INTRODUCTION

Hoffman and Bielchowsky (1900) described two components of the vergence system. The first component which is the fast vergence mechanism, was thought to be responsible for changing the angle of binocular vergence from one amplitude to another. The second component was the slow tonic mechanism which sustained a given amplitude to binocular vergence for longer time periods.[1] Jones described the two as transient fusion initiating phase and a fusion sustaining component. The transient response is driven by the fast controller, the sustained exhibits the personality of a slow controller.[2] The fast and slow vergence mechanisms are classified by their gain and time constant. The fast mechanism having a longer time constant and low gain, whereas the slow has a shorter time constant and higher gain.[3] The slow fusional vergence does not respond directly to retinal image disparity, but responds to the output of the fast mechanism. The fast mechanism responds directly to the retinal disparity.

The accommodative system sends signals to the vergence system by means of the accommodative convergence(AC), and the convergence accommodation(CA) sends signals from the vergence to the accommodative system. The AC and CA are shown to originate from the optical reflex accommodation and disparity vergence controllers. These fast controllers are followed by tonic adapters located after the cross link.[4] Figure 1 shows the entire system. The accommodative loop (upper) is composed of the blur detector (eye-brain), ciliary muscle and lens. The vergence system (lower) is composed of the extraocular muscles, fast vergence mechanism(F.V.A.), and the slow vergence mechanism (S.V.A). The S.V.A. has both convergence and divergence associated with it. The location of the crosslinks between the two systems has been challenged in the past. Schor (1979) showed that the AC enters after the F.F.V. and before the ouput from the accommodative controller.[1]

Another way to analyze the disparity components is by the fixation disparity curve. Shor suggested that the flat central portion of the curve is a function of a properly operating S.V.A., and the width represents the operating range of the S.V.A. The more verticle ends of the Fixation Disparity curve are a function of the F.V.A.[5] One must keep in mind that a response defect in the F.V.A. will effect the S.V.A. as the location of the S.V.A.is directly after the F.V.A.[3,1] The importance of fusion contour in performing Fixation Disparity has been shown not to be a factor when testing your "normal" patient, but a symptomatic patients' curve will tend to rotate around the x axis when fusion contours are not used.[3]

Most recent studies have proposed the idea of a slow accommodation adaptation (S.A.A.) response in the upper loop of the system. No studies have proved the existance of such a system nor have they analyzed where it comes into play, and what strength and time constant is "tagged" onto it. The next few paragraphs will be spent anayzing both the accommodative and vergence systems, with and without a S.A.A. and looking at the consequences of the AC entering at different places of the system. We do not need to analyze the implications of the CA as we have opened up (broken)

the feedback loop in the vergence system therefore it will not play a role in this experiment, or in the following discussion.

Figure 2 represents the AC being entered into the vergence system before the S.V.A. If the lower system were opened up, then by initiating a minus lens before the patient there would be an initial increase in the esophoric posture. If the patient was allowed to keep viewing through the lens there would be an even greater increase in esophoria as the S.V.A. would be allowed to "turn on" and the response would be amplified. We would expect the same response with a plus lens except exophoria would be found. Figure 2A demonstrates this phenomena.

Figure 3 shows the AC entering after the S.V.A. Since the AC is bypassing the S.V.A. there would be no amplification. Upon initiating lens to this system, you would achieve the initial increase in phoric posture, yet with adaptation time there would be no further increase or decrease, this is shown in figure 2A.

Observing the system as if a S.A.A. exists creates a more complex picture. Once again we must consider the AC and where it belongs. Another consideration is the strength of the S.A.A. versus the S.V.A. Figure 4 depicts the scene as if the AC enters before the S.V.A., if that is the case then figures 4A, 4B, and 4C portray the actions possible depending on the strength of the S.A.A. Figure 4A demonstrates what would occur if the S.A.A. and the S.V.A. were equal in gain and time constant. With the addition of plus or minus lens there would be an initial increase in the phoria, but after a period of time there would be no further changes in phoric posture. This is because the two systems would actually cancel each other's actions out. If the S.A.A. was higher in gain and/or shorter time constant the initiation of a lens would produce the same initial increase in phoria as showed before, but after an adaptation period the phoria would actually decrease. The reasoning behind this being that the S.A.A. would eventually take the load off the system due to the S.V.A. being weaker, therefore causing the phoria to decrease. This is shown in figure 4B. If the S.A.A. was less strong than the S.V.A. the opposite case would occur. With the introduction of lens the phoria would show the initial increase, but after adaptation to the lens was allowed, the phoria would increase even more. The S.A.A. would not be able to handle the load of the system and would rely on the S.V.A. therefore causing the phoria to increase. This is represented in figure 4C.

Figure 5 demonstrates the system with the S.A.A. but now the AC is entering in after the S.V.A. Now the system would respond as if the S.V.A. were not even there. Introducing a lens into this system is therefore much less complex. As in all the other cases there would be your initial increase (esophoria with minus lens and exophoria with plus lens), but after time elapses the phoria would actually decrease as the S.A.A. would take the load off of the system, this is depicted in figure 5A.

The purpose of this pilot study is to further investigate the location of the AC in relation to the S.V.A. At the same time I shall attempt to determine if a S.A.A. exists, how it works and fits in with the rest of the system in both asymptomatic and symptomatic individuals.

METHODS

Subjects were randomly selected between the ages of 19 and 30. A brief history was taken on each individual concerning and symptoms such as headaches, asthenopia, refractive error and if they do wear correction, how often. All patients were correctable to 20/20 by Snellen Acuity.

Von Graphe phorias were performed through their spectacle prescription with a Rx Master Phoropter and Risley Prism. A 6 PD BD was placed in front of the OD and used as the dissociating prism. 15 PD BI was placed before the OS. The target was a 20/20 block of letters. The patient was told to focus on the upper block of letters and to keep them clear. BO was dialed into the Risley Prism until the patient reported alignment (that number was recorded), more BO was dialed in until alignment was broken, then BI was dialed until alignment was achieved again(this number was also recorded). Three such like recordings were taken for each trial. An average of the three were used as the final phoria reading for that trial. These phorias were taken at distance with a -1.00 trial lens held over the OD, and the phoria was taken quickly (within 5 sec), not allowing the patient to adapt to the lens. Fifteen seconds were given between each of the three readings. A -1.00 was then dialed into the phoropter and the patient allowed to adapt for one minute. Three readings were taken at 20 second interval between recordings. The exact same recordings were taken at near (40 cm.) with direct illumination on the target.

Accommodative facility was then tested binocularly with plus and minus 1.50 flippers. The patient was told to focus on the 20/20 line of letters, and to report when clarity was achieved after each lens insertion. Ten cycles were performed (one cycle being plus to minus to plus). Any decay, fatigue or slowness on either lens was noted. The normal range was considered to be 30 to 40 sec for 10 cycles.

A lateral fixation disparity curve was performed with a disparometer and graphed on 17 of the 25 patients. The patient held the disparometer at 40 cm. and was given a pair of poloroid glasses to wear over their perscription. They were told to fixate on the letters to either side of the nonius lines and to keep them clear. They rotated the knob which moved the nonius lines until vertical alignment. The patient would then go beyond that point on either side and bracket until a position was determined. This was done with no prisms in place, with 4 PD BI and BO, and 8 PD BI and BO. The patient was allowed no more than 30 seconds for each setting of the nonius lines.

RESULTS

Table 1 shows a list of all responses from 25 different

individuals, excluding their fixation disparity curves. Taking into account only near responses the results can be placed into four groups, which are shown in table 2.

Group A, composed of 14 of the 25 patients, with only 1 of the 14 experiencing any symptoms. This group after adaptation to the lens became more esophoric with the minus lens and more exophoric with the plus lens. All 14 patients were able to perform flippers within normal limits. Fixation disparity curves were too variable to compare within this group.

Group B represents those patients that after adaptation to the minus lens become more esophoric and after the plus lens also become more esophoric. This group consisted of 3 of the 25 patients examined. Of the three 2 couldn't perform flippers, one having trouble with the plus and the other trouble with the minus. All three of these patients were esophoric at near and all had a high AC/A ratio. Fixation disparity curves between these individuals showed no correlation.

Group C had 6 of the 25 patients. This group became more exophoric when allowed to adapt to both plus lens and minus lens. Of the six, five could not perform flippers, and were exophoric at near. Calculated AC/A ratios of five of these subjects ranged from 1.9/1 to 3.8/1. Three of the six suffer from headaches and asthenopic complaints, and two of the three usually are uncorrected refractively. In addition three of the six subjects also become more exo at a distance with adaption to the minus lens.

Group D was the group that became more esophoric after adaptation to the plus lens, and more exophoric after adaption to the minus lens. Two of the 25 belonged to this group. One of the two suffered from symptoms but also goes uncorrected refractively. This same individual had trouble on the plus side of the flippers, and the fixation disparity curve was very steep with the extremes on either side being of a very high value.

DISCUSSION

The majority of the patients fit into group A which indicated an increase in phoria with the lens (both plus and minus). This scene was also shown in fig 2A and 4C. All of these patients seemed to have normal binocular function and no symptomology except for one that had headaches. By assumption then we could say that "normal" patients have a system with 1) no S.A.A. but AC enters in before the S.V.A. or 2) has a S.A.A. which is not as strong as the S.V.A. and the AC comes in before the S.V.A.

Six of the 25 showed a pattern that was "leaning" toward "exo" side and three of 25 showed a pattern "leaning" toward "eso" side. Of these nine, seven could not perform flippers and six of the nine are symptomatic. Since they are "one-sided", the problem with these individuals is within the S.V.A. A defect in the forward controller would show problems on both sides. If there was a problem in the divergence (+) S.V.A. a patient may have problems diverging , and the opposite would hold true for converging (-). Assuming that group A is "normal", group B shows an abnormal response to plus lens. The lower half of their graph mimics figure 4B where the S.A.A. is assumed to be stronger

than the S.V.A. Perhaps in these individuals their divergence S.V.A. is weakened and appears less strong than S.A.A. Group C also shows an abnormal response, but to the minus lens. The upper half of their graph mimics figure 4C. Perhaps in this case the convergence component of the S.V.A. is weakened and makes it appear that the S.A.A. is stronger. Group D is showing an abnormal response to both lens. A possible explanation to this is that a problem exists in the forward controller, as a problem in the S.V.A. would tend to only show a abnormality with either the plus or minus lens.

It would be interesting to test a group of convergence insufficients before treatment and then to retest them after visual therapy had been done. What sort of graph would these patients show before and after? What would happen if the number of subjects tested were greater? Would we still see the distribution already mentioned?

CONCLUSION

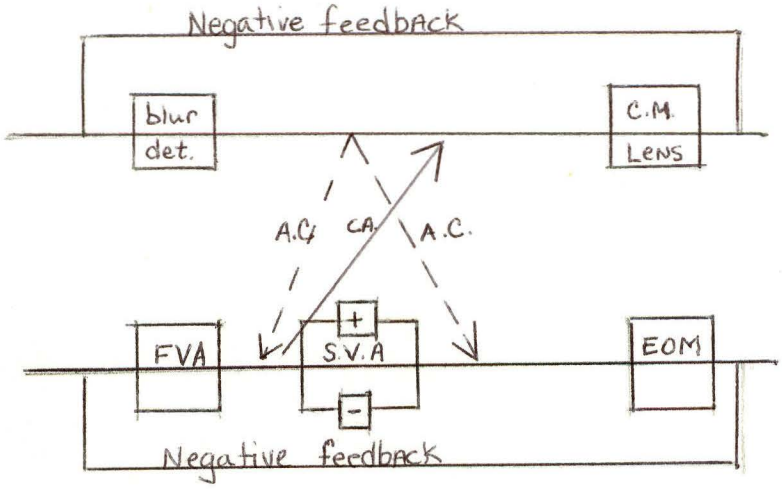
The results of this research data indicated agreement with Shor's research (1986) that the AC enters in before the S.V.A. for the "normal" individual. The research did not prove the existance of the S.A.A., but appeared to indicate that if in existance it is weaker than the S.V.A. In addition, this research study confirmed the role of the S.V.A. in patients who have a convergence or divergence problem. More in depth studies need to be performed on this topic. As a pilot study, this should provide the initiative in the research of this topic, and bring on questions and concerns regarding it.

Table 1

#	DPH	DPH -1.00	DPHA -1.00	NPH	NPH +1.00	NPHA +1.00	NPH -1.00	NPHA -1.00	ACA	FL
1	3X	1.5X	2.3X	6.2X	11X	11.8X	2.5X	3.5X	4.8	-
2	1.8X	3.3X	5.5X	2.3X	6.3X	6.8X	2X	3.2E	5.5	-
3	0	1.7E	0.5E	2X	5.7X	5.7X	2.7X	1X	5.2	-
4	9.3E	16.7E	18.7E	14.8E	9.2E	5.5E	14.8E	15.3E	9.2	-
5	1.7E	1E	1.3E	1.7E	0.7X	2.0X	3E	5.3E	6	-
6	2E	5.5E	6.0E	2.7E	3.7X	6.5X	2.7E	6E	6.3	-
7	0	0	1.2E	4.2X	4.2X	5.2X	4.5X	2.3X	5.6	-
8	0	2.2E	2.5E	0.5X	2.5X	6.7X	0.8X	4.7E	6.2	-
9	3.8X	1.4E	2.2E	7.5X	13.3X	14.5X	11.7X	10.3X	4.5	-
10	4X	2.7E	6.2E	2.7E	5.8X	8.5X	5E	7.2E	8.6	-
11	2.2E	6.7E	9.2E	8.7E	8.2E	4.5E	10.7E	11.7E	8.6	-
12	1.7X	3.7E	7.5E	3X	4.3X	5X	2.3E	5.5E	5.5	-
13	1.7X	3X	0.7X	4.5X	7.7X	9X	5.3X	3.7X	4.8	-
14	0	1E	2E	8X	9.7X	11.5X	9.8X	7.8X	2.8	-
15	0.3X	2E	2.3E	5E	0.5E	1E	2.5E	5E	8.1	-
16	1X	.3E	1.7E	1.5E	2.7X	2.2X	0.8X	3E	7.1	+
17	2.2E	6.8E	9.5E	7.3E	7.5E	9.7E	18.2E	20+E	8	+
18	0	8E	6.2E	0.5E	4.7X	8.3X	2.3E	2.2E	6.2	+
19	8X	5X	8.7X	18.2X	18.5X	20X	13.2X	13.8X	1.9	+
20	4.5X	4.0X	3.0X	13X	14.7X	16X	16.5X	16.8X	2.6	+
21	3X	4X	3.3X	12.3X	15.5X	17.3X	12.3X	14.3X	2.3	+
22	2.3X	2.7X	0.2X	8.5X	9.2X	11X	6.7X	8.7X	3.5	-
23	0	2E	0.7E	5.5X	8.7X	8.8X	7X	9X	3.8	+
24	3.2E	3.5E	9.3E	2.5E	0.3E	2E	3.3E	3.2E	4.4	-
25	0.7X	1.2E	2.8E	4.7X	9.3X	7.3X	3.7E	2.7X	5.5	+

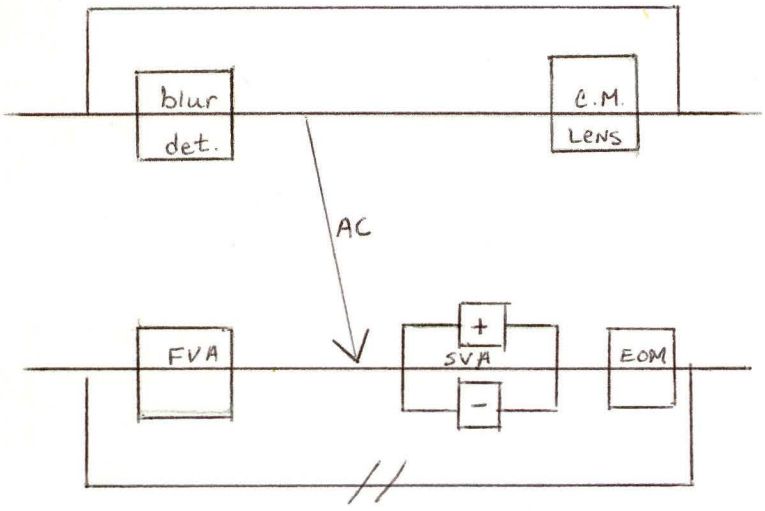
Table 1 is a cummulation of all the data collected excluding the fixation disparity curves. DPH is the distance phoria. DPH -1.00 is the distance phoria with a -1.00 trial lens and DPHA -1.00 is the distance phoria after adapting to a -1.00 trial lens. NPH is the near phoria. NPH +1.00 is the near phoria with a +1.00 lens and NPHA +1.00 is the near phoria after adapting to a +1.00. NPH -1.00 is the near phoria with a -1.00 lens and NPHA -1.00 is the near phoria after adapting to a -1.00 lens. ACA is the accommodative convergence to accommodative ratio. FL is the + or - 1.50 flippers and a + in that column indicates a problem with the flippers, a - in that column indicates that the patient was able to perform them with in the normal limits.

Figure 1



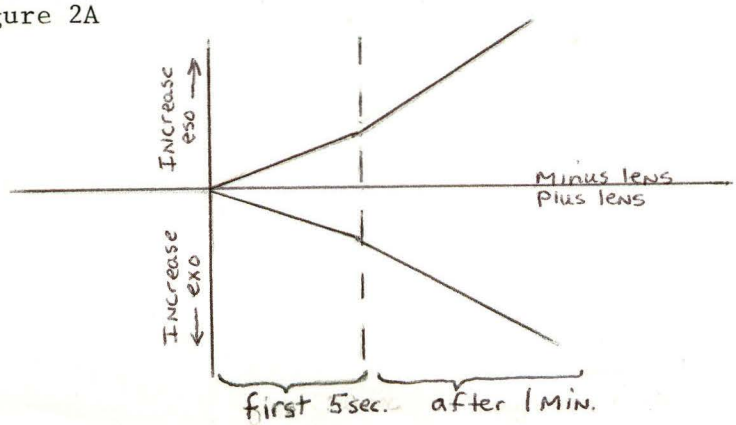
This diagram shows the accommodative loop (upper) consisting of the blur detector(eye-brain), ciliary muscle and lens. The lower loop is the vergence system. It consists of the extraocular muscles, fast vergence adaptation, and slow vergence adaptation(which has convvergence and divergence, indicated by the minus and plus sign). The relationship between the two systems is achieved by the accommodative convergence (AC) and convvergence accommodative (CA). The AC has been shown here with a slashed line as the position of it is not fully determined.

Figure 2



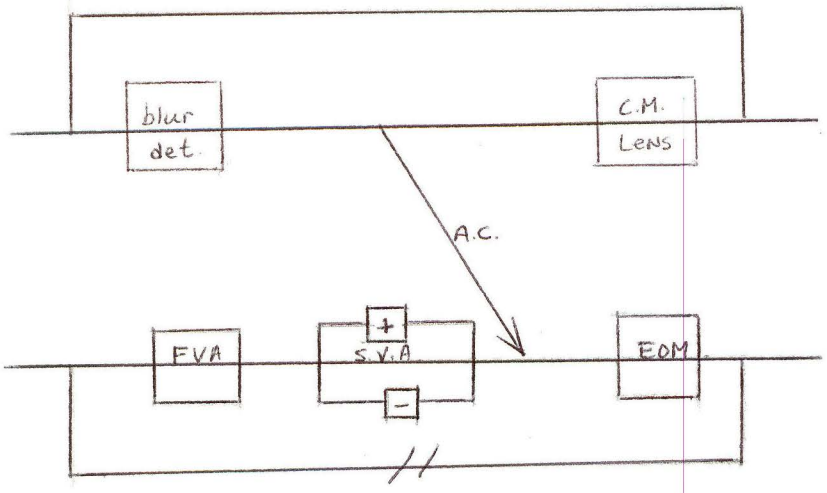
This diagram represents the system with the AC entering before the S.V.A. We can ignore the CA response as we have opened up the feedback loop of the vergence system (this is shown here by the slashed line in the lower loop)

Figure 2A



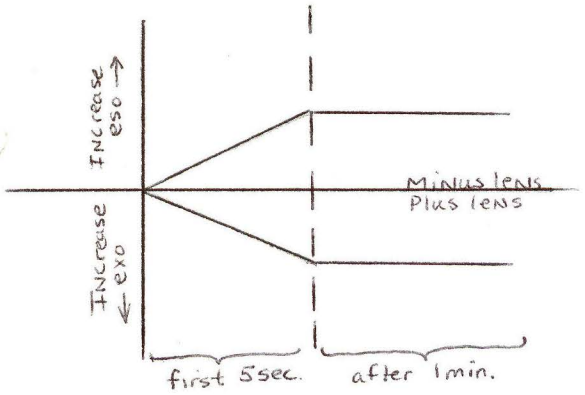
This represents initiating a lens intc the above system. With a minus lens there is an initial increase in esophor that further increases with adaptation. With the plus lens you will get an initial increase in exophoria that will increase even more with time.

Figure 3



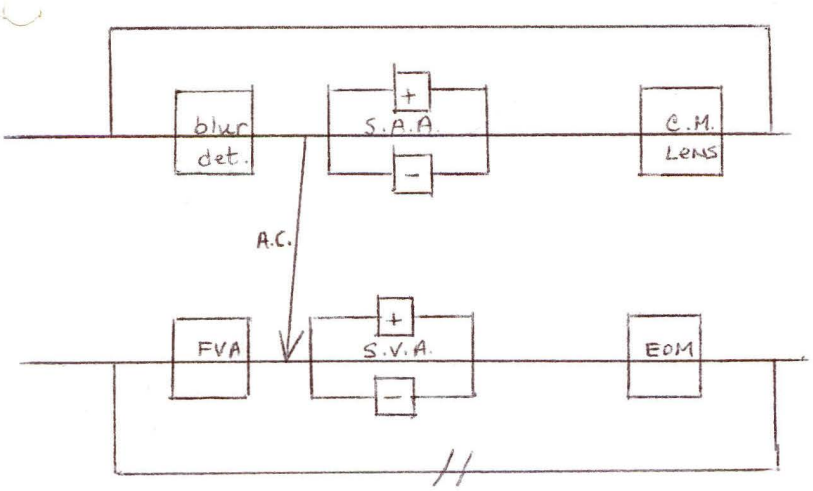
This system is the same as figure 2, except that the AC is introduced after the S.V.A. Once again the lower loop has been broken.

Figure 3A



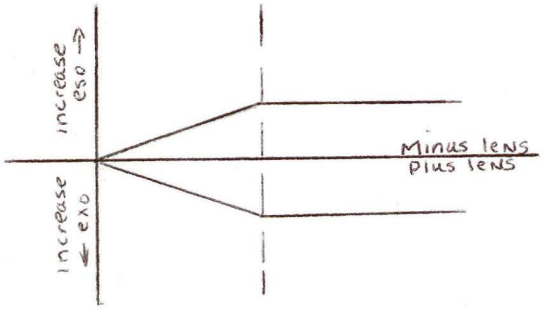
This represents the initiation of a lens into the above system. There is an initial increase in the phoria that has no change after adaptation.

Figure 4



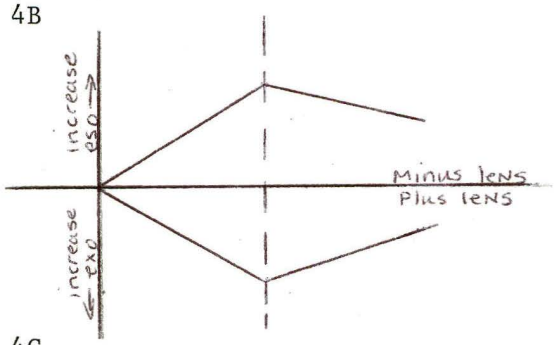
This diagram shows the initiation of the slow accommodative adaptation(S.A.A.) into the upper loop. It also shows the AC entering the vergence loop after the F.V.A but before the S.V.A. The vergence loop is still broken by dissociation.

Figure 4A



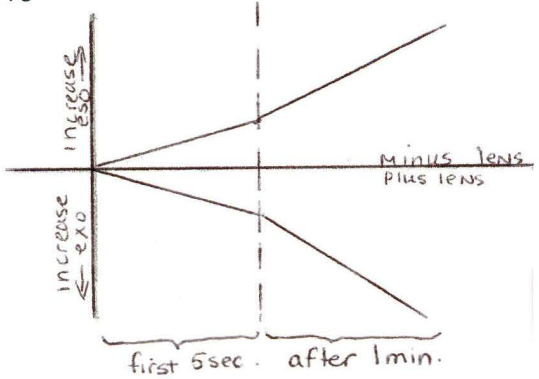
This figure shows what the results would be if the S.A.A. and the S.V.A. have the same strength and time constant. There is an initial increase in the phoria and with adaptation there is no increase or decrease beyond what already seen.

Figure 4B



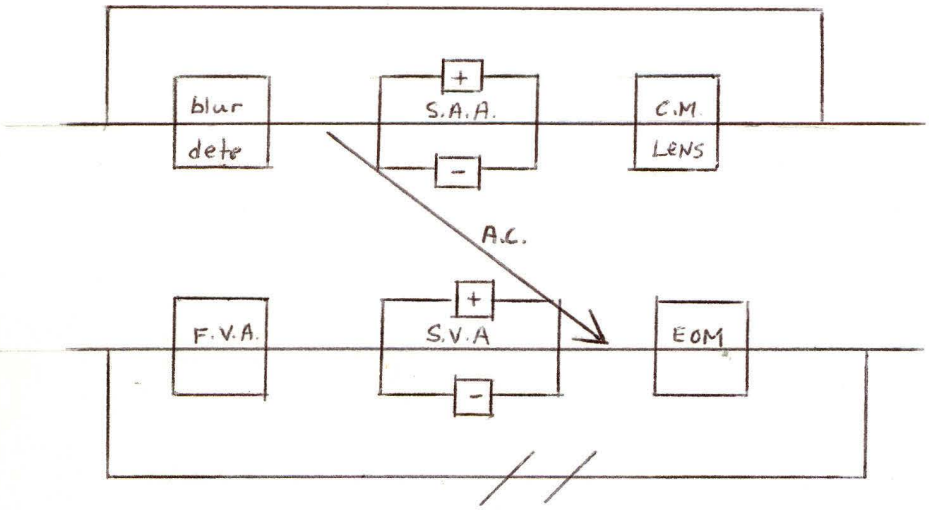
This figure demonstrates what would result if the S.A.A. had a shorter time constant and/or higher gain compared to the S.V.A. There is a decrease in each phoria after adaptation. This is due to the S.V.A. not being able to project all that was put into it.

Figure 4C



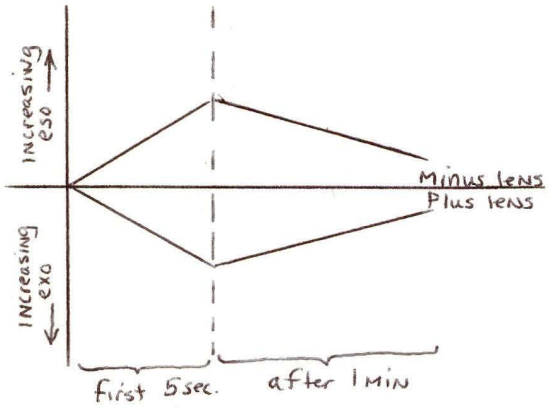
This figure demonstrates what would happen if the S.A.A. is not as strong and/ or longer time constant versus the S.V.A. There is an initial increase in each phor which becomes even greater with time.

Figure 5



This is the same as figure 4 except here the AC enters in after the S.V.A. Once again the lower loop is open.

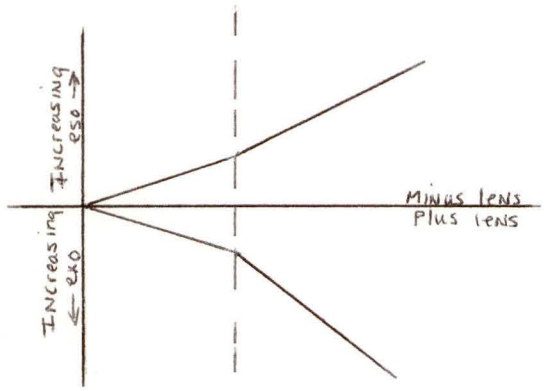
Figure 5A



This diagram represents what would happen if lens were introduced into the above system. There would be the initial increase in phoria, that with time would decrease.

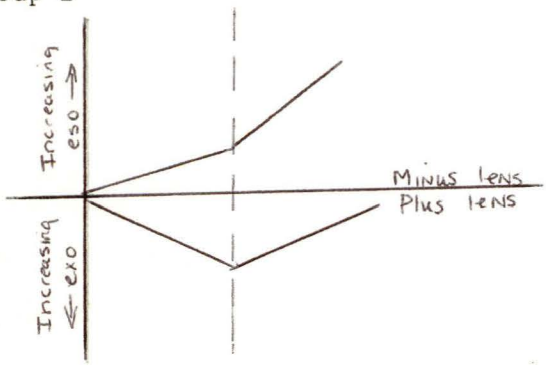
Table 2

Group A



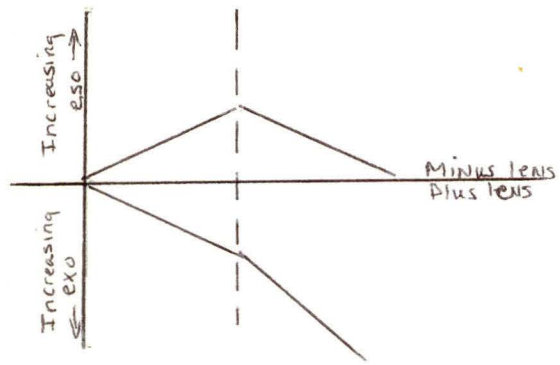
This group shows an increase in esophoria with adaptation to minus lens and an increase with a plus lens. 14 of the 25 subjects were in this group. All subjects could perform flippers. One of 14 are experiencing headaches and 3 of 14 became more exophoric at distance with a minus lens.

Group B



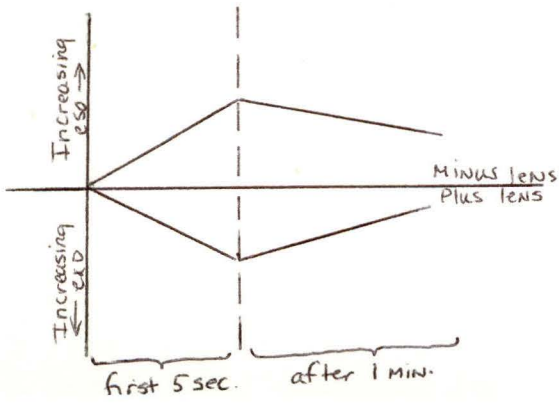
This group shows an increase in esophoria with adaptation to minus lens and an increase in esophoria with adaptation to plus lens. Three of 25 subjects were in this group. Two of those 3 couldnot perform flippers. All three of these subjects were esophoric at 40cm and had a high AC/A.

Group C



This group became more exophoric with adaptati to both plus and minus lens. Six of the 25 belonged to this group, and of those 6, 5 coul not perform flippers. Three of the 6 go uncorrected and suffer headaches and asthenopia. Three of the 6 also became more exophoric at distance with adaptation to -1.00 lens.

Group D



This group represents those that became less esophoric with adaptation to -1.00 and less exophoric with adaptation to +1.00. Two of th 25 fit into this group, 1 of the 2 suffer asthenopia.

1. Schor,C. The relationship between fusional vergence eye movements and fixation disparity. Vis Res 1979;vol 19:1359-1360.

2. Semmlow J, Hung GK, Ciuffreda K. Quantitative assessment of disparity vergence components. Invest Ophth Vis Sci 1986; 27:558-564.

3. Saladin JJ, Carr L. Fusion lock diameter and the forced vergence fixation disparity curve. Am J of Opt and Phy Optics 1983;vol 60 #12:933-943.

4. Kotulak J, Schor C. The dissociability of accommodation from vergence in the dark. Invest Ophth Vis Sci;1986:544-551.

5. Schor C. Fixation disparity: a steady state arror of disparity induced vergence. Am J Optom and Phy Optics 1980; 57:618-631.