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PERSONALITY, PERCEPTUAL REACTANCE, AND AROUSAL

by

ANGUS MCDONALD

Dissertation

Submitted to the Faculty of Graduate Studies through the
Department of Psychology in Partial Fulfillment
of the Requirements for the Degree of
Doctor of Philosophy
at the
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ABSTRACT

Several theories of personality suggest that different individuals react either more or less to the same objective stimulation than others. This characteristic "perceptual reactance" level is thought to determine what amount of environmental stimulation is regarded as pleasant for an individual. One way of changing this perceptual style is the use of drugs, and presumably by knowing what perceptual type a person is one could predict what kind of drug response he will have.

The amphetamine drugs seem to have widely varying effects on different people. Though they often increase intellectual abilities as measured by certain tests, they do not do so with perfect predictability. This study attempted to predict which individuals would benefit from the drug on the basis of their original perceptual reactance. The latter factor was found to be irrelevant to improvement on the Otis Group Intelligence Test, which was statistically significant for a group of 36 university students.

PREFACE

Like most research, this contribution owes much to many people -- especially Dr. Cornelius J. Holland and Dr. A. A. Smith, as well as the other members of my thesis committee. Like most theses, it may even owe too much to too many people. Such are the trials of the university system.

TABLE OF CONTENTS

PREFACE.....		iii
TABLE OF CONTENTS.....		iv
LIST OF TABLES.....		vi
LIST OF FIGURES.....		viii
Chapter		
I	INTRODUCTION.....	1
	Perceptual Reactance and Personality.....	1
	Changes in Perceptual Reactance and	
	Personality Change.....	12
	Arousal, Perceptual Reactance, and	
	Personality.....	22
	Predictions to be Tested in this	
	Study.....	28
II	METHODS AND PROCEDURE.....	32
	Subjects.....	32
	Apparatus.....	33
	Procedure.....	35
III	RESULTS.....	41
	The Block Test for Perceptual	
	Reactance.....	43
	Voice Amplitude.....	46
	Involuntary Pauses While Finger	
	Tapping.....	46
	Extraversion Scores.....	46
	Otis Intelligence Test Scores.....	51
	Additional Measures.....	51
IV	DISCUSSION.....	78
V	SUMMARY AND CONCLUSIONS.....	94

APPENDIX 1.....	97
APPENDIX 2.....	98
APPENDIX 3.....	99
REFERENCES.....	100
VITA AUCTORIS.....	104

LIST OF TABLES

Table		Page
1	Kinesthetic Perceptual Reactance Scores.....	45
2	Means and Standard Deviations of Decibel Levels of Loudest and Least Loud of the Numbers Counted by Subjects Under Drug and Placebo.....	47
3	Means and Standard Deviations of Number of Pauses During Finger Tapping For Subjects Under Drug and Placebo.....	48
4	Means and Standard Deviations of Extra- version Scores on Eysenck Personality Inventory for Subjects Under Drug and Placebo.....	49
5	Means and Standard Deviations of Neuroticism and Lie Scores on the Eysenck Personality Inventory for Subjects Under Drug and Placebo.....	50
6	Mean Otis Group Intelligence Test Scores for Subjects Under Drug and Placebo.....	52
7	Analysis of Variance Made on Otis Group Intelligence Test Subscores.....	54
8	Otis Scores.....	55-56
9	"t" Tests Made Comparing 5 of the Sub- test Means of the Otis Group Intelligence Test for Subjects Under Drug and Placebo.....	57
10	Means of Subtests of the Otis Group Intelligence Test Which Differed Least From Drug to Placebo Conditions.....	58
11	Pulse Readings.....	61
12	Analysis of Variance Made on Pulse Rate Readings Under Drug and Placebo.....	62

13	"t" Test Made Between Estimates of the Length of a Minute (in seconds) Between Drug and Placebo	63
14	"t" Test Made Between Number of Taps in a Minute in Drug and Placebo Conditions	64
15	Means and Standard Deviations of Number of Millimeters of Paper Covered by the Count from one to ten in the Drug and Placebo Conditions	66
16	Mean Perceptual Reactance Scores of the 5 Most Extreme Introverts Compared with the 6 Most Extreme Extraverts in the Placebo Condition	68
17	Mean Extraversion Scores on the Eysenck Personality Inventory of the Most Extreme 3 Reducers and the Most Extreme 2 Augmenters in the Placebo Condition	69
18	"t" Test Made Between Perceptual Reactance Scores of First Testing and the Same Scores on Later Retesting	71
19	Mean Number of Pauses During Finger Tapping, and Mean Number of Taps in a Minute of Extreme Extraverts and Extreme Introverts	73
20	"t" Tests Made Between Subjects who Relatively Augmented on the Drug and Those who Relatively Reduced on Three Measures	75
21	"t" Test Made Between Extraversion Scores of First Born and Later Born Sons	77
22	Sample Record of a Reducer as Given by Petrie	81
23	Sample Record from this Study	82

LIST OF FIGURES

Figure		Page
1	Schematic Representation of Testing Procedure.....	40
2	Distribution of Extraversion Scores.....	42
3	Distribution of Perceptual Reactance Scores.....	44
4	Otis Subtest Scores.....	53
5	Pulse Rate.....	60

CHAPTER 1

INTRODUCTION

Perceptual Reactance and Personality

The terms "perceptual reactance", "figural after-effect", and "adaptation with negative after-effect" all refer to very similar phenomena. Gibson (1933) was the first to work in this area; it was he who used the term "adaptation with negative after-effect". Kohler and Wallach (1944) later used the term "figural after-effect". Petrie (1967) originated a new term which she called "perceptual reactance". Perceptual reactance will be the term used here because Petrie's book Individuality in Pain and Suffering is probably the most complete summary of the research in this area, and is likely to be a standard reference for some time. In addition, perceptual reactance has a more specific meaning than earlier terminology, as it refers not to after-effects in general, but to those related to the apparent magnitude of the stimulus in question.

Perceptual reactance refers to the amount to which a given individual tends to augment or reduce incoming stimuli. For example, the size of a block of wood held in the hand of a blindfolded person does not seem to remain the same, but gradually changes. Augmenters tend, in their perception, to enlarge the object. Reducers tend gradually to perceive it as smaller. Given individuals tend to be very consistent in the degree to which they augment or reduce their perception of the size of an object, says Petrie. Some people, whom she calls

moderates, neither particularly augment nor reduce the perceived size of objects. The range from augments to moderate to reduces represents an approximately normal distribution for various samples she has tested.

That the tendency to augment or reduce the apparent size of objects is consistent has been reported by Petrie, who using a split-half measure of reliability found correlations between the halves of .97+ for two small samples. Spitz and Lyman (1960) report a test-retest reliability of +.74. Still, this would be a matter of no great importance in itself unless this tendency seemed to represent a generalized pattern of receiving and responding to stimulation. Petrie's original interest in this area was stimulated by observations of individual variations of people in response to pain. Two individuals having similar injuries may differ dramatically in how they respond to the pain that each feels, which is clearly a function of more than the physical injury itself. In 1952 Petrie noted that pain tolerance could be changed by surgery, and that there was a corresponding personality change. She hypothesized that pain is the equivalent of sensory excess; it follows that a person who characteristically augments incoming perception would have a lower pain tolerance than one who characteristically reduced it. On the other hand, one would predict that the discomfort felt from sensory deprivation would be much more acute on the part of the reducer, as such an individual is in effect moderately sensorily deprived in his normal state.

The first actual test of the relation of pain tolerance to augmentation or reduction as measured kinesthetically (using the perceived size of wooden blocks) was done by Petrie, Collins, and

Solomon (1958). The results were as predicted, augmenters reporting the least pain tolerance, and reducers reporting more than either augmenters or moderates. This result has been verified by Poser (1960), Ryan and Kovacic (1966), and Ryan and Foster (1966). But whereas reducers and augmenters differed in pain tolerance, they did not differ on pain threshold. Similarly, while prefrontal lobotomy increases pain tolerance, it does not reduce pain threshold (Petrie, 1952).

If a high pain tolerance is the result of a tendency to minimize incoming stimulation, the same individual who has this high tolerance should be relatively intolerant of situations in which incoming stimuli are minimized. Petrie (1967, p. 28) reports on the results of two groups of subjects subjected to sensory deprivation in an iron-lung type apparatus. In this situation, as expected, reducers could not tolerate the sensory lack nearly as well as augmenters. The difference between augmenters and reducers was significant at the .01 level. It was noted that those subjects who were unable to withstand much sensory deprivation believed that they could withstand pain better than most people, and the opposite was true for those who withstood deprivation relatively well.

It would be reasonable to predict that reducers generally would hold up less well than augmenters in any kind of situation involving inactivity or boredom. Petrie (1967, p. 29) reports on a study comparing a group of pregnant women on two dimensions; how well they withstood their confinement just prior to childbirth, and how well they withstood the pain of actual childbirth. One could predict just the opposite results for augmenters as for reducers. Augmenters should

tolerate the confinement rather well, but the pain at the time of birth not very well, and conversely for reducers. Tolerance of the confinement was measured by ratings of hospital staff on the amounts of complaints and general uncooperativeness of the women. The pain experienced with actual birth was rated by the doctor and nurse present at that time. In no case were the raters aware of perceptual reactance measures on these subjects. Again, the results were as expected and the level of significance high (.01).

Solitary confinement is a situation toward which one could readily predict differential tolerance between one person and another. Some individuals give every indication of liking this state, whereas others will turn toward inflicting pain on themselves as the preferable alternative to no stimulation at all. Petrie (1967, p. 30) noted that among boys in a reformatory in Massachusetts where solitary confinement was sometimes used, every reducer said that, given a choice, he would prefer pain to confinement.

Perhaps one of the most significant questions about perceptual reactance is whether it is primarily constitutional or environmental in origin. There is no definite answer to this question as yet, but there is much relevant information concerning it. Melzack and Scott (1957) reported on a dog that had been reared under conditions of extreme sensory deprivation. The dog would curiously stick his nose into the flame of a lighted match, giving no indication of feeling any pain at all. It is possible that a chronic insufficiency of stimulation during the course of development might produce such a need for compensatory stimulation later that what would ordinarily be perceived as painful is

interpreted only as interesting and perhaps satisfying. The development of masochistic personalities might be related to this, and one might predict that reducers would be more likely to fit into this category.

At least one kind of atypical perceptual reactance appears to be one that is "trained out" in the ordinary course of development, and appears only rarely in normal subjects. This type is called by Petrie the "stimulus governed", and appears in approximately 17% of juvenile delinquents. Normally the estimation of the size of wooden blocks is tested with blocks of two sizes, presented alternately. A stimulus governed response is one in which the subject tends to change his estimation of the size of these blocks toward some mean point in between them. Instead of consistently estimating both blocks to be larger or smaller than they actually are, the large one is estimated to be smaller, and the smaller one is estimated as larger. A person with this kind of perceptual reactance would presumably perceive things in his environment in a much less stable fashion than the "normal" type. Petrie reports that stimulus-governed delinquents have been involved in more delinquencies than have controls, and are more often described as immature and unpredictable. She also provides evidence that normal children display progressively less tendency to be stimulus-governed as they age (Petrie, 1967, p. 76-77). Other evidence that contrast effects become trained out with experience is that they are seen significantly less often in the dominant rather than the non-dominant hand (Petrie, 1967, p. 79). Fatigue is also a factor with increasing fatigue increasing the likelihood of stimulus-governed responses.

There is considerably more evidence, however, that perceptual

reactance affects learning rather than vice versa. Frank (1961) found that delinquents were less conditionable than normal controls. It seems likely that reducers would condition less readily than augmenters since they would be less affected by the same objective stimulation. Similarly, one might predict that reducers, who do not tolerate sensory lack very well, would be less able to tolerate the restrictions of the usual school situation. Augmenters, on the other hand, should have less difficulty with this kind of situation since they receive relatively more stimulation from the same setting. Petrie's (1967, p. 86) summary of her findings comparing delinquents and controls are quite striking:

"..(1) juvenile delinquents were twice as likely to be pronounced reducers as were those in the control group; (2) conversely, controls were three times as likely as juvenile delinquents to be pronounced augmenters; and that (3) there was a subcategory of delinquents whose reduction was so extreme that no control subjects were comparable."

Petrie (p. 101) also reports the results of a study comparing augmenters and reducers with respect to the grades received in school by presumably normal children. As expected, augmenters had the highest grades, moderates less high, and reducers least high of all three groups.

Petrie makes surprisingly little reference to H. J. Eysenck considering the degree to which the work of these two individuals overlaps. Before going on to discuss drug effects on perceptual reactance, it would be appropriate to discuss in some detail Eysenck's notions of inhibition/excitation and introversion/extraversion. Eysenck's book Crime and Personality (1964) closely parallels Petrie's book, and the book Experiments with Drugs (1963), which he edited, is

particularly relevant to this study.

Eysenck is considerably more theoretical than Petrie, sometimes, it would seem to this writer, unnecessarily so. He is fairly comfortable with Hullian terminology and refers to it more often than is agreeable to most current readers, since Hull has lost much of his past popularity. Most significant are the terms "inhibition" and "excitation", first used by Pavlov (1927) and elaborated on by Hull (1943). Eysenck admits at the outset that these terms cannot be precisely defined, but he finds them useful nonetheless and gives them tentative definitions.

Inhibition, says Eysenck, "refers to a process within the central nervous system which interferes with the ongoing perceptual, cognitive, and motor activities of the organism". There are two main types of inhibition: temporal and spatial. Temporal inhibition refers to "accumulation of a performance decrement as a result of the performance itself", and corresponds to reactive inhibition as postulated by Hull. Eysenck believes that such findings as those on reminiscence phenomena, adaptation, and the maintenance of vigilance in attending are all a function of temporal inhibition. Spatial inhibition refers to distraction; i.e., to performance decrement as a result of the interference of some other stimuli. Extinction is considered to be the result of spatial inhibition.

In contrast with inhibition, excitation "refers to a process within the central nervous system which facilitates the ongoing perceptual, cognitive, and motor activities of the organism". It is considered by Eysenck that each person has some characteristic inhibition/excitation balance, and that his personality type -- particularly the extraversion/introversion dimension, is a function of this

balance. Excitation is essentially synonymous with arousal, presumably centrally determined.

During the course of everyday activity, sensory and proprioceptive stimulation sets up stimulus-produced inhibition which dissipates at varying rates. Subsequent stimulus-produced effects are altered according to how much the inhibitory effects of the earlier stimuli are dissipated. The inhibition/excitation balance, then, determines how much we react to incoming stimuli. In short, it determines our perceptual reactance. Though this type of explanation might seem unnecessarily abstract, Eysenck does manage occasionally to put it to good use. He begins by suggesting a fairly direct connection between inhibition/excitation and the personality dimension of extraversion/introversion. Extraversion, he says, is the result of strong and persistent inhibitory processes, with weak and irregular excitatory ones. For introversion the reverse is true. Introverts have strong excitatory processes, with weak inhibitory ones, and so they react more to the same objective stimulation.

Eysenck describes at some length the so-called typical extravert and introvert as follows:

"..the typical extravert is sociable, likes parties, has many friends, needs to have other people to talk to, and does not like reading or studying by himself. He craves excitement, takes chances, often sticks his neck out, acts on the spur of the moment, and is generally an impulsive individual. He is fond of practical jokes, always has a ready answer and generally likes change; he is carefree, easygoing, optimistic and likes to 'laugh and be merry'. He prefers to keep moving and doing things, tends to be aggressive and loses his temper quickly; altogether his feelings are not kept under tight control and he is not always a reliable person.

...the typical introvert is a quiet, retiring sort of person, introspective and fond of books rather than people; he is

reserved and distant except with intimate friends. He tends to plan ahead, 'looks before he leaps' and distrusts the impulse of the moment. He does not like excitement, takes matters of everyday life with proper seriousness and prefers a well ordered mode of life. He keeps his feelings under close control, seldom behaves in an aggressive manner and does not lose his temper very easily. He is reliable, somewhat pessimistic and places great value on ethical standards."

It is from these and similar characatures, factor analytically derived, that various questionnaires have been developed for determining a given individual's rating on the extraversion/introversion dimension. Probably the most important such questionnaire in this context is the Eysenck Personality Inventory, developed by Eysenck and Eysenck. If such personality types really do correspond to some kind of constitutional level of arousal, there should be predictable differences between the two types. Here the overlap of the extraversion/introversion dimension with Petrie's reduction/augmentation continuum becomes obvious.

Objectively equal amounts of stimulation, says Eysenck, should not be experienced as equal by extraverts and introverts. He therefore predicts: (1) that extraverts will show greater pain tolerance than do introverts; (2) that extraverts will show less tolerance for sensory deprivation than do introverts; and (3) that extraverts will show shorter perceptual after-effects. He makes other predictions, some of which will be referred to later. The question here arises as to the possibility that the factors discussed by Eysenck and Petrie might be identical. At the least, they are very similar, and the references used to support each overlap a great deal. With reference to pain tolerance, studies by Petrie (1960), Poser (1960) and Lynn and Eysenck (1961) have all found respectably high correlations

between pain tolerance and extraversion. Concerning the tolerance for stimulus deprivation, Petrie, Collins, and Solomon (1960) found the predicted results. Support for the prediction concerning perceptual after-effects is provided by the majority of the studies reported in Eysenck's book Experiments with Drugs.

In his discussion of optimum levels of stimulation Eysenck's approach becomes even closer to that of Petrie. He predicts that extraverts should experience a relative stimulus hunger in their usual state, whereas introverts should be inclined toward stimulus avoidance. Among the many relevant studies are the following: extraverts smoke more cigarettes (Eysenck et al., 1960), make larger physical movements (Rachman, 1961), and have more illegitimate children (S.B.G.Eysenck, 1961).

Petrie also presumes that perceptual types who are relatively insensitive to pain are less likely to be concerned about health hazards, hence, should smoke more. Also, the more a person is a reducer, the more likely he is to seek out socially acceptable ways of stimulating himself. Cigarettes might be one obvious choice. Among delinquents Petrie found that the age at which smoking began was significantly earlier for reducers than for augmenters. Also about 50% of the augmenters questioned said they did not enjoy smoking very much, whereas none of the reducer group expressed this feeling. Forty per cent of the reducer group had never attempted to quit smoking, and only 12% of the augmenters never attempted to quite (Petrie, 1967, p. 95).

It seems apparent that a person's characteristic way of reacting to stimulation would be likely to influence the development of his

personality. There is much evidence that the origin of perceptual reactance either is, or becomes, central in nature. It was earlier mentioned that there was a characteristic personality shift resulting from operations to change pain tolerance (Petrie, 1952). This shift is in the direction of increased extraversion. A similar shift does not occur in brain operations (like temporal lobectomy) which do not affect pain tolerance. It has been demonstrated by Petrie that reducers score markedly higher on extraversion scales than do augmenters, who appear correspondingly introverted (Petrie, 1967, p. 36).

A somewhat different theoretical approach to this area is provided by Michael Wertheimer (1955), especially in the article titled "Figural after-effect as a measure of metabolic efficiency". Wertheimer uses the Kohler and Wallach (1944) approach to explain after-effects. Briefly, the theory is that there is a "polarization" of cortical tissue during perception which changes subsequent perception. Figure-ground reversals, for example, can be considered the result of the rate at which such tissue is polarized (satiated). According to Wertheimer, figural after-effects depend on the ease with which changes in polarizability occur, and that this depends on "metabolic efficiency". Higher efficiency should produce larger figural after-effects. Unfortunately his measures of metabolic efficiency are all rather debatable; for example, he suggests that a mid-range basal metabolic rate is more efficient than either a faster or slower one. More convincingly, he shows a strong correlation between quick reaction time and size of figural after-effect, and also between

visual and kinesthetic figural after-effects. Still, these correlations are only about .50 to .60. Also it is difficult to compare his results to those of the majority of such studies, since his measure of kinesthetic figural after-effect is different than most.

Changes in Perceptual Reactance and Personality Change

The earlier part of this paper was an attempt to support the notion that perceptual reactance is a generalized way of responding to external stimuli, and that characteristic personality patterns emerge as a result of this perceptual style. This information, if valid, leads to at least two possible kinds of response. We can, by being aware of a given individual's perceptual style, shape his environment accordingly. An obvious example is to see to it that extreme reducers are not subjected in youth to schoolroom situations which they cannot possibly tolerate or learn from. Such individuals could gain much more from a relatively noisy environment. A second kind of response is to think in terms of changing a person's perceptual reactance. Brain operations are one way of doing so. Another way is to manipulate the amount of stimulation in the environment at the time of interest. A third is the use of drugs.

In describing how to take measures of kinesthetic perceptual reactance, Petrie greatly emphasizes that stimulation experienced before testing can cause spurious results. Subjects are required to sit for some time without using their hands prior to testing in order to minimize extraneous influences. Perceptual reactance also seems to vary according to the time of day (fatigue), sickness, menstrual changes, and

a host of other factors. Particularly for the augments, it seems that a short period of intense stimulation reduces augmentation considerably. A subject coming in to be tested from a snowstorm at zero degrees is likely to score spuriously low if he is an augments. Perhaps the most impressive work on this topic is that of Petrie on audioanalgesia (Petrie, 1967, pp. 52-56).

Audioanalgesia refers to a technique of reducing the pain felt by dental patients by bombarding them with white noise through earphones. The technique has not gained overwhelming popularity because only some people seem to benefit from it, whereas some find the pain of the dental work to be preferable to the noise. Petrie predicted that stimulation with the white noise would cause a defensive reduction in perceptual reactance among augments, and relatively little change on the part of reducers. After being subjected to the noise, subjects are tested in their estimation of the size of wooden blocks. Her results are quite impressive; the difference between the scores of the augments before and after sound stimulation were different at a .001 level of probability in favor of the hypothesis. Yet there was no difference among the reducers or the moderates. The observed change in augments persisted for at least a quarter of an hour after the stimulation. Among this group, the greater the individual's usual augmentation, the more is his defensive reduction as a result of the noise. It is possible that the augments may develop ways of controlling his relatively exposed sensitivities, to produce a temporary and perhaps relieving reduction in subsequent sensation. It is interesting to note in this regard that the schizophrenics studied by Petrie appeared to be fairly often strong augments who

seem to have "frozen" into a state of defensive reduction, which again reverses itself with recovery. Many of those schizophrenic patients who were tested kinesthetically periodically changed from strong augmentation to strong reduction. Schizophrenics are generally considered remarkably pain resistant. One could note that Van Gogh, as his mental state deteriorated, painted pictures with progressively more intense colors; and that schizophrenics in general tend to use very intense colors in art works.

On the other end of the continuum, one could predict that individuals who develop with some kind of objective sensory lack (like the deaf) might be more likely to augment their other sensory intake as a compensative factor. Petrie reports very convincing evidence of differences here (1967, p. 68). Of a group of subjects born deaf, none were reducers; but subjects who became deaf later in life did not differ from normal samples. So it appears that subjects may adapt both to excess stimulation, or to an insufficiency.

There is perhaps no better study to illustrate the actual distinctness of supposed extraverts and introverts than a simple study done by Spielman and reported by Eysenck in Crime and Personality. It was predicted that if extraverts really have strong "reactive inhibition", they should perform quite differently from introverts on a simple measure of finger tapping by making more pauses. The measure used in this instance was not the rate of finger tapping, as is more commonly the case, but the number of involuntary pauses occurring when the subject is tapping as fast as he can. Eysenck does not make it too clear exactly how the study was done, but it involved

tapping with a metal stylus on a metal plate, and used automatic recording devices. Data analyses on the results were hardly required; there was no overlap at all between the extraverts and the introverts. During a one minute period the average number of pauses for the extravert group was eighteen; for the introverts, the average was one. Here is a case where Eysenck's theorizing seems to contribute something in addition to what Petrie could offer in explanation of such results. Presumably reducers have more involuntary pauses. Eysenck would undoubtedly suggest that such subjects are reducers because they are slower in counteracting reactive inhibition. With regard to the kinaesthetic measure (Petrie's block test), Petrie says that the longer subjects feel the stimulating block, the greater is the reducing effect (in reducers), whereas a rest pause counteracts this effect.

Both Eysenck and Petrie are concerned with how the perceptual differences they discuss affect the formation of personality. Petrie says that juvenile delinquents are more often reducers; Eysenck says that adult criminals are more often extraverts. It is not difficult to imagine what might be the connection. If incoming stimuli are reduced, or have less effect, in some individuals, they would presumably have less reason to pay attention to these stimuli. This might hold true for the rewards and punishments used by parents to shape the behavior of their children, as well as for simpler cases of classical conditioning. Cleckley (1950) suggests that there is a "semantic personality disorder" accounting for why the disturbed do not respond to verbal reinforcement. But Eysenck (1963, p. 15) reports a study in which extraverts and introverts were conditioned using the eye-blink response to a puff of air. As he predicted, introverts

conditioned much faster than did extraverts, and the differences were statistically significant. This would suggest a general under-reactivity to external stimulation rather than a semantic problem.

Drugs may also be sources of change in perceptual reactance. The terms "stimulant" and "depressant" drugs have persisted in spite of their obvious ambiguity. There is no drug which is purely stimulating or purely depressing, and a drug may, for example, cause psychological stimulation by depressing the function of some organ or part of the nervous system. In spite of this ambiguity, some drugs do seem to be a great deal more stimulating than depressing, or vice versa. Meprobamate, for example, tends to reduce physical activity, decrease the amount of speech, and possibly produce sleepiness in most subjects. Dextro-amphetamine (d-amphetamine), on the other hand, more often increases verbal output, willingness to engage in physical activity, and may even produce mild insomnia. Unfortunately it should be remembered that there are many drugs which are much more ambiguous in their effects.

Eysenck suggests that drugs influence the excitation/inhibition balance. To be more specific, he says that stimulant drugs increase excitation and have an introverting effect, while depressing drugs increase inhibition and therefore have an extraverting effect. If it is true that the extraversion/introversion continuum is correlated with such things as pain tolerance, kinesthetic perceptual reactance, etc., then drugs may change personality by changing perceptual reactance. There is much evidence that this is true.

Petrie (1967, pp. 91-93) reports on an interesting and relevant

study on alcoholics. Alcohol is a common depressant. It might be predicted that a reducer, whose incoming stimulation is already relatively low, would be less likely to like drinking alcohol than moderates or augmenters. Petrie tested a group of alcoholics in a hospital, faced the difficult task of making sure that her subjects were not drinking on the ward (which would presumably make them appear to be reducers) and found the expected results. Almost every subject was an augments or a moderate. The rest were of the stimulus-governed type, and none were reducers.

Alcohol has long been used for an anaesthetic, i.e., to increase pain tolerance. One might predict, therefore, that it would cause subjects to reduce more on measures of kinesthetic perceptual reactance. This was also tested by Petrie, and the evidence was favorable. Analyzing data by groups on the augmentation/reduction continuum, however, produces most interesting results. It appears that in reality only the augmenters were affected by a dose of 2 ounces of alcohol. The difference between augmenters tested before and after drug was significant at the .001 level; but there was no difference among moderates, and only a slight difference among reducers in the opposite direction as among augmenters. Reducers tended to reduce somewhat less. The conclusion, if Petrie's reasoning is correct, is that alcohol should be an anaesthetic only to augmenters. If this kind of effect is found with other drugs, it would throw considerable light on the results of many drug studies. All too often doctors will observe clinically apparently dramatic results in patients given some drug. Yet when a controlled study is done, with random assignment of subjects to treatment groups, the originally impressive results fail

to appear. This could be the result of combining within one group of subjects people of different constitutional types, only one of which is affected in the way desired. If other subgroups of subjects are not affected, or are affected in the reverse way, an overall difference among the groups may not appear.

Petrie predicted that alcohol would not only reduce the effect of pain, but of physical discomforts in general. She tested subjects with and without alcohol for the length of time that they could hold their leg out without support. If this capacity is reduced by the psychological sensation of fatigue, and if this is reduced by alcohol, the ability should be increased. This was found to be the case (Petrie, 1967, p. 43). This is a particularly interesting result because it is a case where Eysenck's theorizing about reactive inhibition would lead one to expect just the opposite result of what was found. Alcohol should shift a person more toward the extraversion end of the extraversion/introversion continuum and increase the effects of reactive inhibition. More compatible with both theoretical approaches is the common observation that people tend to speak louder as they drink more. This could be the result of decreased augmentation, such that the person is merely correcting for the fact that his own voice now sounds less loud to him. Also Petrie (1952) has noted that the effects of alcohol are sometimes similar to the effects of frontal lobotomies, which again seem to shift the personality toward extraversion. Among other things, both lobotomies and alcohol increase pain tolerance and decrease psychological inhibitions.

Some years ago when frontal lobotomies first became popular, the results seemed to be very favourable and the method came all too close

to being a standard method of treatment. Later, objective studies provided little support for their efficacy. Why the change?

Petrie suggests that it might have been due to a selection bias in the early years in favor of augmenters. Augmenters, having a lower pain tolerance, are more likely to display symptoms in need of more drastic treatment. Also the operation appeared more successful with cases of anxiety and depression, more common in introverts. This could parallel the earlier observation made here about drug studies; i.e., early treatment of a select population looks very favorable, but later studies with a more mixed population show no objective difference between treatment and no treatment.

With alcohol, of course, there is such an elaborately developed social ritual in much of its use that it could always be argued that it is the latter factor which produces the result. This is not the case with aspirin, however, yet the results are closely parallel. Petrie (1967, pp. 46-48) tested subjects for kinesthetic perceptual reactance one-half hour after aspirin or placebo. The results, even with a small sample, were marked. Augmenters as a group almost totally ceased to augment, but moderates and reducers did not change. The results for the augmenters were so marked that there were overall group differences at the .02 level. Here is a clear case where a generalization like "aspirin is an effective pain reliever", though shown "objectively" in an experiment, is clearly misleading. Most of the population would not find the statement true, at least in its personal application. More important, it would seem that by knowing a person's characteristic perceptual reactance, one could predict with great accuracy whether or not aspirin would benefit a given individual; and this should be true

without any "physiological" measures. If the same were true of many other drugs, it should be possible to make much more accurate predictions about the effects of a given drug than has heretofore been the case, at least on some measures. It should also provide us with a much better understanding of drug-environment interactions, which are poorly understood at present.

With regard to the effects of d-amphetamine, for example, it was early thought that the drug's toxicity was remarkably low. The LD₅₀ level (dose causing death in 50% of subjects) in mice, however, was found to vary widely, and it was some time before the difference was recognized to be a function of whether the rats were kept in individual or group cages. Moore (1963), for example, found the LD₅₀ level for d-amphetamine to be 25 mg./kg. in rats kept caged in groups of 4. The corresponding figure for animals caged alone was almost four times as high (97 mg./kg.). It has been commonly observed that many animals appear to be greatly stressed if the population density exceeds a certain level, and if it becomes too high large numbers of animals may begin to die from such common stress indicators as ulcers. A recent study by Swinyard et al. (1961) analyzed this matter in some detail. Their LD₅₀ for isolated mice was 125 mg./kg. For rats kept in groups of 3 in a small cage the correspondent figure was 35 mg./kg. This study also tested the effect of varying the amount of time the animals had been kept together prior to administration of the drug ($\frac{1}{2}$ to 4 hours), and found that the fatality rate from the drug was greater the less the amount of time the animals had to adapt to being kept in groups. How might this be explained? In Petrie's terms, the stimulant drug

should cause an increase in perceptual reactance, i.e., incoming sensory stimuli become stronger. Consequently a situation in which there is already a relative excess of external stimuli (from the other animals) becomes even more exaggerated with the effect of the drug. The stress, for some animals, is too much to withstand. Of course, the dose levels in question were very dramatically larger than one would ever use with human beings. The point is that the environmental setting changes the obtained results to a very marked degree.

To extrapolate to human beings, one could think of the personality dimension extraversion/introversion as a direct parallel to the environmental dimension. When one manipulates by choosing to use extraverts in a study, for example, is in principle similar to choosing to do the experimental testing in a soundproofed room. There may be favorable or unfavorable combinations of personality and setting. Suppose, for example that an augments (introvert) becomes sick and is hospitalized in a room with a number of other patients and is, for some reason, given a stimulant -- causing him to augment even further than he would normally do. Or, a more likely case, suppose a reducer (extravert) is given a sedative and put into a quiet room by himself. If the sedative in question really did cause further reduction, the result might almost amount to a sensory deprivation situation. For example, Colquhoun and Corcoran (1964) have demonstrated some environmental effects on the performance of introverts and extraverts on a speeded test consisting of crossing out the letter "e" in some prose material. All subjects were tested both in isolation and in groups. In the isolated condition introverts did better; in the group condition

extraverts did better.

It is possible, of course, that depressants are commonly like alcohol and aspirin, i.e., that they do not cause reducers to reduce even further, but have their effect primarily on augmenters. The reverse could be true for most stimulants. There have not yet been enough studies of this type to make very reliable generalizations. But it may be possible to use drugs much more wisely if there are simple ways of predicting what kind of effect it will have on a certain kind of person.

Arousal, Perceptual Reactance, and Personality

"Arousal" here refers in general to the effects of stimulant drugs such as d-amphetamine, the so-called sympathomimetics. Their effects are presumed to be similar in effect to stimulation of the mesencephalic reticular formation, though whether this is the major or only effect is highly debatable. Fuster (1958) noted that such stimulation induced directly by electrode implants decreased the reaction time and increased the number of correct responses of monkeys to a tachistoscopically presented discrimination problem. These effects are very similar to those often achieved by use of stimulants such as d-amphetamine. Uyeda and Fuster (1962), for example, found the same effects on the same learning task as did Fuster, only as the result of amphetamine. Studies indicating that amphetamine action is mediated by the kind of stimulation described by Fuster include Bradley and Elkes (1957) and Longo and Silvestrini (1957).

It seems likely that arousal in this sense may have much to do with the inhibition/excitation balance as discussed by Eysenck, and

perceptual reactance as discussed by Petrie. The purpose of this study is to clarify these relations, if they exist, and to examine their effects on personality in as far as these are manifested over a short period of time. The reasoning behind predicting such relations will be discussed here in some detail.

The effects of the treatment referred to in the Fuster study mentioned above is primarily cortical stimulation. It has long been known that the cerebral cortex has as one of its main functions the inhibition of unconditioned reflex activity. It is also known that the process of maturation involves a gradually increasing degree of such control. In the case of the mentally retarded, such controls fail to develop to the same extent as in normals. Enuresis, for example, is more common among children with EEG patterns like those of immature children (Hodge and Hutchins, 1952), and is quite common among the mentally retarded. Enuresis tends to occur in children when they are asleep (reduced cortical inhibition), and is more common in children who sleep deeply (Molitch and Poliakoff, 1937). Administration of amphetamine prior to sleeping keeps sleep from becoming as deep as usual, hence more cortical activation, and less enuresis.

Enuretics also appear less able to learn in general, and form conditioned reflexes less easily (Leake, 1958). Amphetamine seems to ameliorate these factors as well as eliminate enuresis. Hyperkinetic children also appear to have an insufficient degree of cortical inhibition of reflexes, resulting in quick movements and extreme distractibility. In short, amphetamines (and perhaps many other stimulants) may produce the necessary degree of cortical stimulation for muscle responses to be slowed, keeping irrelevant stimuli from

immediately capturing the attention, and so improving the attention and response to a learning situation. There are many studies which have found improved performance resulting from amphetamines on tasks involving sustained attention (see Hauty and Payne, 1958).

Two particular facts are especially relevant to this study. First, it is commonly observed that there is a paradoxical effect from amphetamines. Hyperactive children frequently tend to slow down and become more coordinated. Nevertheless lethargic children tend to become more active. The connection between hyperactivity and perceptual reactance is not known, but it seems likely that there is a differential effect from arousal depending on the initial degree of cortical excitation. A second interesting fact was observed by Talland and Gardner (1966), in a study of the effects of methamphetamine (methedrine) on a task involving sustained concentration. Those subjects who improved most from methedrine were the same as those who deteriorated the most from pentobarbital. One explanation might be that these were simply the most drug sensitive subjects, and that the other subjects might have responded similarly if given a larger dose of either drug. Having already considered Petrie's work, however, it seems likely that the subjects in question were reducers, and that the effects achieved were due to changes in their perceptual reactance.

Another relevant study of particular interest is that of Epstein et al. (1968), using d-amphetamine. These authors used 10 subjects, 5 of whom had no known organic damage to the central nervous system, and 5 of whom had some kind of known organic damage. The organic group had shown hyperkinesis from birth, but the non-organic group, while also hyperkinetic, had become so later. Organic damage was due

to intracerebral hemorrhage, meningitis, cyanosis at birth, and premature birth. All subjects were tested for fine motor coordination, Porteus maze performance, and for performance on the WISC. Both the organic and non-organic groups improved significantly in fine motor coordination. Both groups also improved in Porteus maze performance, but the organic group improved significantly more than the others. The authors also found that the organic group appeared to tolerate higher levels of d-amphetamine than the others, and excreted it faster in the urine. The authors concluded that hyperkinetic subjects are heterogeneous, and that while d-amphetamine might be useful for the lot, it is especially useful for those children who have specific motor disabilities due to known organic damage. In this case the nature of the "organic damage" is not explicitly defined, but possible connections with the personality changes observed by Petrie are apparent.

If these differential effects do not apply, or at least do not apply very strongly, to the normal population, we would expect stimulants and depressants to produce certain predictable and opposite effects. For example, the rate of conditioning should be increased by stimulants and decreased by depressants. This has been found to be the case by Franks and Laverty (1955), Franks and Trouton (1958), and Willet (1960). We would also expect performance on vigilance tasks to be improved by stimulants and worsened by depressants. This has been supported by many studies, e.g., Felsing, Lasagna and Beecher (1953) and Treadwell (1960). The size of motor movements should be decreased by stimulants and increased by depressants; this was

supported by Rachman (1961). Pupillary reactions to light should be increased by stimulants and reduced by depressants; this was supported by Eysenck and Easterbrook (1960). Critical flicker fusion threshold should be decreased by stimulants and increased by depressants; among many studies supporting this is that of Holland (1960). There are many other predictions that could be discussed, all of which lend support to the idea that arousal affects the excitation/inhibition balance, perceptual reactance, and figural after-effects in general. In all of the above cited studies, however, subjects were not separated according to their original level of perceptual reactance. Arousal, then, may not only change perceptual reactance, but may change it, and hence personality, in different ways depending on its initial level.

Two studies of particular relevance to this proposal involve the use of amphetamines to facilitate intellectual performance (Vaness and Brown, 1966), and to change responses on the Bernreuter Personality Inventory (Turner and Carl, 1939). As early as 1936 improved intelligence test scores from the administration of amphetamine had been noted (Sargant and Blackburn, 1936), and an apparent increase in general efficiency was often reported by subjects taking the drug. Through the years many other studies were done testing this effect, and while many came out positive, many did not. A partial explanation for the inconsistent results might be that most of the early studies used racemic amphetamine (benzedrine) rather than dexedrine. Vaness and Brown (1966), using a double-blind design, found significant improvement in performance on the Otis Group Intelligence Test using

d-amphetamine. However another reason for the inconsistency might be a differential effect on subjects, improving the performance of some of them while not affecting others. Of the many studies on the effects on intellectual performance of amphetamines, the effect, if any, is almost always positive rather than negative. Studies finding a positive trend that is not statistically significant might have resulted from a combination of beneficial effects on some subjects and no effect on others. For example, Molitch and Eccles (1937) found significant improvement on only one of 3 intelligence tests, with non-significant improvement on the other two. Similarly Andrews (1940) found no significant changes in a syllogistic reasoning test, but the trends, in terms of either speed, accuracy, or efficiency were all positive. Curiously, Andrews also noted that those subjects who had the greatest blood pressure rise were those who improved the most, and he suggested an approach along idiographic lines.

Turning to personality measures, Turner and Carl (1939) tested the effect of benzedrine on attitude self-ratings and the Bernreuter Personality Inventory. Their results generally support the idea that different subjects are affected in different ways. With regard to increased optimism reported by subjects, the experimenters commented that: "The foregoing findings are true only 'on the average'. Individual differences in affective response to the drug are profound." Their comments about the lack of overall changes on the personality test are equally interesting:

"...the findings in the present study might appear to support the contention that personality traits remain unaffected by benzedrine ingestion. It seems to the present writers that the chemical does produce changes in behavior of a

temporary nature that would give the impression of (temporary) changes in personality traits. Since the response to benzedrine tends to be as individualized as it is, and since the Bernreuter Inventory appears to have rather severe shortcomings in validity with individual cases and fails to depict individual traits and their interrelationships, the Inventory as originally scored and interpreted does not seem a promising tool for ascertaining at least the more crucial effects of benzedrine on personality. The problem definitely needs further clarification, and, very likely, experimentation along idiographic lines." (emphasis added).

These writers go on to say that most subjects experience a definite enhancement of mood and an increased willingness to work for extended periods of time, but that other subjects fail to show these effects, and still others are affected in the reverse way. This problem is one that hopefully could be clarified if subject's responses were examined with respect to their original performance on the perceptual reactance dimension. Perhaps there is an optimum level of stimulation, as Eysenck believes, and a drug might be beneficial to the degree that it brings about an approximation to this optimal level.

Predictions to be Tested in this Study

Kinesthetic figural after-effect, or perceptual reactance, is, according to Petrie, one of the more stable measures of a person's general responsiveness to stimulation. She has also provided evidence that this measure changes under the influence of drugs such as alcohol and aspirin. Perhaps the safest prediction of this study then, should be that it would also change under the influence of d-amphetamine. The direction of change should be toward augmentation. Whenever possible, however, this change should be

determined separately for subjects initially classified as reducers, moderates, or augmenters. The greatest change is expected among those initially appearing to be reducers, with less effect on moderates, and little or no effect on augmenters. 1. Kinesthetic perceptual reactance, as measured by Petrie's block test, will increase with d-amphetamine, especially among reducers.

If incoming stimuli seem to increase in intensity, subjects could be expected to compensate accordingly. This lead us to the second prediction, which is that subjects should speak in a lower tone of voice while under the influence of d-amphetamine. An obvious complication here is that if the drug elevates mood or causes some emotional excitement, subjects might speak louder regardless of their level of perceptual reactance. To minimize the effect of the "secondary" mood elevation, subjects will be tested while reading a short list of numbers -- counting from one to ten -- as this seems most unlikely material to generate any emotional involvement on the part of the person reading it.

As before, the data here should be analyzed separately as far as possible, according to the subject's initial level of perceptual reactance. And again; the greatest change could be expected among reducers. 2. The amplitude in decibels of subjects' voices while reading a list of numbers from one to ten will be less under the influence of d-amphetamine than in the control condition.

Eysenck's discussion of the differential frequency of involuntary pauses between supposed extraverts and introverts while tapping leads one to expect that a stimulant should decrease the number of such pauses over the initial level. D-amphetamine should presumably shift

subjects toward the introversion end of the continuum, or increase reactive inhibition, or increase the excitation/inhibition ratio (depending on the preferred theoretical interpretation), with a concurrent decrease in the number of involuntary pauses. Once again, the largest change should be expected among the reducers. 3. The number of involuntary pauses during finger tapping will decrease with d-amphetamine.

If the expected increase in perceptual reactance occurs, d-amphetamine should cause a shift toward the introverted personality type. This is probably the most problematic of the predictions made here, since personality measures typically measure long-term inclinations rather than immediate feelings. Yet a positive finding here, while the least likely prediction to be supported, would perhaps be the most impressive finding if it occurred. It would imply that the drug not only produces momentary changes in perceptual reactance, but that these effects generalize broadly enough to change what are usually fairly constant personality measures. Here again, the greatest change should be expected among those initially classified as reducers.

4. D-amphetamine will increase introversion scores and decrease extraversion scores on the Eysenck Personality Inventory.

Intellectual performance under the influence of amphetamines has long been a subject of contention. Since there is much evidence that the affective response to the drug differs from subject to subject, it seems quite likely that intellectual changes are similarly variable. Though the previously mentioned study by Vaness and Brown provides fairly convincing evidence that facilitation will occur, on the average, no previous study has attempted to predict what these effects

will be for different subgroups of the subjects tested. It is to be expected, according to the line of reasoning previously developed that reducers should be the ones to noticeably improve their performance under the drug condition, with less improvement for moderates, and the least, if any, for the augmenters. 5. D-amphetamine will improve scores on the Otis Group Intelligence Test, especially among subjects previously determined to be reducers.

Only the fifth prediction follows directly from past research as well as through the mediating theories of either Eysenck or Petrie. If it holds, it may be possible to demonstrate that the effect is not equal in degree for all subjects, but is instead a function of change in "perceptual reactance" or the "inhibition-excitation ratio" -- presumably overlapping constructs. Perceptual reactance change would be fairly directly demonstrated by the predicted effect of the drug on Petrie's block test, and more indirectly by the change in voice level. A change in the inhibition/excitation ratio would be indirectly demonstrated by the predicted change in involuntary pauses while tapping. From either predicted change it would follow that there should be a shift toward introversion, hopefully demonstrable by lowered scores on the E scale of the Eysenck Personality Inventory.

CHAPTER 2

METHOD

Subjects

Subjects were 36 male volunteers enrolled in either a first or second year psychology course during the 1969 summer session at the University of Windsor. The age range was restricted to between 21 and 34 years. The 21+ requirement eliminated the need for parental consent, and the upper level was chosen arbitrarily so the age range would not be very large. Subjects with a history of either heart trouble or high blood pressure were excluded, as well as those who are currently taking any drug other than caffeine or nicotine, as these might interact with amphetamine. Hyperthyroid subjects, were also excluded, as well as any overtly anxious person. All volunteers were asked to refrain from using alcohol on the evening prior to a testing day if possible, and to consume as little as possible otherwise. They were told that they would not be allowed coffee, tea, coke, or cigarettes prior to testing on experimental days. They were also encouraged not to hide the fact if they violated such restrictions, but to inform the experimenter so testing could be scheduled at another time.

Some of the subjects for this study were encouraged to participate by the offer of being allowed to drop their lowest test grade in the course they are taking in the psychology department. It

was hoped that this procedure would make sure that the supply of "volunteers" was sufficient, and that the expected bias toward extraverts of asking for subjects with no such compensation might be minimized.

Apparatus

To measure kinesthetic perceptual reactance an exact duplicate of Petrie's block test was used, with the exception that only one stimulating and measuring block was used rather than two. Petrie indicates in her book that this is an acceptable short form of the test when used with "normal adults". The equipment used included a $1\frac{1}{2}$ inch measuring block; a $2\frac{1}{2}$ inch stimulating block; a stand to be used for both of these blocks; a tapered block; and a blindfold. The stimulating, measuring, and tapered blocks are all equipped with finger guides. This apparatus is illustrated in Petrie's book. A more exact description can be found in Petrie's book Individuality in Pain and Suffering.

To measure the amplitude of subjects' voices, a microphone was placed 24 inches from the subject's mouth on the far side of a table in front of him. This microphone feeds into a log audio coupler of an Offner Dynograph, which makes a continuous recording on graph paper representing the volume of the person's voice. Calibration of this graph in decibels was set in advance by using a Hewlett-Packard Audio Signal Generator (model 250AG), which can be used to produce sound of known decibel rating.

To measure involuntary pauses during finger tapping, subjects tap with their index finger on a telegraph key. The telegraph key is

connected to a battery which produces a small current with each tap, which is picked up by the Offner Dynograph and recorded as a blip on graph paper. The graph paper moves at a rate of 10 millimeters per second, so if there is an involuntary pause there will be a space without a blip for whatever the duration of the pause. Generally pauses are rather obvious, as the space without a tap is very noticeably longer than the space between taps. For those cases which are not so obvious, criterion for a pause was set at 2 millimeters or more without a blip. Connected to the telegraph key is a small light which blinks on whenever a tap is completed. Though subjects were told nothing about this light, it was hoped that this device would minimize the chance that subjects might have incomplete taps in which the key is depressed, but not enough to be recorded on the dynograph.

To measure changes in the extraversion/introversion dimension, the Eysenck Personality Inventory was used. This test is an updated version of the earlier Maudsley Personality Inventory, and has 3 scales: 1 - an E (extraversion) scale; 2 - an N (neuroticism) scale; and 3 - an L (lie) scale. There are two forms of this test (A and B), which can be counterbalanced for the drug and no drug conditions. The instructions for the test are printed right on it to be read by the subjects themselves.

To measure changes in performance on an intelligence test, two forms (A and B) of the Otis Group Intelligence Test were used. The administration of the two forms was counterbalanced for the drug and no drug conditions. There are ten subtests in the Otis, each with a separate time limit. It is thus possible to measure not only an overall effect, but the pattern of performance changes on the subtests with the drug, if any. The entire test takes about an hour to administer.

Due to a slow response from the largest producer of commercial d-amphetamine (dexedrine), hand made placebos were used. These were made by grinding up 5 milligram dexedrine tablets and putting the result in a dark green gelatine capsule. Placebos were made by filling the same capsules with lactose powder, a fairly standard substance for use as a placebo. The dexedrine tablets were supplied by Dr. Norman Fretz, who also acted as the medical advisor to this study.

Procedure

Subjects were required to appear for testing on four occasions, all during a period of one week. Half of the subjects were tested on Monday, Wednesday, Friday, and Saturday; and the other half on Tuesday, Thursday, Friday, and Saturday. Testing on the first two appearances began at either 1, 2, 3, or 4 o'clock, and was the same for each person on these two occasions. Time of testing was counterbalanced for drug and placebo conditions. These first two sessions were conducted individually. The last two sessions, on Friday and Saturday, included all the subjects for that week, tested in a group at the same time (12:30 P.M.).

A double-blind design was used, neither experimenter nor subject knowing on which day the real drug had been administered as opposed to placebo. Tests used during the first testing session were identical to those used on the second, only the drug condition being changed. The same was true for the third and fourth testing sessions. Subjects who took the drug on the first day also took it on the third; subjects who took the drug on the second day also took it on the fourth.

During the first two sessions testing included measures of kinesthetic perceptual reactance (Petrie's block test), the finger tapping test, and the test for loudness of voice while counting from 1 to 10. Since a delay is required for the drug to take effect, subjects were allowed to leave the testing room after taking the appropriate capsule and asked to return promptly 45 minutes later. They were then required to sit without using their hands for an additional 45 minutes in order to eliminate the effects of previous use of the hands on perceptual reactance. At the end of this period the subject were blind-folded and the block test began, using a $2\frac{1}{2}$ inch stimulating block and a $1\frac{1}{2}$ inch measuring block. Instructions were very nearly identical to those used by Petrie, as reported in her book.

Briefly, the task presented to the subject was to estimate the size of a wooden block held between the thumb and index finger of the right hand. The subject estimated its size by finding a place on a tapered block that felt just as wide as the block in his right hand. He was asked to do this as quickly and accurately as he could, using his left hand on the tapered block which gets wider the further his hand is moved outward. After a series of baseline estimates were made, the subject was then asked to rub another block (in this case a larger one) with the same two fingers of his right hand, for periods ranging from 90 seconds to 2 minutes. Then further estimates of the size of the original measuring block were made. Presumably the intervening stimulation will cause the subject either to reduce or augment his original estimate, whichever happens to be his natural

propensity. All of this was done blindfolded, so the subject did not know that the block he was feeling was the same each time. Estimates are recorded as the number of inches that the subject moves up the tapered block. Petrie suggests that an average increase over his baseline mean of 1.8 inches or more (using both large and small block stimulation, which is not the case here) be labelled an augments, and that an average decrease of 1.8 inches or more be the criterion for a reducer. According to her testing, these criteria will divide a normal population into 3 equal parts -- augments, moderates, and reducers. Since only large block stimulation was being used here, however, it was not possible to use these criteria. An illustration of the testing apparatus and a more complete description of the procedures involved can be found in Petrie's book.

After the block test was completed, the subject was next asked, before removing his blindfold, to estimate the length of a minute. The experimenter said: "Starting from the time I say now, tell me when you think a minute has passed". The subject was then asked to remove his blindfold.

After the subject removed the blindfold, the experimenter placed in preset positions the telegraph key, microphone, and a board which was used to raise the person's forearm to about the same height as the key. Instructions were as follows: "Please place your forearm on the board in front of the telegraph key as if you were going to tap with your index finger. What I'm going to ask you to do is to tap with your index finger without raising either your wrist or elbow from the board. Starting from the time I say now, please tap as fast as you can until I say stop." As was the case with the voice volume test which follows, the

experimenter was not in the room when the subject carried out the instructions. However the subject was viewed through a one-way glass to insure that he carried them out correctly.

Next the subject was asked to sit upright in his chair. Afterward he was told: "Starting from the time I say now, please count from 1 to 10 in whatever you consider a normal tone of voice." Immediately after the subject did the counting, the recording was calibrated by using the sound generator to set the levels for 70, 80, 90, and 100 decibels on the graph paper.

On each of the first two testing days the pulse rate of each subject was taken by hand four times. The first reading was taken within a few minutes after the subject arrived for testing. His pulse was not taken immediately as such factors as walking quickly to arrive at the testing site on time might introduce too much error. The subject was then given his capsule and asked to return promptly in 45 minutes. A few minutes after his return his pulse was taken a second time. Pulse readings were also taken just before, and just after the administration of the block test. Each time the rate was taken from the wrist by counting heartbeats for 15 seconds and multiplying the result by four.

During the 45 minute waiting period in which the subject was not using his hands, bibliographic information was collected in order to keep the subject from being seriously bored. The content of this form is not very important since it was mainly to occupy time, but the information about the subject's sibling position was used in later analyses. Half of the data was gathered at each session, and the two

halves were counterbalanced between the drug and placebo groups.

On the Friday and Saturday sessions there was no need for the subject to sit without using his hands, so he was given a capsule and asked to return in an hour and a half. When all of the subjects for that week had returned, they were given the Eysenck Personality Inventory (either form A or B) and asked to read the directions and fill it out. Afterward they were given the Otis Group Intelligence Test (either form A or B) with directions very close to those given in the test manual. On the last testing day they were given the alternate form of these two tests.

All testing was carried out during the two summer sessions at the University of Windsor, a period of about 12 weeks. A schematic representation of the testing procedure is shown in Figure 1.

Figure 1

Schematic Representation of Testing Procedure
for One Subject

First day (Monday or Tuesday)

Pulse reading taken;
capsule given; S is
asked to return in
45 minutes

45
→
minutes

S return; pulse
is taken; ½ biblio-
graphical information
collected while he
does not use his
hands

45
→
minutes

pulse rate
taken; block
test given;
time estimation
made; pulse
rate taken again;
finger tapping
test; subject
counts from 1-10

Two days later

An exact repetition of the first day

Friday

Subject is given
capsule; asked to
return in 90 minutes

90
→
minutes

Subject takes one
form of Eysenck
Personality Test

→

Subject takes
one form of
Otis
Intelligence
Test

Saturday

An exact repetition of Friday's testing

CHAPTER 3

RESULTS

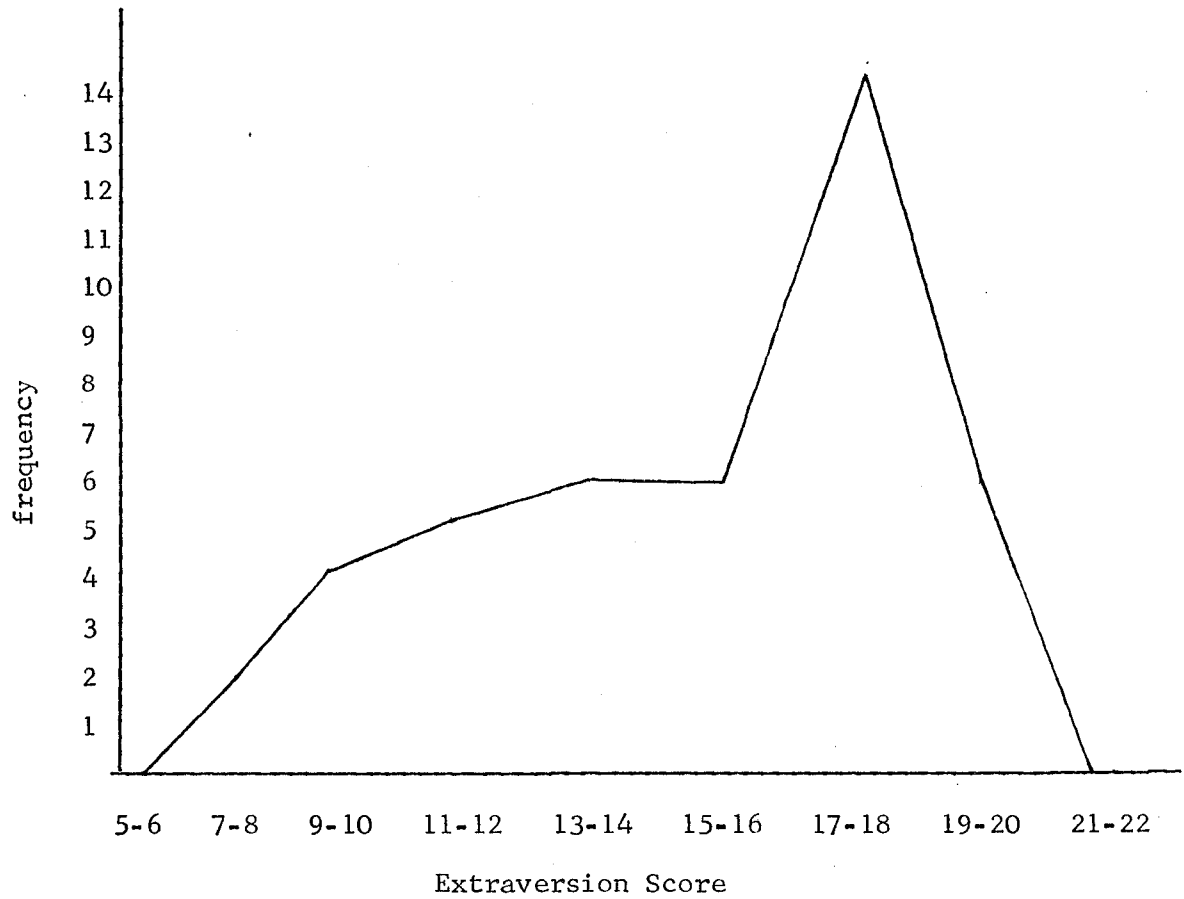
Of the volunteers for this study, only one subject had to be excluded because of complicating features like high blood pressure, taking other drugs, and so forth. This one subject was currently taking tranquillizers for what he described as general nervousness. Five other subjects came to one or more testing sessions but failed to complete the series, and so were excluded from consideration. Thirty-six subjects eventually completed the entire series of tests out of the original 42 volunteers.

Of those who participated, it was anticipated that the use of volunteers would result in a relatively large number of extraverted subjects with relatively few introverts. The distribution on the Extraversion scale of the Eysenck Personality Inventory is shown in Figure 2. There were a total of 16 extraverts (using an arbitrary cutoff of 17 points or more on this scale, approximately two standard deviations above the mean), 12 moderates (scores between 12 and 16), and 8 introverts (scores of 11 or less). The overall group mean in the placebo condition was 14.8. As compared with Eysenck's estimate of 14.1 as the mean for normal college populations (given in the test manual), it appears that this sample was less biased than was anticipated.

Six subjects were accidentally tested using the wrong size stimulus block, and these were excluded from analyses using the

Figure 2

Distribution of Extraversion Scores



augmentation/reduction scores but retained on all other measures.* Since these were the first six subjects, they at least provided extra practice in administering the test smoothly, and the remaining sample of 30 subjects is still larger than that generally used by Petrie. Of the 30 subjects correctly tested, using Petrie's criteria as given for both large and small block stimulation there were no augmenters, only 3 reducers, and 27 moderates (see Figure 3). Of these 30 subjects, 12 were extraverts, 8 were introverts, and 10 scored in the middle range.

I. The Block Test for Perceptual Reactance

Scores on the block test without the influence of the drug varied from -3.27 to +1.18 for 30 subjects. As indicated above, these distributed almost entirely in the middle range with only 3 people reaching the criterion for "reducers" and none at all reaching the criterion for "augmenters".

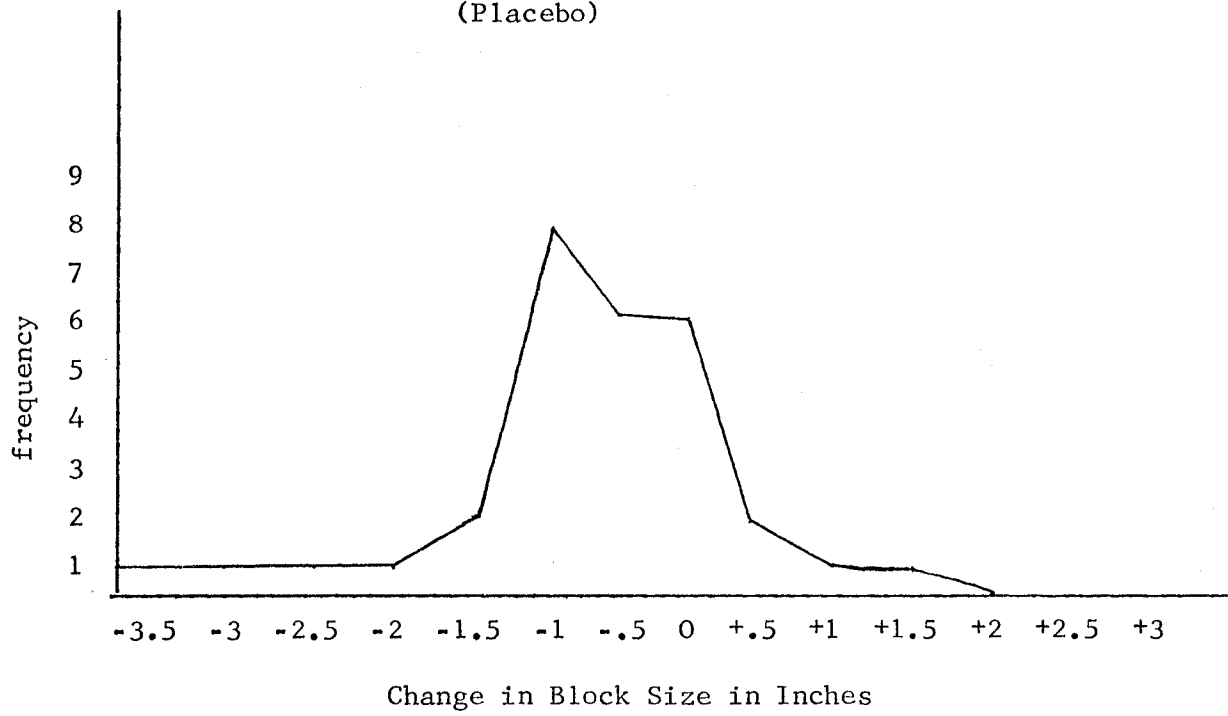
The mean score for the placebo condition was -.688. (See Table 1 for a complete summary of these scores.) For the drug condition the mean score was -.619. Considering the wide variance and a "t" value of .16, there was clearly no drug effect on the estimation of block size. The standard deviations for the placebo and drug conditions were found to be 1.09 and 1.02, respectively, so it is apparent that variability did not increase with the drug.

* In the Appendices these 6 subjects are referred to by their first initial. The remaining 30 subjects are designated by numbers 1 through 30.

Figure 3

Distribution of Perceptual Reactance Scores

(Placebo)



Kinesthetic Perceptual Reactance

<u>Subject</u>	<u>Placebo</u>	<u>Drug</u>	<u>Placebo Baseline</u>
1	- .18	-1.36	7.72
2	+ .45	+ .99	4.84
3	- .04	+ .49	5.44
4	-1.21	-1.98	9.56
5	-1.05	+1.00	7.25
6	-1.42	- .78	8.19
7	+1.45	- .61	9.16
8	-3.27	+1.27	12.09
9	- .87	- .64	6.91
10	- .38	- .02	10.31
11	- .76	-1.57	9.91
12	+1.18	+1.84	7.41
13	-2.90	- .73	8.06
14	-1.12	- .43	10.00
15	- .33	-2.03	7.62
16	- .50	- .92	6.84
17	- .96	- .03	10.34
18	- .25	- .18	6.16
19	+ .13	-1.42	7.50
20	+ .44	-1.86	6.06
21	- .43	-1.19	6.72
22	- .52	-1.05	9.06
23	- .80	-1.09	8.66
24	- .26	- .02	7.22
25	-2.65	- .13	10.84
26	-1.00	-1.45	8.56
27	+ .08	-1.23	7.31
28	-1.81	-1.75	8.59
29	- .11	-1.99	9.38
30	-1.55	- .05	9.47
	<hr/>	<hr/>	<hr/>
Sum	-20.64	-18.56	247.18
Mean	- .688	- .619	8.239
S.D.	1.9	1.02	1.65

II. Voice Amplitude

For each subject the highest volume and lowest volume for any of the numbers from 1 to 10 was recorded under both drug and placebo conditions. The results for the overall group of 36 subjects are shown in Table 2. No analysis was made of these results since the values are almost identical. There was no overall drug effect on the volume of subjects' speech in counting the numbers. (See Appendix 1 for a complete summary of this data for all subjects.)

III. Involuntary Pauses During Finger Tapping

For each subject the number of pauses (defined as an interval of 2 mm. or more on the graph) was recorded. The results for 36 subjects are summarized in Table 3. Again, in view of the wide variability, no analysis was required or performed. The drug did not reduce the number of involuntary pauses. (See Appendix 2 for a complete summary of this data for all subjects.).

IV. Extraversion Scores

Under both drug and placebo conditions subjects took the Eysenck Personality Inventory. The means of their extraversion scores are given in Table 4 for 36 subjects. Once again, the scores are too similar to require an analysis. Extraversion as measured by the Eysenck scale does not change with the drug. Since the Eysenck Personality Inventory also has an N (neuroticism) scale and an L (lie) scale, these values were also recorded, and are summarized in Table 5. In both cases it is clear that the drug had no effect. (See Appendix 3 for a complete summary of this data for all subjects.)

Table 2

Means and Standard Deviations of
Decibel Levels of Loudest and
Least Loud of the Numbers
Counted by Subjects Under
Drug and Placebo

Placebo				Drug			
high		low		high		low	
Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
74.61	5.02	83.44	3.24	74.80	4.39	82.92	3.21

Table 3
Means and Standard Deviation of
Number of Pauses During Finger Tapping
For Subjects Under Drug and Placebo

	Placebo		Drug	
Mean	S.D.	Mean	S.D.	
17.47	28.15	18.47	29.26	

Table 4
Means and Standard Deviations of
Extraversion Scores on Eysenck
Personality Inventory for Subjects on
Under Drug and Placebo

	Placebo		Drug	
Mean		S.D.	Mean	S.D.
14.78		3.69	14.56	3.72

Table 5

Means and Standard Deviations of
Neuroticism and Lie Scores on the Eysenck
Personality Inventory for Subjects under Drug
and Placebo

N	Placebo		N	Drug		L	S.D.
	Mean	S.D.		Mean	S.D.		
8.25	4.91	2.03	4.72	9.08	1.71	1.64	1.73

V. Otis Group Intelligence Test

The mean scores on the Otis Group Intelligence Test are summarized in Table 6. A simple "t" test shows that there is no difference between drug and placebo performance. However, it was noted that of the 10 subtests of the Otis, 9 of them were higher under the drug and only one did not change (see Figure 4). None were higher under the placebo. A χ^2 test indicates that this is more than a chance deviation ($\chi^2 = 3.97$, significant at the .05 level). Consequently an analysis of variance was made so the rather considerable practice effect from first to second testing could be taken into account. The results are summarized in Table 7.

This analysis clearly shows enhanced performance from the drug. The B value can be discounted since the maximum score possible was not the same for each of the subtests. There is, according to the analysis, no AB interaction. However, in looking at the various subtest scores it seems clear that some of the tests are elevated more than others (see Table 8). Separate "t" tests were then made on the difference between placebo and drug score for each of the subtests, using the mean square error value from the analysis of variance. By this means 5 of the subtests are found to be significantly higher under the drug at the .025 level or better. The subtests in question are summarized in Table 9. The remaining 5 subtests differed markedly less in their means from drug to placebo condition, and are summarized in Table 10.

VI. Additional Measures

Four readings of heart rate were taken during the first two sessions. This was taken mainly in case no other effects were

Table 6

Mean Otis Group Intelligence Test Scores
for Subjects under Drug and Placebo

Placebo	Drug
173.50	179.37

Otis Subtest Scores

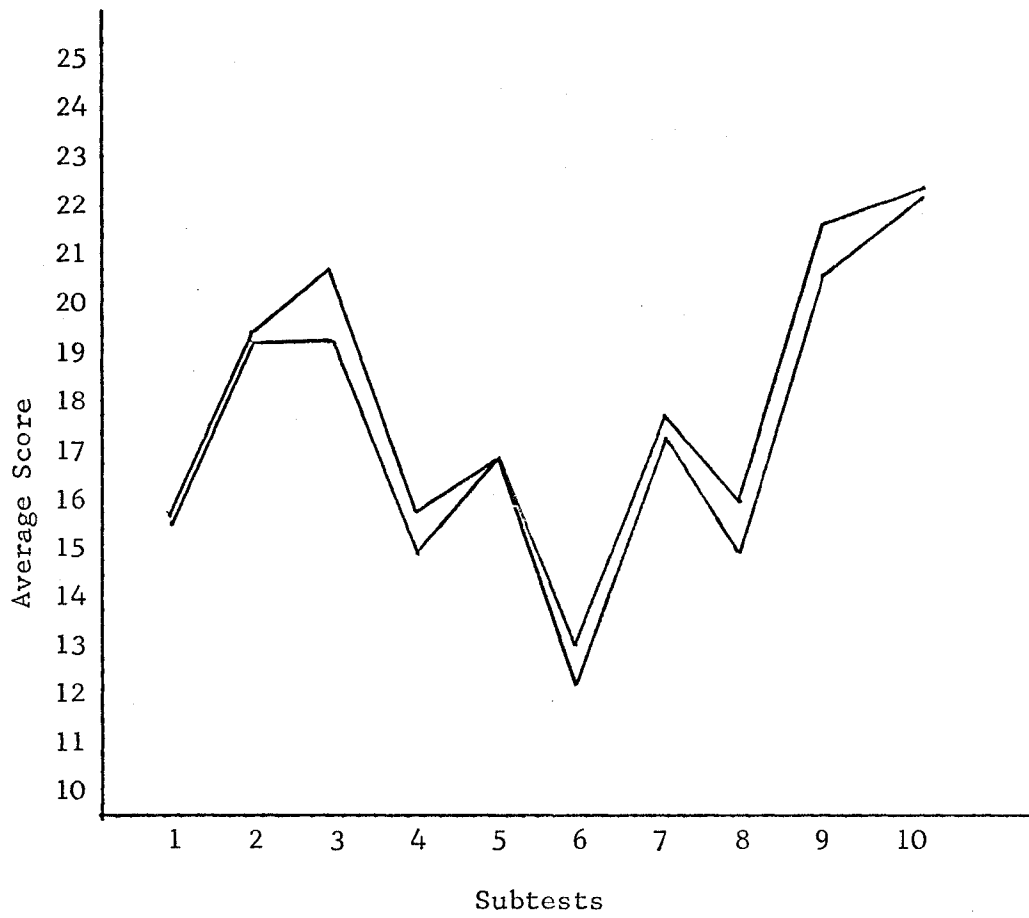


Table 7

Analysis of Variance Made on Otis Group
Intelligence Test Subscores

Source	Sum of Squares	df	Mean Squares	F	P
Total	11495.265	719	15.988		
Between Ss	2560.615	35	73.160		
Within Ss	8934.650	684	13.062		
A (drug)	61.832	1	61.832	13.72	.001
B (subtests)	5837.779	9	648.642	143.92	
AB (int.)	37.902	9	4.211	.93	
Error	2997.137	665	4.507		

Otis Scores
Subtests 1-6

Subject	Subtest											
	1		2		3		4		5		6	
	P	D	P	D	P	D	P	D	P	D	P	D
B	20	20	22	21	19	18	19	18	18	17	16	17
A	13	15	15	12	20	21	11	11	12	12	5	10
M	20	14	23	22	19	17	20	17	20	20	15	13
P	19	18	21	21	16	24	18	19	19	20	9	13
T	13	16	16	23	20	25	19	17	20	20	14	16
R	16	16	17	17	19	13	15	15	16	15	15	11
1	18	13	23	20	25	22	17	19	17	16	14	13
2	15	15	21	21	19	23	10	19	13	12	10	12
3	15	14	19	19	18	18	11	15	18	17	13	15
4	18	16	18	19	22	22	15	18	17	16	12	7
5	19	16	23	23	25	23	20	20	17	16	16	13
6	13	17	22	18	17	17	6	15	15	17	8	12
7	19	19	20	21	21	24	18	15	18	19	12	14
8	12	15	20	21	14	19	17	17	15	17	10	14
9	15	17	20	22	17	24	18	16	16	17	10	15
10	10	10	13	14	18	15	5	12	13	15	9	7
11	11	12	21	20	17	24	9	15	15	16	10	11
12	17	15	21	15	19	17	14	8	16	13	15	13
13	15	16	19	17	21	25	13	11	17	18	13	15
14	16	17	18	20	20	23	12	13	19	18	14	18
15	18	17	22	18	25	14	18	17	19	18	16	15
16	14	17	16	16	19	23	9	8	17	17	9	14
17	16	17	20	18	25	18	20	20	19	18	16	13
18	16	19	18	23	19	24	17	20	17	18	15	16
19	11	11	15	17	16	18	6	5	14	15	13	12
20	15	17	17	21	21	21	9	15	17	17	14	10
21	15	16	21	20	16	25	19	20	15	19	11	11
22	15	15	18	18	16	22	16	12	17	15	12	16
23	12	14	17	20	14	25	12	14	16	16	11	12
24	17	13	18	21	23	22	18	19	18	17	11	8
25	15	15	19	21	23	25	20	19	16	16	12	17
26	17	18	23	21	17	17	17	19	16	16	12	11
27	18	18	19	22	21	21	20	19	15	18	12	12
28	15	16	18	18	14	20	15	14	18	16	8	14
29	19	16	20	19	18	14	14	17	18	16	12	13
30	18	19	22	23	23	25	20	20	20	20	15	15
Sums	565	569	695	702	696	748	537	568	603	603	439	468
Means	15.69		19.30		19.33		14.92		16.75		12.19	
		15.80		19.50		20.78		15.78		16.75		13.00

Table 8
(continued)Otis Scores
Subtests 7-10 and Total Scores

Subject	Subtest 7		8		9		10		Totals	
	P	D	P	D	P	D	P	D	Placebo	Drug
B	20	20	20	18	25	22	22	22	201	193*
A	13	15	12	13	19	24	23	20	143*	153
M	20	19	16	18	22	20	27	24	202	184*
P	22	23	13	16	25	23	22	26	184*	203
T	24	17	12	13	25	25	22	22	185*	194
R	17	14	19	18	22	16	23	20	179	155*
1	19	21	18	16	25	23	24	25	200	188*
2	18	20	18	18	20	21	25	22	169*	183
3	16	19	13	15	23	22	22	21	168*	175
4	18	19	16	17	21	19	27	22	184	175*
5	14	17	16	18	24	23	22	26	196	195*
6	15	11	11	14	9	16	18	17	134*	154
7	20	19	16	18	21	23	26	21	191*	193
8	14	20	11	15	17	22	25	22	155*	182
9	22	18	15	19	18	19	20	23	171*	190
10	14	8	8	2	15	14	19	18	124	115*
11	13	16	10	14	21	24	17	17	144*	169
12	21	14	18	19	22	20	25	23	188	157*
13	15	17	16	17	19	22	26	25	174*	183
14	19	18	13	14	19	18	20	22	170*	181
15	22	22	20	18	25	25	26	21	211	185*
16	15	15	19	19	13	25	20	24	151*	178
17	15	22	15	16	20	20	23	24	189	186*
18	20	24	18	20	22	25	19	26	181*	215
19	14	17	11	15	17	20	16	18	133	148*
20	16	17	19	16	19	21	22	23	169*	178
21	16	17	18	19	11	22	20	23	162*	192
22	18	16	12	23	18	23	23	22	175	168*
23	15	16	5	10	14	23	15	22	131*	172
24	15	16	20	18	18	21	19	19	177	174*
25	15	23	17	19	24	25	27	24	188*	204
26	22	20	18	17	25	25	28	28	195	192*
27	20	15	16	19	21	22	22	23	184	189*
28	11	19	13	16	25	22	19	21	156*	176
29	17	16	16	12	24	21	21	22	179	166*
30	24	21	13	18	24	24	24	27	203*	212
Sums	630	643	545	576	737	775	799	805	6246	6457
Means	17.50		15.14		20.47		22.19			
		17.86		16.00		21.53		22.36	173.50	179.37

* star indicates which test came first

Table 9

"t" Tests Made Comparing 5 of the Subtest Means
of the Otis Group Intelligence Test for Subjects
Under Drug and Placebo

Subtest	Placebo	Drug	"t"	P
#3 Disarranged Sentences	19.33	20.78	4.101	.001
#4 Proverbs	14.92	15.78	2.432	.025
#6 Geometric Figures	12.19	13.00	2.291	.025
#8 Similarities	15.14	16.00	2.430	.025
#9 Narrative Completion	20.47	21.53	2.998	.025

Table 10

Means of Subtests of the Otis Group Intelligence
Test Which Differed Least from Drug to Placebo
Conditions

Subtest	Placebo	Drug
#1 Following Directions	15.69	15.80
#2 Opposites	19.30	19.50
#3 Arithmetic	16.75	16.75
#7 Analogies	17.50	17.86
#10 Memory	22.19	22.36

demonstrable, to see if at least there were some objective effect from the drug. The first was taken a few minutes after the subject first arrived; the second when he returned after the 45 minutes latency; the third just before the block test; and the fourth just after the block test. The results are summarized in Table 11, and represented graphically in Figure 5.

An analysis of variance was made on this data. The results are summarized in Table 12. The drug on the average elevated heart rate about 6 beats per minute, and this change is significant at the .005 level.

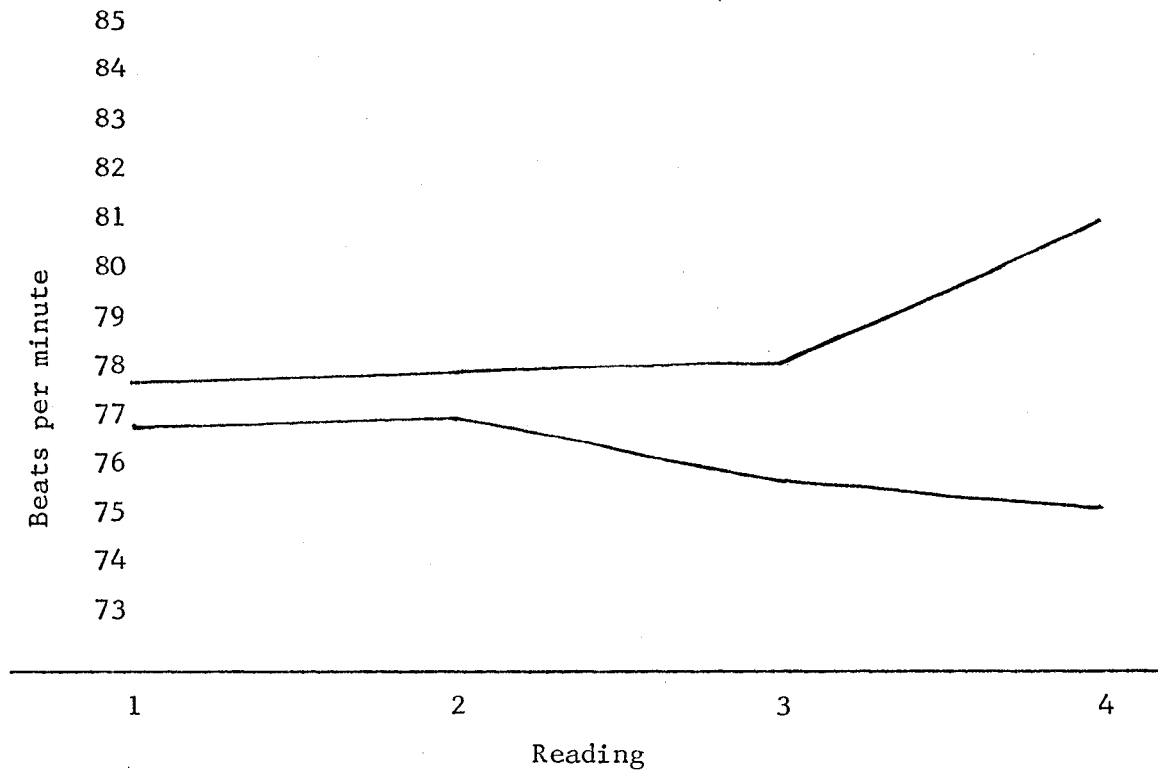
Just after the block test, subjects were asked to estimate the length of a minute while still blindfolded. Estimates varied from 20 to 165 seconds. The average for the drug condition was about 7 seconds shorter than for the placebo condition, so a "t" test was made. The results are summarized in Table 13. The difference in time estimation between drug and placebo conditions is significant at the .05 level.

The number of taps per minute was recorded for each subject under each condition. Rather than count the total number of taps on each record, an estimate was made by taking the average of the first 2 second period, the middle 2 seconds, and the last 2 seconds. The result was multiplied by 30. This procedure was compared with an actual count for several subjects and found to be rather accurate. The results are summarized in Table 14.

Since it is well established that dexedrine decreases reaction time, it was anticipated that the number of taps per minute would increase with the drug. Though this appeared to be true, the difference

Figure 5

Pulse Rate



Pulse Readings

Subject	Readings								Up	Change Down
	1		2		3		4			
	P	D	P	D	P	D	P	D		
B	76	68	64	84	72	92	76	92	12	
A	84	80	76	80	76	72	80	72		6
M	72	84	72	92	68	80	68	88	0	0
P	84	76	88	80	84	76	84	76	0	0
T	60	76	60	80	60	76	60	80	0	0
R	88	92	92	80	96	84	92	88		4
1	80	84	88	84	76	80	80	80	2	
2	116	112	104	100	104	88	120	104		8
3	76	72	76	72	76	72	76	72	0	0
4	80	76	80	76	76	80	80	80	6	
5	72	84	72	80	72	72	72	84		4
6	80	68	76	68	88	76	80	80	4	
7	80	76	88	68	84	72	80	80	6	
8	76	88	80	80	72	76	76	84	0	0
9	72	72	72	68	72	68	72	68		2
10	76	92	80	84	84	80	80	92		6
11	80	72	76	72	68	84	64	84	24	
12	76	76	72	72	72	76	76	84	6	
13	80	76	80	68	80	84	72	80		6
14	56	48	60	48	64	68	52	48	10	
15	84	84	88	80	76	76	76	84	8	
16	64	56	68	72	76	80	72	80	8	
17	80	96	68	88	68	88	60	92	8	
18	84	72	76	76	76	76	76	84	10	
19	80	84	80	92	76	72	84	92		6
20	72	64	68	64	68	68	64	68	8	
21	68	60	72	68	68	72	72	72	8	
22	76	64	68	68	64	64	64	64	6	
23	88	68	84	88	92	84	84	96	10	
24	80	96	80	88	64	96	64	88	16	
25	72	84	80	100	72	100	72	104	14	
26	42	64	60	60	60	56	60	64		11
27	72	84	80	88	92	92	84	92		6
28	80	100	76	92	76	100	76	104	8	
29	88	72	76	68	76	68	76	64	2	
30	72	72	76	68	80	72	84	76		4
Sums	2766		2756		2728		2708			
Means	76.83	2792	76.56	2796	75.78	2820	75.22	2940		
		77.56		77.67		78.33		81.67		

Table 12

Analysis of Variance Made on Pulse Rate Readings
Under Drug and Placebo

Source	Sum of Squares	df	Mean Squares	F	P
Total	35601.320	288	123.616		
Between Ss	21451.820	35	612.909		
Within Ss	14149.500	253	55.927		
A (drug)	528.125	1	528.125	9.794	.005
B (subtest)	95.376	3	31.792	.590	
AB (int.)	368.597	3	122.866	2.278	
Error	13157.402	244	53.924		

Table 13

"t" Test Made Between Estimates of the Length
of a Minute (in seconds) Between Drug and Placebo

MEAN		"t"	df	P
Placebo	Drug			
61.94	54.42	1.820	35	.05

Table 14

"t" Test Made Between Number of Taps in a Minute
in Drug and Placebo Conditions

Placebo	Mean	Drug	"t"	df	P
333.11		346.69	.932	35	NS

was not statistically significant.

A simple possible explanation for the apparently increased sense of time (a minute seems shorter) is increased heartbeat. To test this explanation, estimates of heart rate change were calculated for each subject by the formula:

$$\frac{\text{first 2 placebo readings}}{2} - \frac{\text{next 2 placebo readings}}{2}$$

minus

$$\frac{\text{first 2 drug readings}}{2} - \frac{\text{next 2 drug readings}}{2}$$

This method takes into account that some subjects' heart beat may have gone up during testing even when on placebo, whereas others may have gone down or stayed the same. This provides a better estimate of the actual drug effect on pulse rate than the pulse change on the drug day, since by chance the heart rate may have been higher or lower the second day even before the drug was given.

A rank order correlation was then made between these change scores for each subject and the subject's changed sense of time. The resulting value was $-.054$, which is clearly not significant. A similar correlation was made between heart rate change and the actual estimate in seconds under the drug. This value was $+.130$, also clearly not significant.

Since the estimate of the length of a minute was generally shorter under the influence of the drug, it might be predicted that in counting from 1 to 10, subjects would count faster on the drug day than on the placebo day. This is simply calculated by recording the distance in millimeters from the number 1 to the number 10 as recorded on the dynograph sheets. The resulting values are shown in Table 15.

Table 15

Means and Standard Deviations of
Number of Millimeters of Paper Covered
by the Count from one to ten in the Drug
and Placebo Conditions

Mean	Placebo	S.D.	Mean	Drug	S.D.
68.83			67.17		

These differences are clearly non-significant, as the variability on this measure was considerable.

Contained in the results of this study are some measures which are effectively replications of previous studies done by Eysenck and Petrie. For example, Petrie claims that there is a significant difference between the extraversion scores of augmenters as opposed to reducers, and that the latter are more extraverted. Consequently, a correlation was made between extraversion scores on the Eysenck Personality Inventory and reduction ($n = 30$). The resulting value was $-.007$. The distribution shown in Figure 1 indicates that there is something of an excess of extraverts in this sample, so an extreme groups comparison was made between the most extreme extraverts and the most extreme introverts on perceptual reactance scores. Five subjects scored between 7 and 9 points on the extraversion scale, which, according to the manual, means between the 2nd and 10th per centiles. These five were compared with six subjects scoring between 18 and 20 -- or between the 87th and 97th per centiles. The results are summarized in Table 16. The means between these two groups does not differ enough to warrant performing a statistical test.

One could still compare the most extreme reducers with the most extreme augmenters on extraversion scores in the hope of finding some connection between these two variables. There were no real augmenters in the group studied, but 2 subjects at least were much further in this direction than any of the others. These were compared with the 3 subjects who most reduced (all of these 3 met the criterion for reducers). The results are summarized in Table 17. Once again the

Table 16

Mean Perceptual Reactance Scores of the 5 Most
Extreme Introverts Compared with the 6 Most
Extreme Extraverts in the Placebo Condition

Extreme Introverts	Extreme Extraverts
-.438	-.450

Table 17

Mean Extraversion Scores on the Eysenck Personality
Inventory of the 3 Most Extreme Reducers and the 2 Most
Extreme Augmenters in the Placebo Condition

Extreme Reducers	Extreme Augmenters
16.3	18.5

difference is not great enough for such small samples to warrant doing a statistical test. If anything, the trend is in the opposite direction to that predicted.

On surveying the augmentation-reduction scores, while it is apparent even without a statistical analysis that the drug had no effect, there does seem to be a difference between subjects' performances on the first and second days of testing. This appears evident regardless of when they received the drug. A simple "t" test was made comparing the performance on the first day's testing with later retesting. The results are shown in Table 18. It is evident that there is a practice effect on the block test. When tested for the second time these subjects' estimates were closer to the actual size of the block -- in this case meaning that they relatively augmented as compared with their first testing.

It seemed possible that while extraversion scores do not seem to relate to changes on the block test, that the widely differing baseline estimates of block size might be obscuring a real difference. If a subject, for example, estimated the block's size as very small to begin with, then the degree to which his estimate could change is much more limited than if his original estimate were moderate. So a correlation was made between baseline scores on the block test and extraversion scores. This resulted in a value of $-.001$, which is clearly not significant, indicating that the two variables are not related.

It is possible, though highly unlikely, that the drug had some inconsistent effect even without changing either the overall mean or the standard deviation of the block test. If it is granted that the drug

Table 18

"t" Test Made Between Perceptual Reactance Scores
on First Testing and the Same Scores on Later
Retesting

First Day	Means	Second Day	"t"	df	P
-.936		-.348	2.217	29	.02

had no effect, however, then the two tests can in effect be considered a measure of test-retest reliability. Petrie claims a result for split-half reliability of about $+0.97$. In this study, the correlation between the first and second testing was -0.032 .

Eysenck says that the difference between extraverts and introverts is a result of differing rates of dissipation of "inhibition"; that extraverts dissipate it slowly, and so become more easily bored (distractible) and will have more pauses while engaged in simple motor tasks. So the number of pauses while tapping a telegraph key with the index finger should be greater among extraverts than introverts. A correlation between extraversion scores and the number of involuntary pauses while tapping was made, with a resulting value of -0.083 . Again, since the distribution of this sample was somewhat weighted toward extraversion, an extreme groups comparison was made between the 5 most extreme introverts (scores 7-9) and the 4 most extreme extraverts (scores 19-20). The results are shown in Table 19. If anything there is a slight trend in the opposite direction of the predicted, but they do not approach significance. It should be mentioned that one of the extraverts was excluded from the above, as he was one of two subjects whose finger tapping rate was so slow that between every tap he met the criterion for a pause. The other such subject was an introvert (extraversion score 10), so it seemed fair to exclude both as their performance was completely unlike that of any of the other subjects on this measure.

Both Eysenck and Petrie would agree that the extravert is extraverted because he is relatively stimulus deprived, and seeks to

Table 19

Mean Number of Pauses during Finger Tapping, and
 Mean Number of Taps in a Minute of Extreme Extraverts
 and Extreme Introverts

Extreme Introverts		Extreme Extraverts	
Number of Pauses	Number of Taps	Number of Pauses	Number of Taps
8.8	314	7.0	328

stimulate himself in both social and non-social ways. Petrie also suggests that in the case of alcohol, people are inclined to speak louder under its influence because they are relatively underreacting to the loudness of their own voice. In other words, alcohol causes reduction. If this is true, it should follow that extraverts will generally speak louder than do introverts. Consequently a correlation was computed between extraversion scores and both the lowest and highest decibel level at which any of the numbers from 1 to 10 were read (in the placebo condition). The resulting values are .148 and .216, respectively. Both values are well within the range of chance variation.

Since this study failed to obtain the distribution expected from the Petrie data on the perceptual reactance continuum, a comparison could be made between what changes were obtained on this measure with changes on such measures as the voice level, number of pauses during tapping, and extraversion scores. Three such "t" tests were made, with results as summarized in Table 20. It appears those subjects who relatively augmented (increased) their estimation of block size on the drug did, on the average, speak in a lower volume of voice. There was also a tendency for such subjects to make fewer pauses while tapping. Though both of these results would be predicted from the theory, only the first was significant at the .05 level.

Finally, both Petrie and Eysenck (particularly the latter) imply that perceptual reactance is a more or less constitutional factor rather than the produce of learning. Therefore extraversion/introversion should not particularly relate to nurturance. Irving Harris,

Table 20

"t" Tests Made Between Subjects Who Relatively
Augmented on the Drug and Those Who Relatively
Reduced on Three Measures

	Means for Group With P.R. Up	Means for Group With P.R. Down	"t"	df	P
Change in Ave. Volume in dbs.	-0.812	1.071	1.929	28	.05
Change in # of Pauses	-4.688	5.000	1.459	28	.10
Change in E Score	0.188	0.000	.157	28	NS

on the other hand, in his book The Promised Seed, builds a case for the contrary. Harris says that first borns are generally more introverted than later borns as a function of their parental treatment. Therefore a comparison was made between first born and later born sons in this sample on extraversion scores on the Eysenck Personality Inventory. Though this comparison was made post facto, and was not predicted in the initial proposal for this study, the results were most interesting. They are summarized in Table 21. First born sons are on the average considerably more introverted than later borns in this sample.

Table 21

"t" Test Made Between Extraversion Scores of First
Born and Later Born Sons

First Borns	Later Borns	"t"	df	P
13.667	17.000	2.744	35	.005

CHAPTER 4

DISCUSSION

The most obvious observation to be made about the results of this study is their dramatic dissimilarity from earlier results reported by Petrie and others. First of all it would be appropriate to review the results concerning the five basic predictions made at the outset of this study. The first such prediction is that dexedrine, a stimulant, should cause augmentation. This clearly did not occur. It is therefore not very surprising that the second prediction -- that subjects would speak in a lower voice because of their increased perceptual reactance -- also did not occur. The third prediction is more relevant to Eysenck's theoretical explanation of the cause of over or underreacting to stimuli -- that underreacting is the result of slower rates of dissipation of inhibition. Dexedrine, a stimulant, should increase this rate, producing not only augmentation, but an increased ability to persist in a simple motor task without involuntary pauses. Again, the prediction failed to draw any support, and there was even a small trend in the opposite direction. Fourth, through its augmenting effect the stimulant dexedrine should cause a shift toward the introverted end of the extraversion/introversion dimension. This did not occur. Fifth, performance on the Otis Group Intelligence Test should be better in the drug condition. This prediction was verified dramatically. But any attempt to explain this effect on the basis of

either change in perceptual reactance or the inhibition-excitation ratio draws little support. Since the drug did not cause augmentation in the first place, it is clear that this could not be the mechanism for any improvement in performance. Fortunately a review of these predictions is only a prelude to a much more interesting pattern of results. The drug certainly did have effects, but they were not the effects that one would expect after reading the material of Eysenck and/or Petrie.

On the basis of the results obtained in this study, it seems evident that the so-called measure of perceptual reactance -- the block test -- does not have very much reliability. While the drug had no demonstrable effect nor even a non-significant trend worth looking at, there was a marked difference between performance on the first and second testing. Whatever the deviation from the objective size of the block on the first day, the second day's testing estimates were generally less. In fact, these later estimates were very close to the real block size, showing little trend toward either augmentation or reduction. As a result, the resemblance between initial testing and later testing was so small that the test's retest reliability can be considered almost nonexistent. The correlation between these performances was $-.032$, and the significance of the difference reaches the $.02$ level. Yet Petrie claims split-half reliabilities in the order of $.97$. What might account for these differences? She summarizes at great length the possible factors that could cause variations in performance. Among them are the following: use of drugs; exposure to any excessive stimulation, like loud noises or considerable heat or cold; sickness,

even colds; psychiatric conditions; smoking; allowing the subject to see the testing equipment; and many other factors, all of which were screened for in this study. There were a few subjects who Neuroticism scores on the Eysenck Personality Inventory were rather high, but certainly not enough to obscure differences for the whole group. None of the subjects had colds or obvious psychiatric disturbances. All went the prescribed 45 minutes without using their hands in the same room and with the same external conditions (controlled heat, minimal noise). Subjects were not left alone to be bored, but were asked for some biographical information (standardized) and kept in the company of the experimenter for most of the waiting period. The testing procedures were well practiced in advance and the instructions, though not verbatim, were patterned closely to those described by Petrie. It would have been difficult to make the procedures used any more like those described by Petrie, and yet the results are in no way comparable to hers. Compare, for example, Table 22 and Table 23. Table 22 is a record of a reducer, using large block stimulation as was done in this study, tested by Petrie and supposedly not very atypical. Table 23 is a reasonably representative example of performance obtained in this study -- that of subject number 22 in the no-drug condition. Petrie's sample shows consistently increasing deviations from the baseline average after each period of stimulation. Subject 22 in this study first reduces very slightly, then reduces markedly, and after the third period of stimulation moderately augments. The overall average is in the moderate range. Petrie's example cannot be very common; it is rather very idealized. In addition it is apparent that a split-half

Table 22
 Sample Record of a Reducer as
 Given by Petrie
 (units - inches)

	Practice Trials	1	2	3	4	Total	Ave.	Difference From Base-line Ave.
I Baseline	11 7/8 12 3/8	14 2/8	14 4/8	14 2/8	14 6/8	57.75	14.43	
II After 90 Second Stimulation		12 6/8	13 2/8	13 4/8	14	53.50	13.37	-1.06
III After 180 Second Stimulation		11	13 2/8	12 3/8	12 3/8	49.50	12.37	-2.06
IV After 300 Second Stimulation		9 2/8	10 4/8	11 4/8	12 4/8	43.75	10.94	-3.49

Final Average Modulation = $\frac{-6.61}{3}$ = -2.20

Table 23
 Sample Record from this Study
 (units = inches)

	Practice Trials	1	2	3	4	Total	Ave.	Difference From Base-line Ave.
I Baseline	8 3/8	8 3/8	8 5/8	9 1/8	9 2/8	9 2/8	36.25	9.06
II After 90 Second Stimulation		7 6/8	8 3/8	9 1/8	9 1/8	34.375	8.59	- .47
III After 180 Second Stimulation		6 3/8	7 2/8	7 3/8	7 2/8	28.25	7.06	-2.00
IV After 300 Second Stimulation		8 7/8	9 7/8	11 2/8	9 7/8	39.875	9.97	+ .91

Final Average Modulation = $\frac{-1.56}{3}$ = -.52

type reliability measure is rather meaningless here. If this test relates to a characteristic kind of perceptual response it is reliability over time which is important. That subjects are consistent during one period of time does not necessarily imply that they will be so from day to day, or month to month. Spitz and Lyman (1960) do report a test-retest reliability of $+0.74$, however their testing apparatus and procedure differ somewhat from Petrie's. Also it is interesting to note, as Petrie does not, that they found no correlation between visual and kinesthetic figural after-effects -- a rather surprising finding if perceptual reactance is supposed to be the same through different sense modalities.

Though Petrie says that with normal adult samples testing with either the larger or smaller block alone is sufficient, her own data shows that performance differs markedly as a result. In her book (p. 133) she shows the frequency distribution for some college students tested with large-block stimulation and another sample tested with small-block stimulation. Using her own criteria, none of those tested with the large block were augmenters. Also none of those tested with the small block were reducers ($n = 13$ in both cases). The distributions indicate that the complete lack of augmenters found among 30 subjects in this study is not surprising, since only large-block stimulation was used here. However the further fact that only 3 out of 30 subjects met the criterion for reduction is surprising, and indicates that her sample may not have been very typical. Using both large and small block stimulation might have produced a distribution more like the one she claims is normal, but this seems unlikely since the effects of testing with the two different size blocks would tend to reduce the

overall deviations from objective size.

In hindsight, it is obvious that the criterion of 1.8 inches change has no relevance when only one size stimulating block is used. One could guess that those who increased their estimates the most might reach the criterion for augmenters if tested with both blocks, and conversely for those who reduced their estimates the most. Logically, one could then divide the subjects into three groups based on their performance on the block test and then check for differential effects. But even extreme groups comparisons in tables 16, 17, and 19 show no trend in the predicted direction, so a more complex analysis was not undertaken.

Granted that the test is unreliable, it is hard to conceive how Petrie could have obtained the perfectly neat results she claims as a result of aspirin. The variation in performance from one testing to another is too great, it would seem, for this to be possible. In any case the fact (if it is a fact) that depressants cause reduction by no means necessarily implies that stimulants should cause augmentation. In view of the failure of dexedrine to produce augmentation in this study, a review of some of Eysenck's arguments was made with some most interesting results. In both Experiments in Motivation and Experiments with Drugs, Eysenck has long tables summarizing various studies with stimulants and depressants. In the latter book, for example, he lists the topic "conditioning"; the predictions that stimulants will increase it and depressants decrease it; and three references to support the predictions. Examination of these "supporting references" shows that only one of the three used a stimulant. The other two used depressants only. So it goes throughout the table.

A claim is made that depressants do one thing and stimulants do the opposite, then references are cited which in the majority of cases never used a stimulant at all. The terms "stimulant" and "depressant" have never been very clear in the first place, so to assume that because depressant X does one thing stimulant Y must do the opposite is debatable at best. At worst, it is an outright distortion. Dexedrine has never previously been tested for its effects on perceptual reactance, nor has any other stimulant. It is possible that drugs in general cause reduction, if they have any effect at all on this dimension.

Eysenck's reference in Crime and Personality to an unpublished study on involuntary pauses during tapping again clashes with the results obtained here. Like Petrie's aspirin study, the results claimed are almost too perfect -- no overlap at all between introverts and extraverts. The tasks are somewhat different, so some difference in the results is to be expected. But there is not the slightest support for the idea that extraverts "dissipate inhibition more slowly" and therefore pause more while tapping. The correlation between extraversion scores and number of pauses was $-.083$. Even comparing only the extreme introverts and extraverts no differences appear. In the study Eysenck refers to subjects tapped with a metal stylus on a metal plate, while in this study they tapped a telegraph key with the index finger. It is hardly likely that such a small difference in procedure could completely eliminate results as strong as those Eysenck claimed. Dexedrine, if anything, increased the number of pauses somewhat. This, however, may simply have been the result of a somewhat faster tapping rate with the drug, which is

generally known to decrease reaction time.

The lack of the effect of dexedrine on the Eysenck Personality Inventory could have been expected from the fact that this test, like most personality tests, measure long term inclinations more than immediate feelings. However it is noteable that there was no similarity in results obtained here and the early study by Turner and Carl (1939) using the Bernreuter Personality Inventory. The variation in extraversion scores was no higher under the drug than it was under placebo, nor was there any change in the standard deviation of the neuroticism and lie scales.

It was noted during the course of the experiment that the subjects often reported that they had felt nothing from the drug -- or, if they did feel an effect, it was as often as not on the day that they had taken a placebo. An obvious criticism might be that the dose of the drug was too small, or the latency period insufficient for it to take effect. There is strong evidence to the contrary. There was a statistically significant increase in heart rate with the drug, as shown in Figure 4. In addition the latency period was considerably longer than is usually used as a result of subject's having to sit without using their hands before they could take the block test. A five milligram dose is as large as is used in most studies with dexedrine, and a five milligram tablet as large as any company makes commercially for such purposes as dieting. Corrections for differences in body weight were not taken into account here, which may have increased error variability somewhat. Yet there are the dramatic effects on the Otis Group Intelligence Test, which is certainly the most interesting finding in this study.

Overall, the mean scores between the drug and placebo group are not very great -- only about 6 raw score points. Yet this difference exists in spite of a marked practice effect. The vast majority of subjects did better on the second testing whether or not they had the drug on the first or second day. It is interesting to note that the only two exceptions were both subjects who were tested first with dexedrine and second with placebo. Also the most marked improvement between first and second testing was a remarkable 41 raw score points, this in a subject who was tested with the drug on the second occasion. The smallest improvement among those subjects who did improve the second time was only 1 point, this in a subject who had taken the drug on the first testing. All the evidence makes it clear that the subjects did do better with the drug, with a possible few exceptions that did not change the group trends. Nine of the 10 subtests were higher on the drug; none lower. By analysis of variance this difference is significant at the .001 level, thus corroborating neatly the results of Vaness and Brown (1966).

Even more interesting than the fact that overall differences were found on the Otis is the pattern of subtests which seemed to be the most effected. In previous studies of the effects of dexedrine on intellectual tests it has often been claimed that the effect is only on speed, and that those tests which emphasize "power" rather than speed would probably not change. If anything, the results of this study tend to run counter to this claim. Of the 10 subtests the one which is probably the most simply a measure of speed at a learned task is the Arithmetic test (#3). Probably any, or at least almost any, of the subjects of this study could have done every problem

correctly if he were given enough time. None were particularly difficult or required more than exercising a learned ability quickly. Yet this was the only subtest on which the drug appeared not to have an effect. On the other hand, the subtest most "power" oriented is probably Geometric Figures (#6). Some of the questions on this test could not have been answered correctly by most subjects even if the time limit had been doubled or tripled, as they require the kind of cognitive ability that is difficult to train in. As an example, question 20 of this subtest is: "What is the greatest number of spaces which it is possible to make by overlapping a circle, triangle, and rectangle?" Out of 36 subjects, none answered this question correctly. On this test the effect of the drug was much more marked than on some of the others.

By "t" tests the following subtests appeared to be most affected by the drug: #1 - Disarranged Sentences. On this test the task is to combine a scrambled bunch of words into a recognizable sentence, and then indicate whether the sentence is true or false. For example, the words "uphill, river, flow, all" must be combined into the sentence "all rivers flow uphill". Since the statement is not true, the word false is then underlined. #4 - Proverbs. On this test the task is to find among a given set of statements one which explains certain proverbs, such as "A stitch in time saves nine", for which the proper explanatory sentence is "It pays to attend to troubles before they get worse. #6 - Geometric Figures, previously described. #8 - Similarities. In this test the task is to see the similarity between the first three items, and then select one of five which is most like the first three. The last quarter of the test is geometric

figures. #9 - Narrative Completion. In this test the task is to choose from among sets of 3 alternatives the word which most correctly fits into the context of a story with certain words in it left blank. None of these tests is simply a measure of response speed, but involve the ability to see relationships.

Those subtests showing the least drug effects are the following:

#1 - Following Directions. In this test the alphabet is printed across the top of the page and questions are asked about it, such as "What is the fifth letter of the alphabet", to which the answer is "E".

This test is more a test of response speed than most of the others.

#2 - Opposites. Here the task is simply to choose from among several alternatives the word which means exactly the opposite of some given word. #3 - Arithmetic. This is a test of simple arithmetic problems.

#7 - Analogies. This is a standard type analogy test, with questions resembling some of those on the Miller Analogy Test. #10 - Memory.

In this test subjects are read a story, and then asked questions about what the story did or did not say.

Among those tests showing the most drug effect, none are predominantly speed oriented. Of those showing the least drug effect, only one -- the analogy test -- is fairly obviously a test of the ability to see relationships of the sort required in the aforementioned tests. This is hardly convincing evidence that the drug increases the ability to see relationships rather than merely speed up work rate, but it does provide some evidence in this direction and suggests that further testing of exactly what kinds of intellectual abilities are improved is in order. Testing subjects on the Wechsler Adult Intelligence Scale might be particularly interesting. On the basis of the results

obtained in this study it might be predicted that increased scores would be more likely on the performance tests rather than on the verbal tests.

The effects of dexedrine on pulse rate and blood pressure are known to be less than those of its predecessor, racemic amphetamine or benzedrine. Among the subjects in this study the average heart rate was definitely up, but not to any worrisome degree. The difference was only about 6 beats per minute. A minority of cases had decreased pulse rate, and casual observation makes it appear that there is something of a leveling effect. That is, subjects with initially fast heartbeat tended to slow down with the drug, whereas those with initially slow heartbeat tended to increase. The majority of subjects changed little, and the variability in heart rate with the drug did not differ from placebo to any significant degree.

Having subjects estimate the length of a minute did not particularly relate to either the theories of Eysenck or Petrie. It has often been noted that dexedrine tends to speed up mental activity, and it seemed likely that this might also speed up one's sense of time. People generally think a minute is longer if no activity occurs during that time, and think it is shorter if some intervening activity occurs. Since it was found that the increased sense of time in subjects did not correlate with increase in heartrate, this seems the most likely explanation.

The theory of perceptual reactions as the determiner of such things as how loud one speaks, like most of the other things related to this variable, drew no support. Not only did the drug not affect voice loudness, but voice loudness does not seem to relate to extraversion scores in the first place. If extraverts are extraverted

because they underreact to incoming stimulation, they should speak louder to compensate for this reduced perception. This is Petrie's explanation for why alcohol typically causes people to speak louder, as at parties. But extraverts did not speak louder even with a sample of 36 subjects. And it does not require much imagination to think of other reasons why people at parties might speak louder the more they drink. Correlated with the amount they drink is the amount of time they have been in the group, and probably with the degree to which they have become comfortable with it. Probably people would tend to speak louder at a party whether or not they were drinking, because this is the kind of behaviour one expects in such a situation.

Of all the measures considered in this study, only one provides any support at all for the claim that kinesthetic perceptual reactance -- the block test -- relates to any other perceptual variable. This is the finding that of those subjects for whom perceptual reactance went up (those who augmented) with the drug, the average loudness with which they spoke went down. Correspondingly, those subjects who relatively reduced tended to speak in a louder voice. Even this finding was significant only at the .05 level, and there were many individual subjects who increased their estimate of block size and yet spoke louder, and vice versa. Though estimates of block size may relate to perceptual reactance in general, the relationship is by no means certain. In addition it seems quite possible for a subject to vary dramatically from one testing to another, thus eliminating this variable as a primary explanatory principle for personality traits such as introversion/extraversion. At the same time this finding provides fairly substantial evidence of the validity

of the testing procedures used here. If the testing were merely done sloppily and did not really reflect general perceptual reactance, then those subjects who relatively augmented should not speak in a lower volume of voice, which they do, on the average. This implies that while perceptual reactance may be meaningful at one point in time, it may change from time to time in any given individual and so is rather useless as a method of predicting anything about general behavior. Petrie says that such variability should only occur in abnormal populations, yet, using the Neuroticism scale of the Eysenck Personality Inventory, this sample does not appear to be any more abnormal than the college population in general.

Petrie's description of all the possible complications in administering the block test implies, sometimes fairly directly, that if the obtained results don't correspond to those she has found, something must have been done incorrectly. This is, of course, always possible, but there is another less flattering explanation. Rosenthal's (1966) well known studies on experimenter bias illustrate many ways in which experimenters manage to find what they expect, in spite of the reality of the situation. It is all too common to report the results of studies that support one's initial hypotheses and fail to report those which don't work out. Though this experimenter was initially very positive toward the theories of both Eysenck and Petrie, none of the basic variables relevant to their theories draw any support from the final data. Dexedrine appears to facilitate intellectual abilities, but this seems to have nothing whatsoever to do with either extraversion or perceptual reactance.

Though extraversion appears to be unrelated to perceptual reactance, it is interesting to note the finding that those of this sample who were first born sons were on the average more introverted than those who had one or more older brothers. Five of the 24 first born sons scored less than 10 on the Extraversion scale with placebo. None of the 12 later born sons scored lower than 12. Among this sample it seems that though first born sons may be extraverted, later born sons are almost always extraverted. This isn't very likely a constitutional factor, as there are no known biological differences between first and later-born sons. Yet it has often been observed that parents treat children differently depending on their sibling position. This makes it seem most unlikely that, as Eysenck claims, extraversion is a constitutional factor little affected by upbringing.

CHAPTER 5

SUMMARY AND CONCLUSIONS

Only one of the basic hypotheses of this study was supported. This was the claim that dexedrine would bring about improved performance on the Otis Group Intelligence Test. Dexedrine did not cause agumentation of kinesthetic perceptual reactance, and in turn, subjects did not speak in a lower voice under the influence of the drug. Dexedrine had no effect on Extraversion scores on the Eysenck Personality Inventory; it affected neither group means nor variability.

Though subjects did do better on the intelligence test with the drug than with the placebo, this improvement did not relate to changes in perceptual reactance or to any other measure used in this study. The drug also did not bring about fewer involuntary pauses in subjects tapping a telegraph key as fast as they could, implying that inhibition as discussed by Eysenck was not affected. Nor did subjects tap more quickly on the drug, though previous studies have consistently reported that dexedrine decreases reaction time. It did, however, increase subjective time, causing subjects to relatively underestimate the length of a minute.

There was dramatically little support to be found in the results of this study for either the theories of Eysenck or Petrie. In the first place kinesthetic perceptual reactance does not seem to be at all

a reliable measure, but varies greatly from one time to another. The correlation that Petrie claims between kinesthetic perceptual reactance and extraversion scores simply does not exist in this sample, not even in the form of a non-significant trend. Similarly, the claim by Eysenck that extraverts build up inhibition faster and dissipate it more slowly draws no support. Using the number of involuntary pauses while finger tapping as a measure of the rate of buildup of inhibition, extraverts show no more pauses than do introverts.

That kinesthetic perceptual reactance may reflect general perceptual reactance at a given point in time does draw some support. If a given subject estimates blocks to be larger on one occasion than he does on another, he is likely to also speak in a lower voice, possibly as a result of his changed perception of how loudly he is speaking. Since this measure is so unreliable, however, it seems exceedingly unlikely that perceptual reactance could be a major determiner of personality traits like extraversion.

On the other hand it was found that extraversion does vary with sibling position -- further evidence that this trait is not constitutional but is instead a product of learning. Parents treat first born children differently than they do children who are born later, and this seems to have long term effects on Extraversion as measured by the Eysenck Personality Inventory.

Perhaps the most interesting finding of this study is that those subtests of the Otis which are more measures of the ability to see relationships rather than merely work quickly seem to be more affected

by the drug. Further work on which kinds of mental abilities are improved and why could be most fruitful. In this regard, the theories of Petrie and Eysenck appear not to be relevant.

Voice Volume
(decibels)

<u>Subject</u>	<u>Placebo</u>		<u>Drug</u>	
	<u>lowest</u>	<u>highest</u>	<u>lowest</u>	<u>highest</u>
B	76	83	68	79
A	78	82	76	81
M	78	82	75	83
P	82	86	80	85
T	72	81	74	81
R	77	85	80	84
1	72	80	73	81
2	69	77	68	76
3	71	80	74	81
4	70	88	75	84
5	69	81	72	82
6	78	83	79	85
7	75	80	73	83
8	79	83	77	84
9	80	86	75	82
10	75	85	79	88
11	73	79	77	82
12	78	88	79	87
13	79	83	77	82
14	70	84	75	84
15	75	85	80	86
16	75	84	80	89
17	83	92	75	84
18	85	92	84	90
19	80	85	80	87
20	74	84	76	82
21	64	79	64	75
22	74	84	77	85
23	67	80	70	79
24	80	86	75	83
25	68	81	68	79
26	67	82	69	81
27	70°	83	70	83
28	76	85	70	81
29	79	82	78	81
30	68	84	71	86
Sum	2686	3004	2693	2985
Mean	74.61	83.44	74.80	82.92
S.D.	5.02	3.24	4.39	3.21

Finger Tapping Test

<u>Subject</u>	<u>No. of Pauses</u>		<u>No. of Taps</u>	
	<u>Placebo</u>	<u>Drug</u>	<u>Placebo</u>	<u>Drug</u>
B	1	7	331	300
A	10	14	330	321
M	42	23	309	380
P	0	1	339	321
T	7	10	351	369
R	5	49	320	290
1	4	5	250	220
2	4	0	399	420
3	34	18	350	381
4	4	7	351	351
5	4	3	351	290
6	20	28	441	489
7	12	15	351	399
8	3	3	321	321
9	35	23	411	400
10	9	1	300	351
11	150	105	190	210
12	4	4	420	450
13	20	18	411	420
14	9	10	330	360
15	12	18	339	330
16	9	10	399	411
17	16	20	250	320
18	4	2	369	390
19	3	11	411	411
20	4	14	240	250
21	16	9	309	320
22	4	9	290	309
23	1	0	260	309
24	0	1	290	320
25	60	28	330	429
26	0	12	339	309
27	79	154	250	230
28	3	7	290	320
29	18	19	420	390
30	23	7	350	390
Sum	629	665	11992	12481
Mean	17.47	18.47	333.11	346.69
S, D.	28.15	29.26	57.80	63.81

Eysenck Personality Inventory

Subject	<u>E scale</u>		<u>N scale</u>		<u>L scale</u>	
	Placebo	Drug	Placebo	Drug	Placebo	Drug
B	12	10	7	5	1	2
A	17	17	5	9	5	2
M	17	16	6	6	1	4
P	18	15	5	2	2	0
T	14	12	13	13	1	0
R	20	17	10	10	3	0
1	18	22	4	9	3	1
2	18	14	20	19	1	1
3	16	14	7	9	0	0
4	13	12	10	10	1	4
5	17	16	6	9	4	3
6	8	7	8	8	8	8
7	17	18	18	18	1	1
8	19	19	13	12	2	0
9	17	17	15	19	1	0
10	11	15	12	14	5	4
11	10	10	11	10	3	0
12	20	19	7	7	0	2
13	17	16	10	9	1	2
14	17	14	7	0	3	2
15	11	13	9	10	2	1
16	13	13	10	9	3	1
17	20	21	1	4	2	1
18	16	20	5	10	2	1
19	17	9	1	3	4	4
20	9	10	13	17	0	3
21	9	12	4	7	2	2
22	7	14	2	5	2	0
23	16	17	4	5	0	2
24	9	15	3	3	1	0
25	13	14	6	6	0	0
26	12	7	17	15	2	2
27	20	19	4	7	1	0
28	16	17	0	4	0	0
29	13	9	15	15	4	4
30	15	14	9	9	2	2
Sum	532	524	297	327	73	59
Mean	14.78	14.56	8.25	9.08	2.03	1.64
S.D.	3.69	3.72	4.91	4.72	1.71	1.73

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