

This dissertation has been 64-2654
microfilmed exactly as received

MILLER, Howard Lee, 1937-
THE EFFECTS OF AUDITORY STIMULATION
ON THE CRITICAL FLICKER FUSION FRE-
QUENCY RESPONSE.

University of Hawaii, Ph.D., 1963
Psychology, experimental

University Microfilms, Inc., Ann Arbor, Michigan

THE EFFECTS OF AUDITORY STIMULATION
ON THE CRITICAL FLICKER FUSION FREQUENCY RESPONSE

A THESIS SUBMITTED TO THE GRADUATE SCHOOL OF THE
UNIVERSITY OF HAWAII IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
IN PSYCHOLOGY
FEBRUARY, 1963

By

Howard Lee Miller

Thesis Committee:

A. Leonard Diamond, Chairman
Merle Ansberry
Hubert Frings
William Oakes
Walter Steiger
Warner Wilson

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS.....	ii
LIST OF TABLES.....	iii
CHAPTER I.....	1
CHAPTER II.....	29
CHAPTER III.....	37
CHAPTER IV.....	63
CHAPTER V.....	69
GLOSSARY.....	71
BIBLIOGRAPHY.....	72

LIST OF ILLUSTRATIONS

Figure 1.	Schema of the optical pathway.....	35
Figure 2.	The effect of seven levels of acoustic intensity on the CFF of subject H. Y.	40
Figure 3.	The effect of seven levels of acoustic intensity on the CFF of subject P. C.	42
Figure 4.	The effect of seven levels of acoustic intensity on the CFF of subject P. F.	44
Figure 5.	The effect of seven levels of acoustic intensity on the CFF of subject H. H.	46
Figure 6.	The effect of seven levels of acoustic intensity on the CFF of subject R. C.	48
Figure 7.	The effect of seven levels of acoustic intensity on the CFF averaged over all five subjects.....	50
Figure 8.	The effect of seven levels of acoustic intensity on the CFF averaged over all subjects except R. C.	52

LIST OF TABLES

Table 1.	CFF at seven levels of acoustic intensity; subject - H. Y.	39
Table 2.	CFF at seven levels of acoustic intensity; subject - P. C.	41
Table 3.	CFF at seven levels of acoustic intensity; subject - P. F.	43
Table 4.	CFF at seven levels of acoustic intensity; subject - H. H.	45
Table 5.	CFF at seven levels of acoustic intensity; subject - R. C.	47
Table 6.	CFF at seven levels of acoustic intensity; average over all five subjects.....	49
Table 7.	CFF at seven levels of acoustic intensity; average four subjects.....	51
Table 8.	Individual analysis of variance; subject - H. Y.	53
Table 9.	Individual analysis of variance; subject - P. C.	54
Table 10.	Individual analysis of variance; subject - P. F.	55
Table 11.	Individual analysis of variance; subject - H. H.	56
Table 12.	Individual analysis of variance; subject - R. C.	57
Table 13.	Analysis of variance summary; all subjects.....	58
Table 14.	Combined analysis of variance; all subjects (five).....	59
Table 15.	Combined analysis of variance; four subjects.....	60
Table 16.	F ratios and probabilities of all main effects (five subjects combined) calculated using within cell error variance as the denominator.....	61
Table 17.	F ratios and probabilities of all main effects (four subjects combined) calculated using within cell error variance as the denominator.....	62
Table 18.	Duncan's multiple range test significance of differences of CFF means at each intensity level.....	63

CHAPTER I
INTRODUCTION

Intersensory influence has been regarded as an established fact for many years, and it has been accorded much investigation. Its parameters, however, are surprisingly unknown (London, 1954). It is becoming increasingly clear that these parameters are of more than academic interest. They would not only serve as guides in constructing models and seeking physiological mechanisms but would also prove to be of practical significance in obtaining optimal efficiency of modality responses of human and infrahuman organisms. For if the effects of other modality stimulation on the efficiency of response in one sense modality are known, we will be able to obtain greater efficiency in any setting which is dependent upon responses of these modalities.

Critical flicker fusion frequency (hereafter called CFF) seems an appropriate variable in measuring the effects of stimulation of another modality. It is amenable to precise measurement and is of theoretical importance as an indication of both visual and general response processes (witness, e.g., the extensive review of Landis, 1953, which includes approximately 1200 studies on the relationship between CFF and numerous other variables). Because of its obvious importance, and its physical

The study and physiological model proposed in this paper are due in large part to Donald B. Lindsley, whose suggestions and speculations were responsible for conceiving them. The model represents a slight modification of one created by Doctor Lindsley. Any credit which may accrue is largely due him, but he is in no way responsible for any blame.

I would also like to acknowledge the invaluable suggestions and criticisms of Dr. A. Leonard Diamond, whose assistance was of inestimable value.

characteristics which make possible precise control, audition is chosen as the modality to be variously stimulated. Intensity of sound will thus be the independent variable.

The purposes of this project are twofold. The first is to develop or adopt a physiological model which will embrace the behavioral and physiological evidence now available on intersensory influence. The second is to make additional observations of intrinsic interest which will also test certain specific implications of the model. This introductory section will concern itself with the previous and current behavioral data, the development of the model, the specification of supporting physiological evidence, and the statement of specific predictions.

History

The history of investigation of the effects of auditory stimuli on visual responses is seemingly marked by contradictions. Lack of control and/or reporting of all relevant data has made a clear picture almost impossible. Nonetheless, it is necessary to review these investigations for whatever clues they may contain.

Early Investigations of Intersensory Effects on Vision. In 1888, Urbantschisch noted that different tones seemed to have different effects on perceptions of various colors. His quantification was loose (or unreported). Little of relevance seems to have followed this initial attempt until the later 1920's when work began in earnest on intersensory effects in the USSR. Lazerev (1927) found that the visual sensibility of the periphery (primarily rods) increased under the influence of acoustical stimulation. Yakovlev (1935) in Moscow found that acoustic stimuli apparently increased the area of a colored field for cone vision. Later

(1938) in a detailed study published in the Journal of the Optical Society of America, he reported that the color fields for orange-red (maximum pass 680 m μ as determined by a fairly broad band pass Wratten filter) were diminished, under stimulation by 75 decibels of sound at 780 cps. Red (700 m μ) seemed unaltered.

Kravkov (1936) found that the threshold for rod vision was greatly raised under the influence of a 100 db tone of 2100 cps. The threshold for green (528 m μ) was lowered and orange-red (610 m μ) was raised. He believed that approximately 560 m μ was the dividing point. This seeming conflict with Lazerev will be discussed below. No satisfactory mechanism to "explain" the differences among regions in colors has been offered.

Kravkov (1935) reported also that the flicker fusion threshold increased for central or cone vision and decreased for peripheral (rod) vision. Later (1939) he found that "medium intensity" smells of bergamot oil, camphor, and geraniol raised the sensitivity to green and lowered it to red, as he found with sound. He adds that a reversal of this may sometimes be obtained depending on the intensity of the light.

Lazerev, then, noted a lowered threshold in the periphery and Kravkov found a raised one as a result of auditory stimulation. In commenting on the apparent conflict in the findings of these two investigators, Allen and Schwartz (1940) suggest that the difference might be explained by the fact that Kravkov took most of his data from the macula while Lazerev took his from the periphery. This is not entirely the cause, however, for a conflict in the periphery remains. Another possible explanatory mechanism involves the intensity and the frequency of the stimuli. Kravkov used a sound of 85 db at a frequency of 800 cps. These variables (intensity and

frequency) were not reported in this source for Lazerev's work. A different intensity (or frequency) may have a different effect. In direct support of this possibility, at least as far as intensity goes, we have the work reported by Allen and Schwartz themselves. They felt that their major conclusions supported Kravkov. It was true, however, that they obtained this relationship of an enhancement of the sensitivity to the green portion of the spectrum and a depression of the red (violet was not consistently affected) with their more intense tones. They commented further that "soft" tones reversed this but they failed to give absolute values. Besides sound, Allen and Schwartz reported that stimulation with light and the chemical (taste and smell) senses produced the same phenomena. They further noted that there were a series of reversals of these effects, oscillatory in nature, with the frequency of the oscillatory wave dependent upon the sense organ stimulated. This frequency varied from 40-50 seconds with audition to several minutes with the chemical senses. They hypothesized that whenever any part of the organism is stimulated, all parts that are (functionally) connected to it (which, in the brain means all parts) are thrown out of some sort of hypothetical equilibrium. This equilibrium is restored by "...a short series of oscillations of sensitivity in which the organs are alternately depressed and enhanced in responsiveness or excitability. Though the oscillations appear as a function of time, it is probably a cellular or molecular condition of the central area that fluctuates in activity." (p 119).

Unfortunately, their work is not well quantified, and from their report it is quite difficult to tell what was a reliable result, what an artifact, and what pure chance. Here, again, however, we have a change

of effect which is attributable to the intensity of the stimulation. The directions of the exact effects on various wavelengths (if reliable) still await explanation within a satisfactory theory of color perception.

Bogoslovski (1938) found an increase in CFF for central vision and a decrease for peripheral with an auditory stimulus.

Dobrakovna (1946) found that sweet, warmth, and sound all raised the CFF in the case of orange-red and lowered it to green-blue. Yellow was apparently unaffected. These, again, seem to contradict a number of the above reported results. Once more, however, as we will see below, the relative intensities of the stimuli may provide a possible mechanism to resolve this apparent conflict. Fry and Bartley (1936) found that a steady stimulus applied to one part of the retina raised the CFF (of white light) of another part of the retina. If the intensity of the steady stimulation was raised it reached a critical point (of intensity) after which the CFF began to fall. Earlier, Granit (1930) had reported interaction between distant areas in the human eye. When both were stimulated with intermittent light, the CFF to these stimuli was raised 2.5 cps. Later, with Harper (1930), he found this effect to be greater for peripheral than for central vision.

Later Work on Audition and CFF. More recently, we find three studies which concerned themselves with auditory influences on CFF. Gorrell (1953) studied the effects of high and low tones on the CFF of men, women and children. He found that both levels of frequency (2400 and 270 cycles per second) lowered CFF in all groups. The higher tone affected the response more than the lower tone. Unfortunately the intensity level of

the sound was not specified and it is impossible to estimate it from the information given.

McCrosky (1957) noted a lowering of CFF to a noise accompaniment (whose value was unspecified in the available sources). Levine (1958) tested several intensity levels of a pure tone (1550 cps) on two subjects. Although Levine's measurement of the sound stimulus did not take into account the interaction between the earphone, ambient level, and ear, he indicated in general that auditory stimulation raised the CFF up to some point of intensity and lowered it when the intensity was above that point. The intensity which was associated with the highest CFF differed in the two subjects and only one of the two unequivocally showed the smooth rise then fall of CFF.

From the late 1920's to the present, then (particularly from 1927 to 1945), there are a number of studies dealing with intermodality effects on visual sensitivity in general and CFF in particular. Aside from considerations of wavelength, considering only white light, there seems to be some support for the hypothesis that visual CFF response is raised by light to moderately high stimulation of another modality, and then lowered by higher intensities of such stimulation. (London, 1954, for example, remarks about "the rule of inversion" (p 539) which seems to be a conclusion of much Russian work on any intersensory effect. That is, higher intensity accessory stimulation seems to have the reverse effect of lower intensity accessory stimulation.) But this question in general, and that of auditory effects on CFF in particular, remain, even qualitatively, far from established. A somewhat broader picture of influences on CFF and related phenomena might suggest further hypotheses and/or mechanisms.

Related CFF Work. Knox (1945a) tested CFF in subjects who were instructed to watch for flicker and in subjects who were instructed to watch for fusion. He found a higher CFF in the subjects with a "set" for flicker. In a following article (1945b) he tested flicker fusion under four different auditory situations: 1) silence; 2) coarse auditory flicker of 15/sec.; 3) fine auditory flicker of 30/sec.; and 4) auditory fusion. The sound was reported to be 50 decibels at 1000 cps. He reported that auditory flicker could not create a feeling of visual flicker with a fused stimulus but that flicker already present could be made experientially more pronounced by a flickering auditory stimulus. Knox explains these results by the concept of "behavioral inertia"; i.e., it takes a greater force to change one experiential state to another (here fusion to flicker) than to vary the degree of one existing. He suggests a possible connection between this construct and the Gestalt "isomorphism" (i.e., that flicker is represented by some sort of cortical "oscillation"). It will be seen below how these results can be interpreted within a more physiological frame of reference.

Correlations of CFF with anxiety have also been reported. Goldstone (1955), using his own clinical criteria for anxiety, reported a lower CFF along with a higher intraindividual variability in his high anxiety subjects than in the low anxiety ones. Buhler (1953) also found a lower CFF associated with a high anxiety level. Busch (1953) found that a 'set' to concentrate increased CFF.

Related Physiological Evidence

What structure could account for the intersensory effects noted above? Wang (1934) compared action potentials at the cortex and superior colli-

culus with the electroretinogram and concluded that the similarities he found in flicker and intensity supported the hypothesis that flicker phenomena were determined peripherally. Ross (1935) amplified on a peripheral mechanism speculating that it was a function of the "physio-chemical situation" prevalent in any single receptor and the totality of effective receptors. Hecht and co-workers in a series of studies investigated the parameters of CFF in men and the clam Mya arenaria and concluded that the data could be approximated by a peripheral mechanism (Hecht and Wolf, 1931-32; Hecht, Schlaer, and Verrijp, 1933-34; Hecht and Verrijp, 1933-34a and b; Hecht and Schlaer, 1935-36; and Hecht and Smith, 1935-36). Zoethout (1947) reviews CFF and states that it is a retinal phenomenon.

The explanations, however, now seem to tend at least partly toward a central mechanism in light of recent evidence. For example, the rate of following flashes with evoked potentials is higher at the visual way stations to the cortex than at the cortex (Lindsley, personal communication).

Ireland (1950) reported a significantly higher CFF binocularly than monocularly. He concluded from this that some retinal interaction through the medium of the central nervous system must take place. Baker found essentially the same thing. This conclusion alone (that a central mechanism is involved) is hardly an adequate explanation if we wish to be able to predict further relationships.

Gorrell (1953) speculated on a Gestalt perceptual contention that an irrelevant stimulus to another sense affects the "brightness of the surround". He specified no physiological structures and no specific mechanism. Levine (1958) suggested that the concept of mutually recruitable neurons, that is, neurons which are stimulated by both vision and

audition, might provide the explanation. In effect, the stimulation of one sense could enhance the sensitivity of these neurons until the stimulation is great enough to necessitate a competition for the limited number. This mechanism also is neither very specific nor complete. Levine himself mentions that flexibility in prediction to fit almost any results is a feature of this model.

Where, then, should we start to look for a mechanism? What structure could account for intersensory effects? If we summarize the behavioral data from the first section of this chapter it seems that a fairly consistent thread runs through it. The influences of appropriate attention or "set", and light to moderate intersensory stimulation increases sensitivity (including raising the CFF threshold). High anxiety, inappropriate set, and strong intersensory stimulation result in a lower visual sensitivity and a lower CFF. This seems to suggest a continuum of general physiological activation. That is, raising activation to a point facilitates visual performance (such as higher CFF). Raising it past this point then lowers the performance level. This is in reality only a specific application of the familiar contention that attention and "concentration" improve performance and that excessively high activation states such as in strong emotion are associated with a decline in performance (Hilgard, 1962; Morgan, 1956).

Since activation is a suspected variable, then the reticular formation of the brainstem and the rest of the ascending reticular activating system could be an important structure in this connection (Lindsley, 1956, 1960). In the sense that this system receives collaterals from all of the specific senses, this structure is not entirely

antithetical to Levine's postulation of mutually recruitable neurons. There is indeed evidence that a central mechanism is operating in the determination of CFF. There is also evidence that the reticular formation may be in fact involved. We shall review this evidence below.

Jung (Lindsley, personal communication), working with single neurons in the visual cortex, found that they would cease responding to discrete flashes when the flash rate reached somewhere between five and fifty per second, depending on the neuron. It is interesting to note that at the illumination used (500 lux) the greatest upper limit of any single neuron corresponds to the approximate upper limit of the reported CFF in humans. He finds further correlations of single neuron data with psychological phenomena. For example the Brücke effect (an enhanced brightness response to a flickering stimulus of a particular frequency) corresponds to the mean number of following discharges for units in the visual cortex. Let us assume, then, that this cortical response (the following rate of individual neurons at the visual cortex) is related to behavioral CFF. If this is so, a most important finding of Jung was that this neuronal CFF was raised by electrical stimulation of the reticular formation. (Jung, 1958) A parallel may be drawn from this finding to the raise in subjective CFF associated with attention and arousal states found by investigators reviewed above (Knox, 1945; and Busch, 1953).

Recording from groups of neurons in the visual cortex of the cat, Lindsley (1956) reports a decrease in the two point (temporal) threshold following direct electrical stimulation of the brainstem reticular formation. The criterion was the recording of two distinct responses from the visual cortex. Flashes separated by 50 milliseconds were recorded at the

cortex as one response prior to the reticular stimulation. Two distinct evoked responses were recorded during the interval of 0-10 seconds (especially 8-10) following a brief, direct, electrical stimulation of the brainstem reticular formation.

Cortical Responses and Critical Flicker Fusion. There is, in addition, behavioral evidence that these identifiable evoked potentials from groups of neurons at the visual cortex are significant in the determination of the subjective experience of separation of flashes. Lindsley (1960) and others (see, e.g., Mundy-Castle, 1958) have found that flicker can be "followed" by evoked potentials which correspond to the flicker rate at all "way-stations" up to the cortex. The cortex, however, ceases to "follow" at a rate which corresponds to reported fusion in man and behavioral discrimination of fusion in cats (Lindsley, personal communication). Some following remains up to 10 cps higher than behavioral fusion, but it is sporadic and becomes less and less (proportion of time) the higher the flicker rate. This contradicts the above notions that the determinants of CFF are entirely peripheral and suggests that there is at least some central mechanism.

Knox (1950) coined the term "driving" to refer to this occipital following of flash rate. He demonstrated its occurrence in monkeys. It manifested itself 55% of the time between 2 and 13 flashes per second. Nine years earlier, Toman (1941) had reported the same phenomenon in man recorded by an electroencephalogram (hereafter EEG). He found that subjects showing a low percentage of alpha rhythm also showed the widest range of flicker following. This following was greatest in amplitude and regularity at approximately the frequency of the alpha rhythm. Electrical

reinforcement of potentials can account for the increase in the amplitude of the driving (by summation) but the change in regularity toward matching the alpha rhythm suggests a connection between the response of the visual cortex to stimuli and the alpha rhythm of the cortex. What evidence is there that bears on this possible connection?

Alpha Rhythm and Cortical Responses. Early proposals on the origin of this alpha rhythm entertained the notion that this activity was a summation of spike potentials (Jasper, 1948). The trend in recent years, however, has been clearly toward the conclusion that alpha waves represent a distinctly different process which may be related to excitability. In 1948, Jasper suggested that these waves might be related in some way to spontaneous rhythmic fluctuations of local potentials reflecting the excitability of groups of neurons in the cerebral cortex. He suspected that this activity might be a result of certain specialized cells. Later, Li, McLennan, and Jasper (1952) recorded simultaneous records of action potentials and the slower oscillations of "brain waves". They expanded these notions of fluctuations of local excitability when the evidence they uncovered indicated that the spikes and waves were independent of each other under some circumstances but perhaps not under others. Their evidence will be reviewed below.

At about the same time, Lindsley (1952) proposed that this activity represented a fluctuation in response tendency or excitability of individual neurons (reflected as a change in local potentials). He further suggested that this was recordable as alpha rhythm only when large numbers of these cells were synchronized with regard to this electrical potential change. Lindsley expanded this notion of local rhythmic excitability

fluctuations (1960) and included it in a theory of wakefulness and alertness states.

Other writers, for example, Mundy-Castle (1958), seem to adopt a similar hypothesis in their speculations. It is difficult to find major writers adopting sharply differing views. Local potential changes, related to excitability fluctuations, then, seem to be the direction that is taken by most workers in the area. Before examining recent evidence on this point, however, let us turn to one striking exception to this trend.

Diametrically opposing this excitability hypothesis, Kennedy (1959) asked whether the observed rhythm may not be merely an artifact vitually unrelated to any periods of excitability or refraction. He proposed that these potential changes may be an artifact resulting from three sets of conditions normally found in the brain: "...(a) an electrically charged state, presumed to arise through chemical oxidation-reduction reactions; (b) a gel mass with a co-efficient of elasticity approximated by commercial gelatin, and (c) a periodic mechanical pulse, to set the gel into oscillation at its resonant frequency. It is proposed that the interaction of these conditions in the skull may produce the oscillating potentials recorded from normal human S's by standard electroencephalographic equipment." (Kennedy, 1959, p 352).

In order to demonstrate this mechanism, Kennedy used a mass of commercial gelatin under conditions roughly similar to the human brain. He mechanically pulsed this mass in order to simulate the arterial pulsing of the brain so that it will resonate at its particular frequency. He demonstrated that this resonance is capable of producing rhythmic poten-

tial changes which roughly resemble the alpha wave of the cortical EEG.

As further support, he established an alpha rhythm in a subject in whom it was not previously found by restoring his intracranial pressure to normal through use of a head plate to cover a small wound. These demonstrations are certainly ingenious and worthy of further study. As of now, however, they are not fatal to the hypotheses they are aimed at disproving. Perhaps a certain intracranial pressure is somehow necessary to maintain synchrony. It may be related in some way to the metabolic re-establishing of positive potassium and sodium ion balance. To be sure, there is no certain, immediate explanation in terms of the excitability hypothesis outlined above. Neither does there seem to be a dearth of possible explanations. There is also growing evidence to support the views of Jasper and his followers concerning cyclical excitability changes. It seems appropriate now to present briefly some of the important findings relevant to deciding the nature of alpha rhythms.

Jasper (1948) postulated that alpha rhythms might be related to changes of local excitability in groups of neurons. He rejected spike discharges because the waves are too slow and do not show an all or none character. He then cited the fact that experimental undercutting of the cortex, while diminishing the amplitude of alpha rhythms, does not abolish them. He felt that this belied the possibility of using major reverberating circuits as the explanatory device, for these would have been interrupted by the experimental lesions. He admits, however, that the possibility remained that small intracortical reverberating circuits were responsible for these rhythms. Though this possibility is mathematically demonstrable, it remains to be experimentally verified and Jasper feels

that it is an unlikely explanation. He considered as a greater possibility the electrical field effect (ephaptic transmission) which he demonstrated with Monnier (1938). Arvanitaki (discussed in Jasper and Monnier, 1938) showed that this effect (ephaptic transmission) would synchronize the spontaneous firing of two fibers brought into close proximity to each other.

Further evidence for the notion of cyclical excitability is mostly indirect but voluminous. Barren (1938) recorded slow waves at synapses the cyclical changes in which seemed to be related to synaptic excitability changes. This demonstrated some connection between a wave-like potential change (like alpha rhythms) and the excitability of structures (synapses) that were the locus of these waves. Hoagland (1936) measured the relationship between body temperature and the frequency of the alpha rhythm. This contributed to the original impetus of some of these speculations on excitability and alpha rhythms. Hoagland claimed that the function (alpha rhythms as a function of body temperature) fit the Arrhenius velocity equations for chemical processes. This finding suggested a relationship between a basic respiratory phenomenon of the cortical cells and the alpha rhythm. Hoagland commented, "The frequency of the alpha rhythm appears to be directly proportional to the speed of the underlying respiratory phenomena of the cortical cells which behave electrically like relaxation oscillators." (p 604). Such a relationship to a respiratory process of the cell could readily be associated with excitability.

Bishop (1933) showed some form of rhythmic fluctuation in the excitability of the occipital cortex at the frequency of the 5-6 cycle alpha

rhythm in rabbits. Later (1952) Li, McLennan, and Jasper, using micro-electrode techniques, suppressed spike discharges but not waves from the cortex under a general anesthetic. Kennedy (1959) took their work to indicate that there was no correlation whatever between these spike discharges and alpha rhythms. This does not seem to be a justified conclusion. Although they found no support for the proposition that slow waves result from envelopes of spike discharges or reverberating circuit, they did report some correlation between the occurrences of these spikes and the timing of the waves in animals not under a general anesthetic. Li, McLennan, and Jasper concluded: "Brain waves do seem to be a phenomenon of a different order. The most plausible hypothesis is that they represent synchronized oscillations in membrane potentials, possibly involving small interneurons and dendrites in the cortical matrix, oscillations which would have a definite effect upon neuronal excitability but not dependent upon neuronal discharge." (p 657).

These studies of Jasper, et al, Barren, Bishop, and Hoagland, seem to be the high points of the early support for the excitability hypothesis. Since this is, however, obviously insufficient evidence to ward off the objections of investigators such as Kennedy, we should peruse the more recent literature for further indications.

Walter (1959) reports different functional associations with different rhythms. Numerous investigators (such as reported in Walter, 1959) have found that different rhythm patterns appear in different physiological states. For example, the slow (5-6 cps) delta waves appear in sleep and the fast, shallow, and irregular beta pattern appears during activity. This is certainly not conclusive evidence for an excitability

hypothesis. It does, however, suggest that an artifactual explanation (such as Kennedy's) is quite incomplete without numerous additional unsupported postulates.

Evidence toward such functional relations between alpha rhythms and psychological states is given not only in the well known associations of various rhythms with age and emotional state (Morgan and Steller, 1950) but more directly such as reported by Lindsley (personal communication) on the findings that the blocking time of alpha waves by visual stimulation was correlated directly with the number of cycles of alpha rhythm regardless of the frequency of the cycle. This was also discussed earlier by Mundy-Castle (1958) who speculated on "...the possibility that under equivalent stimulus conditions, subjects with high alpha frequencies may show shorter blocking latencies than subjects with low alpha frequencies...".

The above discussion suggests a relationship between phase of the alpha rhythm (especially in the visual cortex) and the readiness of the cortex to respond to stimuli. Why can we not measure directly a correlation between phase of the rhythm and reaction time to visual stimuli in order to test this? There are difficulties with such measurements. The properties of the retina interfere with accuracy of these measures. It (the retina) acts to some extent to spread out the volley of impulses of a brief flash of light so that this volley down the visual pathway is ragged, spread out over more than 50 msec. even in response to a few microsecond flash. This would allow impulses to be received by central nervous system structures even if they originally occurred in some refractory phase of a cortical cycle because the afferent volley would

outlast the critical period of the cycle.

Despite this, Lansing (1957), meeting this difficulty by large samplings of measurements, found significant correlations between a particular phase of the alpha wave and reaction time. The phase of the alpha cycle which was calculated to be the most excitable was the descending quarter. The constants used to determine this might have been off, however, because of a high interindividual variability. The rest of the cycle seemed to represent a refractory period, whether relative or absolute could not be determined. As mentioned above (Bishop, 1933), this correlation of visual reaction (cortical) and alpha rhythm was found in rabbits. Bartley (1940) also found in rabbits that there was an enhanced response to flicker at about the alpha frequency (Bartley effect). According to Bartley's conclusions, this enhancement was evidently not merely an electrical summation artifact.

These above studies of Lansing, Bishop, and Bartley supply direct evidence that slow waves (alpha rhythms in Lansing's study) are significantly correlated with readiness to respond. As we shall see below, these alpha rhythms are modifiable through the medium of other brain structures. This relationship to other parts of the brain may provide a mechanism for "explaining" the data of intersensory effects.

Ascending Reticular Activating System and the Alpha Rhythm. We have indicated above a relationship between neuronal CFF and the reticular formation from the data of Jung (Lindsley, personal communication, Jung, 1958). Similarly we noted a relationship between the temporal two point (flash) threshold and the reticular formation from the data of Lindsley (1956). Further, in the above section, we have discussed

data which support a connection between the alpha rhythm and cortical excitability. It is interesting to note that there is also a definite relationship between reticular activity and alpha rhythm.

The effects of reticular stimulation on the alpha rhythm are well known. Lindsley (1960) and Mundy-Castle (1958), for example, report the "activation pattern" of the EEG which is found when the reticular formation is stimulated. This pattern is no longer the 8-12 per second (most often close to 10) sine wave of the alpha rhythm but a pattern which becomes shallower and faster, the greater the stimulation (beta rhythm).

Audition and Reticular Activity. At the outset of this chapter, our stated object was to deal with intersensory effects, specifically hearing upon vision, more exactly upon CFF. In order to explore a possible route for such a connection, we digressed at length into evidence tying the reticular activating system with first cortical rhythms and through these to response patterns of the visual cortex and thence to CFF. One step remains and that is a simple one: tying audition with the reticular activating system.

A concise statement of what is known about this relationship is found in Ades (1959).

"Like other sensory systems, the acoustic makes its contribution to the reticular activating system presumably at brainstem levels. Certainly it has been demonstrated that arousal can be induced by auditory stimuli in animals in which the standard acoustic projection pathway has been bilaterally interrupted... Thus, there is through the reticular system another route from brainstem acoustic mechanisms to cerebral cortex, though this is of general rather than specifically auditory distribution. The ascending reticular system seems to be a diffuse and multisynaptic route, so the auditory and other specific modalities of input tend to be swallowed up in the more comprehensive functions of the ascending reticular system. It is impossible to say to what extent, if any, this

system may serve a specific sensory function, though it would appear that this could not be extensive in the light of what we know about reticular function." (p 591).

There is, then, little question that audition contributes to reticular activity. Unfortunately, the precise extent of this contribution is not yet known. Perhaps when the parameters of intersensory effects are better known, a comparison of the relative amount of effect due to audition and the other senses will help provide this knowledge.

The Model

Introduction. We supported (above) a connection between the reticular system and cortical responses, and one between alpha rhythms and cortical responses. A relationship between CFF and alpha rhythms does not seem unlikely. If, indeed, the alpha rhythm represents a cyclical excitability change in cortical cells, then the responses of the cortex to a flickering stimulus would be related to cortical excitability and, therefore, alpha rhythms.

The natures of all of the above relationships are still matters for conjecture. In fact, the nature of the alpha rhythms themselves is still a matter for conjecture. Jung, (Lindsley, personal communication), by indicating that single neurons give off such a rhythmic local potential change, supported the conjectures of Hoagland (1936), Walter (1959), Lindsley (1960) and others that recorded alpha rhythms were a result of a summation of the synchronized individual local potential rhythms of these single neurons. Other than Kennedy (1959) (discussed above) there appears to be almost no major disagreement with this hypothesis. For the purpose of attempting to construct an explanatory model for the phenomena discussed in this section, we shall adopt this assumption that

the alpha rhythm is a result of the synchronized oscillations of local potentials of the individual cortical neurons. If this is true, what could cause this regular rhythm to change to the shallower irregular (beta) rhythm seen with reticular stimulation? Two possibilities suggest themselves: one) that the individual cells change in their rhythm; two) that these cells are desynchronized (that is, no longer beat in phase with each other) so that their local potentials are no longer summed.

The first possibility (that each individual cell changes its rhythm) has been shown to be false by Jung's single neuron recording (Lindsley, personal communication). Since the responses of the individual cells do not become more rapid and shallow, the second possibility (desynchrony) which has been popular since Adrian and Mathews in 1934 (discussed in Hoagland, 1936) seems to be quite likely. Assuming this desynchrony, how could it "explain" the rise in neuronal CFF (following of flashes) found by Jung with reticular stimulation? In order to answer this question and continue our limited model of central nervous system behavior, we must digress for a moment to discuss the neurological basis for the phenomenon of CFF, that is, apparent fusion of a flickering stimulus.

We know from recordings from the optic tract in the frog that the onset of illumination elicits in some groups of neurons spindle like bursts of electrical activity, i.e., the cells' response ("on" firing). Similarly, the cessation of illumination also elicits this spike activity from some other groups of cells ("off" firing). It is reasonable to assume that these fibers are represented in the striate cortex. Generally, the record of activity from the former ("on" firing) is greater

than that from the latter ("off" firing) (Granit, 1956; Hartline, 1935, 1938). These "on" and "off" fibers seem to be mutually antagonistic. They inhibit each other when adjacent (or close) cells discharge simultaneously or close to each other temporally (Granit, 1956). Thus the EEG record of the cortical response to flashes spaced close enough together in time frequently will show only what appears to be a somewhat weakened "on" response (Lindsley, personal communication). It seems reasonable to assume, and for the sake of our model, we shall, that this point of total inhibition of the "off" responses to a flickering stimulus represents the point of perceptual fusion. Keeping the above data of "on" and "off" inhibition and the assumption concerning the nature of fusion in mind, let us return to the question of a mechanism for the effect of reticular stimulation on CFF.

We assumed above that reticular activity serves to desynchronize the neurons in the striate cortex so that their local potential changes and therefore their excitable phases do not coincide. We shall further assume that both these "on" and "off" fibers contribute their local potential changes (reflecting excitability) to the alpha rhythm. Desynchrony between groups of "on" and "off" fibers, then, would make these groups of adjacent cortical areas less likely to discharge at or very nearly the same time since their excitable phases would not coincide. This would, then, reasoning from above, lessen inhibition between "on" and "off" firing. We would, therefore, expect increased on and off responding with this decrease in inhibition. (We would not necessarily expect to find responses to each flash, but this would not seem to be required for flicker.) Therefore, the more the "on" and "off" fibers

are out of phase with each other, the less they will inhibit each other, and the higher the fusion threshold. Why, then, would we find a decrease in CFF with a larger amount of reticular activity?

There seem to be at least two possible answers to this question under our framework. For one, we must assume some additional blocking mechanism which interferes with reticular activity after this activity reaches a certain level. Lindsley (1960) states after drawing the conclusion that moderate interaction (of the senses) could lead to facilitation in the reticular formation, "...whereas excessive bombardment could result in complete blocking of reticular activation of the cortex causing disturbance in attention and awareness, and even complete loss of consciousness as observed in some seizure states." (p 1585). Such an assumption certainly seems to fit the observational data. At this point, however, at least one other possible mechanism seems equally feasible.

We have assumed above the CFF rises with an increase in reticular activity when groups of "on" and "off" fibers go out of phase with each other. It was reasoned that adjacent out of phase groups would not be ready to respond at the same time and would therefore not inhibit each other when the light flashes are rapid. If, now, with greater reticular activity, the desynchrony progressed from between these groups of "on" and "off" cells to within these groups so that the cells within a group were out of phase with each other, both "on" and "off" cells in the entire area would fire in an essentially random manner. We would again have a situation in which some "on" and "off" cells close to each other spatially would be almost or fully in phase with each other and thus excitable at approximately the same time. This would therefore increase

inhibition over the amount found when groups of "on" and "off" fibers were synchronized within themselves but out of phase with each other. This increased inhibition should then lower CFF. It is interesting to note that this desynchrony mechanism's progressing to within groups would not predict a CFF lower (with highest stimulation) than that found when the cells are all in phase (with a low level of stimulation). Lindsley's mechanism of reticular blocking, however, would predict a lower CFF at this highest level of stimulation.

The two above mechanisms (reticular blocking and desynchrony of first between groups and then within groups) are not necessarily antithetical. Reticular blocking could occur at higher levels of stimulation and progressive desynchrony at somewhat lower levels. It is not yet possible to say what combination, if any, of these two mechanisms occurs. The essential need is to include within the model a device to facilitate and then lower cortical response tendency by progressively increasing reticular stimulation.

Specific Postulates and Predictions of the Model. As supported or suggested earlier in this section and in the section on "Related Physiological Evidence" above we have arrived at a set of postulates for the model. These postulates are designed to connect auditory stimulation with CFF. We shall briefly summarize and enumerate these postulates now (with somewhat arbitrary divisions).

1) Local potential changes of cells in the cortex are responsible for the recordable alpha rhythms when they are synchronized or "in phase" with each other. "On" and "off" cells in the striate cortex (see above) specifically contribute to this rhythm.

2) These local potential changes represent cyclical changes in the excitability of the cells.

3) Firing of both "on" and "off" cells is necessary to perceive a flickering stimulus as flickering.

4) Adjacent "on" and "off" cells inhibit each other when they discharge simultaneously or temporally very close together.

5) Activity in the ascending reticular activating system desynchronizes groups of "on" and "off" fibers with regard to these local potential changes. This is reflected on the EEG by the low amplitude, fast, irregular pattern of the "beta" rhythm seen in activity states.

6) This desynchrony between groups of "on" and "off" cells lessens inhibition between them.

7) This lessened inhibition causes CFF to rise by increasing the responsiveness of the groups of "on" and "off" fibers to the flickering stimulus.

8) Excessive reticular stimulation causes either a blocking of reticular activity or a progressive desynchrony to within groups of "on" and "off" fibers which increases inhibition from the former state of desynchrony between groups. Either mechanism would predict a CFF which lowers from its optimal point as a function of increasing stimulation. Reticular blocking predicts a lower CFF at this point than found at the situation of complete synchrony (low reticular activity); progressive desynchrony does not. These mechanisms are not antithetical and both may operate at different levels of activity (blocking, of course, at a higher level).

9) Increasing auditory stimulation increases activity in the reticular formation.

It is obvious that other possible mechanisms, even within this same basic framework as this model, may account for some of these phenomena. For example, the locus of interaction of the specific and non-specific pathways might lie, not at the cortex at all, but at the thalamus where presumably reside nuclei of three different types: specific, non-specific, and associational (Ranson and Clark, 1953). Some sort of interaction here would not necessarily be antithetical to the evidence presented above but could supplement the outline mechanisms. At our present state of sparse knowledge concerning such nuclei, however, it would seem wisest to confine our model building to the somewhat better supported and somewhat more readily testable. If this model successfully predicts future behavioral or physiological data, its usefulness will be demonstrated.

Conclusions and Predictions

We have now constructed a physiological model, based on the effects of the specific senses on the reticular formation and its consequent effect on the specific senses. This model is capable of generating behavioral predictions, and, of course, conversely capable of being modified through behavioral data. Drawing from this mechanism we would expect to find at first a rising CFF as a result of auditory stimulation (corresponding to our desynchronizing effect from reticular stimulation) and a decreasing CFF with further stimulation. Earlier, we reviewed studies, notably those of Allen and Schwartz (1940) and Levine (1958) which found a lowered CFF when a high intensity sound was utilized. (The exact

intensity was unavailable from Allen and Schwartz and differed in the two subjects of Levine but was over 100 decibels in the subject who showed a significant rise.) Because Levine's relative maximum was around 90 db by his mode of measurement, we would expect that within the range of intensities employed in this study (from below 20 to 109.0 db) we would find a rise in CFF and then a lowering from this highest point. Since it is unknown precisely how much effect audition has on reticular activity (see above, Ades, 1959) it is not yet possible to predict from our model where this point of descent (in CFF) will occur. The two mechanisms of reticular blocking and desynchrony within groups offered above would be the model's counterpart to this behavioral prediction. As indicated, however, the model is not at this point capable of predicting the exact point of this descent. Discovering this point of lowering of CFF might suggest a physiological prediction of the relationship between audition and reticular activity.

The experimental part of this study, then, is purely behavioral. Since, however, specific predictions of this study are suggested by the model outlined above, it is capable of throwing some light on the usefulness of this model. Further, it is felt that a careful correlation of such behavioral research with the still to be measured parameters of the physiological systems involved is a most important step in knowledge of the function of these physiological systems. Obviously, the most immediately useful knowledge resulting would be of the psychophysical relationships themselves.

Specific Predictions. It is expected that as the intensity of auditory stimulation (the independent variable) increases, the CFF threshold

(the dependent variable) will increase initially in value until some point of intensity in the auditory stimulus is reached. Beyond this point of intensity further increases in the auditory stimulation will cause a decrease in the CFF threshold. From the data of earlier studies it is anticipated that this point of relative maximum in the CFF as a function of acoustic intensity curve will occur in the range of intensity used in this study.

Summary

The behavioral data heretofore gathered on intersensory influence are both incomplete and partly conflicting but seem to suggest a curvilinear effect of audition on CFF. This project proposes to adopt a model to explain intersensory effects and make further observations of intrinsic value which will test specific behavioral implications of this model.

The model is outlined and supported. Its mechanism involves the effect of audition on the reticular formation and the subsequent effect of this increased reticular activity on the cortical responses related to sensory reactions. The model predicts a diphasic (initial rise then fall) effect of audition on CFF.

CHAPTER II

PROCEDURE

Experimental Room. The experimental room is essentially rectangular, except for a sloping room. The width is approximately 40 inches, the length approximately 80 inches, and the height 79 inches in back and 93 inches in front. Its temperature is maintained at a relatively constant level (70-72 degrees F.) by a GE Thinline one horsepower air conditioner with a capacity of 6600 BTU/hr. Sheets of glass wool insulating material were suspended from the ceiling of the room so that they completely surrounded the head of the subject. They were approximately two inches away in front and one foot at the back and sides, when he was seated in the experimental chair. This experimental chair and all moving equipment were mounted on rubber pads.

The ambient noise level of this room was taken at various times during the day, corresponding to the times used in testing the subjects. All equipment which was in operation during the experimental sessions proper was also in operation during the calibration and measurement of all equipment including the testing of the ambient noise level. The instrument used in this last connection was General Radio model 1551A sound level meter. The ambient level was found to vary from 46 to 51 decibels (hereafter, db) re .0002 dynes/cm² in accordance with the amount of incidental human and equipment activity outside the experimental room.

The subjects were seated in a wooden armchair (which was mounted on rubber pads). They placed their heads on an adjustable chin rest located in front of the eyepiece.

Apparatus. The apparatus can be divided into several parts on a functional basis: 1) equipment for delivering a steady source of illumination; 2) equipment for producing and recording the rate of flicker in the visual stimulus, and 3) equipment for delivering the auditory stimulation.

1) The optical pathway is shown in figure one. The light source is a GE 28 volt, 150 watt DC sealed beam aircraft landing light. This is supplied with current by a U. S. Government Type CCL-211335 DC generator with an AC motor. The lamp actually operated at 25 volts and 4.8 amperes.

The light path travels through a heat absorbing glass which is approximately 1/2 inch from the light source and in turn a rotating sector-ed disc for flicker, a flashed opal diffusing glass, a 4 mm. diameter circular pattern for stimulus, a fixed filter, a 3 mm. artificial pupil and into the eye of the subject. The luminance of the stimulus at the eye was found to be 1.266 log millilamberts. It was measured by a Welch Densichron "10" photometer and compared with the known luminance of a standard light source which was measured under the same conditions.

The pattern, which is located immediately on the subject's side of the diffusing glass is 4 mm. in diameter. A lens is employed to make the rays parallel which maintains the stimulus size from any distance behind it. This lens has a focal length of 25.0 cm. (4 diopters power). The visual angle is therefore 55'. The fixation point was the center of the stimulus, that is, central. This limits the stimulus to the central, rod free portion of the outer fovea (Polyak, 1941; Davson, 1949).

2) The mechanism which produces the flicker is a circular opaque rotating disc 8 inches in diameter. It has two diametrically opposed open sectors, each subtending an angle of approximately 26° , cut through the outer part of the disc to allow the light to pass when these sections cross the pathway. This provides a light/dark ratio of approximately one to six. This disc is driven by a Bodine electric NSH-12R DC motor whose speed is controlled by a variable resistor. A photo-electric cell, placed on the opposite side of the disc from the light pathway of the apparatus is activated by light striking it through the open sectors in the disc. The photocell sends pulses to the Hunter model 120A Klockcounter which records the number of these pulses in one second (timed by an Eagle Signal Corporation "microflex" electric timer) whenever a switch (available to the subject as a button) is depressed. This, therefore, records the flicker rate of the stimulus during the second immediately following his depressing of the counter-switch. The control for the speed of the motor and the resetting of the counter are mounted on a board in the experimenter's room. This control board is available to the experimenter during testing sessions.

The variability of the flicker and recording equipment was measured at three representative speeds (approximately 36, 48, and 58 cycles per second) with the motor running continuously for 50 trials at each speed. The standard deviations of the measured cycles per second of each of the three speeds respectively are $.49 \pm .10$, $.45 \pm .09$, and $.66 \pm .13$ flashes per second at the 95% confidence interval. The apparatus only records in whole cycles per second. Undoubtedly much of the noted variance is due to the fact that the "true" frequency of the flicker was not a whole

number. The recorded frequency, therefore, would vary between the two whole numbers surrounding the "actual" frequency.

3) A Heathkit sine wave radio generator model AG-9A delivers the auditory stimulus to a pair of telephonic WL Recording Company Type COF 49456 PDR padded earphones. The attenuation of the earphones and padding was calibrated by obtaining thresholds in a sound field with and without the earphones in place on the subject's head. It was found on two subjects that the earphones and padding raised the absolute threshold between 30 and 33 decibels. The ambient noise level was therefore assumed to be equivalent to approximately 15-20 decibels when the earphones were in place.

The auditory stimulus was desired to be well within the audible frequency range and not uncomfortable. Eight hundred cycles per second was arbitrarily chosen. The intensity levels were chosen for the widest range possible concordant with ease of delivery (including switching rapidly from one level of stimulation to any other) and safety to the ear.

In order to best approximate the characteristics of the stimulus when the earphones were in place on the subject's ears, the sound was measured by an Allison Laboratories model 300 calibration unit mechanical ear in the situation and place in which the earphones were during the experiment proper. There were seven levels of stimulus in the experiment with the lowest level consisting of the ambient noise as attenuated by the earphones and padding, which was assumed (as above) to be between 15 and 20 db. The other six levels of the stimulus are as

follows (in decibels re: .0002 dynes/cm²): 70.0; 85.5; 90.5; 95.4; 100.0, and 109.0.

Subjects. Five subjects were employed, all drawn from the population of students at the University of Hawaii. All five were males between the ages of 19 and 32. The subjects had normal (20/20) distance vision in the eye which was used in the experiment. This was the right eye for three of the subjects and the left eye for two.

Method. The subjects were instructed to abstain from ingesting stimulants or depressants on the days they were tested. They were specifically warned against coffee, tea, chocolate, alcohol, tranquilizers, or sedatives of any kind. They were also asked to refrain from smoking for at least two hours prior to their being tested.

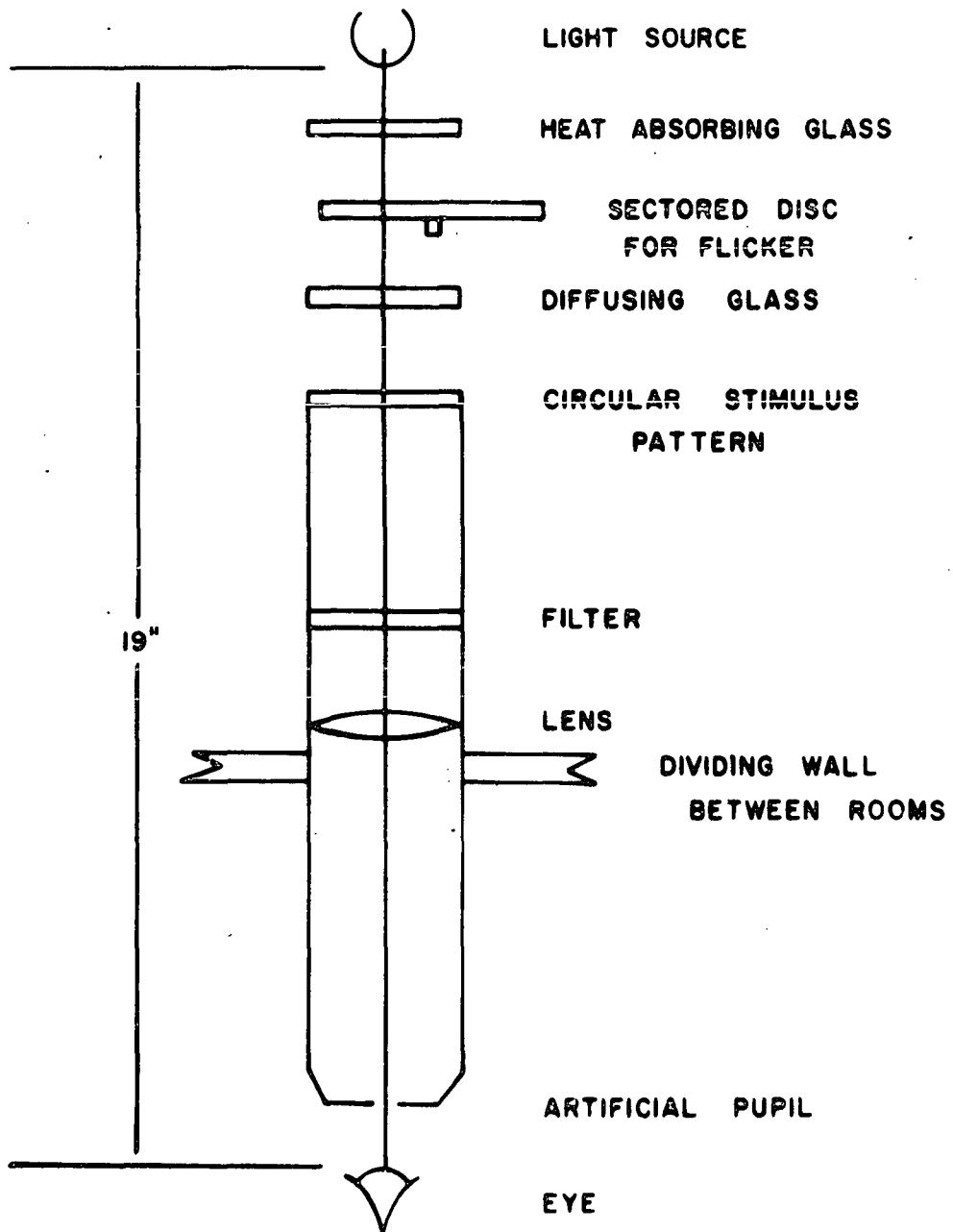
The subjects were seated in the experimental room which was darkened, and allowed to dark adapt without activity for 15 minutes. The apparatus was then tested and a practice trial was taken. This procedure took an average of three additional minutes which varied only a matter of about ten seconds from session to session. The total dark adaptation time (before practice and hence illumination) was, therefore, in excess of 15 minutes. This time is more than sufficient to essentially complete foveal dark adaptation (Hecht, 1921-22; Davson, 1949). The method of determining the frequency of flickering at which the light appeared fused was the psychophysical method of limits. The experimenter increased the speed of the motor until the subject indicated fusion. This frequency was recorded. The motor speed was further increased and then decreased until flicker was now indicated by the subject. The subject indicated fusion and flicker alternately by depressing a button which caused the

apparatus to indicate the frequency at which the light was flickering, as described above under apparatus. One ascending trial (where the frequency of the flicker was increased and thus the threshold for fusion was recorded) and one descending trial (where the frequency was decreased and thus the threshold of flicker was recorded) were averaged and considered to constitute a complete trial. The speed of changing the stimulus (frequency) was varied in an essentially random manner to prevent the subject from utilizing rhythmic cues.

An experimental session included all seven levels of the stimulus. One experimental session, which took a total of approximately 55 minutes was given at the same time every experimental day to each subject until seven sessions were completed. Each level of the acoustical stimulus occurred once and only once in every possible one of the seven positions of testing. Three determinations (complete trials) including both ascending and descending portions) were given at each level and then the stimulus was changed. After the fourth stimulus level, the subjects were allowed to rest for five minutes. The remaining three stimulus levels were then given.

Before the experiment proper, each subject was trained in the technique of making the judgments for at least seven sessions. In addition, two of the subjects took part in pilot studies which provided approximately 20 sessions of extra experience in a similar situation. Under the final conditions of the experiment, significant improvement in the technique of judging required of the subjects, as measured by a decreasing variance, was not noted during the course of the trials ($F = 1.087$, $105/105$ df.). This attests to the adequacy of the previous training.

Fig. 1. Schema of the optical pathway



CHAPTER III

RESULTS

The CFF averaged for each intensity level over all five subjects is highest at the fourth level (90.5 db) and lowest at the first (ambient). The difference is 1.13 cps. The mean CFF at each intensity level and the difference from ambient is given averaged over all 5 subjects in table 6 and presented graphically in figure 7. This information is presented for each subject individually in tables 1-5 and presented graphically in figures 2-6.

An analysis of variance for the significance of the effects of acoustical intensity (the independent variable), position of each set of three trials within an experimental session, and experimental sessions (days) was performed for each subject. The acoustical intensity (treatment) was seen to exert an effect, significant beyond the .001 level, in four of the five subjects. These analyses of variance are presented in tables 8 through 12, and are summarized in table 13. In addition, an overall analysis of variance for all five subjects is presented in table 14. Treatment effects are seen to be significant beyond the .001 level over all five subjects. The interaction terms with subject (one subject per Latin square) are of interest inasmuch as they are a measure of the differences in effect of the main variables from subject to subject. Thus, the interaction between treatment (intensity) and subject represents the variance caused by differences in effects among subjects. This is seen to be significant at the .005 level.

In accordance with standard practice as recommended by Edwards (1960) (who cites Wilk and Kempthorne, 1957), the mean squares for all

interaction terms are tested for significance over the within cell or error variance. Since these are found to be significant, the main effects are tested over this interaction term and the tables are presented on that basis. It is felt that in this instance, however, there is justification for another choice of error term, that is, the within cell variance.

In a meaningful sense, these Latin squares do not represent random replications of the experiment from an infinite population of possible replications. That is, there is ample reason to expect significant individual differences (for example, Levine, 1958). The interaction terms are therefore not merely an estimate of experimental replication error but a direct measurement of the differences in the effects from subject to subject which is attested to by the fact of their significances. This places us in the position of comparing the variance due to main effects not over an extrinsic error variance but a significantly large effect variance which is evidently intrinsic. The pooled within error variance term on the other hand is a measure of experimental error. Accordingly, table 16 presents the F ratios and significances of the main effects of the combined subjects analysis as calculated using the within cell error term as the denominator. (This above argument is largely analagous to that presented by Lindquist, 1953, concerning the inappropriateness of the significant interaction as an error term for factorial designs.)

The testing of subject R. C. was interrupted because of two brief illnesses which interfered with the experiment (an ear infection and an upper respiratory infection). The testing of this subject was

interrupted when these illnesses became known but the actual onset of each was indeterminate. Therefore, the early, undetected stages and lingering effects of the illnesses may indeed have influenced the data of R. C. Accordingly, the analyses and presentation of the data are also presented for the combined subjects, excluding R. C. These are given in figure 8 and tables 7 and 15. The fourth intensity level (90.5 db) is the highest CFF (1.34 cps over the ambient level, the lowest) in these remaining four subjects.

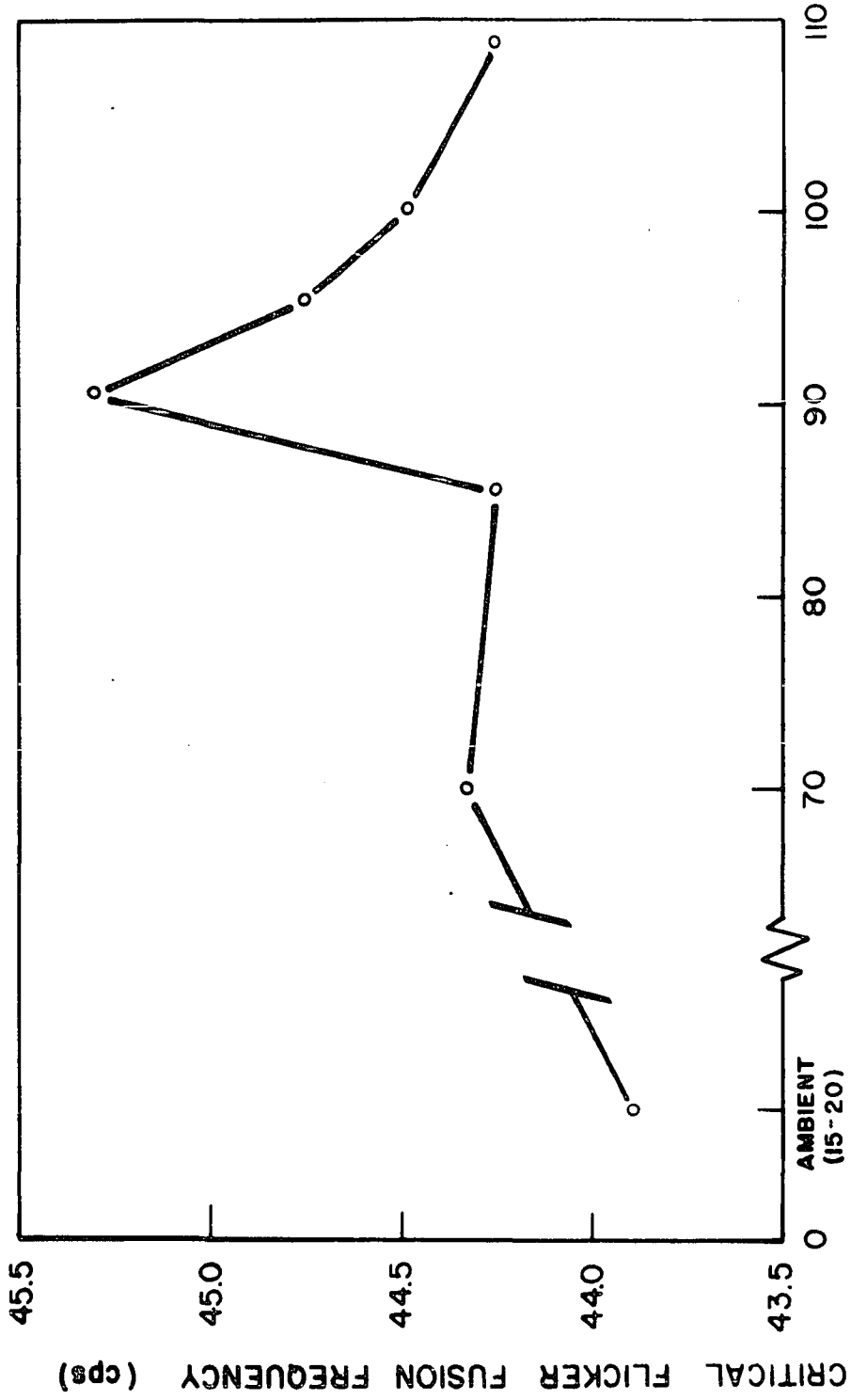
A Duncan's multiple range analysis was performed on the data (Duncan, 1955) in order to test significance of differences among the means (CFF) at each intensity level. The results of this test are presented in table 18 for each of the subjects individually and for the combined data of all subjects. In addition, the data for all subjects combined except R. C. is presented.

A most interesting result of this test can be seen from the averaged data. The CFF at the fourth intensity level (90.5 db) is significantly higher than that at all other intensity levels both more and less intense. The prediction of a significantly curvilinear effect is thus seen to be verified.

TABLE 1
 CFF at Seven Levels of Acoustic Intensity
 Subject - H. Y.

Level of intensity (db)	CFF	Diff. from ambient
Ambient (15-20 db)	43.88	--
70.0 (800 cps)	44.33	+ .45
85.5 (800 cps)	44.26	+ .38
90.5 (800 cps)	45.31	+1.43
95.4 (800 cps)	44.74	+ .86
100.0 (800 cps)	44.48	+ .60
109.0 (800 cps)	44.26	+ .38

Fig. 2. The effect of seven levels of acoustic intensity on the CFF of subject H. Y.

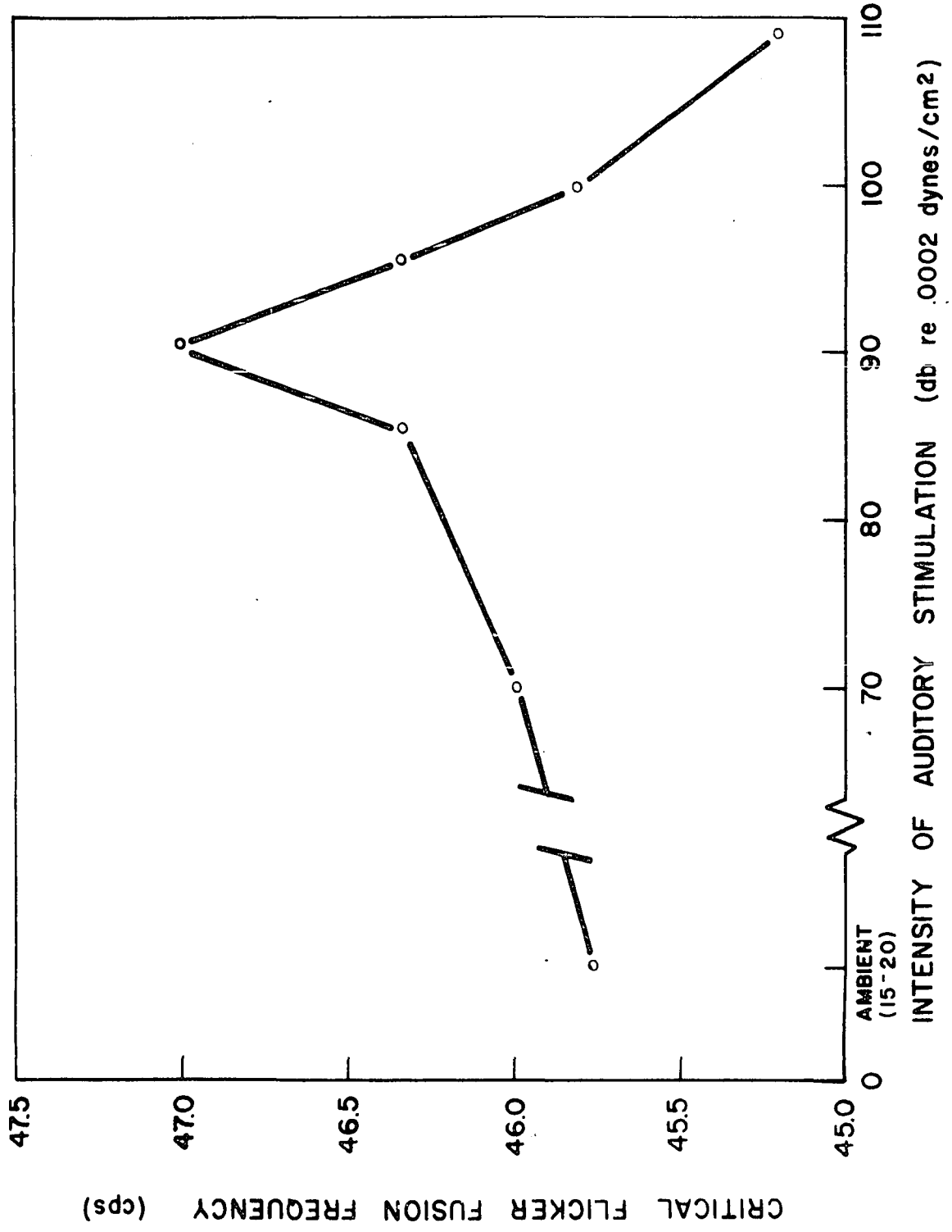


INTENSITY OF AUDITORY STIMULATION (db re .0002 dynes/cm²)

TABLE 2
CFF at Seven Levels of Acoustic Intensity
Subject - P. C.

Level of intensity (db)	CFF	Diff. from ambient
Ambient (15-20 db)	45.76	--
70.0 (800 cps)	45.98	+ .22
85.5 (800 cps)	46.33	+ .57
90.5 (800 cps)	47.05	+1.29
95.4 (800 cps)	46.33	+ .57
100.0 (800 cps)	45.81	+ .05
109.0 (800 cps)	45.21	- .55

Fig. 3. The effect of seven levels of acoustic intensity on the CFF of subject P. C.



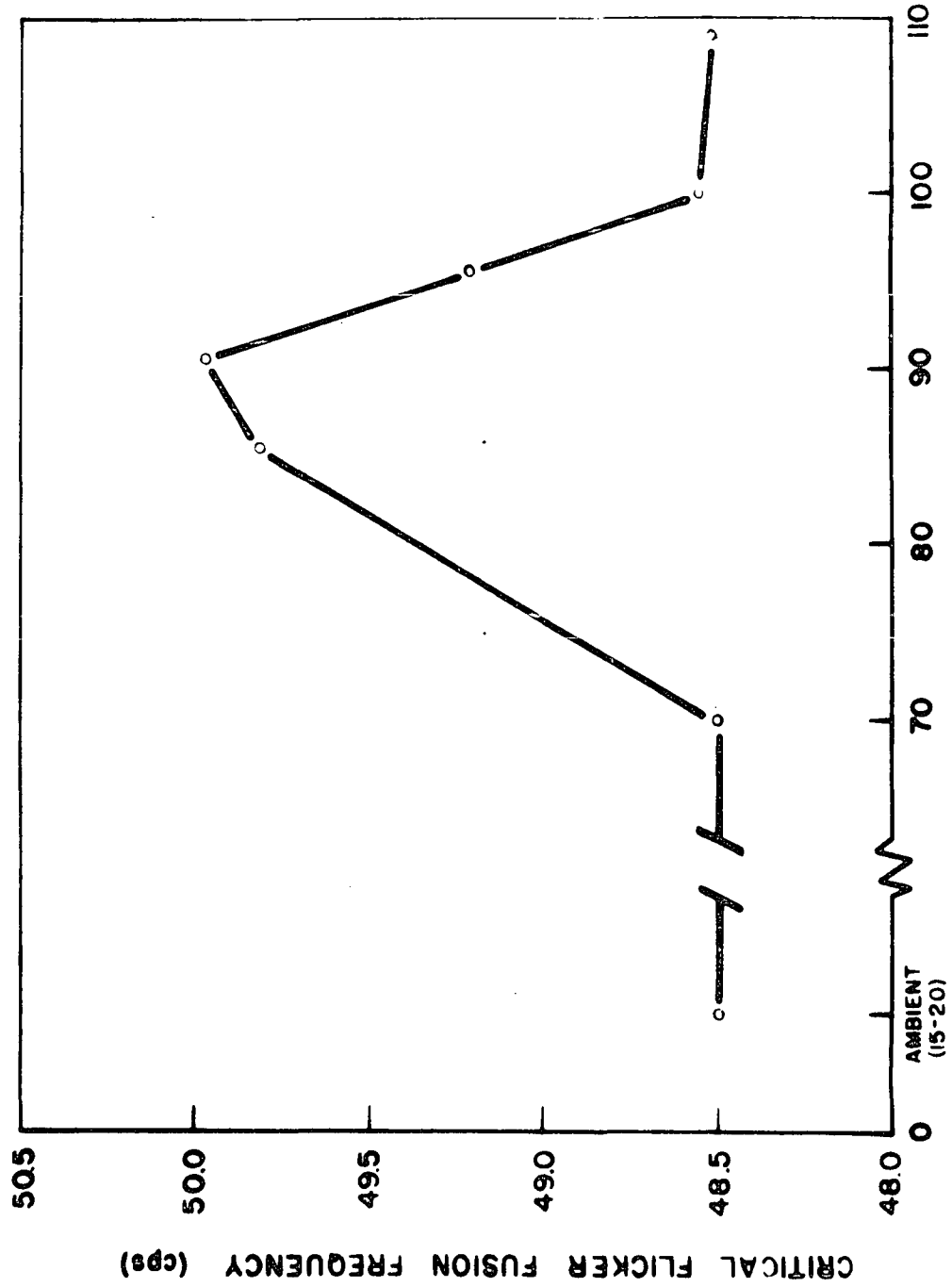
CRITICAL FLICKER FUSION FREQUENCY (cps)

INTENSITY OF AUDITORY STIMULATION (db re .0002 dynes/cm²)

TABLE 3
CFF at Seven Levels of Acoustic Intensity
Subject - P. F.

Level of intensity (db)	CFF	Diff. from ambient
Ambient (15-20 db)	48.50	--
70.0 (800 cps)	48.50	0.0
85.5 (800 cps)	49.81	+1.31
90.5 (800 cps)	49.98	+1.48
95.4 (800 cps)	49.21	+ .71
100.0 (800 cps)	48.55	+ .05
109.0 (800 cps)	48.52	+ .02

Fig. 4. The effect of seven levels of acoustic intensity on the CFF of subject P. F.



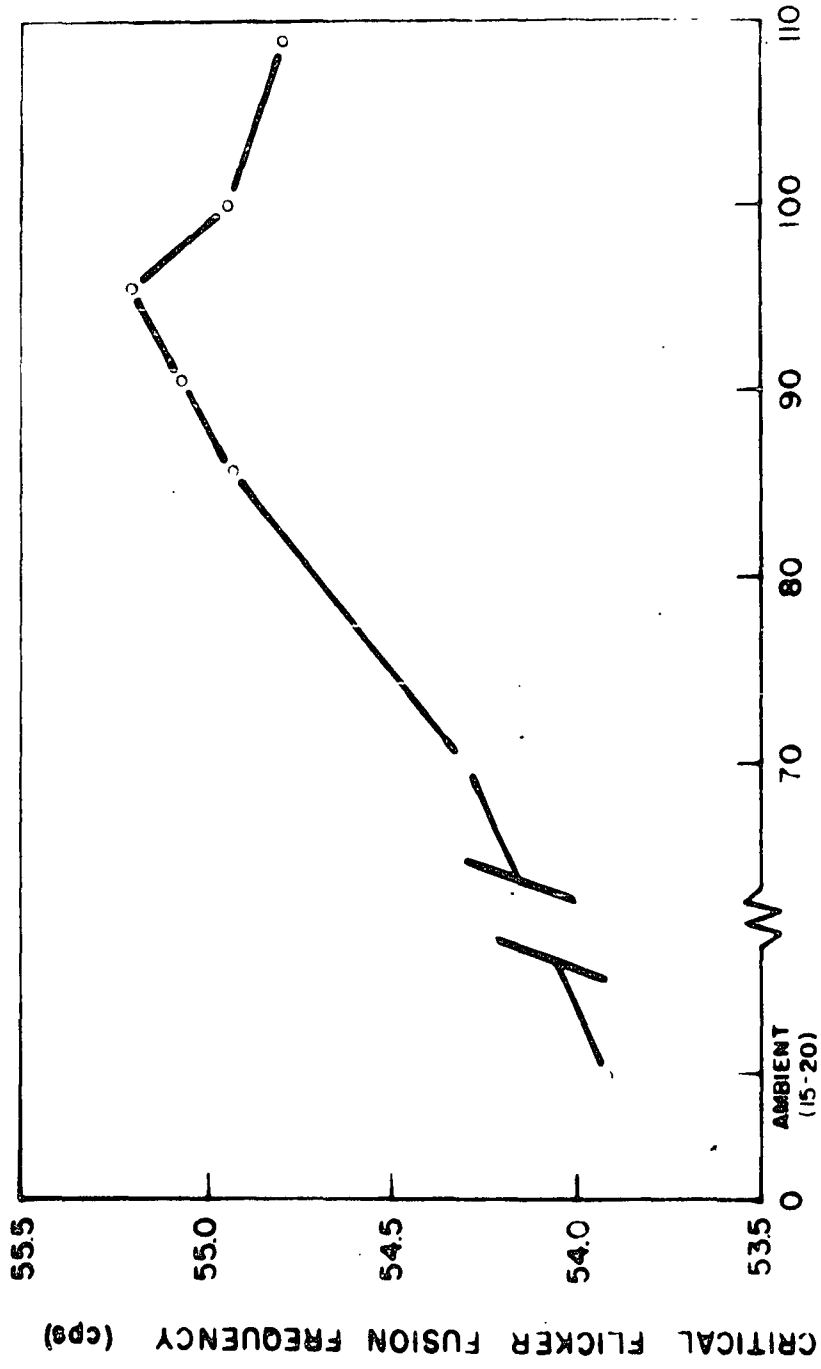
INTENSITY OF AUDITORY STIMULATION (db re .0002 dynes/cm²)

CRITICAL FLICKER FUSION FREQUENCY (cps)

TABLE 4
CFF at Seven Levels of Acoustic Intensity
Subject - H. H.

Intensity (db re .0002 dynes/cm ²)	CFF	Diff. from ambient
Ambient (15-20 db)	53.92	--
70.0 (800 cps)	54.31	+ .39
85.5 (800 cps)	54.93	+1.01
90.5 (800 cps)	55.07	+1.15
95.4 (800 cps)	55.21	+1.29
100.0 (800 cps)	54.95	+1.03
109.0 (800 cps)	54.81	+ .89

Fig. 5. The effect of seven levels of acoustic intensity on the CFF of subject H. H.



INTENSITY OF AUDITORY STIMULATION (db re .0002 dynes/cm²)

CRITICAL FLICKER FUSION FREQUENCY (cfs)

TABLE 5
CFF at Seven Levels of Acoustic Intensity
Subject - R. C.

Intensity (db re .0002 dynes/cm ²)	CFF	Diff. from ambient
Ambient (15-20 db)	42.93	--
70.0 (800 cps)	43.45	+ .52
85.5 (800 cps)	43.00	+ .07
90.5 (800 cps)	43.26	+ .33
95.4 (800 cps)	42.81	- .08
100.0 (800 cps)	43.21	+ .28
109.0 (800 cps)	42.60	- .33

Fig. 6. The effect of seven levels of acoustic intensity on the CFF of subject R. C.

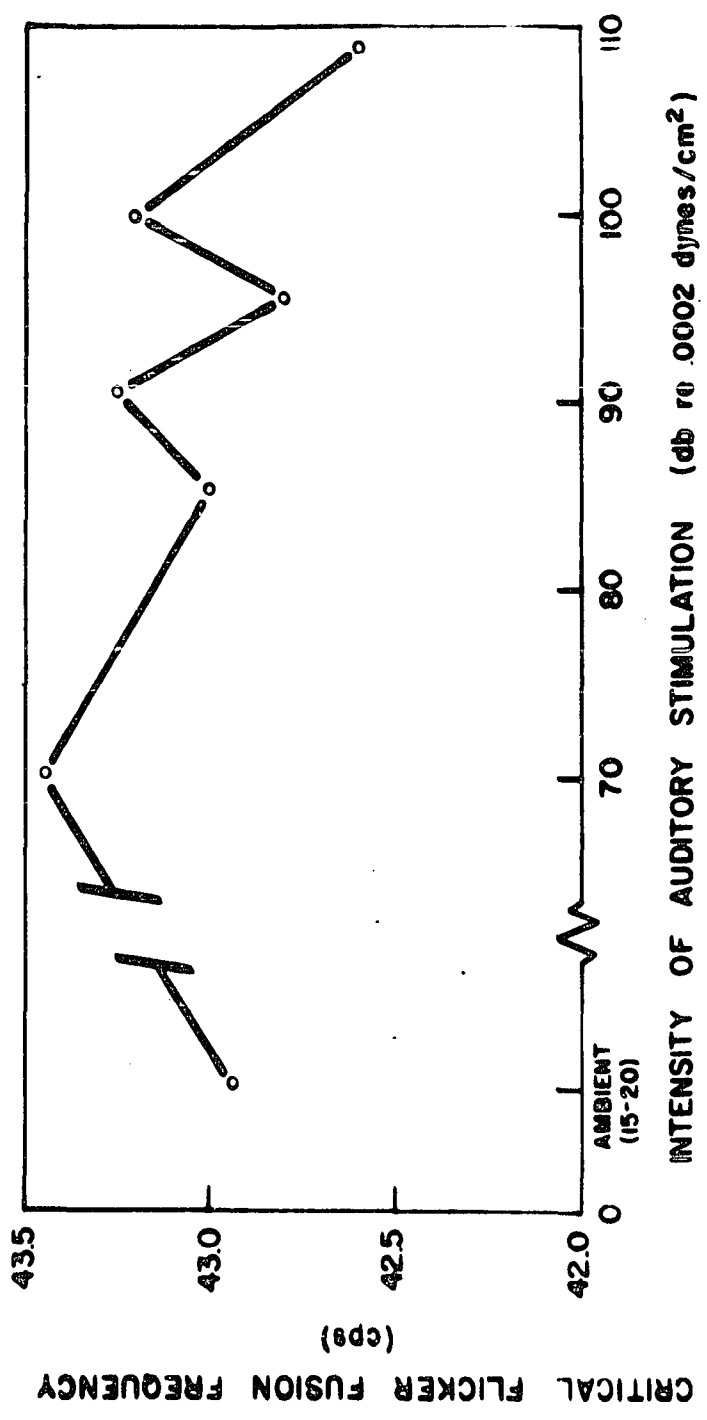


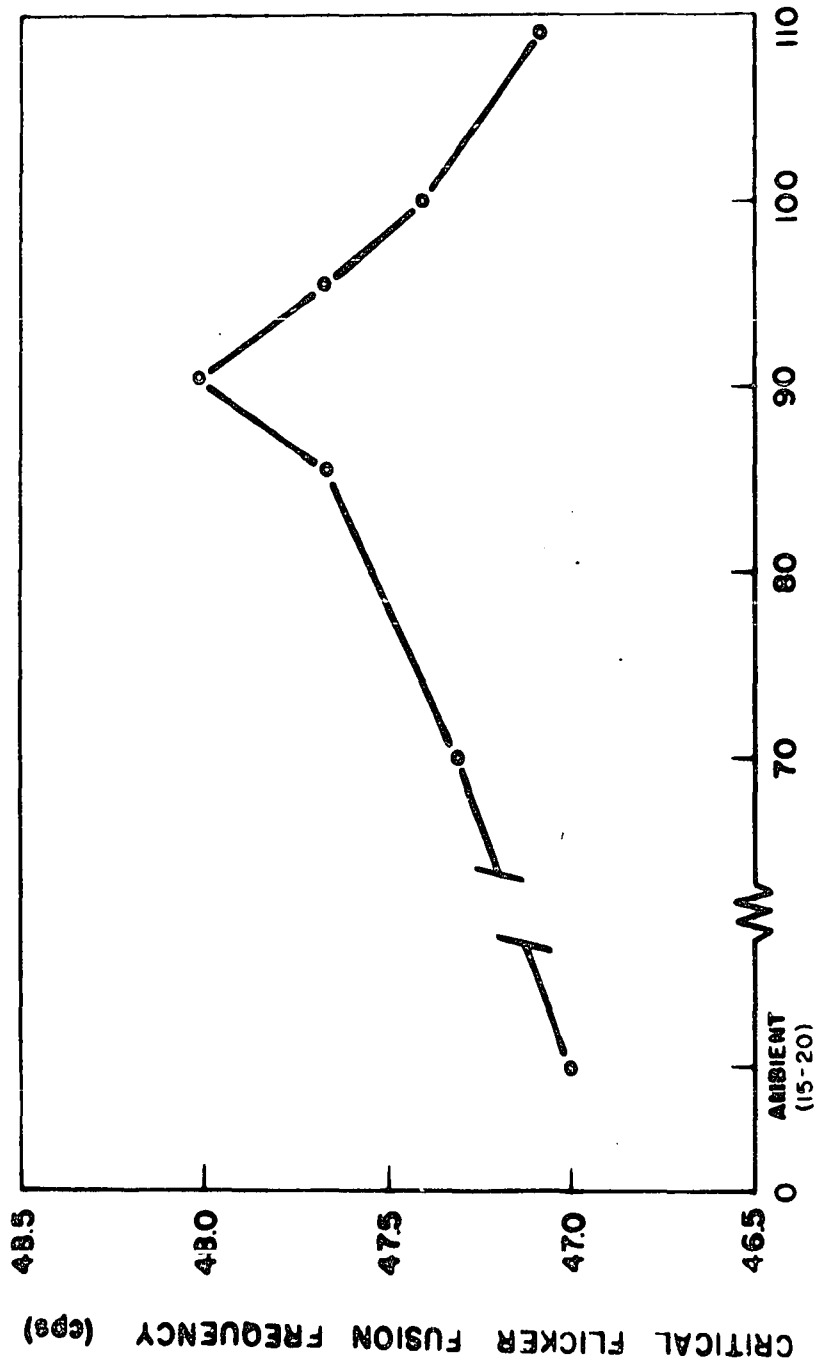
TABLE 6

CFF at Seven Levels of Acoustic Intensity

Average Over All Five Subjects

Intensity (db re .0002 dynes/cm ²)	CFF	Diff. from ambient
Ambient (15-20 db)	47.00	--
70.0 (800 cps)	47.31	+ .31
85.5 (800 cps)	47.66	+ .66
90.5 (800 cps)	48.13	+1.13
95.4 (800 cps)	47.66	+ .66
100.0 (800 cps)	47.40	+ .40
109.0 (800 cps)	47.08	+ .08

Fig. 7. The effect of seven levels of acoustic intensity on the CFF averaged over all five subjects.



INTENSITY OF AUDITORY STIMULATION (db re .0002 dynes/cm²)

TABLE 7
CFF at Seven Levels of Acoustic Intensity
Average Four Subjects

Level of intensity (db)	CFF	Diff. from ambient
Ambient (15-20 db)	48.01	--
70.0 (800 cps)	48.27	+ .26
85.5 (800 cps)	48.83	+ .82
90.5 (800 cps)	49.35	+1.34
95.4 (800 cps)	48.87	+ .86
100.0 (800 cps)	48.44	+ .43
109.0 (800 cps)	48.20	+ .19

Fig. 8. The effect of seven levels of acoustic intensity on the CFF averaged over all subjects except R. C.

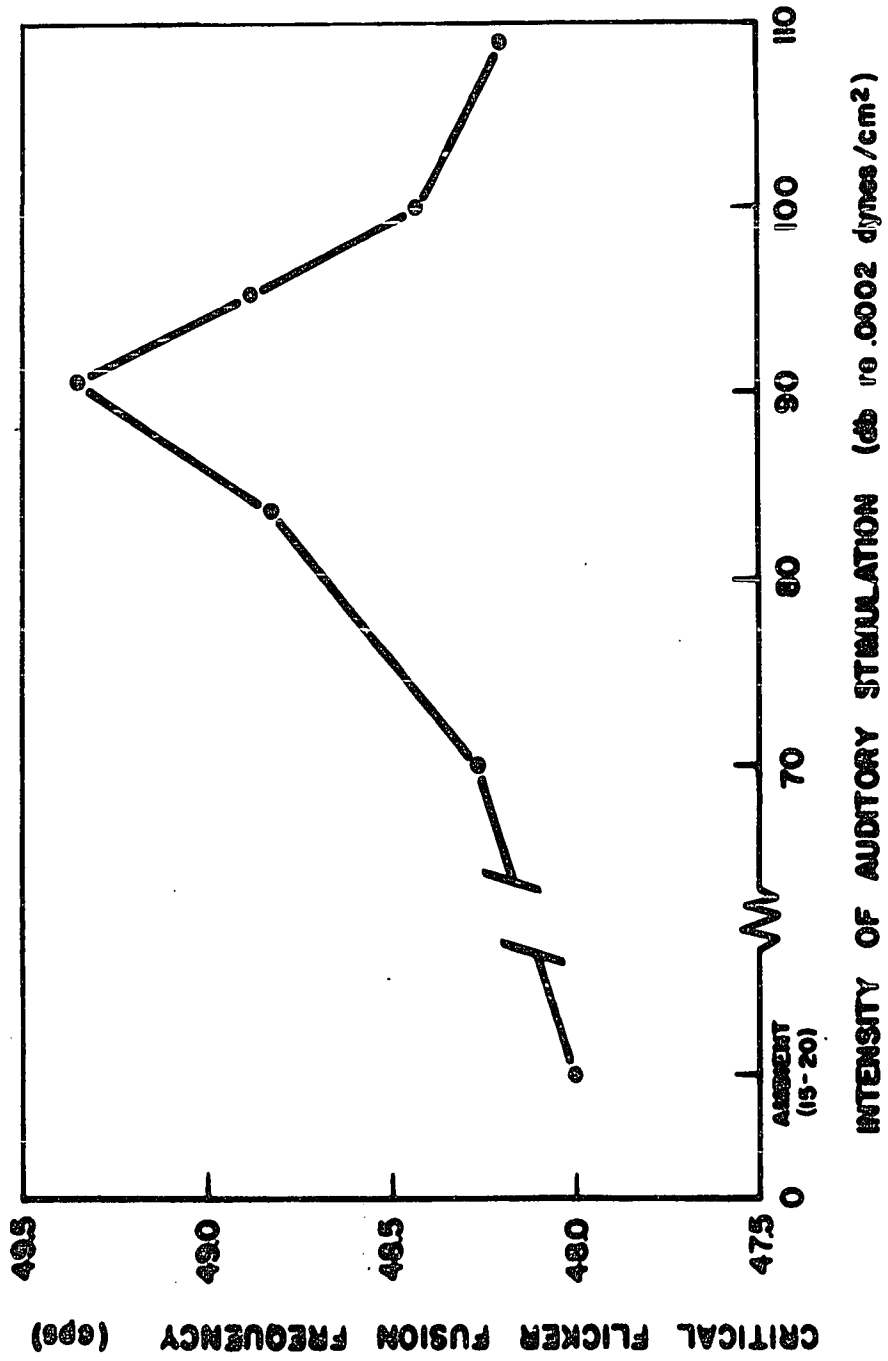


TABLE 8
Individual Analysis of Variance
Subject - H. Y.

Source of variance	df	Mean square	F	P
Total	146	--	--	--
Treatment	6	17.3	5.39	<.001
Position	6	53.8	16.7	<.001
Days	6	52.7	16.4	<.007
Within (error)	128	3.22	--	--

Note.--The analysis of variance was computed (for convenience) without averaging the ascending and descending trials for each threshold determination (see Chapter II) but simply using their sum as a single score. This is true for all analysis of variance tables.

TABLE 9
Individual Analysis of Variance
Subject - P. C.

Source of variance	df	Mean square	F	P
Total	146	--	--	--
Treatment	6	27.83	9.55	<.001
Position	6	20.67	7.09	<.001
Days	6	43.17	14.81	<.001
Within (error)	128	2.91	--	--

TABLE 10
Individual Analysis of Variance
Subject - P. F.

Source of variance	df	Mean square	F	P
Total	146	--	--	--
Treatment	6	36.17	4.27	<.001
Position	6	65.17	7.69	<.001
Days	6	26.38	31.12	<.001
Within (error)	128	8.48	--	--

TABLE 11
Individual Analysis of Variance
Subject - H. H.

Source of variance	df	Mean square	F	P
Total	146	--	--	--
Treatment	6	17.5	5.01	<.001
Position	6	9.16	2.62	<.025
Days	6	10.3	2.95	<.01
Within (error)	128	3.49	--	--

TABLE 12
Individual Analysis of Variance
Subject - R. C.

Source of variance	df	Mean square	F	P
Total	146	--	--	--
Treatment	6	7.17	.865	--
Position	6	10.5	1.27	--
Days	6	26.5	3.19	<.01
Within (error)	128	8.29	--	--

TABLE 13
 Analysis of Variance Summary
 All Subjects

Latin square	Sums of squares				df			
	Subject	Treatment	Position	Day	Within	Treatment	Position	Day
H. Y.	104	323	316	412	6	6	6	128
P. C.	167	124	259	373	6	6	6	128
P. F.	217	391	1583	1085	6	6	6	128
H. H.	105	55	62	497	6	6	6	128
R. C.	43	63	159	1061	6	6	6	128
Σ	636	956		3378	30	30	30	640

TABLE 14
 Combined Analysis of Variance
 All Subjects (Five)

Source of variance	df	M. S.	F	P
Subjects	4	12653.	1209.68	<.001
Treatments	6	64.17	6.135	<.001
Position	6	74.0	3.47	<.02
Day	6	169.17	2.98	<.03
Subjects x Treatments	24	10.17	1.98	<.005
Subjects x Position	24	21.33	4.04	<.01
Subjects x Day	24	56.83	10.77	<.001
Within (error)	640	5.273	--	--

Note.--The denominators for the F ratio of the main effects given here and in table 15 are the interaction between the Latin square (subject) and that particular main effect. For explanation see text (Chapter III).

TABLE 15
 Combined Analysis of Variance
 Four Subjects

Source of variance	df	M. S.	F	P
Subjects	3	12078.	2610.3	<.001
Treatments	6	73.5	8.71	<.001
Position	6	98.5	5.87	.005
Day	6	213.67	46.18	<.001
Subjects x Treatment	18	8.14	1.82	.05
Subjects x Position	18	16.78	3.63	<.001
Subjects x Day	18	52.11	11.26	<.001
Within (error)	512	4.627	--	--

TABLE 16

F Ratios and Probabilities of All Main Effects (Five Subjects Combined)

Calculated Using Within Cell Error Variance as the Denominator

Source of variance	F	P
Treatment	12.16	<.001
Positions	14.02	<.001
Days	32.05	<.001
Subjects	2397.4	<.001

TABLE 17

F Ratios and Probabilities of All Main Effects (Four Subjects Combined)

Calculated Using Within Cell Error Variance as the Denominator

Source of variance	F	P
Treatment	15.89	<.001
Positions	21.29	<.001
Days	46.18	<.001
Subjects	2610.3	<.001

TABLE 18

Duncan's Multiple Range Test Significance of Differences of CFF Means at Each Intensity Level

	Subject							
Intensity CFF	H. Y.	Amb. 43.88	109.0 <u>44.26</u>	85.5 <u>44.26</u>	70.0 <u>44.33</u>	100.0 <u>44.48</u>	95.4 <u>44.74</u>	90.5 <u>45.31</u>
Intensity CFF	P. C.	109.0 45.21	Amb. <u>45.76</u>	100.0 <u>45.81</u>	70.0 <u>45.98</u>	85.5 <u>46.33</u>	95.4 <u>46.33</u>	90.5 47.05
Intensity CFF	P. F.	Amb. 48.50	70.0 <u>48.50</u>	109.0 <u>48.52</u>	100.0 <u>48.55</u>	95.4 <u>49.21</u>	85.5 <u>49.81</u>	90.5 <u>49.98</u>
Intensity CFF	H. H.	Amb. 53.92	70.0 <u>54.31</u>	109.0 <u>54.81</u>	85.5 <u>54.93</u>	100.0 <u>54.95</u>	90.5 <u>55.07</u>	95.4 <u>55.21</u>
Intensity CFF	R. C.	109.0 42.60	95.4 <u>42.81</u>	Amb. <u>42.93</u>	85.5 <u>43.00</u>	100.0 <u>43.21</u>	90.5 <u>43.26</u>	85.5 <u>43.45</u>
Intensity CFF	Mean 5 S's	Amb. 47.00	109.0 <u>47.08</u>	70.0 <u>47.31</u>	100.0 <u>47.40</u>	85.5 <u>47.66</u>	95.4 <u>47.66</u>	90.5 <u>48.13</u>
Intensity CFF	Mean 4 S's	Amb. 48.01	109.0 <u>48.20</u>	70.0 <u>48.27</u>	100.0 <u>48.44</u>	85.5 <u>48.83</u>	95.4 <u>48.87</u>	90.5 <u>49.35</u>

Note.--All means underlined with the same line are not significantly different from each other. All means not underlined by the same line are significantly different from each other.

CHAPTER IV

DISCUSSION

The Results and the Predictions. It can be seen from tables 8 through 12 that auditory intensity in this experiment was shown to exert a significant effect on CFF in four out of five subjects individually. This treatment effect is seen from table 18 to be curvilinear. The rise and then fall of the CFF as a function of auditory intensity is statistically significant as demonstrated by the fact that the CFF at the fourth intensity level (90.5 db) is significantly higher than at any other intensity level. This is true in three of the four individual subjects on whom a significant effect was demonstrated and over the average of all subjects. The hypothesis of a curvilinear effect of auditory intensity is thus considered to be upheld from these data.

No significant treatment effect was demonstrated on subject R. C. As stated above, the testing of this subject was interrupted by an upper respiratory infection and an ear infection on separate occasions. It is felt that either of these variables could have influenced the data and, therefore, that no conclusion can be drawn concerning the effect of auditory intensity on CFF in this subject.

It is interesting to note that in the combined subjects (four and five) the CFF even at the highest intensity level (109 db) was not demonstrated to be lower than that recorded at the lowest intensity level (ambient). In fact, although the difference is not significant at the 5% level, the CFF at the ambient level was actually lower than at any other, including the highest intensity. As seen from table 18, the CFF

at the ambient level was significantly lower than all levels except the highest (109 db) and the lowest 800 cycle tone (70.0 db). As mentioned above, the CFF was lower than these two levels also, but not significantly.

The Results and the Model. What implications do these findings have for the model presented in Chapter I? It was noted that reticular stimulation increased response amplitude (Granit, 1955) and discharge rate (of following flashes) (Jung, 1958, Lindsley, personal communication). This was assumed to be due to reticular activity's desynchronizing groups of "on" and "off" neurons which tended to inhibit each other when synchronized, thus disinhibiting them. This was shown above to lead to the prediction of an increasing CFF as a result of increasing reticular activity (which activity would result from an increase in auditory stimulation). It is seen from the above data (figures 2 through 8 and tables 1 through 14) that this prediction is significantly varied.

It may be recalled that the effects of further reticular activity were expected to lower CFF from the maximum value obtained by the stimulation. From this it was predicted that increased auditory stimulation would exert a diphasic effect on CFF first raising and then lowering it. Two possible mechanisms were suggested that might account for this. One was a result of an additional assumption's extending the desynchronizing model. The other included an additional postulate concerning possible reticular blocking from excess stimulation.

The first of these two, it may be recalled, suggested that the advantage of disinhibition gained by having groups of "on" and groups of "off" fibers synchronized within themselves, but desynchronized with each other would be lost when the synchrony within a group was lost (see

Chapter I). This would predict that as this desynchrony progresses to include within groups, we would have a progressive drop in CFF with additional stimulation; but it does not predict that CFF would ever drop below that obtained under conditions of low reticular activity (synchrony of local potentials of all neurons, that is, alpha rhythm).

The second of these possible mechanisms (that of reticular blocking) does predict that excessive bombardment of the reticular formation results in a blocking of reticular activity below the point necessary for normal activation (Lindsley, 1960). Therefore, this mechanism strongly suggests that excessive stimulation of the reticular formation (in this case with audition, if the audition is intense enough) would result in a CFF lower than that found under normally relaxed conditions. As remarked above, with the intensities tested in this experiment, this was not demonstrated to be the case. The present data alone, therefore, do not seem to require that the assumption (reticular blocking) of the second alternative above be invoked.

This is not to say, however, that a further decrease in CFF to below that found in the ambient situation would not be obtained with higher intensities. The fact that several investigators did report this lowered CFF with high auditory stimulation levels (Levine, 1958; Gorrell, 1953; Allen and Schwartz, 1940) indicates this possibility. In fact, at times in the above studies, this decreased CFF was obtained with intensities reported to be lower than those used here. This conflict will be discussed below.

The present data, then, cannot alone definitively decide which or what combination of the two hypotheses (desynchronization progressing

within groups and reticular blocking) provides a better model. As it was mentioned in Chapter I, the two hypotheses are not necessarily antithetical. It is entirely possible to conceive of progressive desynchronization's accounting for the initial fall in CFF scores and still predict that even greater stimulation would result in a further decrease due to some reticular blocking mechanism. It may also be that no amount of auditory stimulation is capable of raising the activity in the reticular formation sufficiently to reach the postulated "blocking" point.

Conflict with Previous Investigations. The conflict between the present results (which do not show a lower CFF for the condition of most intense stimulation than for the condition of low intensity (15-20 db ambient noise) and the studies of Allen and Schwartz (1940), Gorrell (1953), and Levine (1958) may be due to a number of factors, all of which were unreported in the available publications of each of the above studies but were controlled in the present one. The possibility that stimulants or depressants taken by the subjects (such as coffee or alcohol) may have affected the scores (Bjerver and Goldberg, 1950; Cranston, Zubin, and Landis, 1952) was not ruled out. In addition, a difference in the room temperature (or a fluctuation in the temperature) may conceivably have contributed to any conflict in results. Possibly more important, however, was the measurement of the stimulus and the control of ambient noise. Here, and in other studies, the acoustic intensity was measured and the possibility exists that the phenomenological loudness curves differed among the subjects. In addition, the measurements of the intensity were made differently from the previous studies.

The stimulus levels reported in this present study were measured with the aid of a mechanical ear to simulate best the actual condition of stimulation when the subject was wearing the padded earphones (see Chapter II). In the above studies, the stimulus levels were either reported to be taken at the earphone apparently without benefit of this device (Levine, 1958), unreported altogether (Gorrell, 1953), or were merely implied in a qualitative description of the sound producing apparatus (Allen and Schwartz, 1940).

In addition, the use of earphone padding in the present experiment (calibrated for its attenuation of white noise) lowered the stimulus level at the ambient noise condition well below any levels which were reported in the above studies. An additional possible explanation of difference lies in the use of different frequencies in this study from those used in the above studies. It seems quite reasonable to conclude that any of these differences (but primarily the measurement of the stimulus and the ambient level) could account for the observed discrepancy in the results of this study and those of Allen and Schwartz (1940); Gorrell (1953); and Levine (1958). As mentioned above, it is entirely possible that still higher levels of intensity than the highest one used here (109 db) might have lowered the CFF to below that found at the ambient level.

Differences Among Subjects and Possible Practical Applications.

It is interesting to note that the interaction between subjects and treatments (auditory intensity) is sizable and significant ($F = 10.77$ over the within cell variance) when the data of all five subjects are included. It is much smaller ($F = 1.82$) when the questionable data of subject R. C.

(see above) are omitted. This 1.82 is barely significant at the .05 level with 18 and 512 degrees of freedom. It is small enough, however, that the F ratio of the four subject treatment effect over this interaction is 8.71 (significant beyond the .001 level). This indicates that although it seems likely ($P = .95$) that the treatment affected the subjects differently, this difference is exceedingly small.

In any case, the plotted individual curves (figures 2 through 6) seem striking in their similarity. Since there is also a significant treatment effect in the combined Latin squares, even over the interaction, it may indicate that the effect is similar enough (at least in populations with the degree of homogeneity of this one) among individuals to be of practical significance in such situations as industrial work settings. That is, the possibility of finding levels of acoustic intensity that increase the efficiency of human beings with enough generality to be of immediate practical use is suggested (the amount of change in CFF in the four subjects presented in table 7 is 2.79% with the intensities used). The change is relatively small, however the range of stimuli used was quite restricted and it seems reasonable to expect a possibly greater change may be found with other stimuli. Further work along the lines of investigating what other responses are affected similarly to CFF seems desirable. Investigations of how far and how heterogeneously the results of this study can be generalized are also indicated.

CHAPTER V

SUMMARY

Since we are constantly bombarded by stimuli which simultaneously excite more than one sense, knowledge of the nature of intersensory effects is important. The present study investigates the effects of varying the intensity of auditory stimulation on a visual measure --critical flicker fusion frequency.

A theoretical physiological model is described and supported which incorporates the known physiological relationships of the anatomical structures involved and predicts a curvilinear effect of acoustic intensity on CFF. This model involves the effect of auditory stimulation on the reticular formation, the effect of reticular activity on cortical responses, and the relationship of cortical responses to CFF. It postulates that auditory impulses stimulate reticular activity and that this reticular activity in turn desynchronizes groups of "on" and groups of "off" fibers (from each other) in the visual cortex. The model assumes that this desynchrony is related to the change (faster and shallower) in alpha rhythms which occurs with reticular stimulation. The additional postulate is made that this cortical desynchrony first leads to a disinhibition between groups of "on" and "off" neurons which increases CFF and then progresses with increasing reticular stimulation to desynchrony within these groups of cells which again lowers CFF.

Past studies of auditory influences on CFF often conflict. Frequently the measurements of the stimuli are incomplete. Nevertheless, the studies seem to indicate some support for the contention that high intensity tones lower CFF and low intensity tones raise it. The present

experiment utilizes a Latin square experimental design to investigate the effects of seven levels of acoustic intensity on the foveal flicker fusion threshold of a white intermittent visual stimulus of one to six light dark ratio and a luminance at the eye of 1.266 log ml. The lowest level of acoustic intensity consisted of approximately 15-20 db of ambient noise. The other six ranged from 70 to 109 db of an 800 cycle tone.

The CFF of four of the five subjects tested was significantly (beyond the .001 level by analysis of variance) affected by the variations in acoustic intensity. This effect was similarly significant over all subjects and was significantly curvilinear with the predicted initial rise and then fall in CFF. The highest CFF occurred at 90.5 db and the lowest was recorded at the ambient noise level (15-20 db). Only one of the four subjects on whom the effect was significant displayed a lower CFF at 109 db than at the ambient level. The fifth subject, on whom no significant effect was demonstrated, was interrupted twice during the course of the experiment due to illness and it was felt that no conclusion concerning the effect of auditory stimulation on the CFF of this subject could be drawn. The rise in CFF over the ambient level found at 90.5 db was 1.34 cps over all subjects excluding the one who became ill.

The curves (CFF over acoustic intensity) of the remaining four subjects were sufficiently similar (interaction of subject and treatment equalled 1.82 times the pooled within cell error variance) to suggest the possibility of a practical generalized application for this inter-sensory effect.

GLOSSARY

- Activation: General activity level of the organism; now considered by some (e.g., Lindsley, 1956) to be a function of electrical activity in the ascending reticular formation.
- CFF: Critical flicker fusion frequency; that frequency at which a flickering visual stimulus appears to be the observer to be a fused steady source.
- Lambert: A unit of luminance (photometric brightness) equal to the brightness of a perfectly diffusing surface, normal to and one centimeter away from a one candlepower source.
- Latin square: An experimental design wherein each level of each variable is presented once with each level of every other variable. In this experiment acoustical intensity appears for each subject once in every testing position and once on each testing day, a different position on each day.
- Lux: A unit of illumination equal to one lumen per square meter; $9.29 \cdot 10^{-2}$ foot candles.
- Modality: A specific sense, such as hearing or vision.
- "Off" Fibers: Fibers in the retina, optic tract and visual cortex which respond with a burst of impulses to a sudden lowering in the brightness of a visual stimulus. This high output rate tapers off after the initial outburst.
- "On" Fibers: Fibers in the retina, optic tract, and visual cortex which respond with a burst of impulses to a sudden rise in the brightness of a visual stimulus. Some of these fibers maintain a considerably lower level of output during the duration of the visual stimulus.

BIBLIOGRAPHY

Ades, H. W., Central auditory mechanisms in, Field, J., Magoun, H. W., and Hall, V. E. (ed) Hdbk of Physiol. - sec. 1 Neurophys. Vol. I Amer. Physiol. Soc, Washington D. C., 1959, 591.

Allen, F. and Schwartz, M., The effect of stimulation of the senses of vision, hearing, taste, and smell on the sensibility of the organs of vision. J. gen. Physiol., 1940, 24, 105-121.

Baker, C. H., The dependence of binocular fusion on timing of peripheral stimuli and on central processes. I. Symmetrical flicker. Canad. J. Psychol., 1952, 6, 1-10. (Abstracted in Landis, 1953.)

Barren, D. H., The interrelation of potential changes in the spinal cord., J. Physiol., 1938, 92, 276.

Bartley, S. H., The relation between cortical response to visual stimulation and changes in the alpha rhythm. J. exp. Psychol., 1940, 27, 624-639.

Beck, H., Anstieg der verschmelzungsfrequenz bei erregung im electroretinogramm, Klin. Wschr, 1951, 29, 446-448. (Abstracted in Landis, 1953.)

Bishop, G. H., Cyclic changes in the excitability of the optic pathway of the rabbit., Amer. J. Physiol., 1933, 103, 213.

Bjerver, K., and Goldberg, L., Effect of alcohol ingestion on driving ability, Quart. J. Stud Alcohol, 1950, 11, 1-30.

Bogoslavski, A. I., Changements de la frequence critique des papillotements lumineux a caractere de reflex conditionne. Arch. Opthol. Paris, 1938, 2, 219-227. (Abstracted in Landis, 1953.)

Buhler, A. B., Flicker fusion threshold and anxiety level, Columbia Univ. (Dissert. abst., 1954, 14, #1255.)

Busch, Gerhard, Der Einfluss eines bewissten sichkonzentrurens und sichentspannem auf die flimmerverschmelzungsfrequenz. Psychol. Beitr., 1953, 1, 312-323 (Psychol abst., 1955, 29, #8255).

Cranston, R. E., Zubin, J., and Landis, C., The effect of small doses of thonzylamine, dexedrine, and phenobarbitol on test performance and self-rating of subjective states, J. Psychol., 1952, 33, 209-215.

Davson, H., The Physiology of Vision, Philadelphia, the Blakiston Co., 1949.

- Dobrokova, O. A., The influence of taste, temperature and sound stimuli on the critical flicker frequency of monochromatic light. Probl. physiol. Opt. Acad. Sci. U. S. S. R., 2, 81-84 (Psychol abs., 1946, 20, #671.)
- Edwards, A. L., Experimental Design in Psychological Research, Rev. Ed. N. Y., Rinehart & Co., Inc., 1960.
- Fry, G. H. and Bartley, S. H., The effect of steady stimulation of one part of the retina upon the critical frequency in another. J. exp. Psychol., 1936, 19, 351-356.
- Goldstone, Flicker fusion measurements and anxiety level, J. exper. Psychol., 1955, 49, 200-202.
- Duncan, D. E., Multiple range and multiple F tests. Biometrics, 1955, 11, 1-42.
- Gorrell, Raymond B., The effect of extraneous auditory stimulation on critical flicker frequency. Unpublished doctoral dissertation, Clark University, 1953.
- Granit, R., Comparative studies on the peripheral and central retina, I. On interaction between distant areas in the human eye. Amer. J. Physiol., 1930, 24, 41-50.
- Granit, R., and Harper, P., Interactions between distant areas in the human eye. J. Physiol., 1930, 69, xvii.
- Hartline, H. K., Impulses in single optic nerve fibers of the vertebrate retina. Amer. J. Physiol., 1935, 113, 59-60.
- Hartline, H. K., The response of single optic nerve fibers of the vertebrate eye to illumination of the retina, Amer. J. Physiol., 1938, 121, 400-415.
- Hecht, S. and Wolf E., Intermittent stimulation by light. I. The validity of Talbot's law for Mya. J. gen. Physiol., 1932, 15, 369-389.
- Hecht, S., Schlaer, S., and Verrijp, C. D., Intermittent stimulation by light II. The measurement of critical fusion frequency for the human eye, J. gen. Physiol., 1933, 17, 237-249.
- Hecht, S. and Verrijp, C. D., Intermittent stimulation by light III. The relation between intensity and critical fusion frequency for different retinal locations, J. gen. Physiol., 1933, 17, 251-265.
- Hecht, S. and Verrijp, C. D., Intermittent stimulation by light IV. A theoretical interpretation of the quantitative data of flicker. J. gen. Physiol., 1933, 17, 266-282.

- Hecht, S. and Shlaer, S., Intermittent stimulation by light V. The relation between intensity and critical frequency for different parts of the spectrum., J. gen Physiol., 1936, 19, 965-979.
- Hecht, S. and Smith, E. L., Intermittent stimulation by light. VI. Area and the relation between critical frequency and intensity, J. gen Physiol., 1936, 19, 979-991.
- Hilgard, E. R., Introduction to Psychology 3rd ed., N. Y., Harcourt, Brace & World Inc., 1962, 178-183.
- Hoagland, H., Pacemakers of human brain waves in normals and in general paretics, Amer. J. Physiol., 1936, 116, 604-615.
- Ireland, F. H., A comparison of critical flicker frequency under conditions of monocular and binocular stimulation, J. exper. Psychol., 1950, 40, 282-286.
- Jasper, H. H., Charting the sea of brain waves. Scienc, 1948, 108, 343-347.
- Jung, R., Excitation, inhibition, and coordination of cortical neurones, Experimental Cell Research, Suppl, 1958, 5, 262-271.
- Jung, Richard, Microphysiology of the cortical neurons and its significance for psychophysiology, manuscript presented at M. I. T. under Walter Rosenbluth as reported by Lindsley, D. B., at U. of Hawaii, 1961.
- Kennedy, J. L., A possible artifact in electroencephalography. Psychol. Rev., 1959, 66, 347-352.
- Knox, G. W., Investigations of flicker and fusion. I. The effect of practice under the influence of various attitudes on the CFF. J. gen Psychol., 1945 a, 33, 121-129.
- Knox, G. W., Investigations of the flicker and fusion III. The effect of auditory stimulation on the visual CFF, J. gen Psychol., 1945 b, 33, 139-143.
- Knox, G. W., The control of occipital brain wave frequency, voltage, and wave forms by means of flashing light stimuli, Amer. J. Optom., 1950, 27, 345-349.
- Kravkov, S. V., Action des excitations auditives sur la frequence critique des papillotements lumineux. Acta ophthal. Kbh., 1935, 210-272, (abstracted in Landis, 1953).
- Kravkov, S. V., The action of aural stimuli on flicker fusion. Fiziol. ZH., S. S. S. R., 1935, 19, 826-834, (Psychol abs., 1936, 10, #4363).

- Kravkov, S. V., The influence of odors on color visions. Acta ophthal. Kbh., 1939, 14, 426-442, (abstracted in Landis, 1953).
- Kravkov, S. V., The critical frequency of flicker and indirect stimuli. C. R. Acad Sci. U. R. S. S., 1939, 22, 64-66, (psychol. abs., 1940, 14, #5351).
- Lansing, R. W., Relation of brain and tumor rhythms to visual reaction times. E. E. G. Clin Neurophysiol., 1957, 9, 497-504.
- Lazerev, P. P., The influence of an acoustic stimulation upon the light sensitivity of the eye, Proc. Acad. Sci., U. S. S. R., 7, 18, 1927, Russian (cited by Allen and Schwartz, 1940).
- Levine, Bernard, Sensory interaction: The joint affects of visual and auditory stimulation on critical flicker fusion frequency. Unpublished doctoral dissertation, Columbia University, 1958.
- Lewanow, M. N. (Rhythmical stimuli and the interrelationship of cortical areas. I.) (German abstract) Fiziol Zh. S. S. S. R., 1940, 28, 183-194, (abstracted in Landis, 1953).
- Li, C., McLennan, H. and Jasper, H., Brain Waves and unit discharge in cerebral cortex. Science, 1952, 116, 656-657.
- Lindquist, E. F., Design and Analysis of Experiments in Psychology and Education, Boston Houghton Mifflin Company, 1953.
- Lindsley, D. B., Basic perceptual processes and the E. E. G. Psychiat. Res Reports, 1956, 6, 161-170.
- Lindsley, D. B., Attention, Consciousness, Sleep and Wakefulness. In Hdbk Physiol. - Neurophysiol. III., Wash., D. C., Amer. Physiol Soc., 1960, Chap. 64, 1553-1593.
- London, I. D., Research on Sensory Interaction in the Soviet Union. Psychol. Bull., 1954, 51, 531-568.
- McCroskey, L, A research note on the effect of noise on flicker fusion frequency. U. S. N. Sch Aviat med res rep Proj. No. N. M. 180299 Sub. 1, no. 7, 1951 (Psychol. abst. 1958, 32)
- Morgan, C. T., Introduction to Psychology, N. Y., McGraw Hill Book Co., Inc., 1956, 257-266.
- Mundy-Castle, A. C., An appraisal of electroencephalography in relation to psychology. Nat. Inst. Person Res. Mon. Sup. 2, 1953.
- Polyak, S. I., The Retina Chicago: Univ. Chicago Press, 1941.

Porter, T. C., Contributions to the study of flicker, III. Proc. Royal Soc., 1912, 86A, 495-513 (abstracted in Landis, 1953).

Ranson, W. R. and Clark, S. L., The Anatomy of the Nervous System, Philadelphia, W. B. Saunders Company, 1953.

Roback, G. S., Krasno, L. R., and Ivy, A. C., Effect of Analeptic drugs on the somnifacient effect of seconal, and antihistamines as measured by the flicker fusion threshold. J. appl. Physiol., 1952, 4, 566-574.

Ross, R. T., Contribution to the theory of retinal reaction to intermittent stimuli Psychol. Bull., 1935, 32, 712.

Simonson, E., and Enzer N., Effect of pervitin (desoxyephedrine) on fatigue of the central nervous system, J. industr. Hyg., 1942, 24, 205-209.

Toman, J., Flicker potentials and the alpha rhythm in man. J. Neurophysiol., 1941, 4, 51-61.

Urbantschitsch, V., Uber den Einfluss einer Sinnerserregung auf die ubringen sinnesempfindugen, Arch, ges Physiol., 1888, 42, (cited in Allen and Schwartz, 1940).

Wang, G. H., Action potentials of the visual cortex and the superior colliculus induced by stimulation of the retina with light. Chinese J. Physiol., 1934, 8, 121-144 (abstracted in Landis, 1953).

Yakovlev, P. A., Visual sensations and perceptions, Moscow, 1935, 127, (Russian) cited in Allen, Schwartz, 1940.

Yakovlev, P. A., The influence of acoustic stimuli upon the limits of visual fields for different colors., J. opt. Soc. Amer., 1938, 28, 286.

Zoethout, Z. D., Physiological Optics 4th ed., Chicago Professional Press, 1947, 1-430 (abstracted in Landis, 1947).