A STUDY OF SLOW VERGENCE ADAPTATION

USING

FORCED VERGENCE FIXATION DISPARITY CURVES

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Introduction

There is a general belief that the slope of the standard forced-vergence fixation disparity curve is an indication of patient comfort, with the steeper curves more often associated with asthenopia. When fixation disparity measurements are taken, it usually takes at least 15 seconds to generate each point on the curve. The works of Carter (1963, 1965) and of Schor (1979[1&2]) have suggested that this is enough time for a slow prism adaptation cycle to be set in motion. Carter (1965) assumed that fixation disparity is proportional to the stress on fusional vergence. It therefore seems likely that persons with efficient prism adaptation mechanisms will produce flatter fixation disparity curves. Indeed, Schor (1979[1]) did find a high correlation between efficient slow vergence adaptation and small amounts of fixation disparity.

A difference between the slopes of the prism induced fixation disparity curves before and after slow prism adaptation has been initiated should be easy to demonstrate. The amount of the difference between the two curves should be related to the efficiency of the patient's slow vergence adaptation mechanism.

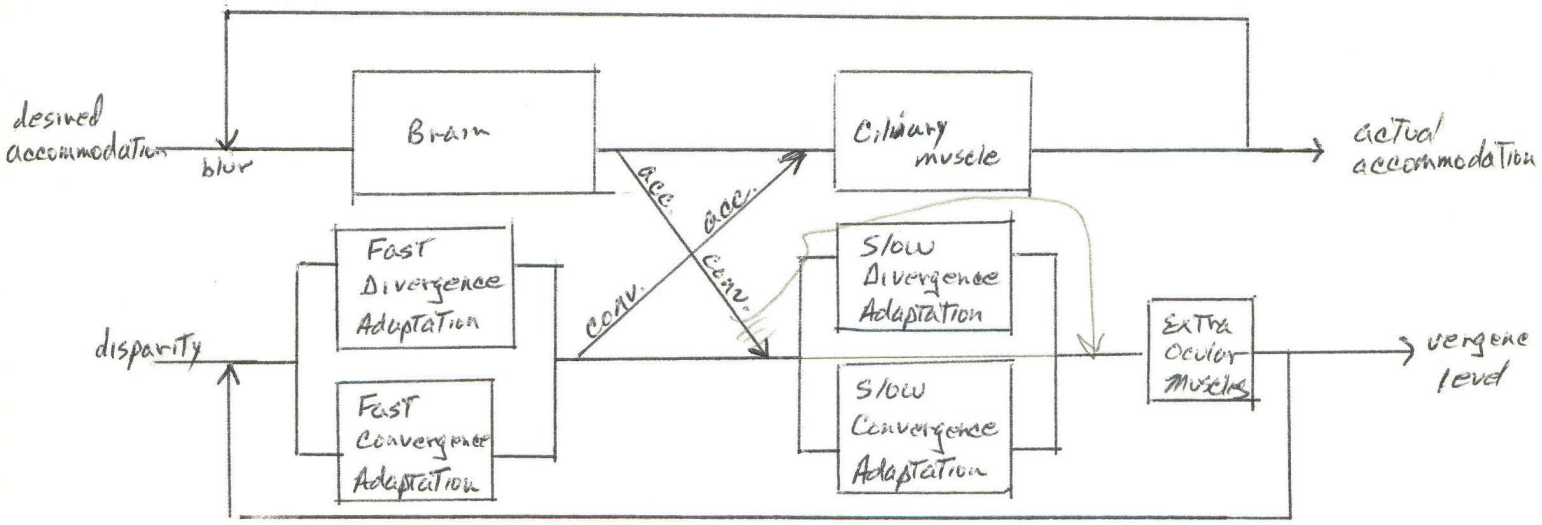
Prism Adaptation

Prism adaptation was described by Carter (1963) as "a shift of the fusion free position of the eyes so as to maintain through

the prescription the same demand on fusional convergence that existed prior to the wearing of the prism." In other words, after wearing a given prescription for a while, the patient demonstrates the same phoria and fixation disparity as before the prescription was worn. This prism adaptation is dependent on the presence of adequate sensory fusion. Carter (1957) showed that adaptation of the fixation disparity to both base in and base out prisms occured when foveal sensory fusion was permitted. (Mitchell and Ellerbrock (1955) had previously found adaptation of the fixation disparity to base in prism that was not demonstrated to base out prism. This difference is presumably because they used targets with peripheral fusion contours which did not allow adequate foveal fusion.)

That sensory fusion must be obtained for adaptation to occur is demonstrated in patients with an induced tropia following the removal of prism which has been worn for a prolonged period. The tropia recovers (returns to the original phoria position) very slowly because there is no binocular vision and hence, poor adaptation. Additionally, a prism-induced phoria can still be present after eight hours of sleep (after prism removal), but disappears completely if single binocular vision is maintained for about 20 minutes (Carter, 1965). These observations caused Carter (1965) to postulate that poor sensory fusion is indirectly the cause of high heterophoria, rather than the high phoria causing the poor sensory fusion.

The Model



Whenever the desired vergence level is not equal to the resting state of the extraocular muscles, some motor imput to vergence is required to maintain fusion. This innervation comes from a combined effort of the fast and slow neuronal integrators diagrammed above. The initial response (that which causes fusion to be obtained) is accomplished by the fast neuronal integrator. As we know the stimulus to fusion is retinal disparity. We can, therefore, think of disparity as the input to this fast integrator. Its output is a stimulus for continued vergence innervation (i.e. fusion is maintained).

As the diagram shows, the output from the fast integrator need not go directly to the extraocular muscles. It may instead be routed through the slow neuronal integrator.

In a healthy binocular system, the fast integrator will normally only produce a maximum output for a few seconds. Beyond this the slow integrator is employed. It acts as an amplifier to the output of the fast integrator. Vergence is then maintained

primarily by output from the slow integrator feeding into the extraocular muscles. Since the slow integrator amplifies the amount of vergence innervation to the extraocular muscles, less output is needed from the fast integrator to maintain the required amount of innervation. Negative feedback, therefore, decreases the amount of disparity, the stimulus to fast fusional vergence, so that less innervation is fed into the slow neuronal integrator. The cycle is repeated until the desired steady vergence level is achieved.

Since disparity is the input to the fast neuronal integrator, it is obvious that there must always be some disparity to provide the necessary stimulus. This disparity is fixation disparity. If the disparity is reduced to zero, there is no input to the vergence system. Fixation disparity, therefore, can be described as a "steady state error that acts as a stimulus to maintain convergence" (Schor, 1979[2]).

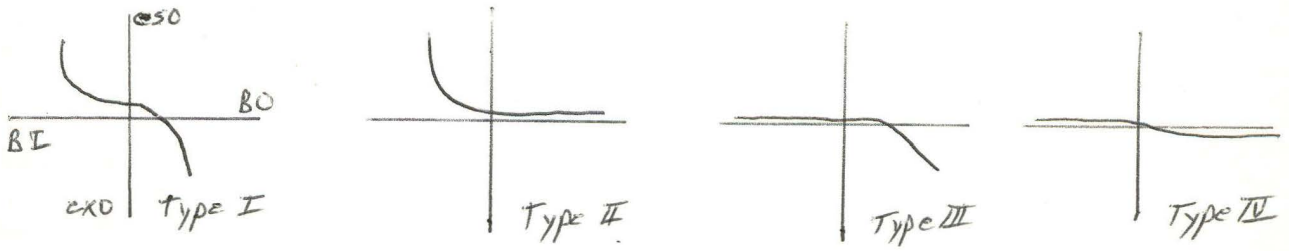
There is a high correlation between the minimum amount of fixation disparity and the maximum amount of slow fusional vergence (Schor 1979[1]). In other words, for a vergence demand where the slow vergence adaptor is efficient, the fixation disparity will be small.

The Curve

A fixation disparity is generally considered an incomplete vergence response. Carter assumes that fixation disparity is due to stress on the fusional vergence system. A subject with a high exophoria, for example, would be expected to exhibit an exo fixation disparity, the magnitude of his vergence response being just large enough to maintain binocularity (i.e. within Panum's fusional area).

Fixation disparity has been called "a microstrabismus existing during normal binocular vision."

Fixation disparity curves have historically been classified into four types, based upon their shapes. The Type I Curve demonstrates divergence and convergence errors that are approximately equal in magnitude. Convergence errors are smaller than divergence errors in a Type II curve. Conversely, Type III curves have smaller errors of divergence than of convergence. The Type IV curve has small errors of both divergence and convergence, and is usually associated with asthenopia.



The assumption that fixation disparity is due to stress on fusional vergence can be used to explain the shape of the Type I forced vergence fixation disparity curve. The introduction of prism changes the magnitude of the needed vergence response. As the response becomes more difficult to obtain, the increased stress can be observed as an increase in fixation disparity.

The relatively flat central portion of the curve represents an area where the fusional demand is being adequately met. Schor (1979[2]) postulates that this flat central portion is due to efficient slow fusional vergence taking the stress off the fast vergence system. The steep slopes at the end of the curves are due to fast fusional vergence working alone, resulting in high fixation dispar-

ities, This explains why patients with steeply sloping curves experience asthenopia. These patients have weak slow vergence adaptors; the fast adaptors are consequently overworked.

Methods

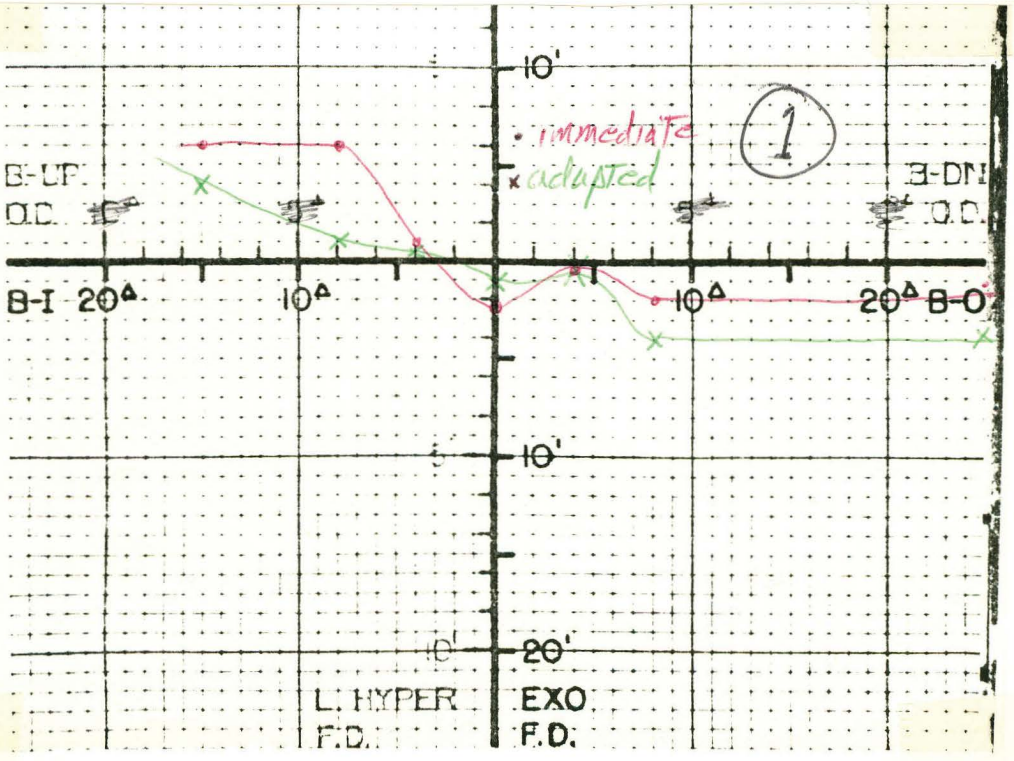
Forced vergence fixation disparity curves were generated using the standard fixation disparometer, under conditions of normal room illumination and with the disparometer usually held in the subject's lap at a normal reading distance. (For a few patients, the disparometer was placed on the phoroptor near point rod at a 16" viewing distance. This did not appear to make any difference.) Prisms were presented before the right eye alternately base in, then base out, proceding from smallest to largest prism power to minimize the effect of any adaptation from the previous stimulus on the desired reading. Measurements were taken using no prism, 4BO, 4BI, 8BI, 8BO, and base in and base out prism near the end of the patient's near vergence ranges. Two curves were generated using the same prism powers but with different viewing times. For Curve I, the immediate curve, the patient obtained single binocular vision through the prism, described the location of the nonius lines, and removed the prism as quickly as possible. This procedure took less than five seconds. The position of the nonius lines was then adjusted and the process was repeated until the subject reported alignment of the lines immediately upon fusion. For CurveII, the adapted curve, fusion was maintained through the prism for one minute. The nonius lines were then adjusted, with the patient still looking through the

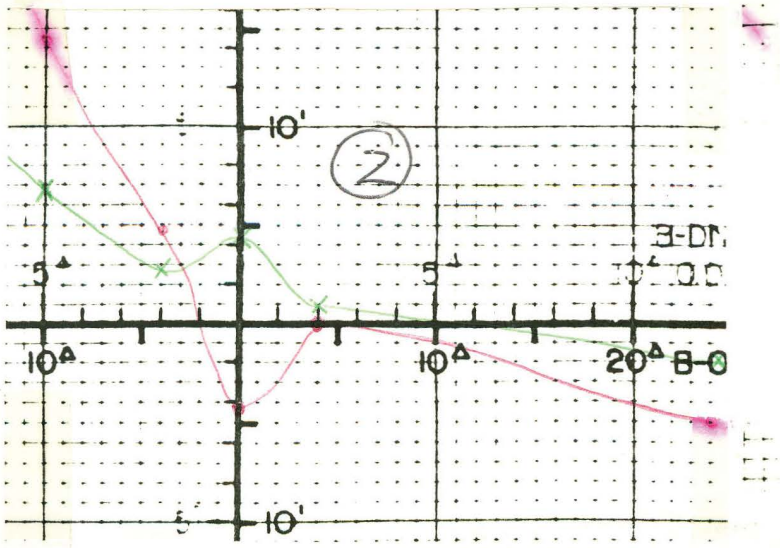
prism, until subjective alignment was again reported.

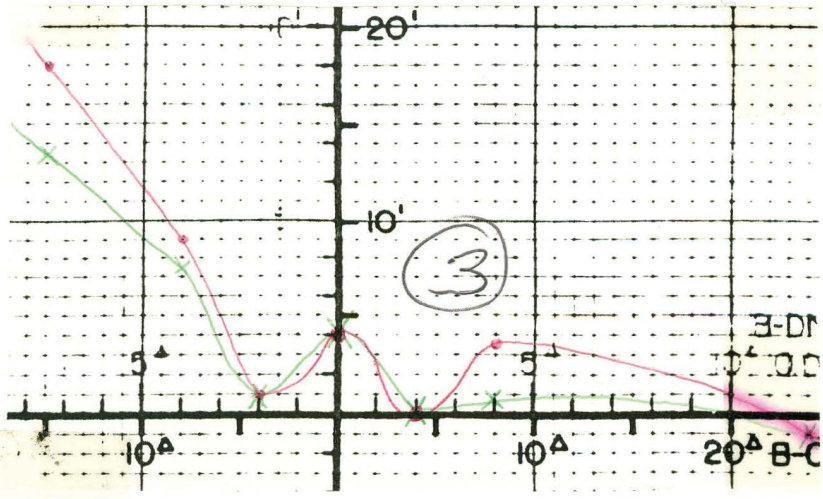
With three subjects, many pairs of curves were generated and the results averaged to yield an average fixation disparity curve. A one-time sampling of curve pairs for 24 clinical patients was analyzed along with these three sets of curves to determine if prism adaptation occured. An attempt was also made to analyze these curves with respect to curve type, objective binocular findings, patient symptoms, and history of previous visual training.

Results

The three subjects studied in detail all show an adaptation to base in, but no change in response to base out. All three of these subjects have very flat fixation disparity curves. While the response to base in is the expected one, it is difficult to draw any conclusion concerning response to base out from such a flat curve.







While only half of the patients tested show the expected adaptation to prism, this is the most common response. The bulk of this group is comprised of type I curves. According to Ogle, the majority of the general opoulation has a type I curve. It is considered a normal curve, seen in patients with good binocular vision and should show adaption. The results show that type I's do adapt better than any other group, both to base in and base out prism. Of the remaining curve types, type II shows a tendancy to adapt. Type III andIV do not.

	Patients(24)		Type I(13)		Type II(8)		Type III(1)		Type IV(2)	
	#	%	#	%	#	%	#	%	#	%
Adapted to BI	12	50	9	69	3	38				
Adapted to BO	14	58	11	85	3	38				
Adapted to both	10	42	8	62	2	25				
Inverted BI	5	21	1	8	2	25			1	50
Inverted BO	6	25	3	23	2	25			1	50
Inverted both	3	13	0	0	2	25			1	50
Same BI	7	29	4	31	1	13	1	50	1	50
Same BO	4	17	1	8	2	25	1	100	1	50
Same both	3	13	0	0	1	13	1	100	1	50

Three curves changed type with adaptation. The immediate classification is used for the above computations. The term "inverted" refers to curves taken after one minute of fusion through prism that show an increase in fixation disparity instead of the expected decrease.

Discussion

The subjects studied in depth do show an adaptation after one minute of fusion. The adaptation to base in is significant, while the degree of base out adaptation is more difficult to interpret owing to the flattness of the base out side of these curves.

Schor (1979[1]) has speculated that the base out portion of type II curves (and the base in portion of type III's) might be due to a very rapid adaptation to prism. It seems more likely to us that this phenomenon is caused by a lack of response to convergence (type II) or divergence (type III) stimuli. This lack of response could be caused by a lack of, or deficiency in, one of the

vergence integrators. Abnormal curves might also be caused by accommodative fluctuations, either involuntary (faulty accommodative system) or voluntary. The voluntary accommodative changes are used to circumvent the impairment to fusion caused by faulty slow vergence integrators and/or lack or weakness in disparity detectors.

Subject 1 observed that the nonius lines tended to oscillate while generating the first few curves, but that the nonius lines were much more stable during later trials. (The curves were produced over a period of weeks.) Presumably, the testing itself had an effect on his slow vergence system. This suggests that a vision training method which involves prolonged fusion through prism should improve the performance of the slow neuronal integrator. The generally accepted method of prism insertion and removal is more likely to train fast fusional vergence.

Subject 2 shows an adaptation to base in prism and perhaps a slight adaptation to base out prism. The eso position of the zero point on the adapted curve is a common phenomenon. The reading previous to this was taken through the largest base out prism (strong convergence response). This subject's slow vergence adaptor for convergence was set in motion very quickly, and the duration of this response was obviously greater than one minute.

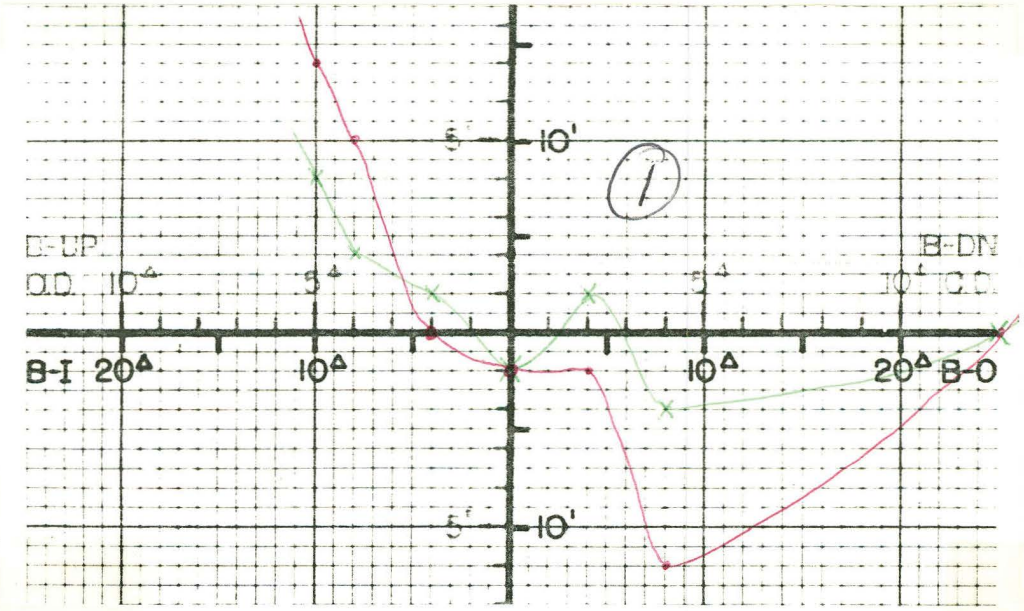
This same phenomenon was observed in Subject 1's curve which may account for the flattness of the base out portion of that curve.

The flat base out portion of Subject 3's curve makes a judgement of prism adaptation difficult to make. Base in adaptation

does occur, possibly because this subject is esophoric and the slow neuronal integrator for divergence has had a chance to develope. Unfortunately it has not developed well enough. Adaptation is slight. This subject wears a plus prescription for reading to relieve esophoric asthenopia. It has long been known that esophores have more symptoms than exophores and are more difficult to train. Is the slow vergence adaptor for convergence naturally stronger or does accommodative convergence account for the difference?

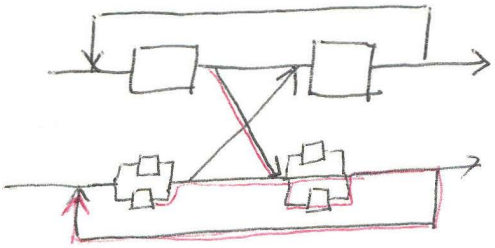
The following is an attempt to explain individual patient responses, based on classification of curve types, objective binocular findings, subjective symptoms, and history of vision training.

Patient 1.

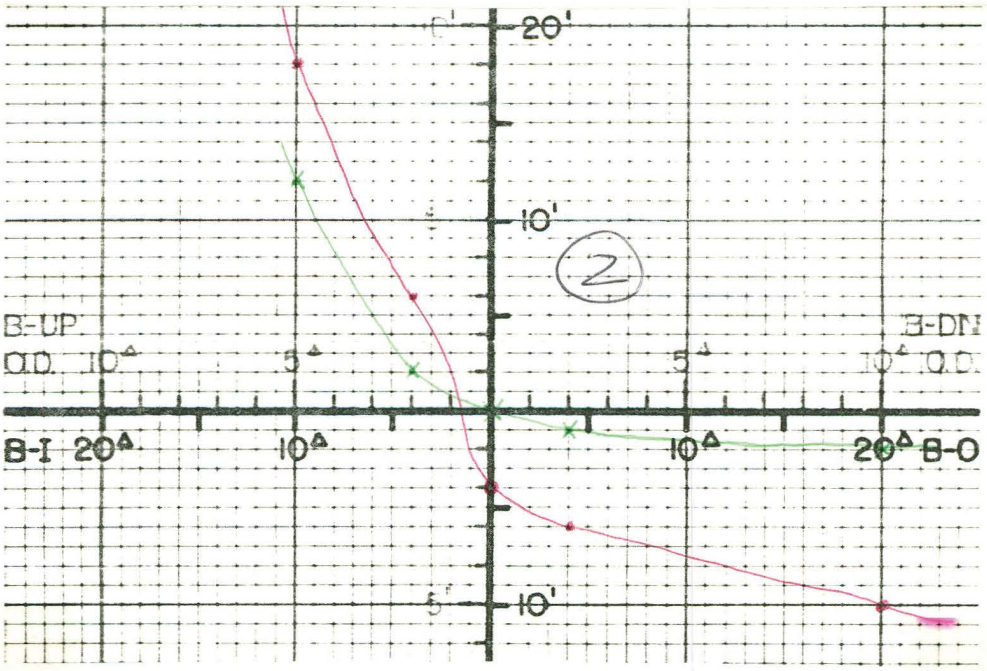


This patient has normal binocular vision with no asthenopia. She has a type I curve with some accommodative fluctuations, but which nevertheless shows the predicted adaptation to both base in and base out prism. The upward slope near the end of the range on

the base out side is probably due to accommodative convergence being used to supplement the vergence system which is being severely stressed. The increased convergence stimulates negative feedback, which in turn decreases the fixation disparity.

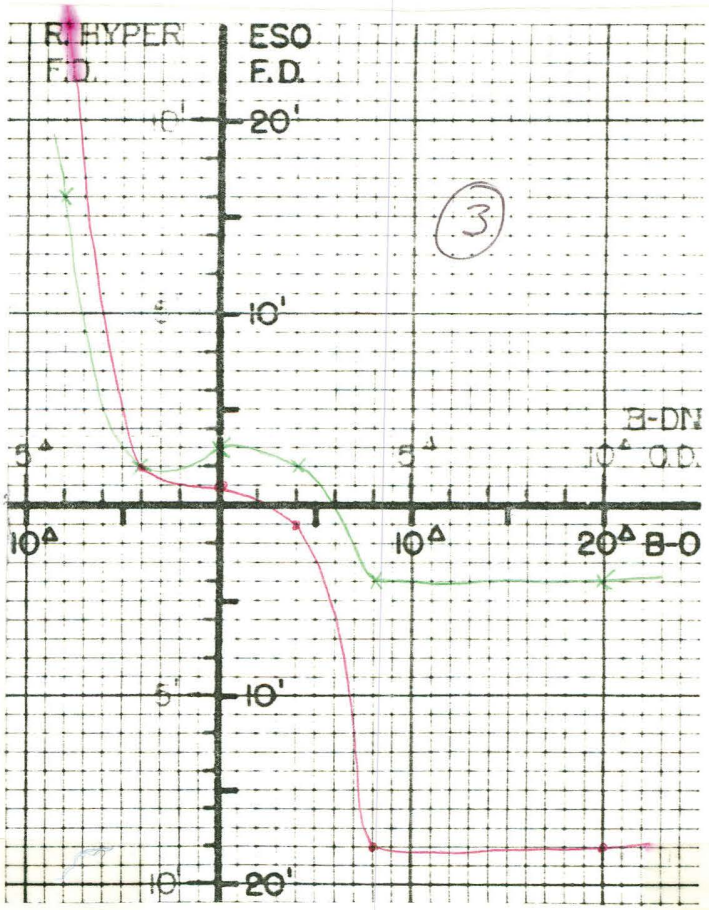


Patient 2.



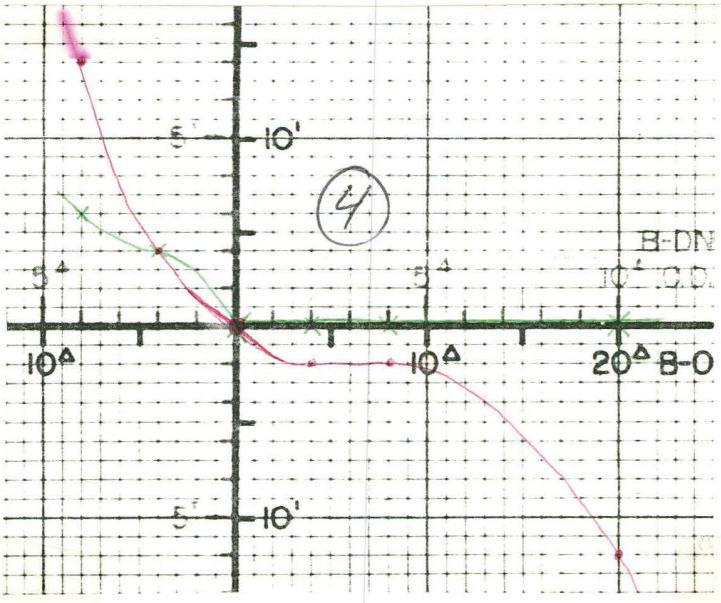
This patient represents the perfect example of the expected result. A type one curve has shown adaptation to base in and base out. Since this patient is a presbyope there are two possible explainations: 1) The patient changes his accommodative innervation to manipulate his vergence posture. If this is an efficient mechanism in presbyopes, we would expect a flat curve.

2) This patient has efficient slow vergence integrators for convergence and divergence.



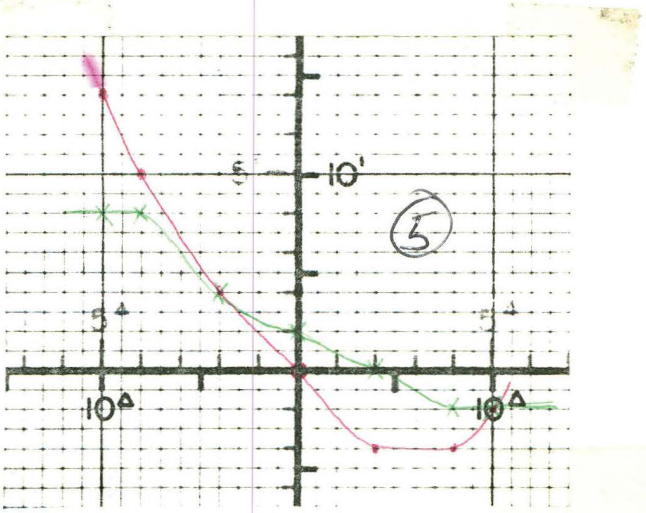
Patient 3.

This is another patient with normal binocular vision whose type I curve adapted to base in and base out prism. Again, accommodative convergence comes into play on the base out side, giving a "tail" to the curve.



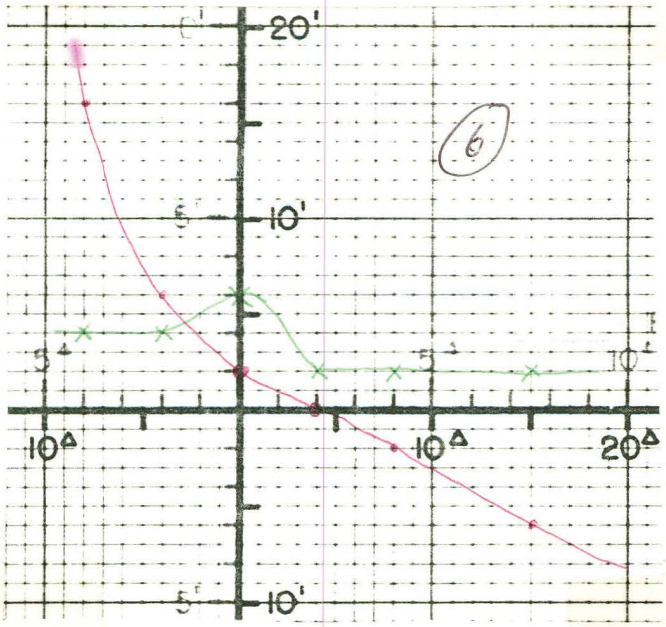
Patient 4.

Type one curve adapted to base in and particularly well to base out.



Patient 5.

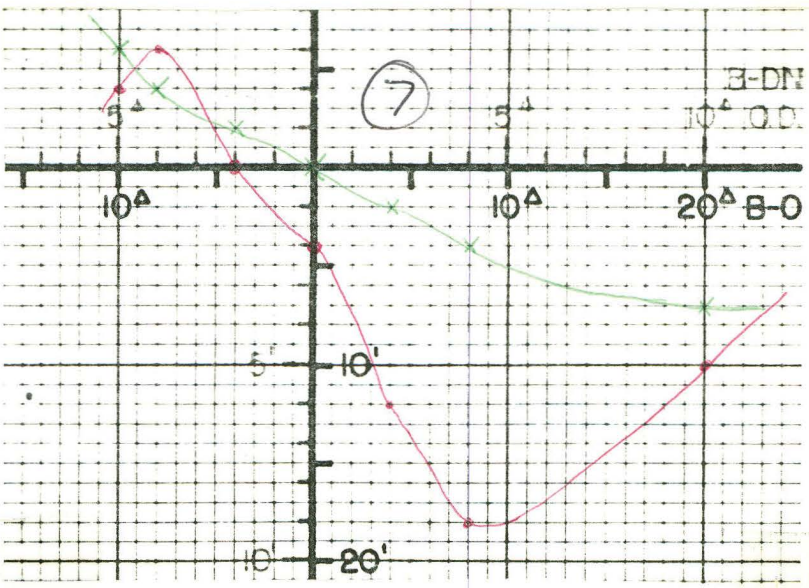
This is a patient who originally presented with poor vergence facility. She was unable to fuse 4BI or 8BO at a vision screening. Subsequent vision training has slightly improved her vergence ranges to those shown by the curve. It is a steep type I with good adaptation to base in and base out prism.



Patient 6.

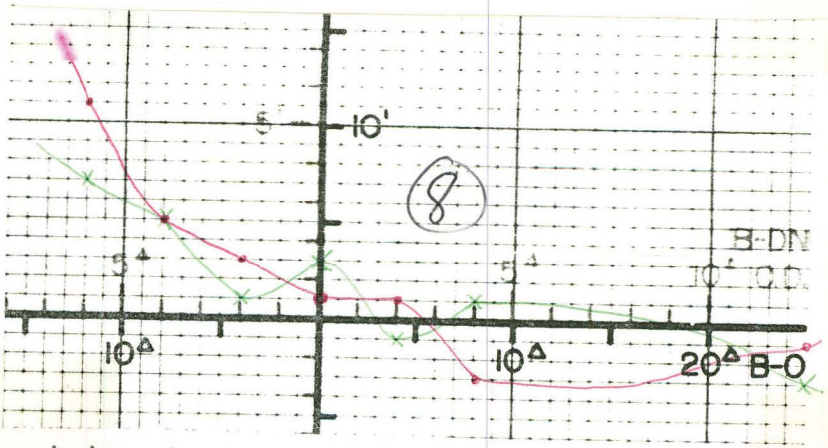
This patient has previously had extensive vision training. The slow vergence integrators have had a lot of practice. Adaptation is e xcellent on both sides.

Patient 7.

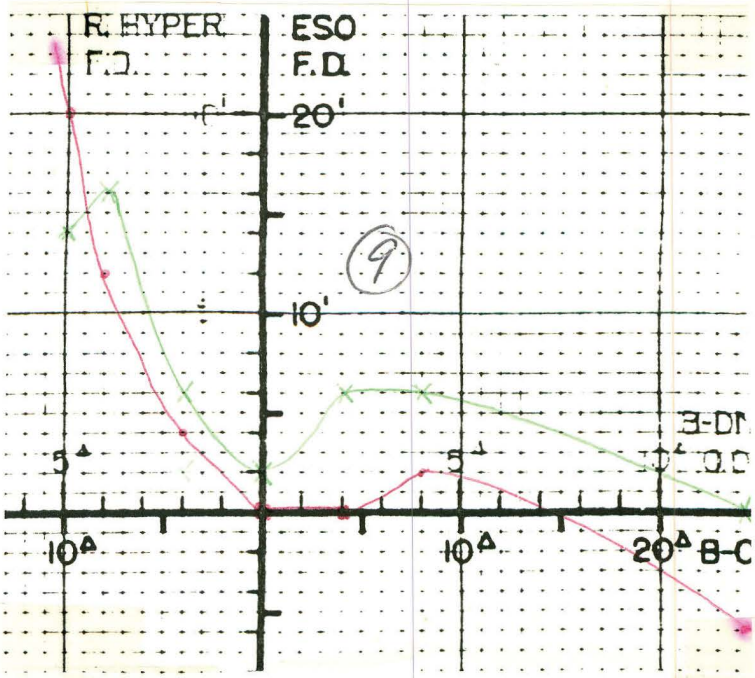


This is a type I curve with no adaptation to base in. The adaptation to base out is obvious. The upward swing of the immediate curve on the base out side is most likely caused by accommodative innervation. This patient is a pre-presbyope. Again we wonder how much of this is prism adaptation and how much is due to using accommodative innervation to manipulate vergence response.

Patient 8.

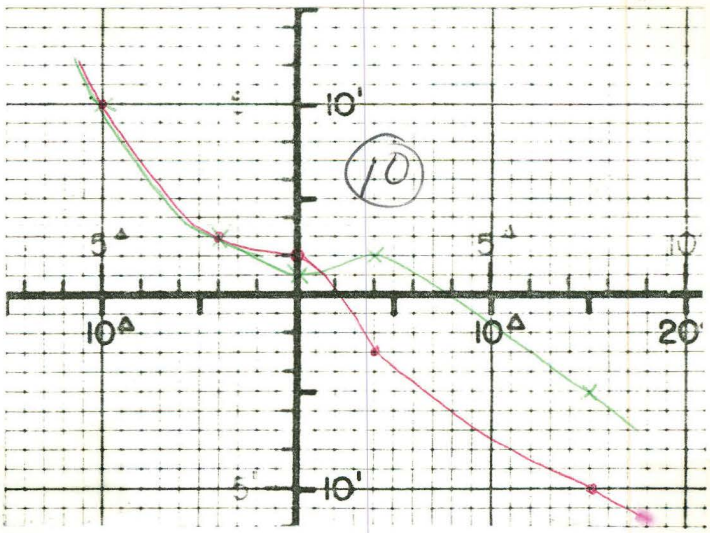


This is a relatively flat type I curve which shows a slight adaptation to base in prism. This patient has had base out training to compensate for convergence insufficiency and has, therefore, learned to use accommodative innervation to manipulate convergence. This accounts for the resemblance of the base out side to a type II curve. There seems to be a very slight adaptation here.



Patient 9.

This patient has a normal type I curve which shows no change in response on the base in side and an increase in fixation disparity on the base out side. The inversion may be due to increased accommodation in response to a prolonged convergence stimulus.

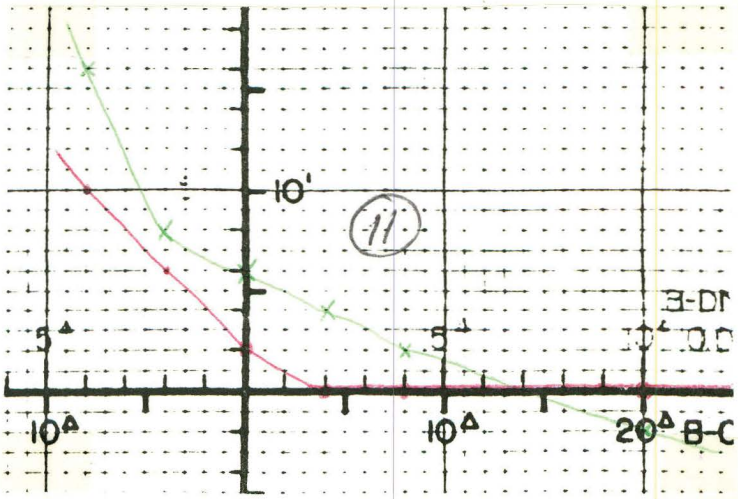


Patient 10.

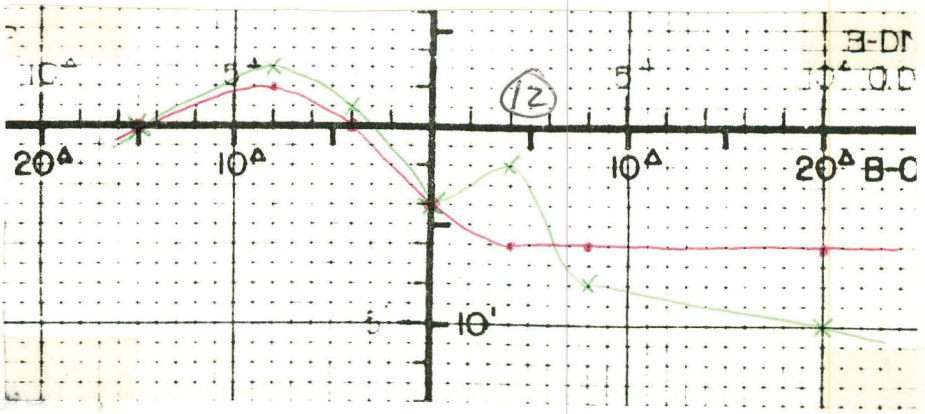
This patient is slightly exophoric at distance and near. For this reason she has never had much need for prolonged divergence.. We, therefore, theorize that the slow neuronal integrator for divergence has not had the opportunity to become efficient. There is no adaptation to base in prism. Prolonged fusional convergence is the normal state for this individual. The slow neuronal integrator for convergence is efficient. Adaption to base out prism occurs.

Patient 11.

Here is a patient whose immediate curve is type II, but whose adapted curve more closely resembles a type I curve.. The fixation

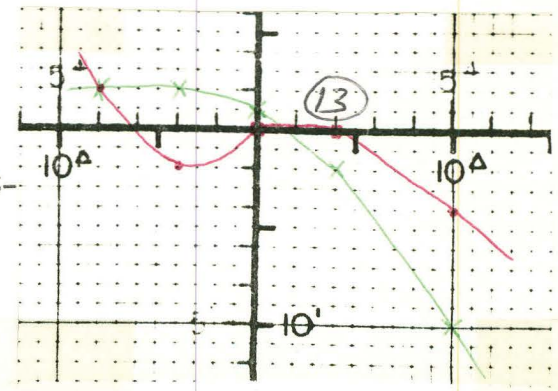


disparity has increased on both the base in and base out sides after adaptation. This phenomenon can possibly be related to the patient's orthophoria at distance and near. His visual system does not undergo "normal" convergence/divergence training because there is no phoria to compensate for. Thus, his slow vergence integrators never get much practice. When his system is stressed by higher amounts of prism and he must continue to maintain single binocular vision for a minute of more, his fast vergence adaptors get tired of trying to keep up with the demand and there is a resulting increase in fixation disparity.



Patient 12.

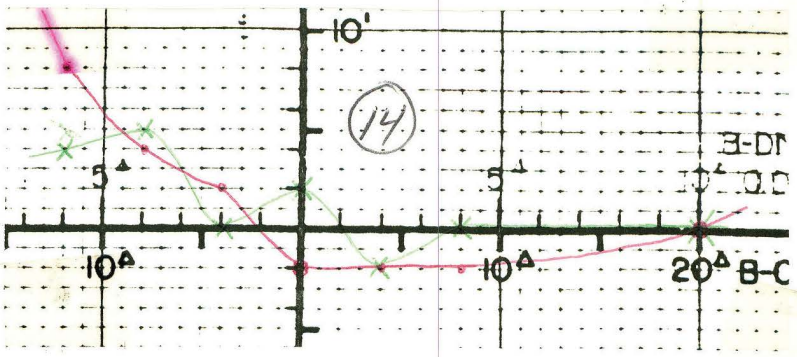
This appears to be a type IV curve. This patient is also orthophoric at distance and near and again shows the double inversion.



Patient 13.

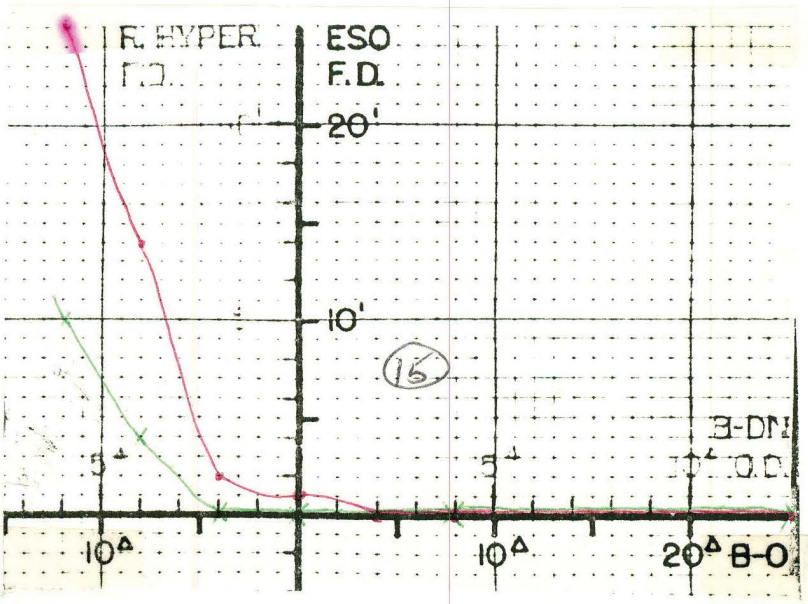
This patient is orthophoric at distance and near and exhibits narrow vergence ranges and lack of adaptation. He

had a great deal of difficulty performing the test due to oscillation of the nonius lines.



Patient 14.

This curve shows adaptation to base in and base out. The amount is very little owing to the fact that the immediate curve is already flat.



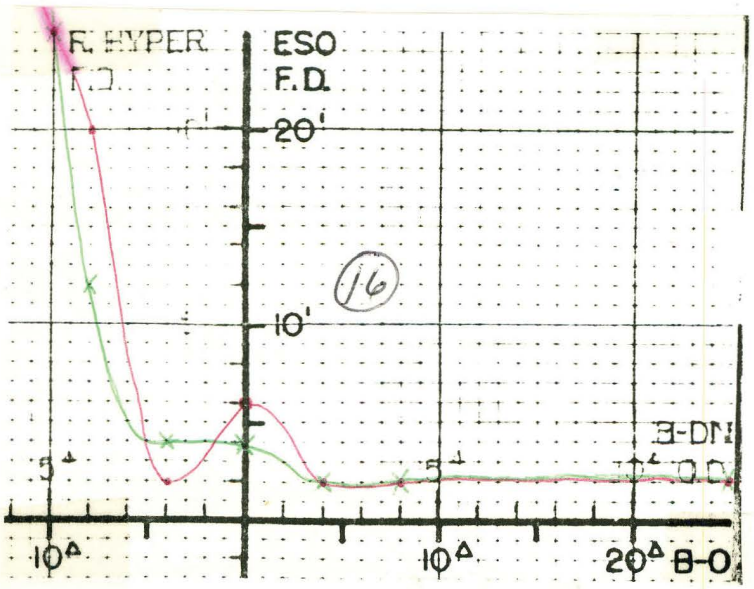
Patient 15.

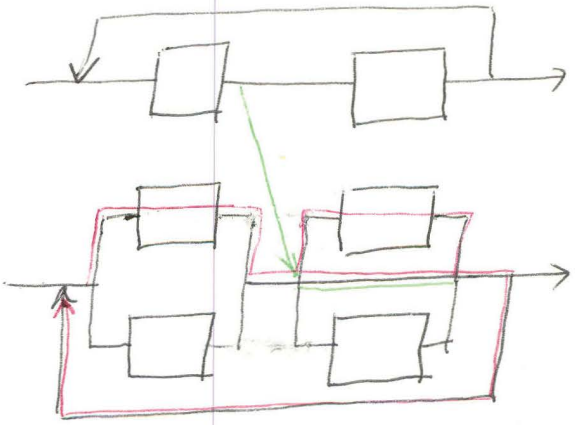
Type II curve adapted well to base in.

Patient 16.

Here is an example of atype II curve which shows basically no adaptation. Interestingly, this patient has an exophoria and an eso fixation disparity. We theorize that this is a patient who lacks convergence disparity detectors. Possibly there are very few such detectors or the detectors have a very low gain compared to the divergence disparity detectors.) The disparity input must go through the fast divergence integrator. Like the fast convergence adaptor, the slow convergence adaptor is lacking or deficient. The fast divergence output must either go through the slow divergence adaptor or go right through the slow

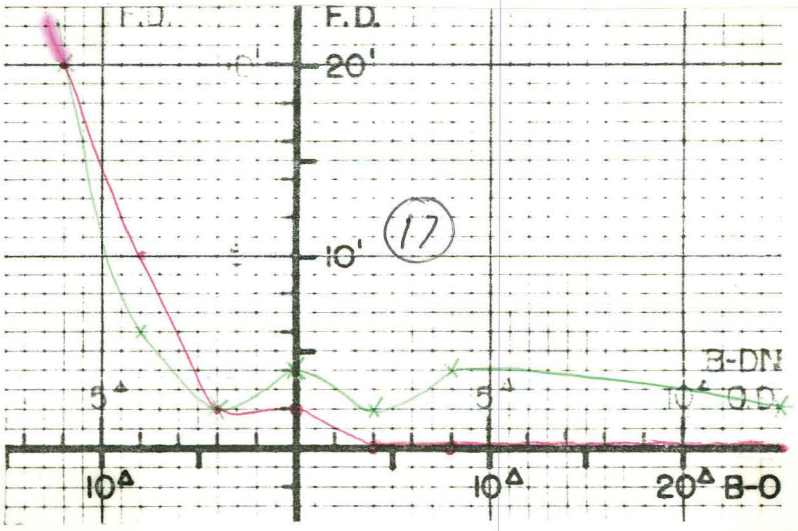
vergence integrator without being amplified. Even if accommodative convergence is fed in to compensate for the divergence, it cannot be amplified efficiently and the resulting divergence innervation causes the patient to be exophoric. This would theoretically cause an exo fixation disparity (innervation to converge), but since this patient has such poor convergence detectors the fixation disparity must remain eso in order to give any innervation to the vergence system.



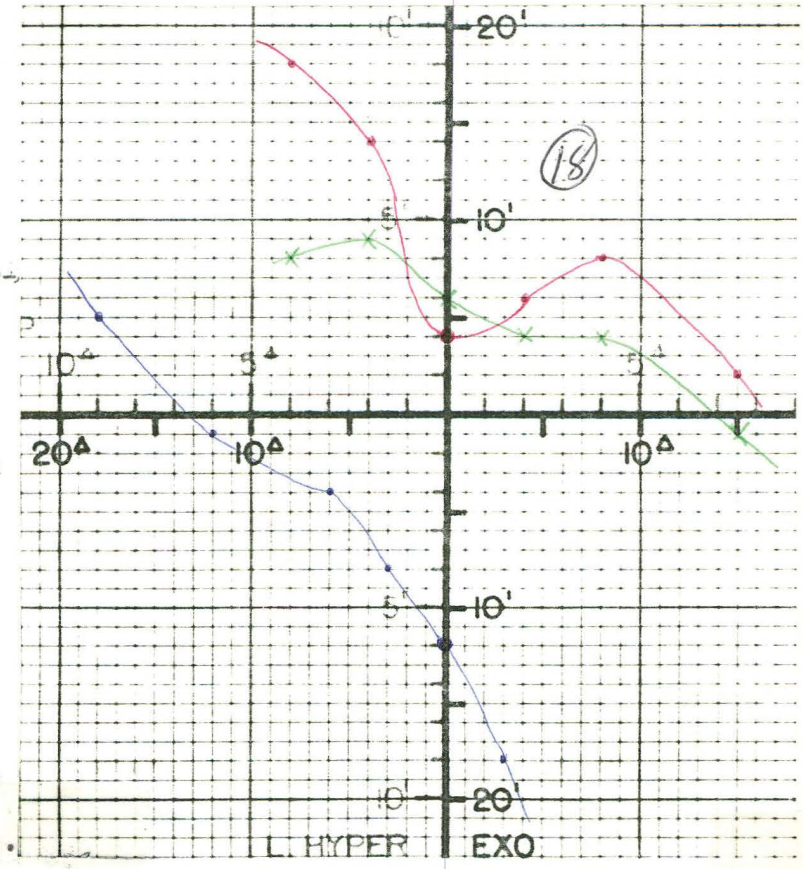


Patient 17.

This type II showed little if any adaptation to base in. The base out side seems to have adapted to the patients habitual level. We have just suggested



that the flat portion of these curves is due to a lack of response owing to a defficiency in convergence detectors. This patient is using divergence detectors and accommodation to maintain fusion.

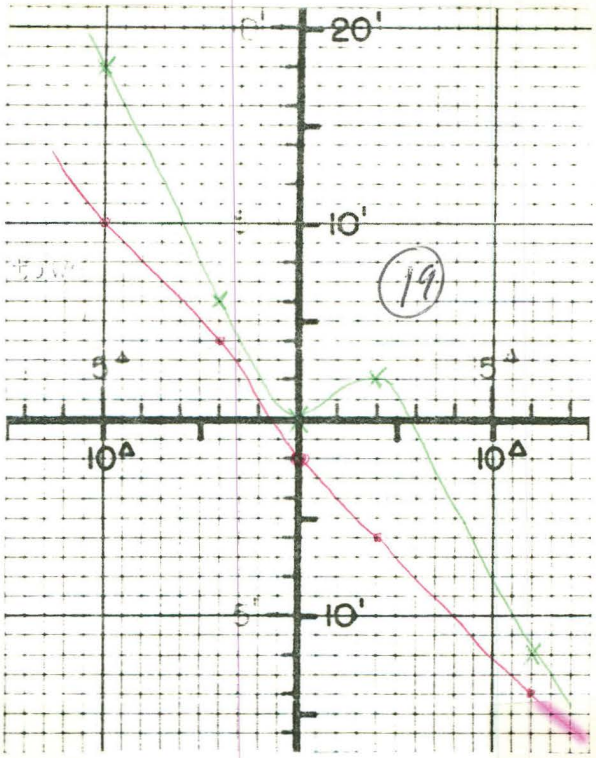


Patient 18.

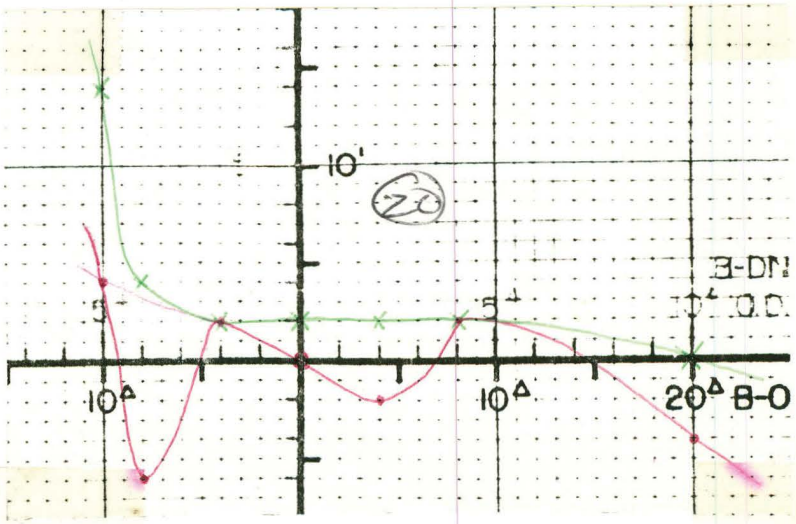
This patient is a 40 year old beginning presbyope who presented with symptoms secondary to convergence insufficiency. The lower curve (blue) wastaken at that time. Following this she underwent several weeks of convergence training which resulted in improved convergence skills and eradication of symptoms. It was at this time that our test was given. The adapted curve shows that this patient's slow vergence integrator has become more efficient. The upward swing on the base out side of the immediate curve, as well as the change of location of the curve after training suggest that this patient has learned to use accommodative innervation to help meet the convergence demand.

Patient 19.

The very steep type I curve shown here is typical in that the



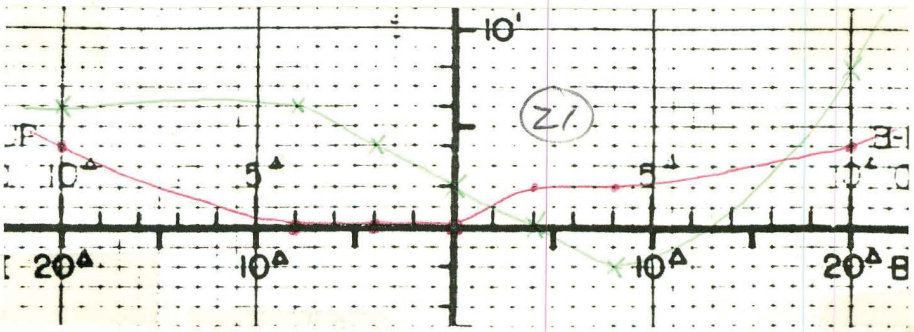
patient is symptomatic. She has some accommodative spasms and may be a pseudomyope. She has some tendency toward (pseudo?)convergence excess and additionally has a small vertical phoria which is uncorrected. The AC/A ratio seems to be slightly high (~7/1) but the exact amount is not presently determinable. There is probably always some accommodative convergence feeding into the vergence system (see the model) due to the high AC/A and/or accommodative spasms; this constant innervation to convergence forces her slow convergence integrators to work. Slight adaptation is, therefore, shown on the base out side. The slow divergence integrator apparently does not work very well. The curve inverts on the base in side, illustrating stress on fast fusional vergence.



Patient 20.

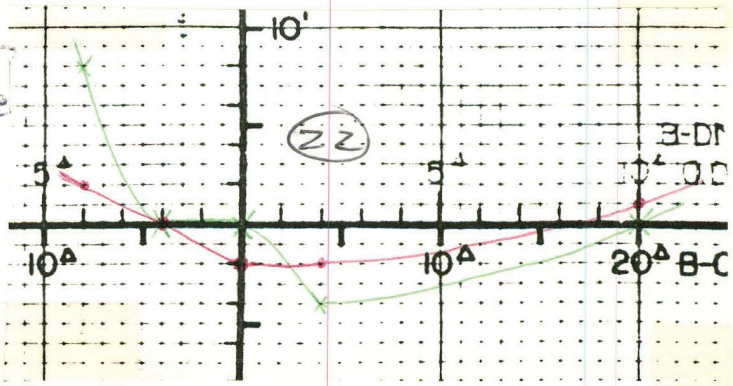
This patient has a very high AC/A ratio and, additionally, appears to have some accommodative fluctuations. The adapted curve is much more stable and appears to show inversion on the base in side and slight adaptation on the base out side. Again, this may be because the high

AC/A causes constant accommodative convergence which works against the slow divergence adaptor to cause base in inversion, and works with the slow convergence adaptor to cause base out adaptation.



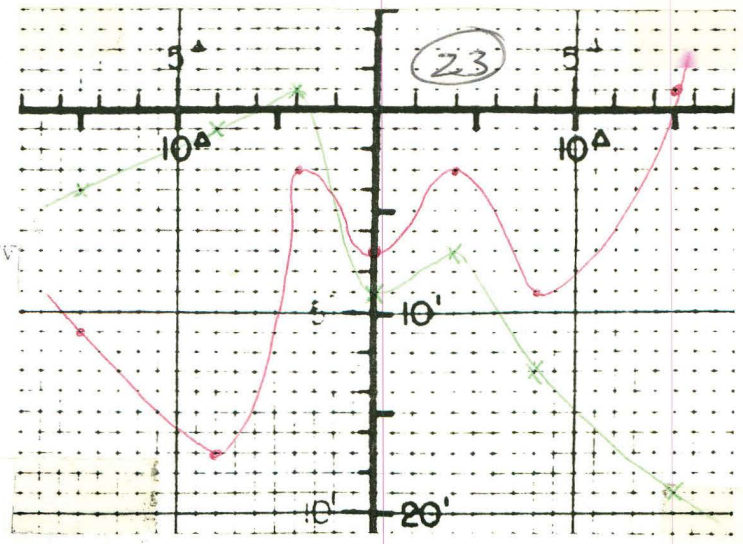
Patient 21 and 22.

These patients are very similar. Number 21 is a basic exo with a tendency toward convergence insufficiency. He has a base out problem which he compensates for by using accommodative convergence. Number 22 is a pseudomyope due to constant over-accommodation. Both are symptomatic. The constant accommodative congergence accounts for the upward slope on the base out side of both curves. This accommodative convergence may also be working cooperatively with the slow convergence integrator to cause a slight adaptation on the base out side. On the base in side, the accommodative convergence works against the slow divergence integrator. As a result the curve inverts, i.e. the fixation disparity increases to give further divergence innervation to the system.

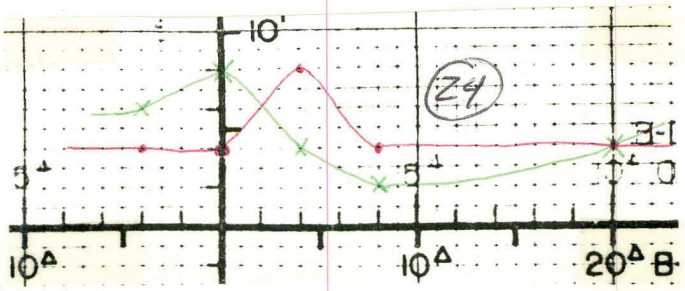


Patient 23.

This patient has problems with accommodative spasms and



infacility. Her curves obviously reflect the constant fluctuations. The adapted curve is a little more stable and, as such, may be a truer measure of the patient's real curve. The immediate curve apparently reflects whatever level of vergence response combined with her accommodative level at that moment to yield the alignment of the nonius. When given a chance to get used to the demand, her accommodation can find its desired response level and give a more stable curve.



Patient 24.

This is apparintly a type IV curve belonging to a patient with a very high AC/A ratio and problems with base in stimuli. He basically shows no adaptation, and this may be because both his divergence and convergence integrators are deficient. (Both curves are almost straight lines, possibly due to very poor vergence disparity detectors.) The high AC/A causes constant convergence innervation which he has no control over and this results in a constant esophoria. Probably because he has no way to release this involuntary convergence, the fixation disparity is also eso. This gives him divergence innervation, but he simply cannot meet the demand because the high AC/A ratio causes him to converge. Consequently, he has a low base in range (difficulty with divergence).

Adaptation With Lenses

Ogle has shown that a fixation disparity curve can be generated using lenses instead of prisms. Since the model shows accommodative convergence entering the vergence loop before the slow neuronal integrators, we should also be able to produce a prism adaptation with lenses.

Previous studies of prism adaptation have used phoria as well as fixation disparity to demonstrate adaptation. As an adjunct to the present study, the following data was collected. The patient's near phoria was measured, followed by his gradient phoria through a +1.00D add. The patient's negative relative accommodation was then determined. Fusion was maintained through these lenses for approximately 15 seconds. (This represents a convergence demand. One could produce a divergence demand by employing positive relative accommodation). The plus lens power was then reduced once again to +1.00D and the gradient phoria remeasured.

Phoria	Gradient-1	Gradient-2
7 exo	12 exo	2 exo
4 exo	8 exo	4 exo
16 exo	18 exo	14 exo
2 eso	ortho	3 eso
ortho	3 exo	5 exo
ortho	8 exo	5 exo
4 eso	2 exo	2 eso
7 exo	11 exo	5 exo
3 exo	6 exo	6 exo
6 eso	2 exo	3 eso

The data suggests that these patients do show varying degrees of adaptation to lens induced convergent stimuli. The most interesting case is the first one. According to the Duane-White system of classification this patient is slight convergence insufficient. The reduction in gradient phoria (12→2) after convergent stimulation demonstrates an efficient adaptation and helps explain this patient's total lack of symptoms.

Conclusion

The present study demonstrates that prism adaptation can be measured with fixation disparity after one minute of binocular fusion. Healthy binocular systems, especially type I's and to a lesser degree type II's, are most likely to show this adaptation. Lack of adaptation can often be explained by deficiency of the slow neuronal integrators, lack of disparity detectors, or accommodative fluctuations. These variations are most often seen in symptomatic patients.

An alternative clinical tool (lenses) for measuring degree of prism adaptation is also suggested.

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