliability of the inhibition sum in this experiment has been seen in the pre-drug measurements for the three groups. The significant differences in the inhibition sums which were found at the forty-five and seventy-minute testings were common to the three groups and cannot, therefore, be rightfully attributed to the action of the drugs exclusively.

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3 Barry, Op. Cit.

4 Kovatch, Op. Cit.

Table VI.-

Data for the t Tests of Significance of the Pre-Drug and Post-Drug Mean Systolic Blood Pressures for the Three Groups.[a]

Groups (N=19)	Readings	Means	σ	σ_m	Diff.	σ_d	t	Sig.
I	Pre-Drug	123.10	10.47	2.586	10.06	3.918	2.5684	.02
	45-minute	113.04	12.42	2.926				
	Pre-Drug	123.10	10.47	2.586	13.84	3.561	3.8865	.01
	70-minute	109.26	12.24	2.448				
II	Pre-Drug	117.80	14.13	3.424	6.27	4.446	1.4102	--
	45-minute	111.53	11.94	2.892				
	Pre-Drug	117.80	14.13	3.424	4.85	4.344	1.1165	--
	70-minute	112.95	10.77	2.672				
III	Pre-Drug	121.88	10.29	2.582	13.30	3.596	3.6985	.01
	45-minute	108.58	9.93	2.503				
	Pre-Drug	121.88	10.29	2.582	13.41	3.287	4.0797	.01
	70-minute	108.47	8.73	2.035				

a Values expressed in millimeters of mercury.

Table VII.-

Data for the t Tests of Significance of the Pre-Drug and Post-Drug Mean Diastolic Blood Pressures for the Three Groups.[a]

Groups (N=19)	Readings	Means	σ	σ_m	Diff.	σ_d	t	Sig.
I	Pre-Drug	77.95	5.61	1.426	0.05	2.306	0.0217	--
	45-minute	77.00	7.47	1.815				
	Pre-Drug	77.95	5.61	1.426	4.27	2.196	1.1944	--
	70-minute	73-68	6.66	1.652				
II	Pre-Drug	71.31	6.84	1.986	0.94	2.752	0.3416	--
	45-minute	70.37	8.01	1.892				
	Pre-Drug	71.31	6.84	1.986	0.27	2.682	0.1007	--
	70-minute	71.58	7.44	1.788				
III	Pre-Drug	75.42	7.62	1.599	3.84	2.350	1.6340	--
	45-minute	71.58	6.84	1.721				
	Pre-Drug	75.42	7.62	1.599	3.84	2.312	1.6609	--
	70-minute	71.58	6.84	1.787				

a Values expressed in millimeters of mercury.

Table VIII.-

Data for the t Tests of Significance of the Pre-Drug and Post-Drug Mean Heart Rates for the Three Groups.[a]

Groups (N=19)	Readings	Means	σ	σ_m	Diff.	σ_d	t	Sig.
I	Pre-Drug	78.15	9.75	2.355	2.70	3.088	0.8743	--
	45-minute	75.45	8.49	1.997				
	Pre-Drug	78.15	9.75	2.355	4.89	3.006	1.6267	--
	70-minute	73.26	7.44	1.868				
II	Pre-Drug	78.95	9.03	2.164	4.42	2.841	1.5558	--
	45-minute	74.53	7.65	1.841				
	Pre-Drug	78.95	9.03	2.164	6.30	2.944	2.1399	.05
	70-minute	72.65	8.61	1.909				
III	Pre-Drug	81.95	10.32	2.401	6.84	3.167	2.1598	.05
	45-minute	75.11	8.91	2.065				
	Pre-Drug	81.95	10.32	2.401	9.30	3.431	2.7106	.02
	70-minute	72.65	9.57	2.449				

a Based on one-minute readings.

An analysis of the individual response curves of the three groups revealed that learning was not operative, the slope of the curves being in an upward direction. The effects of learning are discernible in a declining curve. Another factor considered was the 'set' of the subjects as they performed the experimental task. This was discounted because the basal level of Session II remained at that of Session I.

The hypothesis advanced by the writer to account for the significant mean differences between the inhibition sums of Session I and Session II is erratic performance of the subjects upon retesting as a function of fatigue. Previous reliability coefficients obtained for the inhibition sum were based on a single testing period of a shorter duration. Testing in this experiment extended over a period of ninety minutes during which time each subject was required to perform a total of thirty-six trials on the NAIT apparatus. A perusal of the comments of the subjects collected over the ninety-minute period provided verification of this inasmuch as they reflected fatigue, boredom and, at times, irritation with the experimental task. It may be reasoned therefore, that the subjects' attitudes during the latter part of the experiment influenced and contaminated performance.

Pertinent to this contention is the fact that the drugs failed to produce an offsetting condition of a

'feeling-of-well-being' which generally accompanies the stimulating action. Paradoxically, the drugs produced 'feelings' of relaxation which might be considered as preparatory or preceding actual stimulation. An analysis of the data which was collected regarding possible extraneous stimulation resulting from coffee, tea or tobacco indicated that their possible influence was negligible.

The interplay of the factors which have been discussed in this section undoubtedly must be interpreted in combination with the ineffectiveness of the pharmacological agents. The resultant of such an interpretation clarifies the fact that no statistically significant differences between the pre-drug and post-drug mean inhibition sums were found. On the basis of the obtained results, the null hypothesis that 'there will be no significant differences between the pre-drug and post-drug inhibition sums of groups of subjects after receiving a stimulant drug or a placebo' cannot be rejected.

SUMMARY AND CONCLUSIONS

This paper reported on an investigation of the effects of two stimulant drugs on the negative after-image threshold which was interpreted as being a measure of reactive cortical inhibition. The relatedness of the problem of previous research efforts was confirmed with a pertinent review of studies conducted to date. Various statements regarding the phenomenon of reactive cortical inhibition were presented with both acknowledgement and criticism.

Following this review of previous research, the problem was defined in terms of an experimental hypothesis that 'there will be no significant differences between the pre-drug and post-drug inhibition sums of groups of subjects after receiving either a stimulant drug or a placebo'. An explicit statement of the method followed to test the hypothesis as well as a description of the population and statistical evaluation were presented in the context of the experimental design, an attempt being made to utilize to a maximum the contributions of other authors.

The results of the experiment were then reported and discussed, an attempt being made to account for the unexpected findings. These findings cannot be legitimately evaluated within the perspective of contemporary theories and hypotheses.

The possibilities for future research studies concerning the reactive inhibition phenomenon remain many and varied. Before proceeding further with the method of investigation offered by the NAIT apparatus, however, an endeavour must be made to determine the influence of the subjects' attitudes with regard to the experimental task. Other studies should investigate again the effects of stimulant drugs on the negative after-image threshold. Different types of physiological imbalances may also be approached via the negative after-image phenomenon.

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APPENDIX 1

ABSTRACT OF

The Effect of Stimulant Drugs on the Negative After-Image Threshold[1]

Several theories and hypotheses regarding the nature and mechanisms of reactive cortical inhibition have been advanced and subsequently investigated via after-image phenomena. The definitive description has yet to be written.

This study investigated reactive cortical inhibition by evaluating the effects of ten milligrams of amphetamine and five milligrams of d-amphetamine on the negative after-image threshold. A new apparatus designed to produce the negative after-image was employed as the principal tool. The research was conducted with a population of graduate students and graduate nurses.

The mean inhibition sums determined from the threshold values for each group permitted the pre-drug and post-drug comparisons using *t* tests. On the basis of this method, the null hypothesis was not rejected. The interpretation of the findings considered two relevant factors, the ineffectiveness of the drugs in producing arousal and the subjects' attitudes.

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1 A. Eugene Palchanis, M.A. thesis presented to the School of Psychology and Education of the University of Ottawa, Ontario, June, 1962, vii-40 p.