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Abstract

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THE EFFECTS OF MAGNIFICATION ON NYSTAGMUS AND VISUAL ACUITY

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Introduction

Nystagmus is defined as an involuntary, rhythmic oscillation of the eyes. It can be divided into two major categories, pendular and jerk. Pendular nystagmus is characterized by approximately equal movements in both magnitude and velocity in each direction. Pendular has been further categorized as pure, asymmetric, or pendular with a correcting saccade.[1] Jerk nystagmus consists of a slow pursuit phase followed by a rapid saccadic phase. The nystagmus is named for the direction of the fast phase. Jerk nystagmus can be further classified into two basic subtypes, unidirectional and bidirectional.[1] It is not uncommom to find a mixture of the two types dependent on the direction of gaze.

Gay, Newman, Keltner and Strouds'[2] classification system of nystagmus includes physiological, sensory deprivation, and motor imbalance nystagmus. Physiological nystagmus includes endpoint, optokinetic, and vestibular. Ocular albinism, achromatopsia, blindness, latent oculomotor and miner's nystagmus are all considered sensory deprivation types. The final category of motor imbalance nystagmus includes voluntary, paretic and congenital nystagmus.

All efficient visual systems are characterized by some degree of physiological nystagmus. However, if the magnitude of this nystagmus is excessive, it leads to a decrement in performance.[3] The usual symptoms include decreased visual acuity, a cosmetic disturbance, and in the case of albinism and achromatopsia an increased photophobia.[4] The reduced visual acuity is the greatest deterrent of normal function. It is this decreased visual acuity that this study wishes to investigate regardless of the classification or etiology of the nystagmus.

There have been numerous strategies presented in the literature which attempt to combat this problem. Regardless of the technique used, the goal is to increase visual acuity by dampening the nystagmus.[2] The various treatments include surgery, prism therapy, after-image treatment, red lens, contact lenses, occlusion combined with cycloplegics, barbiturates and magnification.

Many of these strategies utilize the fact that a neutral point or null region exists in many nystagmus patients. It is within this null region that the nystagmus is decreased in amplitude and frequency resulting in the attainment of maximun visual acuity. Untreated patients often discover and utilize this region with a head tilt or a preferred direction of gaze. It is the intent of these techniques to place the eyes in this null region without the cosmetically displeasing head tilt.

Surgical techniques were introduced by two independent authors, Kestenbaum[5] and Anderson[6] in 1953. The objective of surgical treatment is to relocate the eyes so that the patients null point coincides with the straight ahead position. This goal is acheived by simultaneously resecting and recessing the horizontal recti muscles in order to move the eyes away from the nystagmus quiet zone or in the direction of the head tilt.[7]

Kestenbaum attempted to imitate the patients head turn by surgical techniques. Therefore when the patient attempted to turn his eyes to the null point it would result in the eyes being turned to the primary position.[5] Anderson theorized that the slow phase muscles were too strong compared to the fast phase muscles. He therefore recessed the strong muscles to restore equilibrium of the four horizontal muscles.[6] In 1954, Goto[8] reported electromyographic results performed on patients with jerk nystagmus. His results showed that the muscles responsible for the jerk phase showed less muscle tone. His surgical technique involved the resection of these "weak" muscles.

Although the theories of Kestenbaum, Anderson and Goto are not in agreement the end results of their surgical procedures are identical.

The reported results of surgical procedures are varying. Anderson[6] and Goto[8] reported improvements in visual acuity and head turn in their patients. Cooper and Edward[9] reported seven cases of surgical treatment of nystagmus. All seven cases showed improvement in head tilt. However, of the fourteen eyes only two showed improvement in visual acuity. Parks[7] utilized a modified Kestenbaum technique on ten patients. Nine of ten showed a significant improvement in head tilt, no evidence of surgically induced strabismus and fairly normal versions. However, no post operative visual acuities were reported.

In addition to the dampening effect observed with a preferred angle of gaze, a reduction of the nystagmus movement has been noted with increased convergence. Prism glasses or prisms combined with minus lenses can be prescribed to produce necessary eye versions and vergences to reduce the nystagmus and eliminate habitual head tilts. Prism glasses consist of several types. Base out prisms before both eyes may be used to stimulate convergence. Prisms of equal amplitude and base direction are utilized to create a pure versional movement. Prisms of equal base direction and unequal amplitude may be used to produce both a version and vergence movement. This final effect may also be accomplished through the use of prisms combined with minus lenses.[10,11,12]

Metzger utilized this program of treatment as early as 1938. More recently, Dell'Osso, with the use of electro-oculography and the infrared eye movement recorder, was able to record the precise angle of gaze in which the nystagmus amplitude of the two eyes was equal and minimized.[13] Dell'Osso's patients reported fewer headaches, greater working distance, improved visual acuity, and less ocular fatigue upon administration of prism glasses. The transition period needed for adjustment from unaided vision to the prism glasses was eventually reduced to one second.[11]

Others reporting success in this area include Sternberg-Raab,[14] Goddé-Jolly[15] and Leonardi and Lischeti.[16]

Treatment of congenital nystagmus utilizing afterimages was demonstrated by Cüppers in Germany as early as 1958. Professor Hans Goldman of Bern, Switzerland perfected the procedure in 1967. Therese Stohler, who worked with Professor Goldman, was responsible for introducing this method of treatment in the United States.[10]

The procedure is as follows. The patient is given an afterimage with an electronic flash similar to a euthyscope. Next, he is made aware of the movement of the afterimage by observation of a blinking light projected on a white screen. Normally, the congenital nystagmus patient is unaware of movements in the environment and so the afterimage helps him appreciate his eye movements.[2] Once this phenomenon has occurred, the patient's task is to find the head position which most dampens the nystagmus. Upon completion of this stage, the patient tries to dampen the nystagmus while maintaining his head and eyes in the primary position.[17]

Stohler reports the following results with six patients. Four of the six showed a decrease in the number of movements per minute while two displayed an increase in the number of movements. All six exhibited an improvement of 1-4 lines of visual acuity. Finally, four of the six claimed subjective improvement in visual activities.[17] No explanation has been given for the unexpected findings of the two patients exhibiting both an increase in nystagmus and improvement of visual acuity.

At Emory University, Stegall reported a red lens therapy used on patients exhibiting strabismic amblyopia combined with latent nystagmus. The non-amblyopic eye was occluded and a narrow band red transmission filter was placed over the eye with the latent nystagmus.

The results have included a decrease in amplitude and frequency of the nystagmus and an increase in visual acuity.[10] The theory involved in this procedure maintains that light passing through the filter stimulates mainly the cone populated areas. Flynn and Dell'Osso believe the nystagmus movement travels from the fovea to the periphery and back to the fovea with a maximum velocity in the periphery and decreasing as it returns to the fovea possibly stopping momentarily at the fovea.[10] This above model could explain the effectiveness of the red lens therapy.

The application of contact lenses to nystagmus patients has produced mixed results. J. A. Holanda De Freitas et. al.[18] report six cases of congenital nystagmus which resulted in a reduction of the amplitude of nystagmus upon fitting of contact lenses. These results were confirmed by ENG recordings. J. Sédan[19] describe two cases of high myopia combined with nystagmus in which vision was improved with contact lenses. However, the effect on the nystagmus was minimal and possibly adverse.

Although earlier studies contraindicate occlusion therapy in the treatment of latent nystagmus, Windsor, Burian and Milojevic,[20] in 1968, achieved positive results when occlusion was combined with 1% cyclopentolate. Visual acuity showed improvement and a reduction in nystagmus occurred. Calcutt and Crook, in 1972, experienced similar results when combining occlusion with 1% atropine.[10]

Barbiturates have been used for the treatment of nystagmus with varying results. Intravenous injection of barbiturates may induce nystagmus yet when the nystagmus is due to brain stem disease, similar treatment may dampen the nystagmus.[21]

It has been reported by Rosenberg and Werner[4] that perhaps the most satisfactory control of the patient exhibiting nystagmus is the use of low vision aids. They indicate that there are three possible interaction effects between nystagmus, visual acuity and magnification. These three interactions are:

1. Magnification may improve visual acuity in the presence of nystagmus.

2. Magnification may have no effect on visual acuity in the presence of nystagmus.

3. Magnification may reduce visual acuity in the presence of nystagmus.

All three of the results can be witnessed in one patient depending on the amount of magnification used.

Rosenberg and Werner theorize that if the visual acuity improves with magnification the low vision is primary and the nystagmus is due to poor fixation abilities. In these cases the increased acuity results in decreased movement which in turn increases the visual acuity further to result in a superproportional gain.

On the other hand, if the nystagmus is primary then the mystagmus is magnified by the device and the visual acuity remains the same or is decreased.

It is this interrelationship between the amplitude of nystagmus, visual acuity and magnification that this study investigated.

Subjects

Four females and one male ranging in age from sixteen to fifty two years participated in this study. They consisted of: two students and a teacher from the Washington State School for the Blind, a lady from Portland contacted through the Low Vision Clinic in Portland, and a lady from Astoria, Oregon contacted through the Low Vision Clinic in Forest Grove. In all cases, the type of nystagmus exhibited was pendular and each subject had experienced the condition since birth. The various etiologies included: albinism, congenital cataracts, and optic atrophy. One of the subjects had previous experience with telescopic aids and another had been previously fit with contact lenses in an effort to dampen the nystagmus and improve visual acuity.

Procedure

The effects of magnification on visual acuity performance of low vision patients with nystagmus was observed through a series of afocal magnifiers. Initial acuities were taken with the subjects best correction in place in the preferred direction of gaze at a distance of ten feet. This was done to evaluate the subject's performance level under typical environmental conditions.

Acuities were taken under standard room illumination utilizing the Feinbloom Distance Acuity Chart. Habitual acuities were measured and recorded for both eyes. Acuities through each of the magnifiers were measured for only the eye demonstrating the best habitual acuity. In the latter instance the eye not being measured was left unoccluded. The magnifiers consisted of the following afocal telescopes: 1.3X, 1.75X, 2.2X, 2.5X, 3.0X, 4.0X, 6.0X, and 8.0X. Acuities were recorded in both Snellen notation and in minutes of arc. The following criteria was utilized in determining acuity level. If one half or more of a line was achieved, credit was given for the entire line. If less than one half of a line was achieved, no credit was given for that line and the previous line was recorded.

To evaluate the effect of magnification on nystagmus, eye movements were measured utilizing electro-nystagmography (ENG). It was done habitually and through each of the magnifiers on only the eye demonstrating the best habitual acuity. Again, both eyes remained open throughout the testing process. The fixation target consisted of a 20/30 illiterate "E" located one meter from the subject. A +1.00D reading cap was placed in front of the magnifiers in an effort to offset the stimulus to accommodation resulting from target proximity.

Nystagmus amplitudes for each of the magnifiers were averaged and recorded in degrees of arc. In order to make the proper transformation from millimeters of amplitude as indicated on the paper recorder to degrees of arc, a calibration was required prior to testing for each individual. This was needed due to variability in electrode placement, electrical potential differences and individual fixation characteristics. The calibration targets consisted of two red lights 4mm in diameter placed 81cm apart alternately flashing at one second intervals. The subject was instructed to fixate the flashing lights and his/her eye movements were recorded. A degree/millimeter calibration could then be derived by comparing the linear eye movements recorded to the angular subtense of the targets.

Unfortunately, electronystagmography was only performed on three of the five subjects. While testing the three people from the Washington State School for the Blind, an equipment malfunction occurred. This plus the limited availability of these subjects, accounts for the fact that electronystagmographic data was only obtained for one of them.

Results

The results are tabulated in Table I and Figures I-VI. For each subject, expected visual acuity achieved through each of the magnifiers is compared to the actual measured visual acuity. The ratio of these two findings denotes supraproportional gain, if any, that has occurred. Supraproportional gains ranged from 1.5 to 0.8. Two of the five subjects achieved supraproportional gains. The other three achieved linear gains to slightly less than expected increases in visual acuity. Subject O. J. achieved exceptional gains with 1.3X and 1.75X, slightly higher than linear gains with 2.2X, 2.5X, 4.0X, and WITH THE 3.0X and 6.0X displayed linear gains in visual acuity. Subject B. H. showed slightly higher than linear gains with 1.3X, 1.75X, 2.2X, 2.5X, linear gains with the 3.0X and 4.0X and slightly less than linear gain with the 6.0X. Subject J. B. showed linear gains with 1.75X, 2.5X, and 4.0X and less than linear with the 2.2X. J. B. expressed difficulty in utilizing the magnifiers which resulted in no measured visual acuity with the 1.3X and 6.0X magnifiers. Subject D. H. showed less than linear gains for all magnifiers except 2.5X which gave him a slightly better than linear gain. Subject D. E. had a slightly less than linear gain with 1.3X, 2.2X, 2.5X. She had linear gains with 4.0X and 6.0X and slightly greater than linear gains with the 1.75X. These patterns can be observed in Figures I-V.

The three subjects on which the ENGs were run all showed decreases in nystagmus amplitude with all the magnifiers as compared to the habitual. O. J. showed a rapid decrease in amplitude with the 1.3X and 1.75X and a slight decrease until a minimum was reached at 3.0X. A slight increase in amplitude was measured with 4.0X, 6.0X and 8.0X as compared to the minimum. B. H. experienced two instances where a rapid decrease was noted — at 1.3X and 3.0X with a minimum at 3.0X. J. B. showed a rapid decrease with the 1.3X and a minimum amplitude with the 1.75X. The patterns of these amplitudes are illustrated in Figure VI.

Table I.

		O.J.	B.H.	J.B.	D.H.	D.E.
	Age	52	33	16	16	32
Habitual Visual Acuity						
	OD	6.0'	4.0'	70.0'	10.0'	12.0'
	OS	6.0'	4.0'	10.0'	8.0'	16.0'
	OU	6.0'	4.0'	10.0'	8.0'	10.0'
Habitual Nystagmus Amplitude		6.7°	3.1°	4.9°	—	—
1.3X	Expected VA	4.6'	3.1'	7.7'	6.2'	9.2'
	Achieved VA	3.0'	2.5'	—	8.0'	10.0'
	VA Gain	1.5	1.2	—	0.8	0.9
	Nystagmus Amp.	4.6°	2.6°	3.3°	—	—
1.75X	Expected VA	3.4'	2.3'	5.7'	4.6'	6.8'
	Achieved VA	2.5'	2.0'	6.0'	6.0'	6.0'
	VA Gain	1.4	1.2	1.0	0.8	1.1
	Nystagmus Amp.	3.3°	2.2°	2.9°	—	—
2.2X	Expected VA	2.7'	1.8'	4.5'	3.6'	5.4'
	Achieved VA	2.5'	1.67'	6.0'	4.0'	6.0'
	VA Gain	1.1	1.1	0.8	0.9	0.9
	Nystagmus Amp.	3.3°	2.4°	3.3°	—	—
2.5X	Expected VA	2.4'	1.6'	4.0'	3.2'	4.8'
	Achieved VA	2.0'	1.5'	4.0'	3.0'	6.0'
	VA Gain	1.2	1.1	1.0	1.1	0.8
	Nystagmus Amp.	3.2°	2.0°	3.4°	—	—
3.0X	Expected VA	2.0'	1.3'	3.3'	2.7'	4.0'
	Achieved VA	2.0'	1.25'	—	—	—
	VA Gain	1.0	1.0	—	—	—
	Nystagmus Amp.	2.9°	1.2°	—	—	—
4.0X	Expected VA	1.5'	1.0'	2.5'	2.0'	3.0'
	Achieved VA	1.3'	1.0'	2.5'	2.5'	3.0'
	VA Gain	1.2	1.0	1.0	0.8	1.0
	Nystagmus Amp.	3.6°	1.5°	3.3°	—	—
6.0X	Expected VA	1.0'	0.67'	1.67'	1.33'	2.0'
	Achieved VA	1.0'	0.75'	—	1.5'	2.0'
	VA Gain	1.0	0.9	—	0.9	1.0
	Nystagmus Amp.	3.5°	1.5°	—	—	—
8.0X	Expected VA	0.75'	—	—	—	—
	Achieved VA	—	—	—	—	—
	VA Gain	—	—	—	—	—
	Nystagmus Amp.	3.3°	—	—	—	—

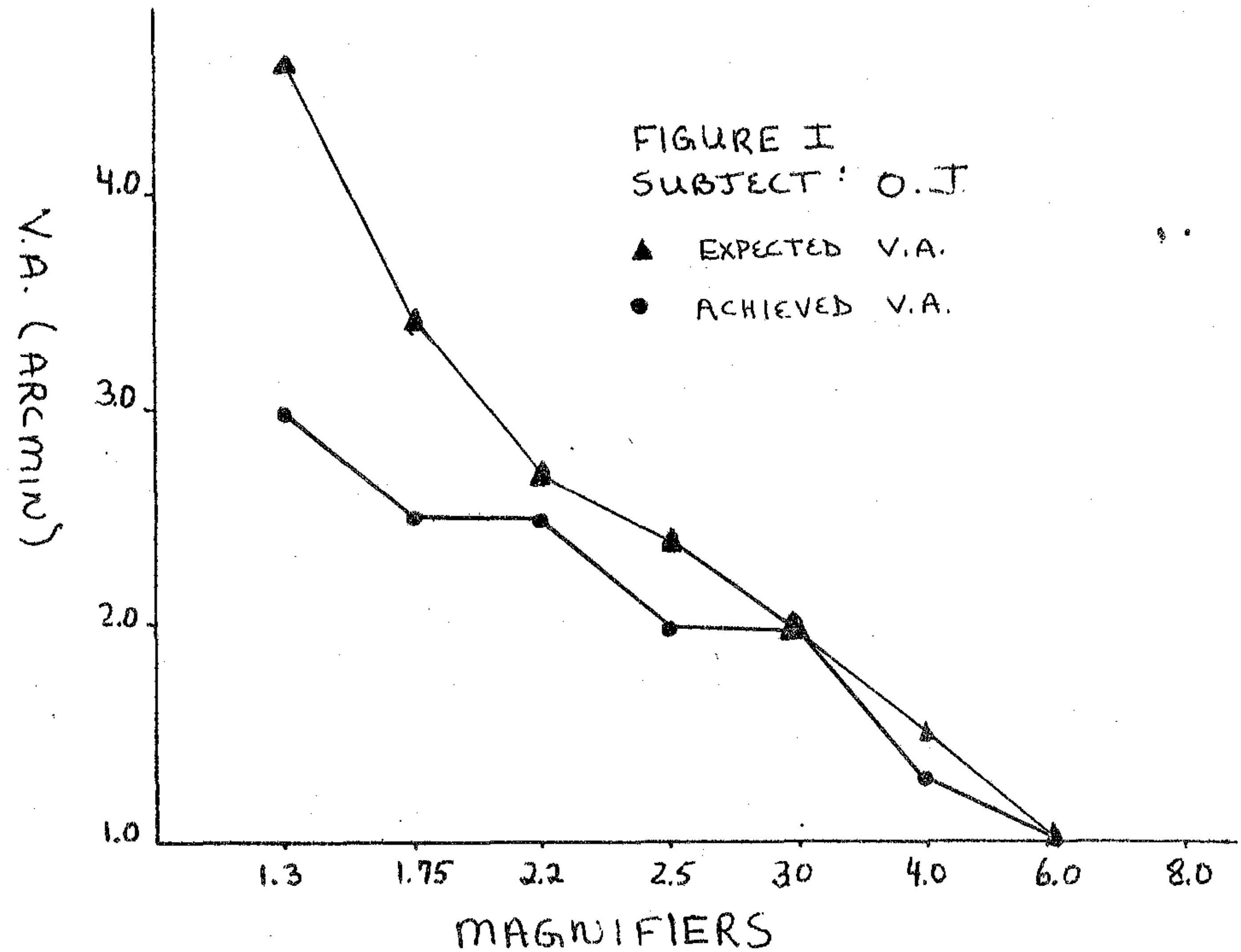
FIGURE I
SUBJECT: O.J.
EXPECTED V.A.
ACHIEVED V.A.
V.A. (ARCMIN)
4.0
3.0
2.0
1.0
1.3
1.75
2.2
2.5
3.0
4.0
6.0
8.0
MAGNIFIERS

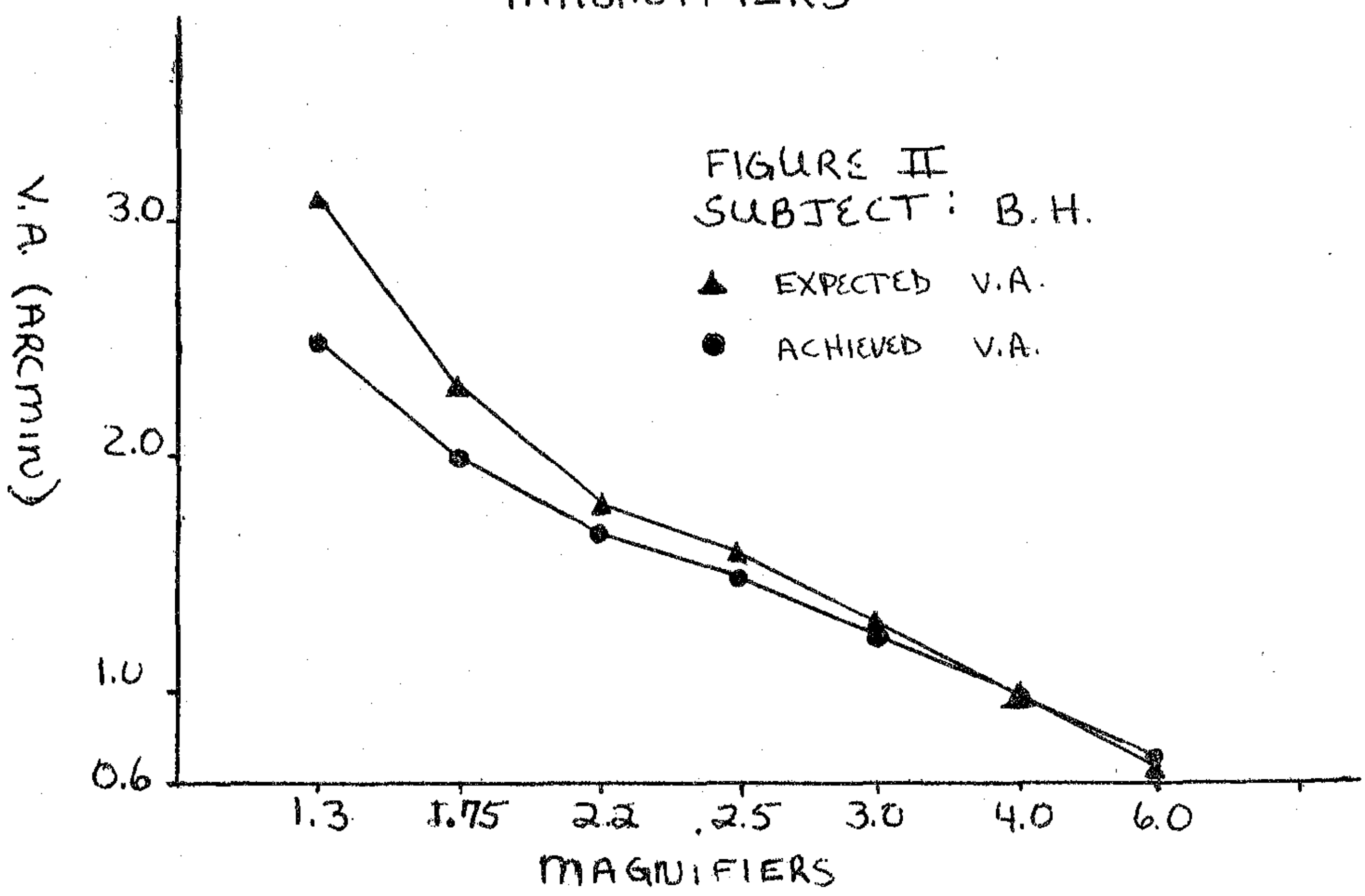
FIGURE II
SUBJECT: B.H.
EXPECTED V.A.
ACHIEVED V.A.
V.A. (ARCMIN)
3.0
2.0
1.0
0.6
1.3
1.75
2.2
2.5
3.0
4.0
6.0
MAGNIFIERS

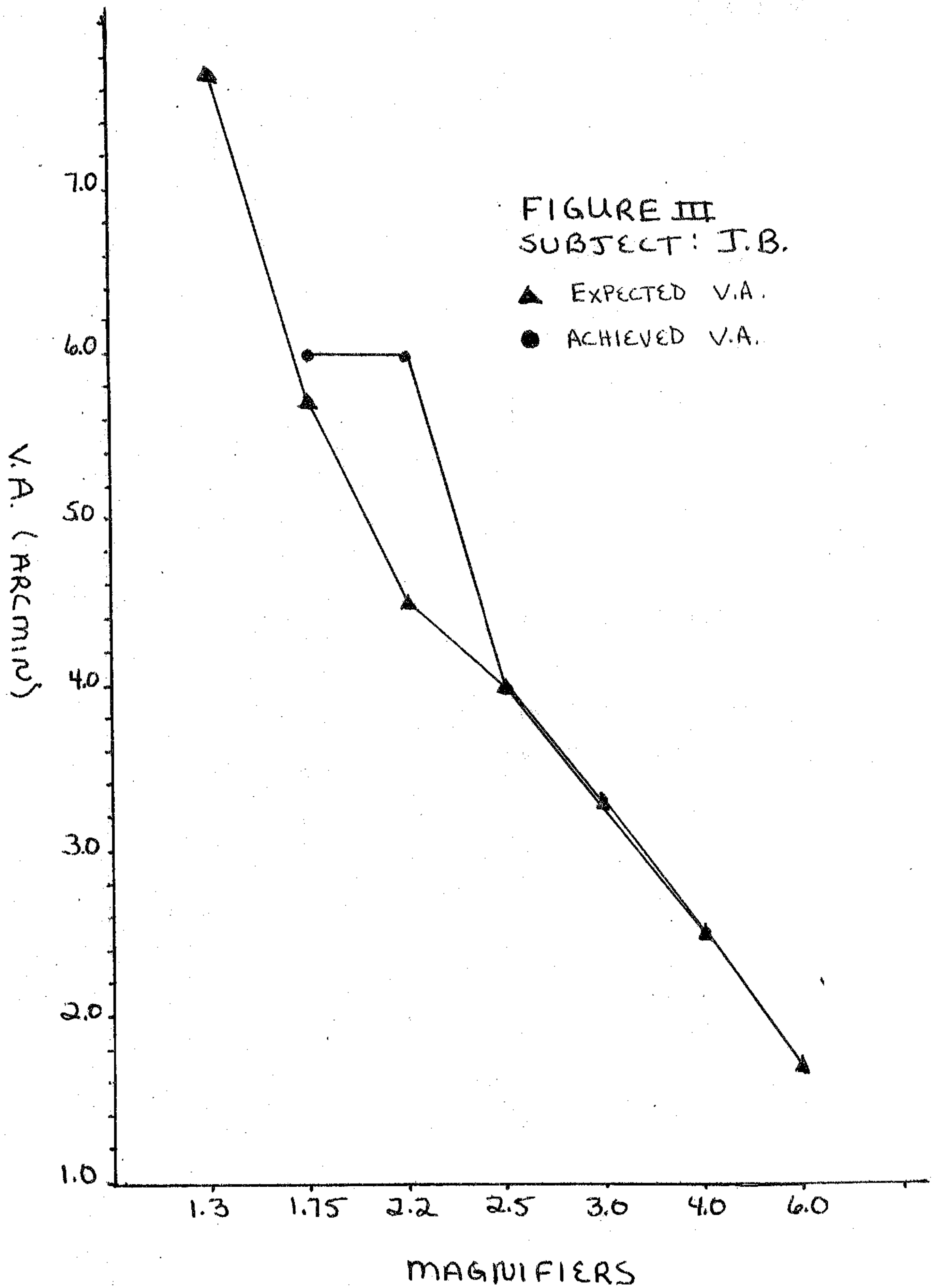
FIGURE III
SUBJECT: J.B.
EXPECTED V.A.
ACHIEVED V.A.
V.A. (ARCMIN)
7.0
6.0
5.0
4.0
3.0
2.0
1.0
1.3
1.75
2.2
2.5
3.0
4.0
6.0
MAGNIFIERS

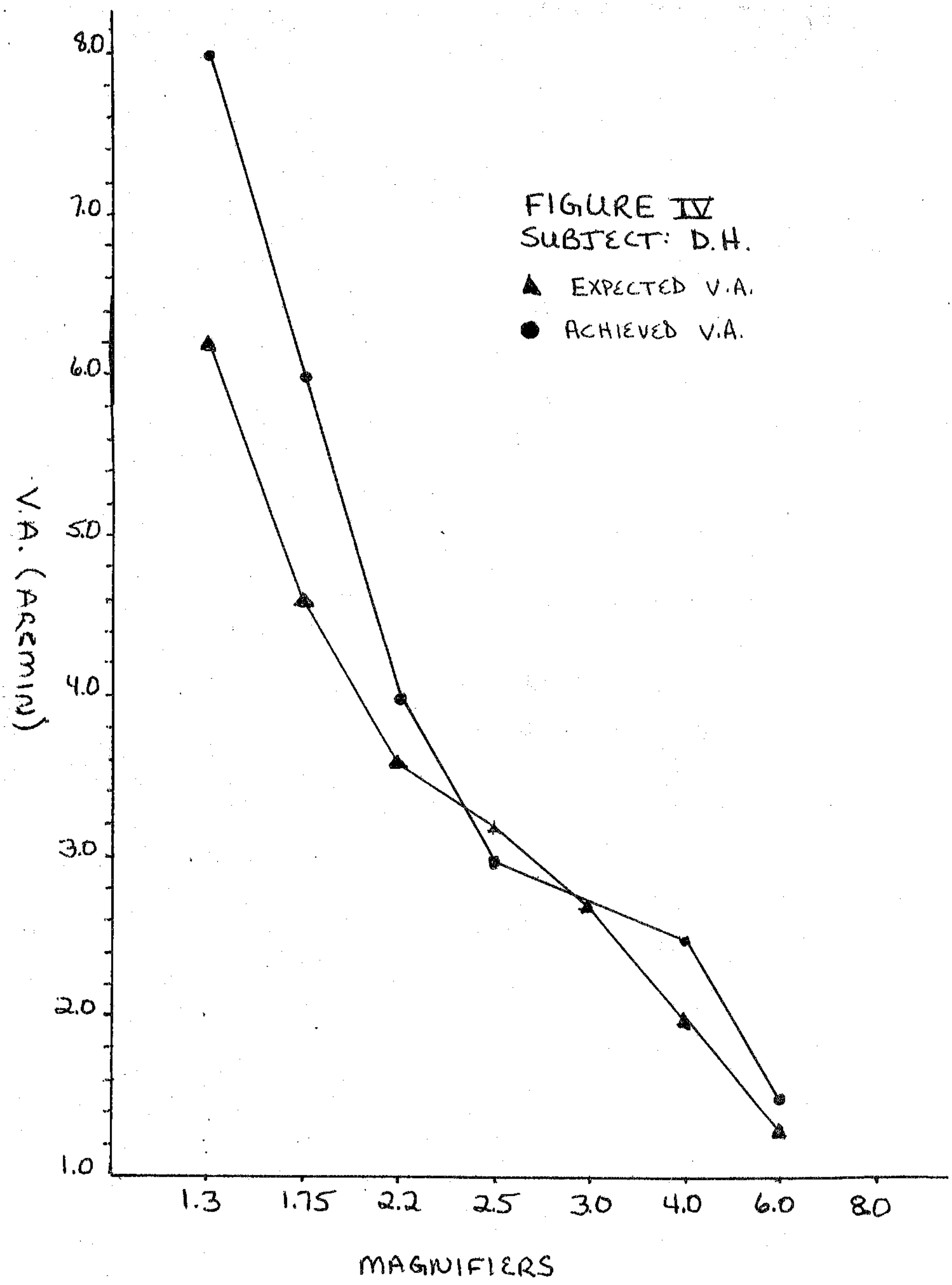
FIGURE IV
SUBJECT: D.H.
EXPECTED V.A.
ACHIEVED V.A.
V.A. (ARCMIN)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
1.3
1.75
2.2
2.5
3.0
4.0
6.0
8.0
MAGNIFIERS

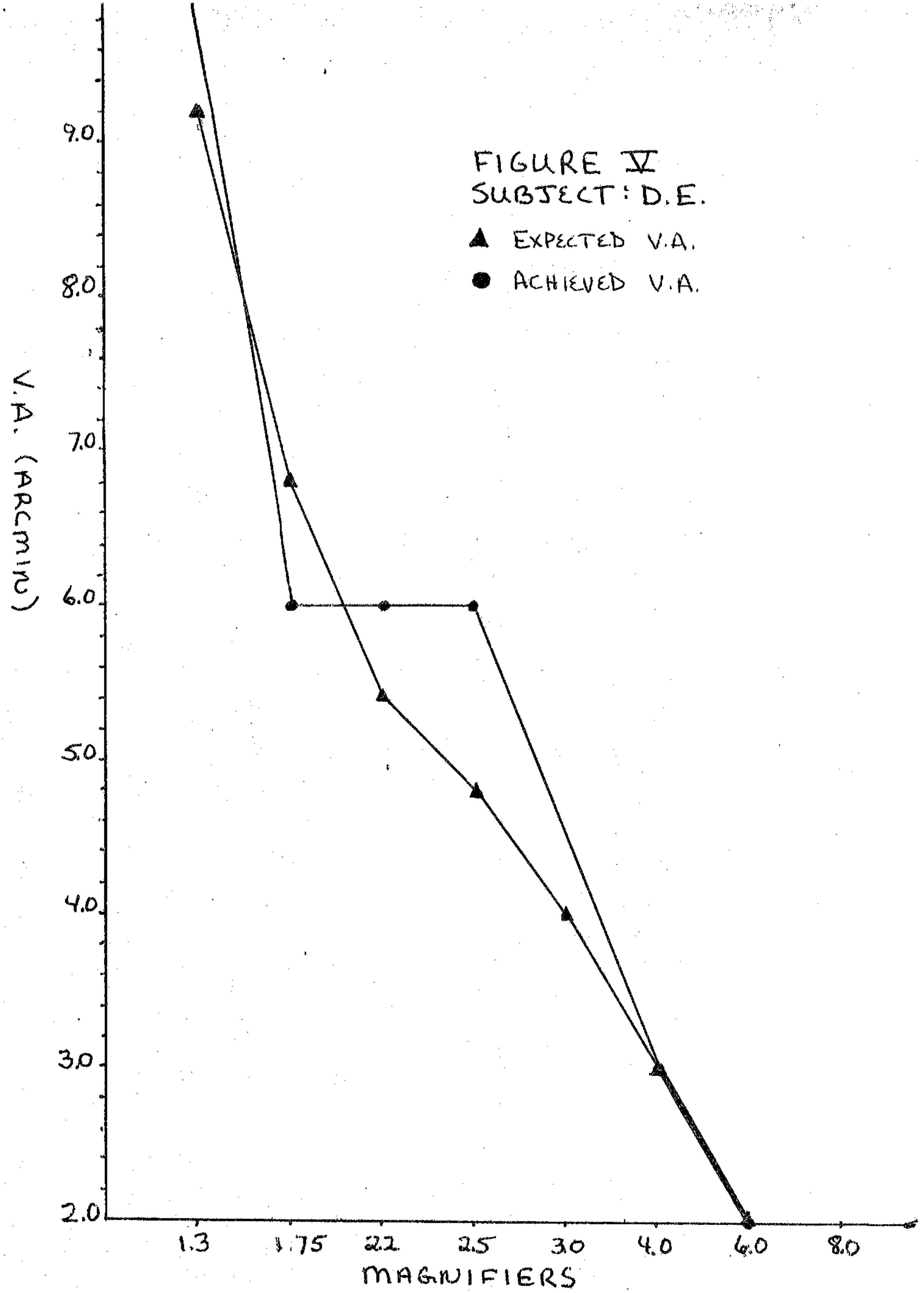

FIGURE V
SUBJECT: D.E.
EXPECTED V.A.
ACHIEVED V.A.
V.A. (ARCMIN)
9.0
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.3
1.75
2.2
2.5
3.0
4.0
6.0
8.0
MAGNIFIERS

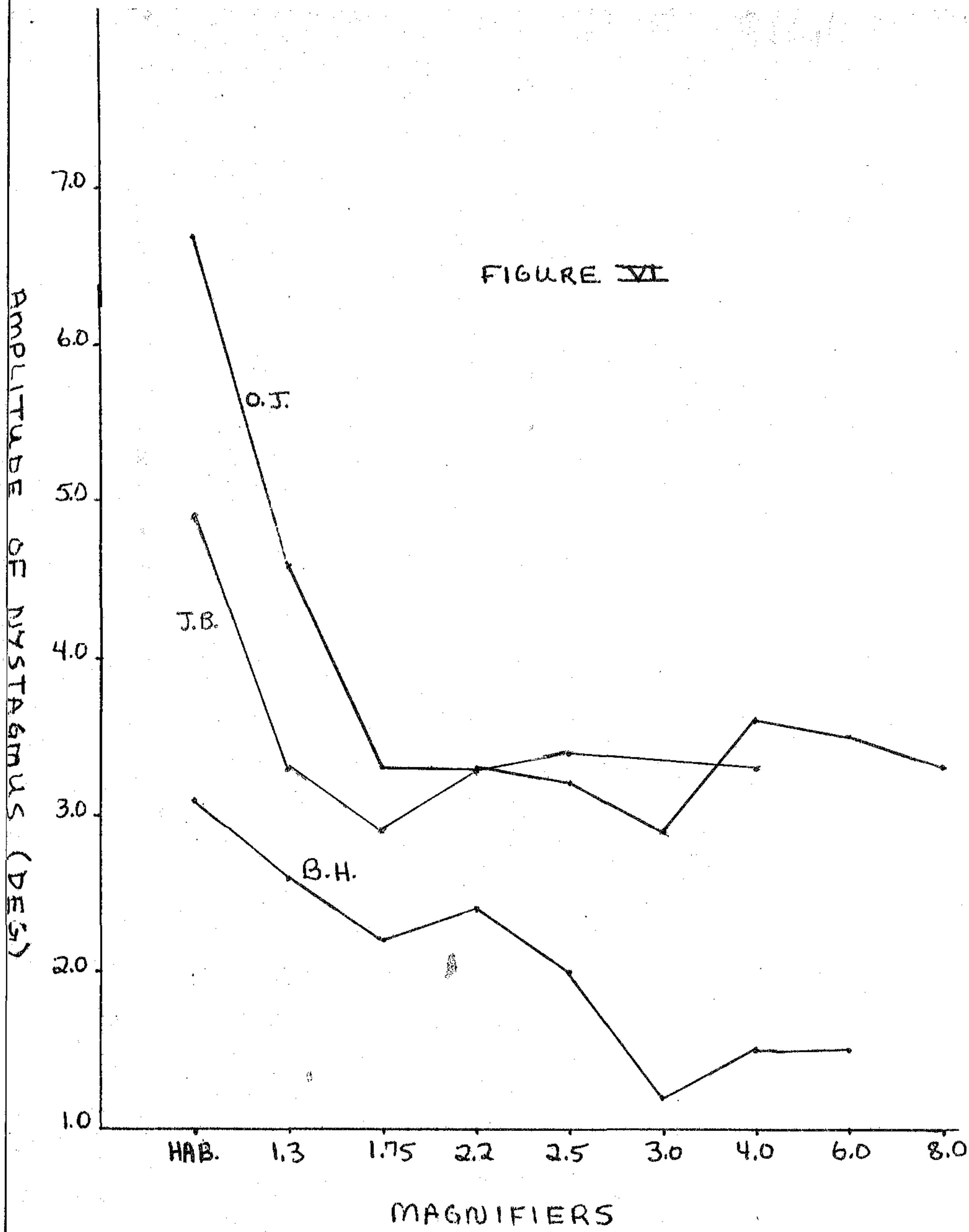
FIGURE VI
AMPLITUDE OF NYSTAGMUS (DEG)
7.0
6.0
5.0
4.0
3.0
2.0
1.0
O.J.
J.B.
B.H.
HAB.
1.3
1.75
2.2
2.5
3.0
4.0
6.0
8.0
MAGNIFIERS

Discussion

The results of this study indicate that a minimal amplitude of nystagmus does not necessarily correlate with the best visual acuity achieved or even the greastest increase in visual acuity. However, in each instance where amplitudes were recorded, it was observed that the greatest gain in acuity was accompanied by a large dampening of the nystagmus amplitude. The greatest amount of supraproportional gain for all of the subjects was achieved with magnifiers of 2.5X or less. Thus, the use of such an aid as a contact lens telescope might prove quite beneficial to such patients. Also, it was shown that a subject's ability to achieve supraproportional gains in acuity cannot be determined with one magnifier. The cause of these differential responses to various magnifiers within one subject can only be speculated. It could possibly be due to the difficulty involved in using the magnifiers of higher powers. Also, it may be the result of only measuring one series of acuities with each patient. Future longitudinal studies could answer whether such results are reliable or merely artifactual. Regardless of such results, in a clinical situation several different magnifiers should be used in the low vision evaluation of nystagmus patients.

In this study, it was found that age was not a limiting factor in achieving supraproportional gains in acuity as the most dramatic increases were obtained by the oldest subject. Similarly, the duration of the nystagmus did not impair a subject's chances of achieving a supraproportional gain in acuity. Finally, it should be noted the greatest increase was achieved by the only subject with previous magnifier experience.

Unfortunately, the size of this sample is quite small and it would be unfair to extrapolate these results to the entire population. However, since the gain in visual acuity for one of the subjects was so dramatic, it is felt this area of research should be investigated further.

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