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THE IMPLICATIONS OF MOTIVATIONAL FACTORS IN CORTICAL PLASTICITY: THEORY AND RESEARCH

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A thesis submitted to the faculty of the
College of Optometry
Pacific University
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Introduction

The human cortex is considered to be very plastic in the early years of childhood. However, the length of time in which one can manipulate the neuronal structure of the brain is a debatable issue. Traditional scientific thought establishes a window of time for cortical plasticity to take place known as the "critical period". Recent scientific research has provided evidence for the ability of the brain to remain or become plastic after the so-called critical period. Examination of this evidence will be done in context of amblyopia, a condition often mistakenly believed to be irreversible after the "critical period".

Amblyopia is defined as a condition of reduced visual acuity not correctable by refractive means and not attributable to any obvious structural anomalies.[1] Amblyopia can be further classified into organic and functional forms. Organic amblyopia is the result of any nutritional, toxic, or congenital abnormalities where no ocular pathology is evident. The term functional amblyopia is reserved for those patients in which the reduction of visual acuity is neither organic nor pathological. Deprivation of adequate visual stimulation early in a child's life results in amblyopia ex anopsia[2], which is defined as a deficit in vision due primarily to neuronal factors in the brain.

The first two years of human life are regarded to be the critical period (sensitive period) for the development of normal vision in the infant. If during this period one of the infant's eyes are prevented from receiving proper retinal stimulation through such anomalies as strabismus,

anisometropia or a high uncorrected refractive error, a functional amblyopia will result.

It is the assumption of many vision scientists that the causes of the reduced visual acuity is the result of an atrophy of the visual pathway, and thus, patients beyond the age of two become very difficult if not impossible to treat, hence an irreversible dysfunction. The clinician has long known that although cases of amblyopia may be difficult to treat beyond the age of two, by no means is the condition irreversible.

The treatment of amblyopia has always been controversial. Determining when and what type of treatment is effective has been based on Worth's proposal in 1903, suggesting that no functional improvement could be observed after the age of six.[3] However, the latest neurophysiological research presented in this paper is evidence for those who are bound by a "deterministic, linear systems analysis approach of western scientific thought"[4] and disbelieve the abundant functional validation of amblyopia "cures". Our purpose is to review literature which supports the role of neurophysiological mechanisms of active inhibition (vs. binocular competition) in amblyopia, the interaction between the reticular formation and the lateral geniculate nucleus (LGN), and the effects of the biogenic amines upon cortical plasticity. We propose: 1. active inhibition in the lateral geniculate nucleus, from both the dominant eye input and from striate cortex feedback, plays a significant role in functional amblyopia, 2. the interaction between the reticular formation and the retino-geniculo-striate pathways provides an accessible behavioral pathway for the treatment of amblyopia, 3. Motivation is a significant factor when predicting the progression and outcome of therapy.

Amblyopia therapy will continue to find success with supporting evidence such as this and with practioners who "believe".

The effects of early visual stimulus deprivation have been established experimentally. Early visual stimulus deprivation was induced in young kittens by surgical lid suture or patching before normal opening of the eye, which is approximately between 7 and 10 days (Hubel and Wiesel[5]). These kittens experienced monocular vision for the first few months of their lives. At the end of this time period, Hubel and Wiesel found that "monocular suture leads to a condition in which only 10% of the [cortical] cells can be activated via the deprived eye". [6] Monocularly deprived animals are those referred to as having stimulus deprivation amblyopia. Since the effects of early environment are crucial to normal development, the question arises as to what is the underlying mechanism to this deprivation amblyopia.

Early theories of this etiology postulated a binocular competition of development. Binocular competition was first proposed by Hubel and Wiesel in 1965 after a comparison study of the effects of unilateral and bilateral lid closure on cortical unit responses in kittens.[6] The effects of unilateral suture are the same as mentioned above; i.e. only 10% of the [cortical] cells can be activated via the deprived eye. Therefore, based on the monocular deprivation effects one might expect that binocular suture would produce very few cortical cells that either eye could activate. However, binocular suture does not produce comparable changes. "Instead, rearing with binocular suture results in cortical neurons that both eyes can activate and relatively few visually unresponsive cells are encountered. Because these alterations seem less severe than those after monocular suture, the effects of monocular suture can reasonably be

ascribed to unbalanced competitive interactions".[7] Even though Hubel and Wiesel in 1963 first pointed out that somata in the deprived A lamina, those receiving retinal afferents from the sutured eye, were about two-thirds as large as their nondeprived counterparts[7] it is important to remember that research has indicated that "neither monocular nor binocular eyelid suture had any observable effects on the features of synaptic development".[8] Therefore, the effects of stimulus deprivation amblyopia suggested a competitive nature between the eyes.

Experiments through the mid-1970's have supported the theory of binocular competition. However, research since 1976 has brought new insight as to the mechanism of stimulus deprivation amblyopia. "First, Kratz et al. showed that if the nondeprived eye is enucleated in monocularly deprived cats, the percentage of striate cortex responding to visual stimulation of the deprived eye increases from 5% to over 30%".[7] In another study, Duffy et al. utilized an intravenous injection of bicuculline which is an antagonist of the inhibitory neurotransmitter gama aminobutyric acid (GABA). This pharmacological manipulation rapidly restored the ability of the deprived eye to drive striate cortex cells. "Over 50% of the cells studied became responsive to visual stimulation of the deprived eye after the injection, and the effect can be observed reversibly".[7] Other, more recent studies indicate that 29%-42% of deprived eye cells become responsive after iontophoretic application of bicuculline treatment directly into the striate cortex. "Control experiments suggest that the effect is due to release from tonic inhibition rather than to nonspecific increases in cortical excitability".[7] The difference noted between enucleation and pharmacological manipulation suggests that there are tonic inhibitory sources from within the LGN and via corticogeniculate

pathways. Therefore, both enucleation of the nondeprived eye and bicuculline treatment indicate that the so called "critical period" is not final in terms of functional change, and that deprivational consequences are certainly not irreversible.

Sherman and Spear refer to the loss of cortical responsiveness to the deprived eye of monocularly deprived cats as a very rapid loss. "...the results in kittens deprived to 4-5 wk of age are nearly identical to those in animals deprived for months or years".[7] Therefore, "...these results indicate that the loss of response to the deprived eye in 4- to 5-wk-old monocularly deprived kittens is due almost entirely to an interaction (presumably suppressive) with inputs from the nondeprived eye. There is no evidence of a loss of inputs or even of a direct change in synaptic efficacy at this age". By 9-10 wk of age, however, the synaptic efficacy and/or connections from the deprived eye have significantly decreased. "Nevertheless many functional connections remain into adulthood".[7]

Recent studies utilizing immunocytochemistry offer reliable procedures for direct visualization of fibers in the CNS.[9,10] The results from these studies provide a neurophysiological framework and a possible model for active inhibitory deprivational amblyopia. Lateral geniculate interneuron cell bodies which occupy one layer of the LGN, project their axons to the layer below (which is the layer associated with the contralateral eye). The adjacent LGN layer also contains cell bodies of interneurons with axonal projections to the layer above (again, the contralateral eye). The interneurons use GABA as their neurotransmitter and thus are assumed to be inhibitory in nature.[11,12] The study by Papadopoulos and Parnavelas clearly showed dopaminergic innervation of the LGN. Dopamine has been implicated to be "an active neurosubstance"

within the LGN who's terminals target presynaptic dendrites of presumably GABAergic interneurons. Therefore, "a modulatory action of dopamine in GABA-mediated inhibition...is highly probable".[9]

Corticogeniculate axons provide direct synaptic input to interneurons in the LGN.[11] The excitatory effects of the corticogeniculate axons on the interneurons may serve as another system of inhibition on the visual pathway.

The LGN interneurons also provide the basic physiological "hook-up" for an active inhibitory model of deprivational amblyopia.[12,13] The ultrastructure of the synaptic connections on the dendritic portion of the LGN relay cell are such that the F cells (interneuron)[12,13] and retinal ganglion synapses are in the proximal region relative to the LGN relay neuron soma, with the F cell closer to the soma than the retinal ganglion synapse. Cortical synapses occur at intermediate and distal portions of the dendritic tree. This hook-up allows for active retinal information modification (including suppression) by the interneurons of adjacent LGN layers. Therefore, "...the position of most of the F terminals, being very close to the retinal terminals, is ideal for lowering the transfer ratio by inhibitory mechanisms. Such inhibition could also affect signals from more distal points on the dendrites where cortical and/or brain-stem afferents make their contacts. Lowering the transfer ratio or the signal-to-noise ratio would presumably reduce the information going to the cortex during conditions such as sleep or inattention."[12] There are three possible origination sources for the GABA containing F cell terminals. Those sources include: dendrites of LGN neurons, axons of interneurons, and axons from neurons in the thalamic reticular nucleus.[12] This research confirms at least three places in which GABAminergic inputs to the LGN originate.

There are afferents to the monkey's LGN from the brain stem,[12] however, the specifics have been studied relatively little. Other researchers[9] indicate that there is brainstem input to the LGN and others[11] have implicated the importance of cortical feedback to the LGN. The non-visual brainstem inputs and feedback systems suggest that there are "other" variables that influence the act of visual processing.

LGN interlaminar inhibition occurs mostly between layers 4 and 5, which are parvocellular layers. Parvocellular layers of the LGN receive mainly foveal input, as opposed to peripheral input. Deprivational amblyopia is a central, foveal phenomenon with decreased high spatial frequency resolution. Therefore, it is reasonable to say that the area in the LGN which is effected by deprivational amblyopia is also the area in which the most interlaminar inhibition takes place.

Therefore, inputs to the LGN from one eye will produce a given amount of inhibition to the adjacent LGN layer (contralateral eye) and visa versa for the other eye. This static level of equivalent inhibition between the two eyes may act as a functional tuning system. However, if the input to the LGN layer from one eye is diminished for some reason or another (e.g. monocular lid suture, ptosis, congenital cataract), the inhibitory output to the adjacent layer is also decreased. Therefore, the outputs of the "good" eye from the LGN are less inhibited and relatively greater in quantity than the "bad" eye. If the inputs are constantly decreased from the "bad" eye, the cortex learns to favor the "good" eye inputs and there's a resultant domino effect where the "good" eye continues to send the larger output and further inhibits the deprived eye. A functional amblyopia is the resulting condition. It is a learned biochemical process. Some future research may show why and/or how.

Examination of the cholinergic and monoaminergic innervation of the lateral geniculate nucleus and other thalamic nuclei in the cat demonstrate cholinergic fibers are present in all thalamic nuclei, but with striking differences in density. "...The lateral geniculate nucleus receives, by far, the greatest density of cholinergic fibers."[10] The sources of these fibers were located in the reticular formation. Determination of monoaminergic[14] brainstem afferent fiber input to the lateral geniculate indicated uniform distribution within the LGN and perigeniculate nuclei.[10] Only the cholinergic projections from the brainstem show a preferential innervation of the LGN. Cholinergic terminals in the lateral geniculate represent the association of cholinergic axons with encapsulated synaptic zones or glomeruli. "The presence of synaptic contacts between cholinergic fibers and dendritic processes in the synaptic glomerulus is significant because it means that these fibers synapse in the same location as retinal terminals, undoubtedly a strategic location for influencing the transmission of activity from retinal afferents to relay cells."[10] Another route by which the cholinergic system exerts an influence over the relay nuclei of the thalamus is via it's dense innervation of the reticular nucleus. "The reticular nucleus is composed entirely of GABAergic neurons and is the source of recurrent inhibition to all thalamic nuclei" therefore, activation of cholinergic neurons in the reticular formation would "presumably reduce the level of recurrent inhibition received by most of the thalamus".[10] The monoaminergic and cholinergic innervations of the lateral geniculate from the midbrain region are the accessible behavioral pathways which set the scene for effective therapy to take place.

The neural circuitry described has functional ramifications on visual information processing. As mentioned above, there is a dense cholinergic

innervation of the lateral geniculate, and uniform monoaminergic innervation across all thalamic nuclei. The source of these fibers arise from the midbrain; i.e. the reticular formation.[10,13] Perhaps the selective cholinergic innervation of visual sensory and motor structures in the thalamus and midbrain reflects the unique role of visually guided behavior in response to an arousing stimulus, where the reticular formation regulates the excitability of all thalamic nuclei.[10] The reticular activating system (RAS) is responsible for the activation of the EEG that accompanies the shift from states of sleep, drowsiness and inattentiveness to alertness.[10,15] Therefore, stimulation of the RAS would mimic this shift, would increase neural input to the thalamic nuclei, and thereby increase the pattern of spontaneous activity. The increased stimulation produces an increase in the single spike-firing of relay neurons and a concomitant reduction of high frequency bursts, that faithfully transmit sensory information to the cortex.[10,13] Raczkowski and Fitzpatrick noted that electrical stimulation of the reticular formation facilitates the response of neurons in the lateral geniculate nucleus to visually evoked stimulation. "At least some of these facilitatory effects are thought to arise from disinhibitory mechanisms since stimulating the brainstem reticular formation with brief electrical shocks eliminates hyperpolarizing potentials in geniculate relay cells."[13] These effects are attributed to the reticular formations cholinergic afferent fibers, assuming that acetylcholine(ACh) has an inhibitory effect on geniculate interneurons. The ACh inhibitory effects are mediated via muscarinic receptors. The facilitation of information through the relay cells is also due to ACh, however, this effect is achieved via nicotinic receptors.[13]

Therefore, based on the presented evidence there are neurological ultrastructures within the LGN and interactions between the LGN and reticular formation which would enable a visual system to learn suppressive behaviors; i.e. amblyopic behaviors. The same evidence suggests there are mechanisms which could be utilized to unlearn a suppressive behavior. This scientific evidence supports the clinician's claim that amblyopia can often be treated beyond the critical age (sensitive period). The question, however, still remains: if it is an inhibitory mechanism responsible for deprivational amblyopia, why is it that some patients show a return to normal vision after treatment, and some do not? What is the intangible element that separates success from failure?

Patient motivation seems to be the factor that most clinicians point to as being the difference between successful and unsuccessful vision therapy. But what is motivation? Does it have a physiological basis? Can it be quantified? Can the effects of motivation be experimentally documented?

Recent research has provided the "scientific" data to demonstrate the physiological basis for motivation. Norepinephrine is a neurotransmitter that is found in catecholaminergic pathways throughout the central nervous system. It is released upon activation of the sympathetic nervous system. Pettigrew[16] describes the origin of norepinephrine in the CNS as being the locus coeruleus of the RAS found in the midbrain. The RAS is responsible for the overall arousal and attention of an organism. Norepinephrine outputs leave the locus coeruleus and project throughout the cortex. These projections are also closely associated with the visual cortex. In fact, the striate cortex has as many inputs from the non-visual

locus coeruleus as it has from the primary visual pathway and the LGN.[16] This suggests that norepinephrine from the RAS is strongly related to the receiving and processing of visual information in the primary visual cortex.

Research by Kasamatsu and Associates [17] has demonstrated that norepinephrine is necessary to the maintenance and enhancement of neuronal plasticity. The release of norepinephrine from the locus coeruleus is only accomplished by stimulating the RAS. Hence, the patient must be attentive, aroused and motivated in order for synaptic reorganization to take place. The initial study done by Kasamatsu used a catecholamine, 6 hydroxydopamine (6-OHDA) as a blocking agent against norepinephrine. The experimental subjects were kittens with artificially induced deprivational amblyopia. Results indicated that kittens injected with 6-OHDA showed virtually a total loss of cortical plasticity, i.e. irreversible amblyopia. Later studies by Kasamatsu showed that enhanced release of endogenous norepinephrine could restore plasticity in the mature cat cortex.

This evidence suggests that norepinephrine released by the locus coeruleus at specific synapses acts as a modulator that directly increases cortical plasticity. Recent work by Aoki and Siekevitz[18] has helped explain this modulatory mechanism. These researchers state that norepinephrine acts as a trigger in combination with a protein called G-protein, which activates an enzyme involved in the production of cyclic adenosine monophosphate (cAMP). The production of cAMP is a necessary step in the dephosphorylation of microtubule associated protein 2 (MAP2). The dephosphorylation of MAP2 is believed to be directly responsible for the changing of the tertiary structure of the microtubule cytoskeleton proteins

which make up CNS neurons. In changing this structure, it is believed that neuronal structure is altered, and in doing so, there is an alteration of biological activity, hence an increase in neuronal plasticity and reorganization.

The presented scientific research provides the neurophysiological mechanisms responsible for the condition "functional amblyopia", it also shows that the RAS maintains biochemical communications with the LGN (to open the suppressed information channels) and to the visual cortex (to enhance neuronal plasticity and reorganization). "These biochemicals do not, themselves, cause the changes in brain processing, but, rather, they set things up so that change can occur".[19]

Based on recent scientific research, we investigated an animal model of motivation and attempted to experimentally demonstrate the implications of motivation on cortical plasticity. We induced a deprivational amblyopia in kittens via monocular occlusion. The kittens were trained to respond to high contrast spatial frequency gratings in order to asses the animal's visual acuity. After the critical period of visual development, the monocular occlusion was reversed forcing the animal to use the "amblyopic" eye. Based on the assumption that switched occlusion would reverse the induced deprivational effects, two experimental groups were formed: with and without the motivational factor. We hypothesized that the group with the motivational factor would take less time to demonstrate the reversal of the amblyopia. We isolated time as the dependant variable, while trying to keep all other variables constant.

Materials and Methods

Apparatus

Figures 1 and 2 illustrate the apparatus used in our experiments, which was a modified Lashley jumping stand. The stand was made of a black plywood box (38.5 x 71 x 166 cm) and cut away in front to 100 cm. Two trapdoors (35.5 x 35.5 cm) located 39 cm above the floor and separated by a central divider were held closed by pressure latches that could also be locked into the closed position by metal pins.

Photographic reductions of commercially prepared (Intergraphics, Kirkland Washington) high contrast, square-wave gratings served as the visual stimuli for the training and testing. Each grating had a homogeneous grey photograph of matching luminance used with it. The gratings and grey photographs (12.5 x 19 cm) were laminated and placed on the closed trapdoors in matched pairs (Figure 3). Uniform lighting was provided by two fluorescent (F40CW) cool white bulbs resting on top of the stand.

A wooden tunnel (38 x 17.8 x 10.7 cm) was centered directly in front of the stand and placed the kittens' eyes 37.5 cm above the stimuli.

Cats and Contact Lenses

The nine kittens used for the study were raised in the Pacific University College of Optometry animal care facility. This is a USDA approved, closed breeding colony. These nine kittens were housed (with their mothers) in a light-deprived room after they were born. They were divided into a control group and two experimental groups in which one had a motivational factor and another without a motivational factor. All kittens were maintained in a dark environment except for six hours per

day, five days per week. During this six hours the experimental animals were allowed to play in a fully-lighted animal colony room with the opaque contact lenses on their right eyes. The control animals were allowed to play without any lenses on.

Contact lenses were ordered using parameters for corneal curvature based on a previous study.[20] The contact lenses were made of polymethyl methacrylate (PMMA). These lenses were made completely opaque by applying two separate coats of black enamel to the front surface. The kittens started wearing the contact lenses at about 5 weeks of age. All Experimental animals wore the contact lenses on the right eye at first. After the critical period, they were occluded in the other eye (left eye) with or without the motivational factor.

Special flashlights were devised in order to apply the contact lenses on the kittens while allowing little or no light to be seen by the kittens. A red (Wratten gel #29) filter was affixed to the front of a flashlight, allowing transmission only of deep red light (625nm or greater). This provided enough light for us to see, but did not yield substantial lighting to the kittens, since cats have little ability to see wavelengths above 600nm.[21,22]

Training Procedure

Training the kittens followed the same protocol as that developed by Feiten and Mace.[23] All of the kittens received six weeks of wearing the contact lens for six hours per day before training was started. This allowed the kittens to get used to wearing the contact lens and to allow monocular visual experience. We began training the kittens at eleven weeks of age and continued until the twentieth week. The training sequence started with one door left open while the visual stimulus was placed over a closed, locked door. The grating was randomly placed on the right and left side with no more than two consecutive placements of the grating on the same side. The kitten was placed into the tunnel of the Lashley jumping stand and encouraged to jump out the other end by blocking the entrance. The kitten was able to choose which side to jump to and correct responses were reinforced with a treat of tuna fish on a semi-random schedule. Incorrect responses resulted in the animal falling one foot to the floor of the enclosed "pit" as a form of negative reinforcement. After the kittens jumped without hesitating to the side with the grating, the door on the side with no grating was closed but remained unlocked. No visual stimulus was placed on this side during this stage of training. If the kitten jumped to this unlocked side with no grating, the trapdoor opened, and the animal dropped to the floor. The kitten was left in the "pit" for approximately 15 seconds before being picked up and petted. The kitten was then placed into the tunnel again. After this stage of training was mastered, the same procedure was followed only with the appropriate homogeneous matched-luminance gray photograph placed on the closed but unlocked, side of the jumping stand. Now the kitten had to chose between jumping to the side with the grating or the side with the gray photograph. Training procedure

was performed Monday through Friday and continued until a 75% accuracy was achieved.

Testing Procedure

We followed the similar testing procedure developed by Feiten and Mace[23], Haley and White[5], with slight modification. Testing was performed Tuesday through Friday. The training procedure was given every Monday to reinforce the testing procedure. During testing, no positive reinforcement was given and both doors were locked. If the kitten was unable to distinguish the grating there was no negative reinforcement. To reinforce the procedure, training-frequency gratings were presented between test gratings. Test gratings were presented an equal number of times on the right and left side. Two different training gratings were presented between each test grating to prevent memorization of a specific training grating. Testing was done from the twentieth to the forty-fifth week of age.

Reversal Procedure with and without Motivational Factor

After the critical period of visual development (ten months), the experimental groups were occluded in the non-amblyopic eye (left eye). Then the same testing procedure was continued for an average of twenty weeks.

In the experimental group without the motivational factor, the test procedure was the same as described above. The group with the motivational factor had a different test procedure. They were deprived of food during housing. Food was given when they achieved a correct

response. Unfortunately, two of three in the motivational group suffered from corneal ulcers and were faded out from the project.

Data Analysis

Data (correct or non-correct responses) were entered into a data program created with Microsoft Excel. The program was created entirely for this project. (See Appendix A for computer spreadsheet and formulas.)

Results

Training

After the training period, each cat was able to respond to the appropriate spatial frequency grating at least 75% of the time. The performance of each cat was variable from one training date to another. Therefore, there were a few instances in which 75% was not achieved five consecutive days.

Testing

Testing was conducted as described in the materials and methods section for a period of twenty-five weeks.

Reversals

1. Spatial Frequency Recognition:

The presentation of the results correspond to a descriptive verses analytical type of statistical analysis with an arbitrary analytical 75% criterion. The average level of spatial frequency recognition for each

group can be inferred from the graphs, Figures 4, 5, and 6, which demonstrate percent of correct responses. The average visual acuity of each group and eye was as follows: control O.D. 3.0cpd O.S. 3.0cpd, experimental O.D. 2.0cpd O.S. 3.0cpd, motivational O.D. 2.0cpd O.S. 2.0cpd, given a criterion of 75%.

The variance of responses between the right and left eye can also be inferred from the graphs (Figures 4 and 5). As the cycles per degree increased the variability of responses increased and the cats would jump randomly with little regard for the gratings or they would jump consistently to one side. The percent of correct responses given by the control group demonstrated equal variance between the right and left eye for 1.0, 1.5, 3.0, 4.0, and 6.0cpd.. The right eye varied slightly more than the left when testing 2.0cpd for the first ten weeks of reversals. When testing 4.0cpd, the right eye indicated poorer performance, and both eyes decreased in performance as the cycles per degree increased. The percent of correct responses given by the experimental group varied more for the right eye during the first several weeks (9-10) of reversals. This is noted especially with 1.0cpd and 2.0cpd. After the first 9-10 weeks, the average percent of correct responses remained at or above the 75% criterion level with less variance. Spatial frequencies of 3.0, 4.0, and 6.0cpd demonstrated increasing variability of both eyes equally. When testing 1.0, 2.0, and 3.0cpd, the right eye suggested poorer performance. The motivational cat (Figure 6) showed an increased variance for the first 10-12 weeks with an equal variance between both eyes when testing 1.0cpd and 1.5cpd. When testing 2.0cpd, the left eye varied more than the right for the first 10-13 weeks, then the variance between eyes resolved and the average percent of correct responses remained at or above the 75%

criterion level with less variance. The magnitude of variance increased correspondingly as 3.0cpd, 4.0cpd, and 6.0cpd were tested. The cats behavior/mood influenced their performance as well. Some days a cat would be more difficult to handle and less attentive to the task.

2. Time to Achieve 75% criterion:

Two of the control cats demonstrated similar performance between the right eye and left eye for spatial frequencies of 1.0cpd, 1.5cpd, and 2.0cpd in terms of time to achieve the 75% criterion. The third control cat showed an increased time to achieve 75% criterion with the left eye for 1.5cpd and 2.0cpd. Both eyes of all three cats exhibited an increased time to achieve 75% criterion for 3.0cpd, 4.0cpd, and 6.0cpd. In addition, there was an increased variability of time between eyes for the higher frequencies (Figure 7).

Within the experimental group, one cat's performance was much like the control group's (Figure 8). There was similar performance between the right and left eyes for spatial frequencies of 1.0cpd, 1.5cpd, and 2.0cpd and then as the cycles per degree increased, there was an increased time to achieve and an increased variance between eyes. The two other cats of the experimental group demonstrated that 2.0cpd took the least amount of time to achieve for both right and left eyes. 1.0cpd and 1.5cpd took slightly longer with little difference between eyes, while testing of 3.0, 4.0, and 6.0cpd demonstrated an even longer time to achieve was necessary with an increased variance between eyes.

The motivational cat's performance was very similar to that of the two alike experimental cats (Figure 9). There was little difference between the right and left eye for all cycles per degree graphically

depicted. 75% criterion was achieved the fastest by 2.0cpd, 1.0cpd and 1.5cpd took slightly longer, while 3.0, 4.0, and 6.0cpd were increasingly longer as the cycles per degree increased.

3. Comparison: Experimental vs. Control

The performance of the experimental group indicated the right eye was slightly poorer (based on the the percent of correct responses) than the left eye when testing the spatial frequency of 1.0, 2.0, and 3.0cpd. The control group did not indicate better performance by either eye except when testing 4.0cpd. However, the spatial frequency 1.5cpd showed no significant performance difference between the two eyes in both groups. The control group indicated slightly better left eye performance when testing 4.0cpd, while the experimental group showed no difference. Average results for either group when testing 6.0cpd did not suggest one eye as better than the other. Observations based on the time to achieve indicate similar performance between the two eyes for spatial frequencies of 1.0,1.5, and 2.0cpd (Figure 10). The control group exhibited an increase in time to achieve and variance between eyes when testing 3.0, 4.0, and 6.0cpd. Whereas the experimental group demonstrated an increase in time to achieve and variance between eyes when testing 1.0, 1.5, 3.0, 4.0, and 6.0cpd. All groups demonstrated an increasingly longer time to achieve the 75% criterion as the cycles per degree increased.

4. Comparison: Experimental vs. Motivational

The experimental and motivational cats demonstrated similar performance.

Discussion

A descriptive type of statistical analysis with an arbitrary analytical 75% criterion was used to present our results. The 75% criterion is considered a standard practice when behaviorally determining the visual acuities in cats.[21,22,24,25] Therefore, we determined the average level of spatial frequency recognition (visual acuity) for each group using Figures 4,5, and 6. The behaviorally determined average visual acuity of the control group, O.D. 3.0cpd O.S. 3.0cpd, is similar to those found by others. The non-deprived left eye of the experimental group also indicated an appropriate average visual acuity of 3.0cpd. The range of visual acuity extends from 3.1 cycles/degree reported by Muir and Mitchell[26] to 6 cycles/degree found by Blake, Cool, and Crawford.[21] The range of acuity measurements may be attributed to the different behavioral techniques utilized or could be representative of the variance within the cat population.[21] A brief discussion of our visual acuity determinations should be noted before we go on to the experimentally altered visual acuities. The visual acuity for each group was found by determining the spatial frequency which demonstrated the majority of correct responses above the 75% cut-off level in Figures 4, 5, and 6. The figures depict the percent of correct responses verses time. The time period shown begins at the time of reversed occlusion. Therefore, the average visual acuity for the experimentally deprived eyes include data which involve the effects of the reversed occlusion. Therefore, the reported average acuities may be skewed towards a higher resolution. The average visual acuities of the experimentally deprived right eyes were 2.0cpd for both experimental groups. The motivational cat's non-deprived visual acuity was 2.0cpd.

This acuity is probably not representative of this population due to the small size of the group. The motivational group was originally matched in size to the other groups, unfortunately, two of the three animals developed corneal ulcers and were faded out of the experiment. The visual acuities of the control group can reasonably be compared to those reported by others since there wasn't any occlusion. Therefore, via the techniques we used, we can safely say that any deviation from the reported 3.0cpd visual acuity is a result of experimentally induced factors.

As mentioned above, the average correct responses include factors that influence the average visual acuity. The factor is the time in which the acuity measurements were taken. Therefore, examination of the responses at different points in time bears notice.

The factor chosen to be evaluated at different points in time is the varying magnitude of responses, and the variance between eyes over time. There is significance in these varying responses. The control group, for example, demonstrated little or no difference between eyes. This is expected since neither eye was deprived of normal stimulation. However, the responses given by the experimental group (without the motivational factor) varied between eyes, especially during the first 9-10 weeks. The varying responses demonstrated the left eye to be more stable than the right. The percent of correct responses were lower for the right eye. Over 9-10 weeks these differences gradually smoothed out and the average responses were at or above the 75% criterion level. This suggests the decreased right eye performance was secondary to the induced deprivational amblyopia. The gradual increase in performance suggests that a reversal of the amblyopia was taking place. This is especially seen in 1.0 and 2.0cpd. 3.0cpd shows an increase performance over time of the

left eye, however, the right eye can not repeat the same performance, more than likely due to the deprivational effects. In most of the graphs, the left eye percent of correct responses is lower at the beginning than one would expect since there was no deprivation of this eye. We can not offer an explanation for this. All three groups' percent of correct responses decreased as the cpd increased, indicating the cpd were approaching the limit of reasonably measurable visual acuity. (Notice the number of data points above the 75% line as the cpd's increase in Figures 4,5, and 6).

The performance of the motivational cat was ambiguous. We expected to see results much like that of the experimental group. However, there was no definite difference between the right and left eye. We also expected to see a quicker time to reverse the effects of deprivation compared to the experimental group without the motivational factor. This was not observed. Again, a single subject limits our findings.

To compare time as a variable we created Figures 7,8,9, and 10. The time to achieve the 75% criterion is an arbitrary analytical criterion used to indicate the point in time which three consecutive correct responses were at or above 75%. We used the variation between eyes of the control group to indicate normal variation (Figure 10). The two experimental groups did not demonstrate the right eye as taking longer to achieve the 75% criterion as would be expected of a deprived eye (Figures 9 and 10). The experimental and motivational animals produced similar results with one striking difference to the control animals. The control group took approximately three to five weeks to achieve the 75% criterion when testing 1.0,1.5,2.0 and 3.0cpd, while the experimental group took four to eight weeks to achieve 75% criterion for 1.0 and 1.5cpd. The increased time to achieve at the lower spatial frequencies by the experimental group

may be attributable to the effects of deprivation. All three groups achieved criterion the quickest when testing 2.0cpd, three to four weeks. The control group achieved 75% criterion in three to four weeks for 3.0cpd, however, the experimental and motivational groups took eight to twelve weeks. We would expect the right eye to have an increased time due to the deprivational effects and not the left eye, however, both took an equally longer time. This is another set of interesting data points we cannot explain. The increased time to achieve criterion when testing suggests the limits of visual acuity again are being reached.

We attempted to experimentally demonstrate the implications that motivation would have on cortical plasticity. Even though conclusions were not drawn from this study, our theory is supported throughout the literature. We have documented an induced deprivational amblyopia and described the progression of reversing the condition. We attempted to include motivation as a factor and measure it in terms of time, however, due to the diminished group size, we were not able to draw an experimental conclusion. Yet, this is another example of being unable to scientifically demonstrate what we know clinically is very important.

All fields of therapy have long recognized the importance of attention, arousal and motivation. The literature presented in this paper helps demonstrate why it is that these factors are crucial to success. With an understanding of how motivation aids in making the brain more pliable and receptive, therapy and new learning can be that much more effective. Motivation is the interaction between the patient and the environment and the therapist as well. The degree of the functional interaction with the environment is governed to an extent by the attentiveness that the individual allocates to the situation. Thus the synaptic organization is

formed by the reticular activating system, and the sensory input system active at that time in space. The scientific data presents strong evidence of the biochemical nature of motivation in securing cortical plasticity. Norepinephrine, which is an essential modulator in the chain of events leading to cortical reorganization is the biochemical link between motivation and successful vision therapy. Therefore, the amount of arousal/motivation could determine not only the behavioral interaction but the possibility of new synaptic connections and new learning.

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Appendix A

Microsoft Excel program created to summarize raw data. Depicted in regular and formula format.

TEST DATA FORM

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
157	Week of														
158	Cat form 1	OD	OD	OD			Cat form 2	OD	OD	OD			Cat form 3	OD	OD
159	Examiner:						Examiner:						Examiner:		
160	Date:						Date:						Date:		
161		Testing??						Testing??						Testing??	
162	Enter 1 if testing »»	0					Enter 1 if testing »»	0					Enter 1 if testing »»	0	
163															
164	Trial	Left	Right	Correct	Wrong		Trial	Left	Right	Correct	Wrong		Trial	Left	Right
165	1	3	N10	0	1		1	N1	T-2	0	1		1	0.25	N9
166	2	N12	1.5	0	1		2	1	N1	0	1		2	N2	6
167	3	T-.5	N10	0	1		3	N1	8	0	1		3	8	N1
168	4	8	N1	0	1		4	N10	3	0	1		4	T-2	N1
169	5	N10	3	0	1		5	0.25	N9	0	1		5	N1	1
170	6	N1	T-2	0	1		6	1.5	N12	0	1		6	0.25	N9
171	7	6	N2	0	1		7	N1	8	0	1		7	12	N1
172	8	0.25	N9	0	1		8	T-.5	N10	0	1		8	N10	T-.5
173	9	T-2	N1	0	1		9	N5	4	0	1		9	N12	1.5
174	10	N12	1.5	0	1		10	6	N2	0	1		10	1	N1
175	11	4	N5	0	1		11	N1	T-2	0	1		11	N9	0.25
176	12	N9	0.25	0	1		12	1.5	N12	0	1		12	T-.5	N10
177	13	N10	3	0	1		13	4	N5	0	1		13	4	N5
178	14	6	N2	0	1		14	N1	12	0	1		14	N1	T-2
179	15	12	N1	0	1		15	T-.5	N10	0	1		15	8	N1
180	16	N10	T-.5	0	1		16	12	N1	0	1		16	3	N10
181			Subtotal:	0	16				Subtotal:	0	16				Subtotal:
182															
183	Week of														
184	Cat form 1	OS	OS	OS			Cat form 2	OS	OS	OS			Cat form 3	OS	OS
185	Examiner:						Examiner:						Examiner:		
186	Date:						Date:						Date:		
187		Testing??						Testing??						Testing??	
188	Enter 1 if testing »»	0					Enter 1 if testing »»	0					Enter 1 if testing »»	0	
189															
190	Trial	Left	Right	Correct	Wrong		Trial	Left	Right	Correct	Wrong		Trial	Left	Right
191	1	3	N10	0	1		1	N1	T-2	0	1		1	0.25	N9
192	2	N12	1.5	0	1		2	1	N1	0	1		2	N2	6
193	3	T-.5	N10	0	1		3	N1	8	0	1		3	8	N1
194	4	8	N1	0	1		4	N10	3	0	1		4	T-2	N1
195	5	N10	3	0	1		5	0.25	N9	0	1		5	N1	1
196	6	N1	T-2	0	1		6	1.5	N12	0	1		6	0.25	N9
197	7	6	N2	0	1		7	N1	8	0	1		7	12	N1
198	8	0.25	N9	0	1		8	T-.5	N10	0	1		8	N10	T-.5
199	9	T-2	N1	0	1		9	N5	4	0	1		9	N12	1.5
200	10	N12	1.5	0	1		10	6	N2	0	1		10	1	N1
201	11	4	N5	0	1		11	N1	T-2	0	1		11	N9	0.25
202	12	N9	0.25	0	1		12	1.5	N12	0	1		12	T-.5	N10
203	13	N10	3	0	1		13	4	N5	0	1		13	4	N5
204	14	6	N2	0	1		14	N1	12	0	1		14	N1	T-2
205	15	12	N1	0	1		15	T-.5	N10	0	1		15	8	N1
206	16	N10	T-.5	0	1		16	12	N1	0	1		16	3	N10
207			Subtotal:	0	16				Subtotal:	0	16				Subtotal:

	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE
157																
158	OD															
159																
160																
161																
162																
163				SUMMARY	Week of			OD								
164	Correct	Wrong			# OF TEST	#OF CORRECT	# OF WRONG	%OF CORRECT								
165	0	1		0.25	0	0	0	#DIV/0!								
166	0	1		0.50	0	0	0	#DIV/0!								
167	0	1		1.00	0	0	0	#DIV/0!								
168	0	1		1.50	0	0	0	#DIV/0!								
169	0	1		2.00	0	0	0	#DIV/0!								
170	0	1		3.00	0	0	0	#DIV/0!								
171	0	1		4.00	0	0	0	#DIV/0!								
172	0	1		6.00	0	0	0	#DIV/0!								
173	0	1		8.00	0	0	0	#DIV/0!								
174	0	1		12.00	0	0	0	#DIV/0!								
175	0	1		TOTAL	0	0	0	#DIV/0!								
176	0	1														
177	0	1														
178	0	1														
179	0	1														
180	0	1														
181	0	16														
182																
183																
184	OS															
185																
186																
187														FOUR WEEKS' DATA SUMMARY		
188										COMPARISON	Week of			Week of	TO	Week of
189				SUMMARY	Week of			OS			% OF CORRECT	% OF CORRECT			% OF CORRECT	% OF CORRECT
190	Correct	Wrong			# OF TEST	#OF CORRECT	# OF WRONG	%OF CORRECT			OD	OS			OD	OS
191	0	1		0.25	0	0	0	#DIV/0!		0.25	#DIV/0!	#DIV/0!		0.25	#DIV/0!	#DIV/0!
192	0	1		0.50	0	0	0	#DIV/0!		0.50	#DIV/0!	#DIV/0!		0.50	#DIV/0!	#DIV/0!
193	0	1		1.00	0	0	0	#DIV/0!		1.00	#DIV/0!	#DIV/0!		1.00	#DIV/0!	#DIV/0!
194	0	1		1.50	0	0	0	#DIV/0!		1.50	#DIV/0!	#DIV/0!		1.50	#DIV/0!	#DIV/0!
195	0	1		2.00	0	0	0	#DIV/0!		2.00	#DIV/0!	#DIV/0!		2.00	#DIV/0!	#DIV/0!
196	0	1		3.00	0	0	0	#DIV/0!		3.00	#DIV/0!	#DIV/0!		3.00	#DIV/0!	#DIV/0!
197	0	1		4.00	0	0	0	#DIV/0!		4.00	#DIV/0!	#DIV/0!		4.00	#DIV/0!	#DIV/0!
198	0	1		6.00	0	0	0	#DIV/0!		6.00	#DIV/0!	#DIV/0!		6.00	#DIV/0!	#DIV/0!
199	0	1		8.00	0	0	0	#DIV/0!		8.00	#DIV/0!	#DIV/0!		8.00	#DIV/0!	#DIV/0!
200	0	1		12.00	0	0	0	#DIV/0!		12.00	#DIV/0!	#DIV/0!		12.00	#DIV/0!	#DIV/0!
201	0	1		TOTAL	0	0	0	#DIV/0!		TOTAL	#DIV/0!	#DIV/0!		TOTAL	#DIV/0!	#DIV/0!
202	0	1														
203	0	1														
204	0	1														
205	0	1														
206	0	1														
207	0	16														

	A	B	C	D	E	F	G
157	Week of						
158	Cat form 1	OD	OD	OD			Cat form 2
159	Examiner:						Examiner:
160	Date:						Date:
161		Testing??					
162	**Enter 1 if testing »»**	0					**Enter 1 if testing »»**
163							
164	Trial	Left	Right	Correct	Wrong		Trial
165	1	3	N10	0	=1-D165		1
166	2	N12	1.5	0	=1-D166		2
167	3	T-.5	N10	0	=1-D167		3
168	4	8	N1	0	=1-D168		4
169	5	N10	3	0	=1-D169		5
170	6	N1	T-2	0	=1-D170		6
171	7	6	N2	0	=1-D171		7
172	8	0.25	N9	0	=1-D172		8
173	9	T-2	N1	0	=1-D173		9
174	10	N12	1.5	0	=1-D174		10
175	11	4	N5	0	=1-D175		11
176	12	N9	0.25	0	=1-D176		12
177	13	N10	3	0	=1-D177		13
178	14	6	N2	0	=1-D178		14
179	15	12	N1	0	=1-D179		15
180	16	N10	T-.5	0	=1-D180		16
181			Subtotal:	=SUM(D165:D180)	=SUM(E165:E180)		
182							
183	Week of						
184	Cat form 1	OS	OS	OS			Cat form 2
185	Examiner:						Examiner:
186	Date:						Date:
187		Testing??					
188	**Enter 1 if testing »»**	0					**Enter 1 if testing »»**
189							
190	Trial	Left	Right	Correct	Wrong		Trial
191	1	3	N10	0	=1-D191		1
192	2	N12	1.5	0	=1-D192		2
193	3	T-.5	N10	0	=1-D193		3
194	4	8	N1	0	=1-D194		4
195	5	N10	3	0	=1-D195		5
196	6	N1	T-2	0	=1-D196		6
197	7	6	N2	0	=1-D197		7
198	8	0.25	N9	0	=1-D198		8
199	9	T-2	N1	0	=1-D199		9
200	10	N12	1.5	0	=1-D200		10
201	11	4	N5	0	=1-D201		11
202	12	N9	0.25	0	=1-D202		12
203	13	N10	3	0	=1-D203		13
204	14	6	N2	0	=1-D204		14
205	15	12	N1	0	=1-D205		15
206	16	N10	T-.5	0	=1-D206		16
207			Subtotal:	=SUM(D191:D206)	=SUM(E191:E206)		

	H	I	J	K	L	M	N
157							
158	OD	OD	OD			Cat form 3	OD
159						Examiner:	
160						Date:	
161	Testing??						Testing??
162	0					**Enter 1 if testing »»**	0
163							
164	Left	Right	Correct	Wrong		Trial	Left
165	N1	T-2	0	=1-J165		1	0.25
166	1	N1	0	=1-J166		2	N2
167	N1	8	0	=1-J167		3	8
168	N10	3	0	=1-J168		4	T-2
169	0.25	N9	0	=1-J169		5	N1
170	1.5	N12	0	=1-J170		6	0.25
171	N1	8	0	=1-J171		7	12
172	T-.5	N10	0	=1-J172		8	N10
173	N5	4	0	=1-J173		9	N12
174	6	N2	0	=1-J174		10	1
175	N1	T-2	0	=1-J175		11	N9
176	1.5	N12	0	=1-J176		12	T-.5
177	4	N5	0	=1-J177		13	4
178	N1	12	0	=1-J178		14	N1
179	T-.5	N10	0	=1-J179		15	8
180	12	N1	0	=1-J180		16	3
181		Subtotal:	=SUM(J165:J180)	=SUM(K165:K180)			
182							
183							
184	OS	OS	OS			Cat form 3	OS
185						Examiner:	
186						Date:	
187	Testing??						Testing??
188	0					**Enter 1 if testing »»**	0
189							
190	Left	Right	Correct	Wrong		Trial	Left
191	N1	T-2	0	=1-J191		1	0.25
192	1	N1	0	=1-J192		2	N2
193	N1	8	0	=1-J193		3	8
194	N10	3	0	=1-J194		4	T-2
195	0.25	N9	0	=1-J195		5	N1
196	1.5	N12	0	=1-J196		6	0.25
197	N1	8	0	=1-J197		7	12
198	T-.5	N10	0	=1-J198		8	N10
199	N5	4	0	=1-J199		9	N12
200	6	N2	0	=1-J200		10	1
201	N1	T-2	0	=1-J201		11	N9
202	1.5	N12	0	=1-J202		12	T-.5
203	4	N5	0	=1-J203		13	4
204	N1	12	0	=1-J204		14	N1
205	T-.5	N10	0	=1-J205		15	8
206	12	N1	0	=1-J206		16	3
207		Subtotal:	=SUM(J191:J206)	=SUM(K191:K206)			

	O	P	Q	R	S	T	U	V
157								
158	OD	OD						
159								
160								
161								
162								
163					SUMMARY	=A157		
164	Right	Correct	Wrong			# OF TEST	# OF CORRECT	# OF WRONG
165	N9	0	=1-P165		0.25	=B162*2+H162*1+N162*3	=SUM(D172+D176+J169+P16	=T165-U165
166	6	0	=1-P166		0.5	=B162*2+H162*2+N162*2	=SUM(D167+D180+J172+J17	=T166-U166
167	N1	0	=1-P167		1	=B162*0+H162*1+N162*2	=SUM(J166+P169+P174)	=T167-U167
168	N1	0	=1-P168		1.5	=B162*2+H162*2+N162*1	=SUM(D166+D174+J170+J17	=T168-U168
169	1	0	=1-P169		2	=B162*2+H162*2+N162*2	=SUM(D170+D173+J165+J17	=T169-U169
170	N9	0	=1-P170		3	=B162*3+H162*1+N162*1	=SUM(D165+D169+D177+J16	=T170-U170
171	N1	0	=1-P171		4	=B162*1+H162*2+N162*1	=SUM(D175+J173+J177+P17	=T171-U171
172	T-.5	0	=1-P172		6	=B162*2+H162*1+N162*1	=SUM(D171+D178+J174+P16	=T172-U172
173	1.5	0	=1-P173		8	=B162*1+H162*2+N162*2	=SUM(D168+J167+J171+P167	=T173-U173
174	N1	0	=1-P174		12	=B162*1+H162*2+N162*1	=SUM(D179+J178+J180+P171	=T174-U174
175	0.25	0	=1-P175		TOTAL	=SUM(T165:T174)	=SUM(U165:U174)	=SUM(V165:V174)
176	N10	0	=1-P176					
177	N5	0	=1-P177					
178	T-2	0	=1-P178					
179	N1	0	=1-P179					
180	N10	0	=1-P180					
181	Subtotal:	=SUM(P165:P180)	=SUM(Q165:Q180)					
182								
183								
184	OS	OS						
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186								
187								
188								
189					SUMMARY	=A183		
190	Right	Correct	Wrong			# OF TEST	# OF CORRECT	# OF WRONG
191	N9	0	=1-P191		0.25	=B188*2+H188*1+N188*3	=SUM(D198+D202+J195+P19	=T191-U191
192	6	0	=1-P192		0.5	=B188*2+H188*2+N188*2	=SUM(D193+D206+J198+J20	=T192-U192
193	N1	0	=1-P193		1	=B188*0+H188*1+N188*2	=SUM(J192+P195+P200)	=T193-U193
194	N1	0	=1-P194		1.5	=B188*2+H188*2+N188*1	=SUM(D192+D200+J196+J202	=T194-U194
195	1	0	=1-P195		2	=B188*2+H188*2+N188*2	=SUM(D196+D199+J191+J201	=T195-U195
196	N9	0	=1-P196		3	=B188*3+H188*1+N188*1	=SUM(D191+D195+D203+J194	=T196-U196
197	N1	0	=1-P197		4	=B188*1+H188*2+N188*1	=SUM(D201+J199+J203+P203	=T197-U197
198	T-.5	0	=1-P198		6	=B188*2+H188*1+N188*1	=SUM(D197+D204+J200+P192	=T198-U198
199	1.5	0	=1-P199		8	=B188*1+H188*2+N188*2	=SUM(D194+J193+J197+P193	=T199-U199
200	N1	0	=1-P200		12	=B188*1+H188*2+N188*1	=SUM(D205+J204+J206+P197	=T200-U200
201	0.25	0	=1-P201		TOTAL	=SUM(T191:T200)	=SUM(U191:U200)	=SUM(V191:V200)
202	N10	0	=1-P202					
203	N5	0	=1-P203					
204	T-2	0	=1-P204					
205	N1	0	=1-P205					
206	N10	0	=1-P206					
207	Subtotal:	=SUM(P191:P206)	=SUM(Q191:Q206)					

	W	X	Y	Z	AA	AB	AC	AD
157								
158								
159								
160								
161								
162								
163	CD							
164	% OF CORRECT							
165	=U165/T165							
166	=U166/T166							
167	=U167/T167							
168	=U168/T168							
169	=U169/T169							
170	=U170/T170							
171	=U171/T171							
172	=U172/T172							
173	=U173/T173							
174	=U174/T174							
175	=U175/T175							
176								
177								
178								
179								
180								
181								
182								
183								
184								
185								
186								
187							FOUR WEEKS' DATA SUMMARY	
188			COMPARISON	=T189			=Z32	TO
189	OS			% OF CORRECT	% OF CORRECT			% OF CORRECT
190	% OF CORRECT			CD	OS			CD
191	=U191/T191		0.25	=W165	=W191		0.25	=(Z191+Z139+Z87+Z35)/4
192	=U192/T192		0.5	=W166	=W192		0.5	=(Z192+Z140+Z88+Z36)/4
193	=U193/T193		1	=W167	=W193		1	=(Z193+Z141+Z89+Z37)/4
194	=U194/T194		1.5	=W168	=W194		1.5	=(Z194+Z142+Z90+Z38)/4
195	=U195/T195		2	=W169	=W195		2	=(Z195+Z143+Z91+Z39)/4
196	=U196/T196		3	=W170	=W196		3	=(Z196+Z144+Z92+Z40)/4
197	=U197/T197		4	=W171	=W197		4	=(Z197+Z145+Z93+Z41)/4
198	=U198/T198		6	=W172	=W198		6	=(Z198+Z146+Z94+Z42)/4
199	=U199/T199		8	=W173	=W199		8	=(Z199+Z147+Z95+Z43)/4
200	=U200/T200		12	=W174	=W200		12	=(Z200+Z148+Z96+Z44)/4
201	=U201/T201		TOTAL	=W175	=W201		TOTAL	=(Z201+Z149+Z97+Z45)/4
202								
203								
204								
205								
206								
207								

	AE
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176	
177	
178	
179	
180	
181	
182	
183	
184	
185	
186	
187	
188	=Z188
189	%OF CORRECT
190	QS
191	=(AA191+AA139+AA87+AA35)
192	=(AA192+AA140+AA88+AA36)
193	=(AA193+AA141+AA89+AA37)
194	=(AA194+AA142+AA90+AA38)
195	=(AA195+AA143+AA91+AA39)
196	=(AA196+AA144+AA92+AA40)
197	=(AA197+AA145+AA93+AA41)
198	=(AA198+AA146+AA94+AA42)
199	=(AA199+AA147+AA95+AA43)
200	=(AA200+AA148+AA96+AA44)
201	=(AA201+AA149+AA97+AA45)
202	
203	
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Figure legends

Figure 1: The modified Lashley jumping stand with entrance tunnel visible from the front.

Figure 2: A top view of the jumping stand showing tunnel, entrance, and a set of photographs.

Figure 3: Top view of the matched grating and grey photographs placed on the trap doors.

Figure 4: Averaged percentage of correct responses vs. time (weeks) for the control group.

Figure 5: Averaged percentage of correct responses vs. time (weeks) for the experimental group.

Figure 6: Percentage of correct responses vs. time (weeks) for the motivational cat.

Figure 7: Time to achieve 75% criterion vs time (weeks) for the control group.

Figure 8: Time to achieve 75% criterion vs time (weeks) for the experimental group.

Figure 9: Time to achieve 75% criterion vs time (weeks) for the motivational cat.

Figure 10: Averaged time to achieve 75% criterion vs time (weeks) for the control and experimental groups.

Figures

Figure 1: The modified Lashley jumping stand with entrance tunnel visible from the front.

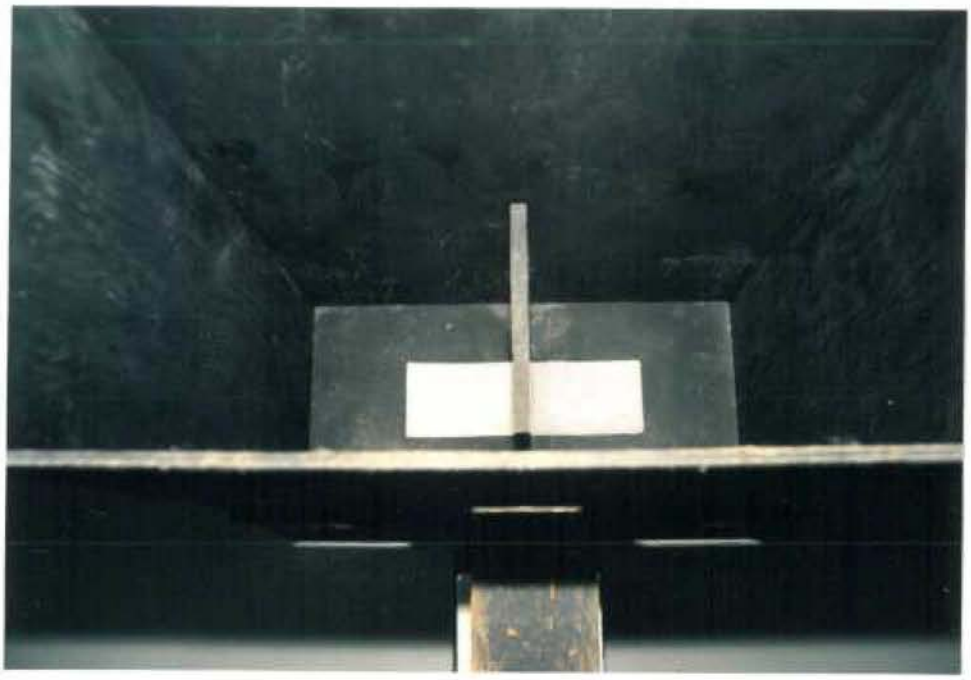


Figure 2: A top view of the jumping stand showing tunnel, entrance, and a set of photographs.

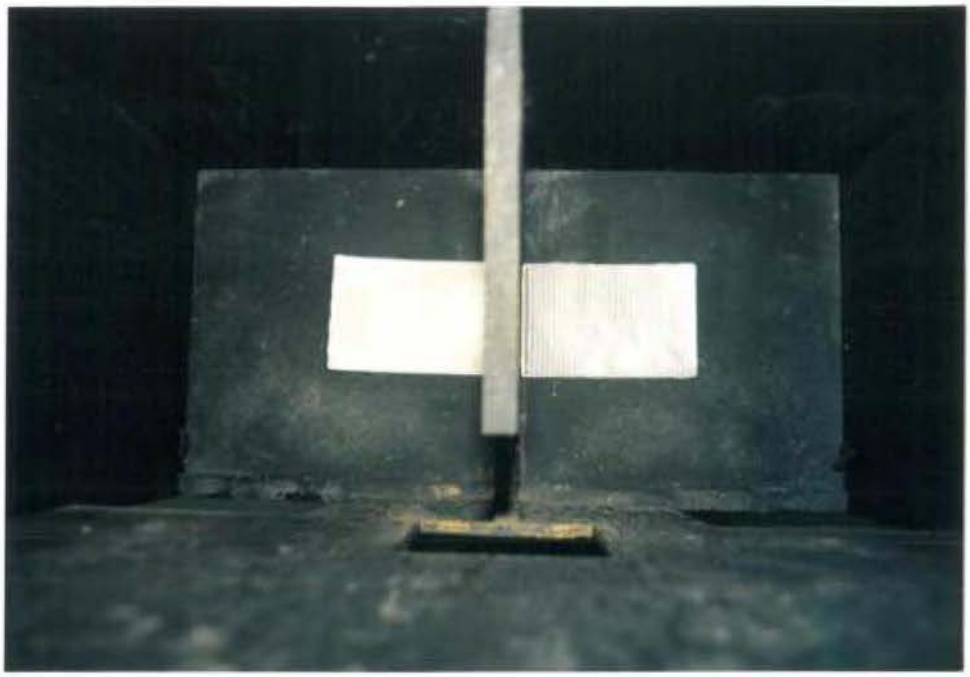


Figure 3: Top view of the matched grating and grey photographs placed on the trap doors.

Fig. 4: Averaged "Control" Data

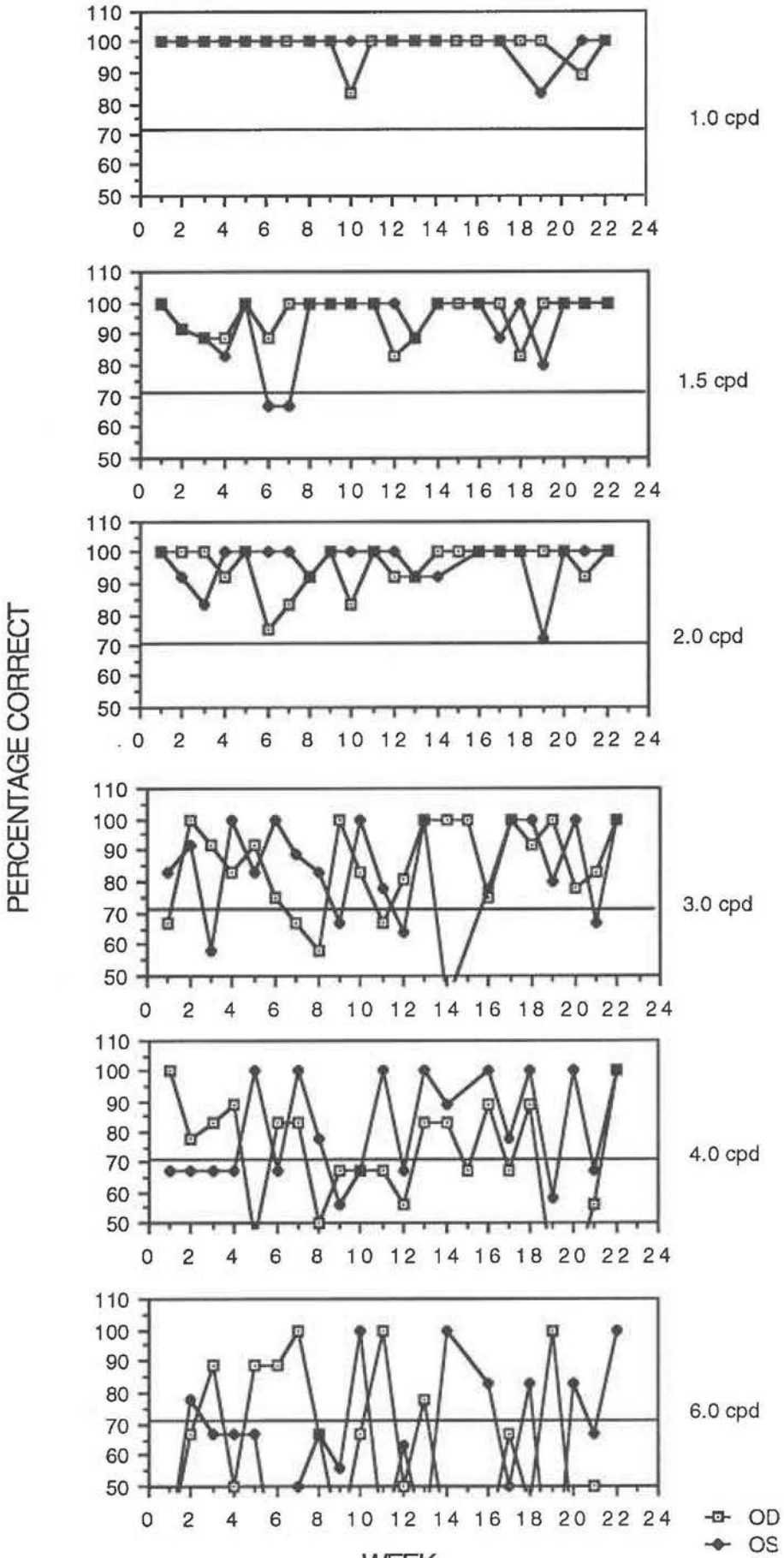


Fig. 5: Averaged "Experimental" Data

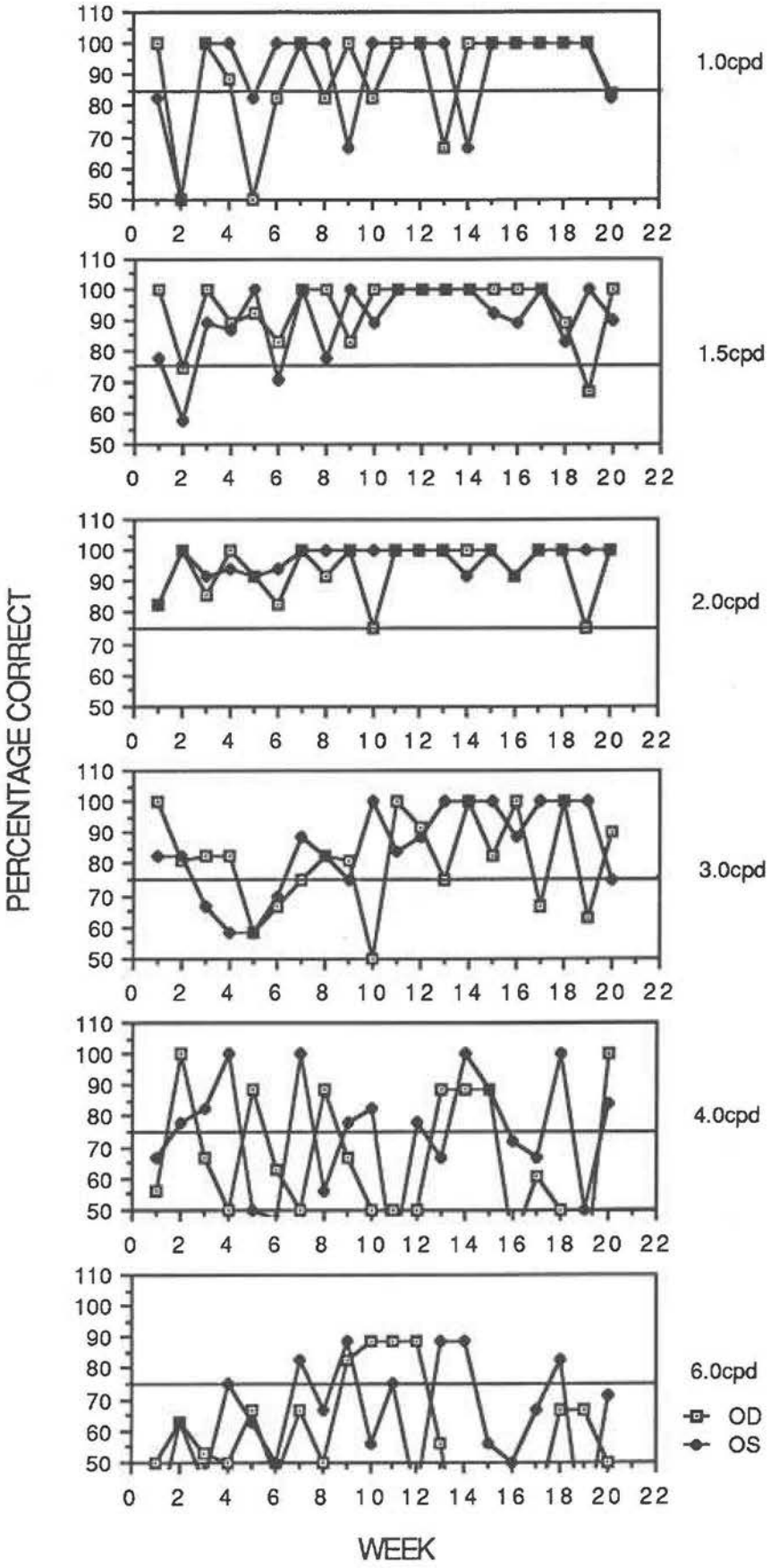


Fig. 6: "Motivational" Data

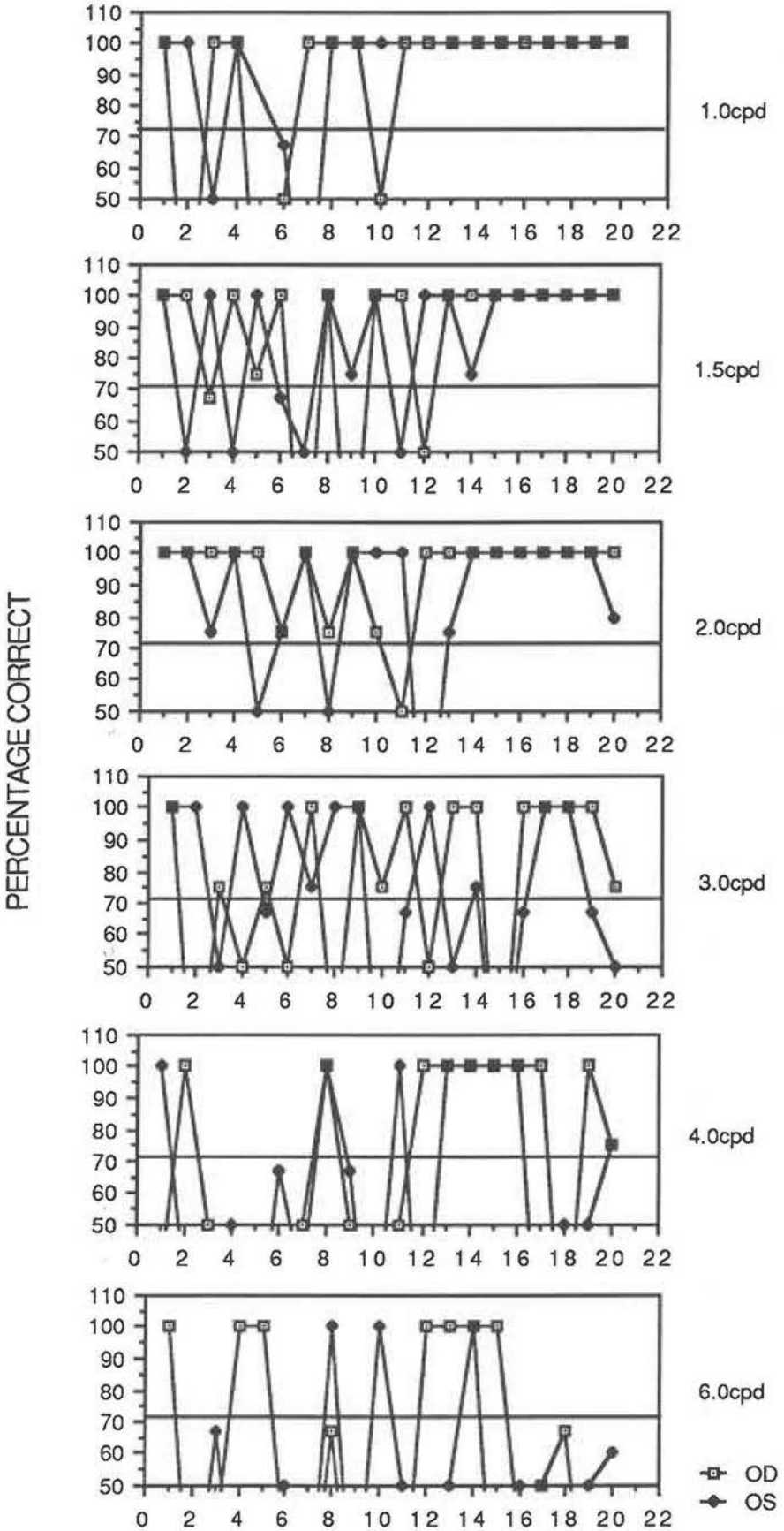


FIGURE 7: TIME TO ACHIEVE 75% CRITERION FOR CONTROL GROUP

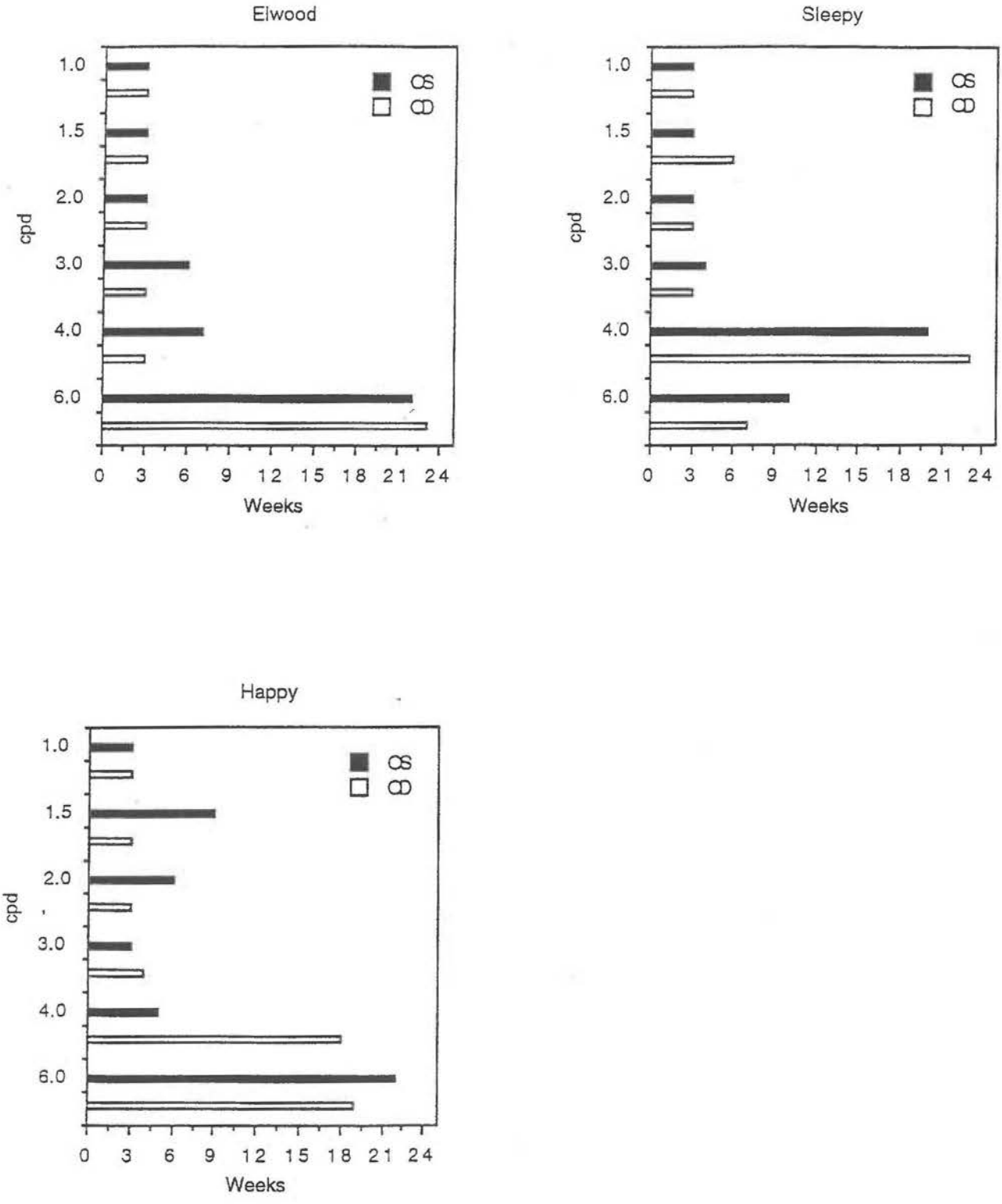


FIGURE 8: TIME TO ACHIEVE 75% CRITERION FOR EXPERIMENTAL GROUP

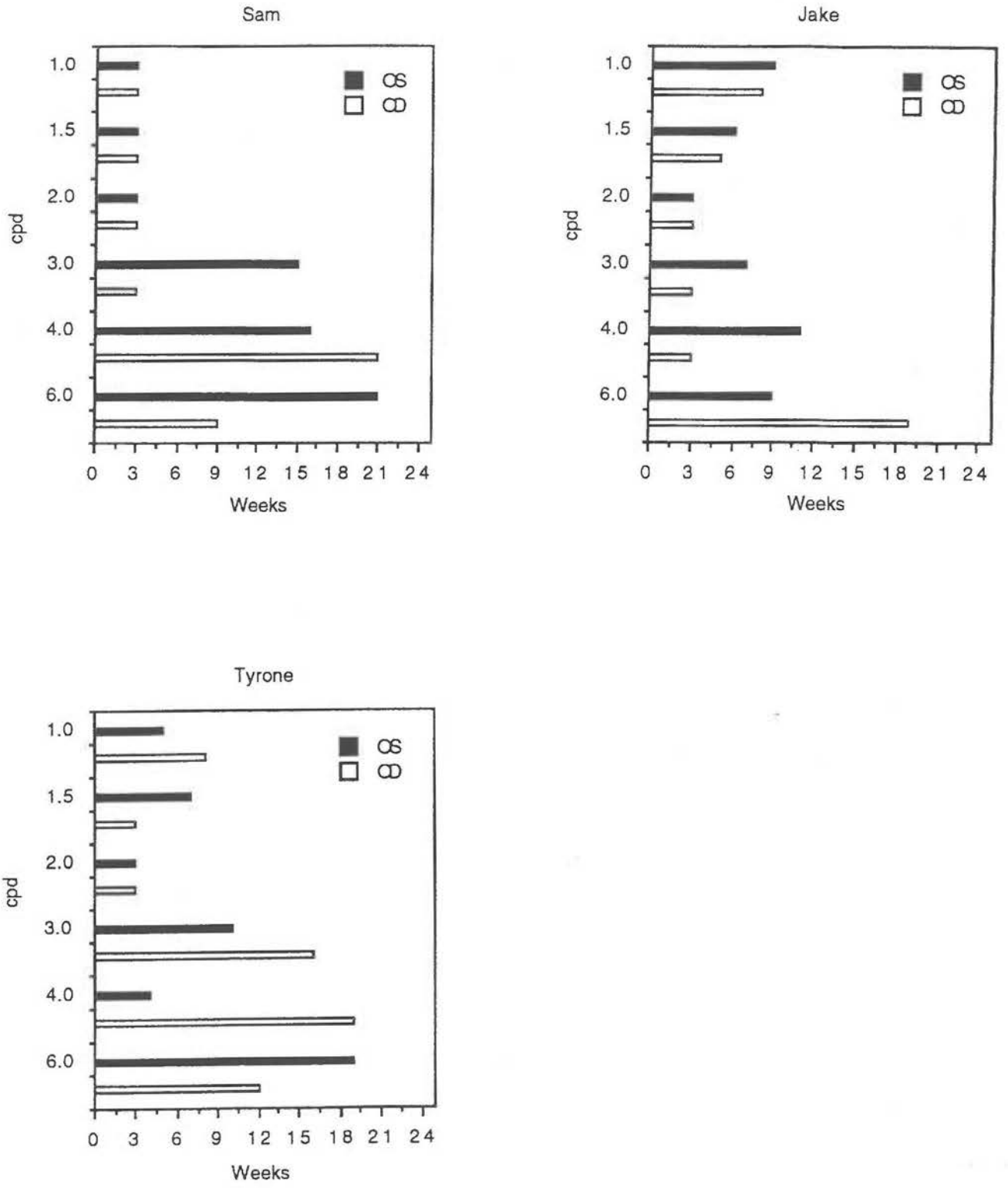


FIGURE 9: TIME TO ACHIEVE 75% CRITERION FOR MOTIVATIONAL GROUP

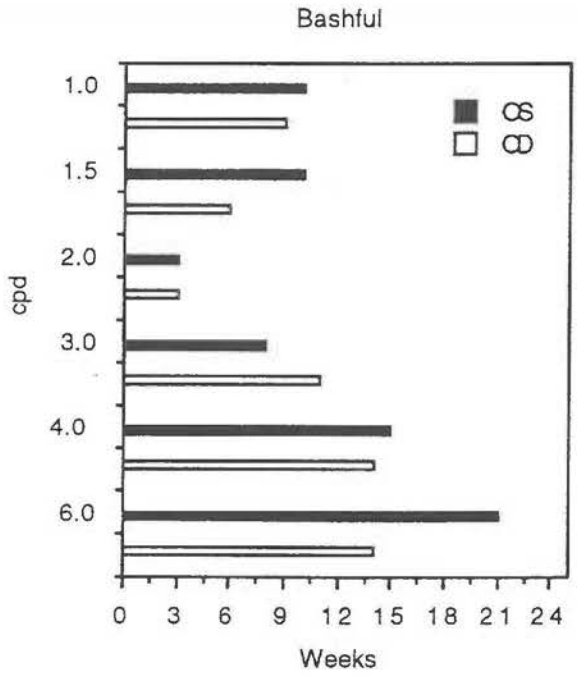
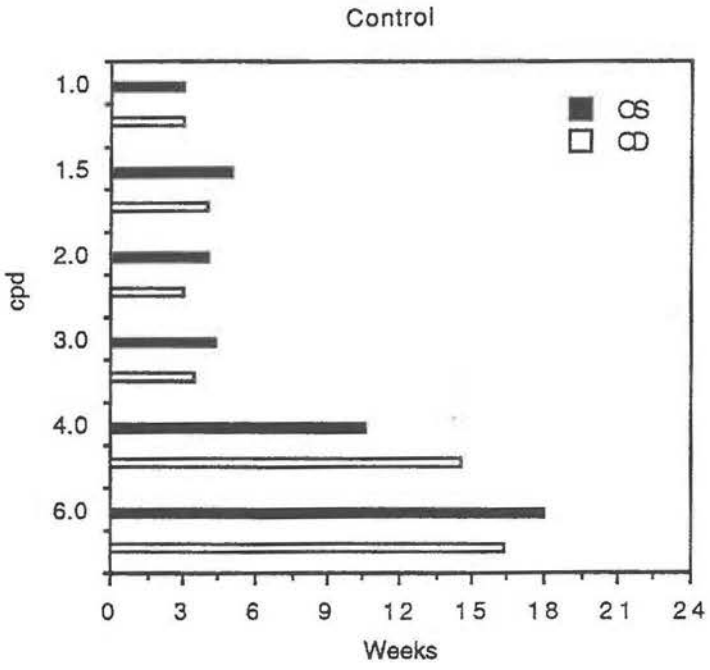
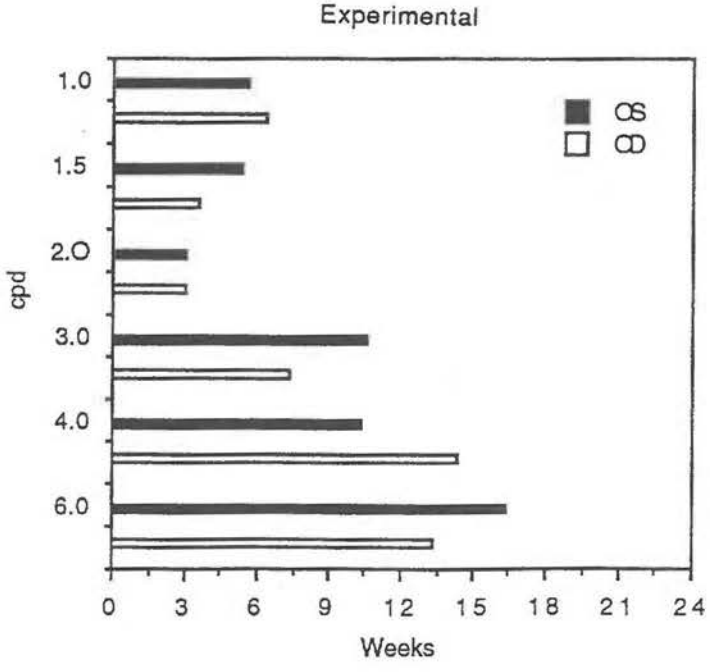


FIGURE 10: AVERAGED TIME TO ACHIEVE 75% CRITERION FOR CONTROL AND EXPERIMENTAL GROUPS





Author Biographies

William L. Hills attended the University of Nevada, Reno receiving a Bachelor of Science degree in pre-medicine. His four years of optometric education included the SUNY internship in vision therapy, as well as a preceptorship at Beale Air Force Base, CA. After receiving his Doctor of Optomtery degree in May of 1992, Bill will become an associate with an established optometrist in Sparks, Nevada. His future goals for private practice in primary care include subspecialties in vision therapy and low vision.

Ning Lin is a special student of optometry from the People's Republic of China, and is a member of the Chinese Medical Association (Ophthalmology), AOSA, AAO and ARVO. He has published 22 papers since 1983, and have two posters presented in ARVO Annual Meeting in 1987 and 1992. [1,11]

Dr. Lin graduated from the Medical School of Sun Yat-sen University of Medical Sciences in China where he received a M.B. degree (M.D. equivalent), then was a resident of ophthalmology in Zhongshan Ophthalmic Center which is the largest ophthalmic research and eye care facility in China. From 1986 to 1987, he worked and studied as a Visiting Assistant Professor of Ophthalmology in the University of Texas Southwestern Medical Center at Dallas. His interest in clinic was corneal transplantation and IOL implant. Four research projects [1-8] have been done during one and half years. After he returned to China, he developed a tissue culture lab in the Zhongshan Ophthalmic Center and cultured corneal epithelial, corneal endothelial, lens epithelial, and retinal pigment epithelial cells *in vitro*. From 1987, cytotoxicity of drugs such as neomycin, gentamicin, polymyxin B and amphotericin B was studied using cultured corneal epithelial and endothelial cells [9-11]. His investigation was the first report in studying the cytotoxicity of the drugs to corneal epithelium and endothelium *in vitro*. In September 1989, he was awarded a graduate fellowship from Pacific University College of Optometry and came to study optometry. He would like to continue his research career in vision science after graduation.

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Nancy Lee Barnard received her Bachelor of Science degree in Visual Science from Pacific University, after completing the course work at Valley City State University in 1989. She will receive her Doctor of Optometry degree from Pacific University College of Optometry in May 1993.

Nancy's future intentions are to enter private practice in the western United States practicing full scope optometry with special interests in vision therapy, sports vision, and multidisciplinary practice.