Fusion Lock Diameters and the Forced Vergence Fixation Disparity Curve in Symptomatic Subjects

by

Randall Riemer

Steven Bierlein

Advisor: J. James Saladin, O.D., Ph.D.

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Abstract

There have been previous studies investigating the effect of fusion lock diameters on forced vergence fixation disparity with both asymptomatic and symptomatic patients. A recent study showed no effect with variable fusion lock diameters with asymptomatic patients while symptomatic patients showed a noticeable change in the disparity curves. The intent of this investigation is to determine characteristic patterns of forced vergence fixation disparity curves with variable fusion lock diameters in symptomatic patients.

Introduction

Forced vergence fixation disparity curves (f.d. curves) have long been a topic of research. These disparity curves have been used to explain the physiological mechanisms of how the two eyes function together and also as a clinically oriented diagnostic tool in assessing binocular anomalies. Saladin and Sheedy[1] have stated that phoria, vergence, and f.d. curve parameters are the most valid measures of oculomotor balance. Those with abnormal oculomotor balance may have symptomatic complaints of asthenopia, headaches, blur, and diplopia.[2]

Early studies by Ogle indicated that the fixation disparity curve would pivot clockwise and decrease its horizontal width as the fusion contour diameter is increased.[3] It was presumed that by changing the fusion lock diameter, a given amount of vergence stress would result in larger fixation disparities as the fusion lock diameter was increased. Explanations for this are based upon the concept that larger diameter fusion locks correspond to larger Panum's areas thus allowing more "retinal slip", thus resulting in greater disparity before a diplopic response is noted. The second is that the locomotor sensitivity of the retinal elements decrease with increasing retinal eccentricity. It was assumed, at this time, that the associated and dissociated phorias were directly correlated in both quantity and direction. But Ogle[3] noticed that not only was there a poor correlation in the size of the phorias, but also in the direction.

Following Ogle's publication, Shepherd found that fusion contour size had no effect on fixation disparity. With all this new evidence that contradicted the prevailing theory of fixation disparity, Ogle[4] repeated his original study on the effect of fusion contour diameter on the f.d. curve. He concluded by saying that some subjects showed an effect and others did not, and he really never gave an explanation for this.

Carter[5] compared f.d. curves using foveal versus peripheral fusion locks and found that the f.d. curve becomes steeper when only the peripheral fusion lock is used.

In 1967 Ogle[4] presented new ideas on fixation disparities. He used a model consisting of accommodative convergence and convergence accommodation and their interactions with the fixation disparity curve. He suggested that fixation disparity may act as a stimulus to the fusion mechanism. Stark[6] has stated that it is not defocused images that drive accommodation, but rather that the disparate images drive vergence and vergence drives accommodation.[7] This has been found to be the main mechanism for change of focus.

A control systems analysis was designed to elucidate the actions of disparity and the fusion mechanism. Works on negative feedback control systems of accommodation and disparity vergence mechanisms were summarized by Toates[8,9] and the results were combined into a unified model as typified by Semmlow and Venkiteswaran.[10] The model shown in figure 2 is a version of the model proposed by Shor[11] that has been modified by Saladin to emphasize two concepts developed by Shor[12] in another paper. In Shor's control system design, he has established four vergence mechanisms which include fast and slow convergence adaptation and fast and slow divergence adaptation. The fast vergence adaptation (F.V.A.) mechanisms are in the forward controller portion of the disparity vergence loop, act as a fast neural integrator with a time course of a few seconds or less, and receive input through their accompanying disparity detection mechanisms. Jones[13] has summarized the evidence for these separate disparity mechanisms. The slow vergence adaptation mechanisms are found before the input for the accommodative loop. The flat central portion of a normal fixation disparity curve represents the slow vergence adaptation, and the width of this flat portion is indicative of the strength of the operating range of the slow vergence adaptation mechanism.[11] The more vertical ends of the f.d. curve are functions of the fast vergence adaptation mechanism. The input to the slow adaptation mechanism is the output from the fast vergence adaptation mechanism, and the purpose of this slow mechanism is to relieve the fast mechanism of long term duty. There are two possible ways of obtaining a steep fixation disparity curve. The first way results when the fast vergence adaptation mechanisms output is weak causing input to the slow vergence system to be deficient. The other way occurs when the slow vergence system is weak; consequently, the curve must be largely dependent upon the fast vergence adaptation mechanisms.

A binocular dysfunction such as suppression may lead to an inability to utilize the disparity signals resulting in diminished input to the fast vergence system. Another point to note is that in certain individuals with a large heterophoria, the fixation disparity curve may have a steep y-axis intercept yet have a displaced flat central portion. The large heterophoria is the underlying cause for this abnormal f.d. curve. Saladin and Sheedy[1] have stated that a flat slope of the f.d. curve was associated with a lack of symptoms. They[2] also said the y-intercept is the single most discriminative fixation disparity variable.

Saladin and Carr[14] recently concluded in a research report that subjects with normal binocular vision show little if any f.d. curve pivoting about the x-axis intercept as eccentricity of the fusion contour is increased. They also noticed that subjects with abnormal binocular vision may pivot. They attempted to prove that a defect in the forward controller output would affect both the convergent and divergent slow vergence mechanisms while a defect in only the slow vergence adaptation, would tend to be one sided. Their results from the few subjects with abnormal binocular vision was indeed consistent with this hypothesis, but a larger population was needed to substantiate this principle.

Our goal in this research project is to test a larger population with abnormal binocular vision to determine if there are any characteristic patterns of forced vergence fixation disparity curves with variable fusion lock diameters. If there are characteristic patterns, we will attempt to relate them to the control systems model for clinical application.

METHODS

The subjects chosen were from a clinical population presenting with subjective symptomatic complaints. All subjects were between the ages of 20 to 30. The subjects were binocular in origin, but had abnormal binocular systems based on the fact that they had fixation disparity curves with y-axis intercepts of more than 4 minutes of arc eso or 6 minutes of arc exo and/or having slopes of more than one minute of arc per prism diopter at one meter with a $1\frac{1}{2}^{o}$ fusion lock.

The apparatus consisted of two oppositely polarized vertical nonius lines which were located 1 meter from the subject. Each nonius line was 7' high by 1.7' wide and was illuminated from the back with a 7 watt incandescent bulb. A 1' high horizontal strip of tape painted with green-yellow fluorescent tempura paint was used to separate the two nonius lines. The upper nonius line was movable by the observer by using a screw drive mechanism attached to a cable. A potentiometer and voltmeter were used to read the lateral displacement of these nonius lines. Two horizontal lines were placed below the lower nonius line in order to stabilize accommodation. These horizontal lines were 34 minutes of arc in length and 1 minute of arc high. Both the horizontal lines and lower nonius lines were flashed on for ½ second and off for 1½ seconds. See figure 1 for a diagrammatic representation. The size of the fusion contour was controlled by placing a series of masks containing an annulus to surround the nonius lines. Four masks were used with an annulus of 6 degrees, 3 degrees, 1½ degrees, and 3/4 degrees, and each annulus was painted with the green-yellow fluorescent paint. The background was painted with a flat black. The ratio of outer annulus diameter to inner annulus diameter was held constant at five to three.

A Burton lamp was placed directly over the subject's head which caused the green-yellow paint to fluoresce and all room lights were extinguished to avoid miscellaneous targets acting as fusion locks. The subject was at a distance of one meter while viewing the annular targets through his habitual prescription. Risley prisms were incorporated in the determination of the curves.

The subject's task was to fixate the upper nonius line and move it over the lower nonius by using a knob at hand level. The subject was instructed to place the upper nonius line over the lower nonius line by coming from the right side and then the left side in thirty seconds or less. The investigator recorded the midpoint of the bracketed range.

The order of presentation for the Risley prisms was 0, 3 B.I., 3 B.O., 6 B.I., 6 B.O. followed by whatever prisms were necessary in order to determine a characteristic fixation disparity pattern for each annulus. The annulli were presented in a random order.

RESULTS

Analysis of the fixation disparity curves of symptomatic patients allows one to take note of characteristic patterns that do exist. Eleven convergent insufficient subjects were analyzed and these individuals show one of two types of curves. The predominant f.d. curve they show is one that is extremely steep on both the base in and base out side. The second type still shows steepness on the base in side; however, the base out side tends to become less steep, becoming somewhat irregular, and may even flatten out. We concluded that this latter effect is brought about by the subject using voluntary vergence to meet the base out prism demands.

Three convergent excess subjects all had Type II fixation disparity curves and showed almost no exo disparity regardless of the fusion contour or prism demand level used.

The curves of all the subjects are found in this paper with brief explanations found on the curves and subsequent explanations made in the discussion section.

Three fixation disparity curves were taken on one basic exo subject. If the data is analyzed by the Fisher analysis of variance test and this yields an α level of $\alpha = .009$ which is significant. This supports the fact that pivoting is occurring on both the base in and base out sides of the fixation disparity curve. This individual has such poor F.V.A. that he relies heavily on his accommodative convergence in meeting fusional convergence demands.

Three f.d. curves were taken on another basic exo. In this subject pivoting was only found on the base out side of the fixation disparity curve which is supported by the Fisher analysis of variance resulting in an $\alpha = .022$ value. In this individual, $F = 6.97$ with 3/6 degrees freedom.

Four fixation disparity curves were taken on a convergence excess patient. This individual had a very unstable accommodative system; consequently, there was no significant α value. The subject has a classic F.V.A. suppression problem resulting in a degradation to the S.V.A. input.

DISCUSSION

As with all other physiological phenomena, there is no clear-cut distinction between normal binocular systems and abnormal binocular systems, but rather there is a gradual continuum between these two groups. This must be considered when analyzing the data in this study. Not all symptomatic patients will have curves that behave similarly or show commonalities. It becomes extremely difficult to generalize or summarize; therefore, we intend to select commonly observed behaviors of the f.d. curves, and we will attempt to explain them.

Fixation disparity curves of symptomatic patients are not nearly as predictable and easily interpreted as the f.d. curves of asymptomatic patients. This is largely because the true f.d. curve behaviors are masked by interactions between disparity vergence and voluntary accommodative convergence. Both accommodative convergence and convergence accommodation play a role in the vergence system and are sometimes heavily relied upon by patients with disparity vergence anomalies. Accommodative convergence interactions are indicated when there is a sudden variable change in the form of the f.d. curve such as the classic "hump" as seen in figures 6, 9, 11, and 17. Accommodative humps are fairly common among the convergent insufficients on the B.O. side of the f.d. curves. Figure 2 would explain this phenomenon in two ways. If a patient has a high threshold (insensitive or weak) for convergent F.V.A. and has normal convergent S.V.A., then a high exo disparity is needed to drive the F.V.A., thus causing a steep curve. This can be augmented by utilizing accommodative convergence thus taking the stress off the convergent F.V.A. and convergent S.V.A.; therefore, reducing the exo disparity. If the patient has a normal threshold for convergent F.V.A. but has a low gain output on the convergent S.V.A., there will be a high exo disparity needed to maintain and sustain fusion. Again, the accommodative convergence stimulation will decrease the output needed from the convergent S.V.A. and decrease the stress placed on the convergent F.V.A. resulting in fusion with a lower exo disparity. In summary, the accommodative convergence hump is used to relieve the need for a large disparity input to the disparity driven vergence system for maintenance of fusion.

The use of accommodative convergence during forced vergence disparity measurements appears to be influenced by the fusion contour target size. In general, the larger the annulus, the greater the tendency to use voluntary convergence. One would expect this since the eccentric retina is less sensitive in blur interpretation; therefore, the larger the fusion contour, the easier it may be to have either convergence accommodation or accommodative convergence.drive the system resulting in an accommodative hump.

There are exceptions to this pattern where there seems to be no relation between annular size and the degree of accommodative convergence in play as seen in figures 22 and 9. When an individual has an unstable vergence system or a sensory block, such as an uncorrected refractive error, the subject may lose his sensitivity to blur interpretation. This loss of feedback to the accommodative loop allows an increased ability to use the accommodative convergence mechanism to drive the vergence system for both large and small target sizes. This explains the importance of blur interpretation and how it can hinder the success of a vergence training. This individual must once again learn how to interpret blur so that fusional convergence can replace the presently used accommodative convergence.

As previously theorized by Saladin and Carr, normal binocular f.d. curves show no pivoting about the x-axis intercept with increasing fusion contour target size, whereas abnormal binocular system f.d. curves pivot. We have noticed similar pivoting with many symptomatic f.d. curves, but not with all of them. Once again, the degree of intensity of the symptoms must be considered. The subjects with the most intense symptoms show the largest degree of pivoting and this would be expected.

Whether or not pivoting occurs depends largely on the plant load (phoria), and the ability of the system to support that load. A pivot occurs when the system breaks down and is unable to support the load. Either there is too large a load, or there is insufficient innervation; furthermore, both will produce the same effect. The reason for insufficient innervation is either a poor gain or long time factor in the S.V.A. or F.V.A. systems.

The majority of the patients who were easily classified into a Duane-White syndrome support the theory that only the deficient areas of the vergence system show the pivoting phenomenon. In general, the convergent insufficients (figures 6-16) have a convergent F.V.A. that cannot handle the large plant load at near. This results in a pivoting of the B.O. side. The steep slope of convergent insufficients indicates that there is little if any functional S.V.A mechanism. The convergent excess (figures 17-19) has a divergent F.V.A. that cannot handle the plant load at near, which results in pivoting of the B.I. side. The basic exo (figures 3 and 21) has a large plant load at both distance and near. In this circumstance, the F.V.A. cannot handle the plant load at any distance; therefore, voluntary convergence is used to sustain fusion without even attempting to use the F.V.A. mechanism. This is why the curve generally appears jagged and fairly flat. The basic eso (figures 22-24) also has a large plant load at all distances but cannot use voluntary convergence to overcome the disparity. These individuals show a pivoting on the B.I. side in addition to a large variation in the amount of accommodative convergence incorporated into the vergence system. It appears that the subject uses the least amount of accommodative convergence as possible to sustain fusion, yet have a minimally blurred target.

There also appears to be some patterns associated with all of the curves in general. Many of the curves demonstrate a characteristic pattern whereby the $3/4^{\circ}$ and $1\frac{1}{2}^{\circ}$ fusion targets produce very similar curves, whereas the 3° and 6° fusion targets also produce similar curves; however, these curves show a steeper pattern. This can be observed in figures 3, 4, 5, 6, 8, 21, and 24. This correlates with the locomotor sensitivity function as described by Hampton and Kertesz[15].

While collecting our data, it was noticed that a few subjects initially could not fuse the target in a forced vergence situation; however, with time, they could slowly bring the targets to a fused position. This was particularly noted on the B.I. forced vergence side of the f.d. curve, and was independent of the fusion contour size. One would have to consider three possible reasons for this occurrence. The first and most common reasoning would be that the subject is simply relaxing voluntary accommodative convergence until finally the targets are close enough together to fuse. Another possibility to consider is the subject is allowing time for the divergent S.V.A. to build up to the point where fusion is possible. Thirdly, in our forced vergence sequence, we alternated B.I. and B.O. prisms. In doing so, the subject was always returning from high B.O. forced vergence to a high B.I. forced vergence, and the delay in B.I. fusion could have been proportional to the decaying time of the convergent S.V.A. just built up from the previous B.O. forced vergence. If the latter is true, then this means there is some connection between decay and build up times of the convergent and divergent S.V.A.'s, which is why the sequence of forced vergence prism presentation order makes a difference in the shape of the f.d. curves.

Normally the fixation disparity is in the same direction as the measured phoria, but it was noticed in the past that some of the population have a fixation disparity in the opposite direction of the phoria. We have noticed this very same phenomenon as seen in figures 7 and 8. It appears that this occurs only in individuals with abnormal binocular systems. One would ask why such an occurrence would exist. An exophore would have to overconverge in order to get an eso-disparity. When accommodative convergence is activated to sustain fusion, the individual really has no way of knowing exactly how much accommodative convergence to use other than the detection of blur. Therefore, the subject may place more accommodative convergence into play than is actually needed, causing the need for fusional divergence to counter the excessive accommodative convergence. The result of this is an eso-disparity. If this individual was to be trained to use fusional convergence rather than accommodative convergence, then one would expect the curve to develop into an exo-disparity pattern.

CONCLUSION

In summary, we make the following conclusions:

1. Subjects with abnormal binocular vision show a pivoting of their f.d. curves with increasing eccentricity of the fusion contours. The pivoting tends to occur only where the vergence system is deficient.
2. Each classification of the Duane-White syndromes shows definite characteristic patterns in the forced vergence fixation disparity curves. These patterns may be difficult to interpret due to interactions of the accommodative convergence mechanism with the fusion vergence mechanism.
3. The behaviors of the f.d. curves support the clinical application of the servo-mechanism theory to the oculomotor system.
4. Further studies should be made on ways to better develop or train specific deficient areas of the oculomotor servo-mechanism for a more efficient clinical training program.

FIGURE CAPTIONS

Figures 3a - 3c	L.H., a 23-year-old W.M., is a basic exo. The three sets of f.d. curves had the following results of statistical analysis of variance: $F = 10.36$ with 3/6 degrees of freedom. This shows that the patient has an extremely high degree of consistency and repeatability. Each set of curves show an atypical rotation of both the B.I. and B.O. sides. Note the relationships of the $3/4^{\circ}$ to the $1\frac{1}{2}^{\circ}$ and the 3° to the 6° fusion targets. 6m. phoria 22^{Δ} exo; 40 cm. phoria = 28^{Δ} exo.
Figures 4a - 4c	R.V., a 23-year-old W.M. is a convergent insufficient. The three sets of f.d. curves had the following results with the statistical analysis of variance: $F = 6.97$ with 3/6 degrees of freedom. This shows that the patient has a high degree of consistency and a repeatability. Each set of curves show a classical B.O. pivoting effect about the x-axis intercept. 6m. phoria = 2^{Δ} exo; 40 cm. phoria = 6^{Δ} exo.
Figures 5a - 5d	S.B., a 24-year-old W.M., is a convergent excess. This patient could not repeat the f.d. curves with much consistency. The inconsistency appears to be the result of varying degrees of voluntary convergence incorporated into each set of f.d. curves. 6m. phoria = ortho; 40 cm. phoria = 6^{Δ} eso.
Figure 6	M.W. is a 25-year-old W.M. and a convergence insufficient. The curve shows that the patient is well adopted to the insufficiency as the consistency of the curves show. Note the accommodative convergence is used in the center of the $3/4^{\circ}$ and 6° curves and also the pivoting of the curves on the B.O. side. 6m. phoria = 10 exo; 40 cm. phoria = 7^{Δ} exo.
Figure 7	S.V., a 22-year-old W.M. is a symptomatic convergence insufficient as indicated by the steep curves and the poor B.O. acceptance. Here is an example of a patient with an exophoria and an eso disparity. We explain this by saying that the patient is using an excessive amount of voluntary accommodative convergence. 6m. phoria = 3^{Δ} exo; 40 cm. phoria = 9^{Δ} exo.
Figure 8	R.C., a 27-year-old W.M. is a convergence insufficient with a steep slope typical of symptomatic convergence insufficients. This patient also has a disparity in the opposite direction of the phoria. The B.O. pivoting appears to rotate about the y-axis intercept rather than the typically pivoted x-axis intercept. 6m. phoria = 1^{Δ} exo; 40 cm. phoria = 5^{Δ} exo.

Figure 9	K.W., A 21-year-old W.M., is a symptomatic convergent insufficient. This individual does not show the classic steep curve that the typical convergent insuffient shows because she is using voluntary vergence to drive the BO.O system. In analyzing the curves on the B.O. side, one may note the presence of accommodative humps which indicate the use of accommodative convergence. Pivoting is present on the B.O. side. 6m. phoria = ortho; 40 cm. phoria = 6^{Δ} exo.
Figure 10	B.H. is a 21-year-old W.M. who is a symptomatic convergent insufficient. The curve became steeper upon presenting successive annuli. This subject has extremely poor B.O., F.V.A. since he broke fusion with 6 prism diopters B.O. This poor F.V.A. results in a weak S.V.A. system. 6m phoria = 6^{Δ} exo; 40 cm. phoria = 10^{Δ} exo.
Figure 11	R.R. is a 23-year-old W.M. and is a symptomatic convergence insufficient. Note the order of presentation of the fusion contour targets (6°, $3/4^{\circ}$, $1\frac{1}{2}^{\circ}$, 3°) and how the patient adapted to the B.O. side by going from a high exo disparity to an eso disparity. This patient has either learned to completely support the weak S.V.A. with voluntary accommodative convergence or his S.V.A. had a very long time constant. 6m. phoria = 4^{Δ} exo; 40 cm. phoria = 9^{Δ} exo.
Figure 12	S.M., a 22-year-old W.M., is a convergent insufficient with a steep slope on the fixation disparity curve. The order of presentation was $3/4^{\circ}$, 6°, $1\frac{1}{2}^{\circ}$, 3° and this individual has boosted the voluntary vergence to compensate for B.O. prism. 6m. phoria = 2^{Δ} exo; 40 cm. phoria = 7^{Δ}exo.
Figure 13	B.H., 29-year-old W.M., is a convergence insufficient with a poor B.O. acceptance. The B.O. side is pivoting slightly and is not steep because this individual is probably using voluntary convergence to maintain fusion. 6m. phoria = 2^{Δ} exo; 40 cm. phoria = 10^{Δ} exo.
Figure 14	B.W., a 23-year-old W.M., has an elevated convergent insufficient type fixation disparity curve. This symptomatic subject shows a very steep slope which shows that the F.V.A. is heavily relied upon if a disparity exists. For some reason, the S.V.A. has grown weak in this individual, and it may be from a lack of use. 6m. phoria = 2^{Δ} exo; 40 cm. phoria = 8^{Δ} exo.

Figure 15	B.R., a 27-year-old W.M., is a convergent insufficient. This curve is very steep and has a slope greater than one. This individual has poor S.V.A. and also poor B.O. acceptance. There is a definite pivoting on the B.O. side. 6m. phoria = 1^{Δ}exo; 40 cm. phoria = 6^{Δ}exo.
Figure 16	M.K., a 23-year-old W.M., is a classic symptomatic convergent insufficient. This individual shows a very steep slope and a y-intercept of greater than 6 exo. There is pivoting on both the B.I. and B.O. side. The S.V.A.'s effectively non-existent and the subject relies totally on his F.V.A. to sustain fusion under forced vergence conditions. 6m. phoria = 1^{Δ}exo; 40 cm. phoria = 5^{Δ} exo.
Figure 17	L.J., a 24-year-old W.M., is a convergence excess. Both ends of the curves are fairly consistent, whereas the central portions vary with a distinct pattern. With the increasing fusion contour diameter, the f.d. curves progressively cross the y-axis at a lower amount of eso disparity. This occurrence is easily explained by the decrease in accommodative demand for larger, more eccentric fusion contours. 6m. phoria = ortho; 40 cm. phoria = 4^{Δ} eso.
Figure 18	P.M., a 24-year-old W.M., is a symptomatic convergence excess. Note the mild pivoting effect on the B.I. side with the B.O. side being flatter and less variable. 6m. phoria = ortho; 40 cm. phoria = 3^{Δ} eso.
Figure 19	R.H., a 23-year-old W.M., is a symptomatic convergence excess with an eso disparity at the y-intercept. With the exception of the 6° annulus f.d. curve, there is marked B.I. pivoting about the x-axis intercept. The 6° annulus f.d. curve is not at all what would be expected, as if some threshold was finally reached whereby tonic vergence relaxed to allow a lower disparity with the B.I. forced vergence. 6m. phoria = 1^{Δ} eso; 40 cm. phoria = 11^{Δ} eso.
Figure 20	M.R., a 21-year-old W.M., is a symptomatic mild exophore at both distance and near. All four curves are identical except for the vertical displacement. The presenting order of the fusion contour diameters was 3°, 1½°, 6°, 3/4°, and this suggests that the patient progressively incorporated voluntary convergence to attempt to obtain zero disparity. 6m. phoria = 2^{Δ} exo; 40 cm. phoria = 3^{Δ} exo.

Figure 21	J.M. is a 24-year-old W.M. who is an asymptomatic basic exo with unstable accommodation. This curve shows variable B.O. responses with fairly stable B.I. responses. This individual appears well adopted to the exo pattern with the use of accommodative convergence. 6m. phoria = 6^{Δ} exo; 40 cm. phoria = 6^{Δ} exo.
Figure 22	S.Br., a 25-year-old W.M., is a symptomatic basic eso with an eso fixation disparity predominating. This individual is using his F.V.A. the majority of the time since there is little S.V.A. present. The hump that is present with the 3/4° annulus at 3^{Δ} B.O. is brought about by voluntary vergence. 6m. phoria = 6^{Δ} eso; 40 cm. phoria = 5^{Δ} eso.
Figure 23	R.S., a 25-year-old W.M., is a symptomatic basic eso with atypical pivoting. Both sides of the curves pivot and pivot about the same point, just above the x-axis intercept. The B.I. side shows the typical pivoting of increased disparity with increased annular diameter; however, the B.O. side pivots in the opposite direction. 6m. phoria = 4^{Δ} eso; 40 cm. phoria = 4^{Δ} eso.
Figure 24	D.F., a 25-year-old W.M., is a basic eso and the f.d. curves show the classical pivoting effect about the x-axis intercept on both the B.I. and B.O. sides. This suggests an F.V.A. problem since both sides pivot, resulting in both convergent and divergent S.V.A.'s being deficient. 6m. phoria = 3^{Δ} eso; 40 cm. phoria = 3^{Δ} eso.
Figure 25	W.F., a 27-year-old W.M., is a symptomatic mild esophore at both distance and near. If the x-axis intercept is inferred through extrapolation, then there is an excellent example of pivoting about the x-axis intercept. 6m. phoria = 2^{Δ} eso; 40 cm. phoria = 2^{Δ} eso.
Figure 26	W.W., a 21-year-old W.F., is a divergence excess. This subject has a large amount of exophoria at distance and appears to be using so much B.O. fusional vergence at distance that she is unable to release the positive fusional vergence at near, thus resulting in a reduced B.I. acceptance. 6m. phoria = 11^{Δ} exo; 40 cm. phoria = 2^{Δ} exo.

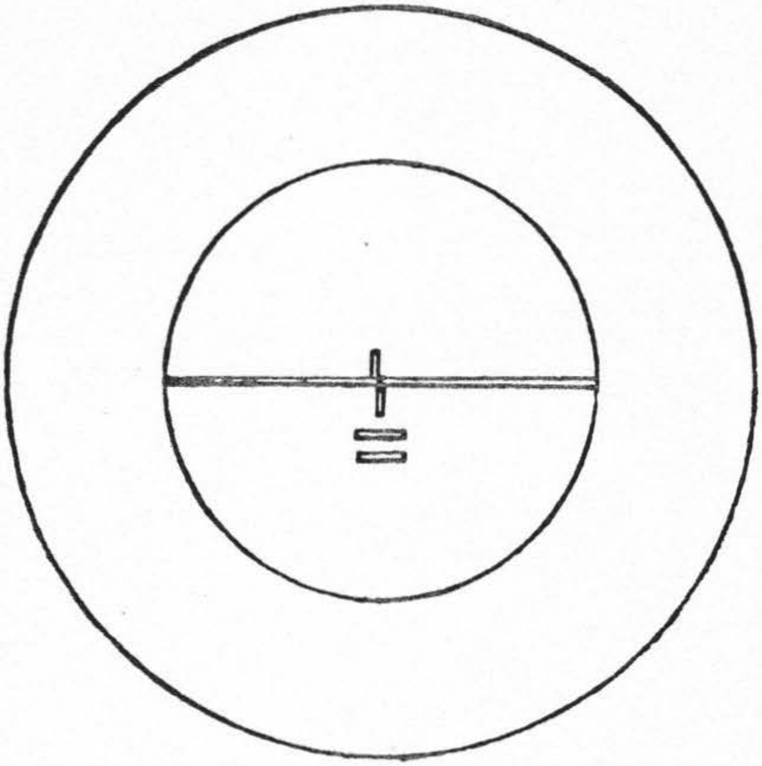

Figure 1

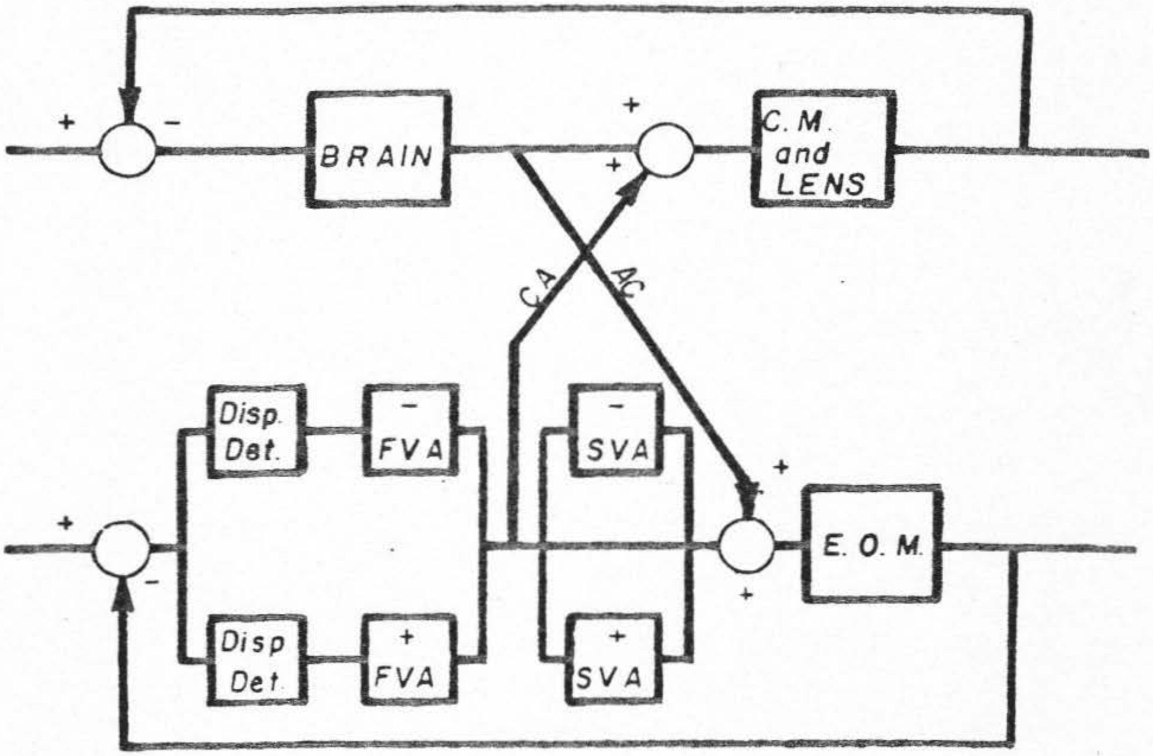

Figure 2

Figure 3a

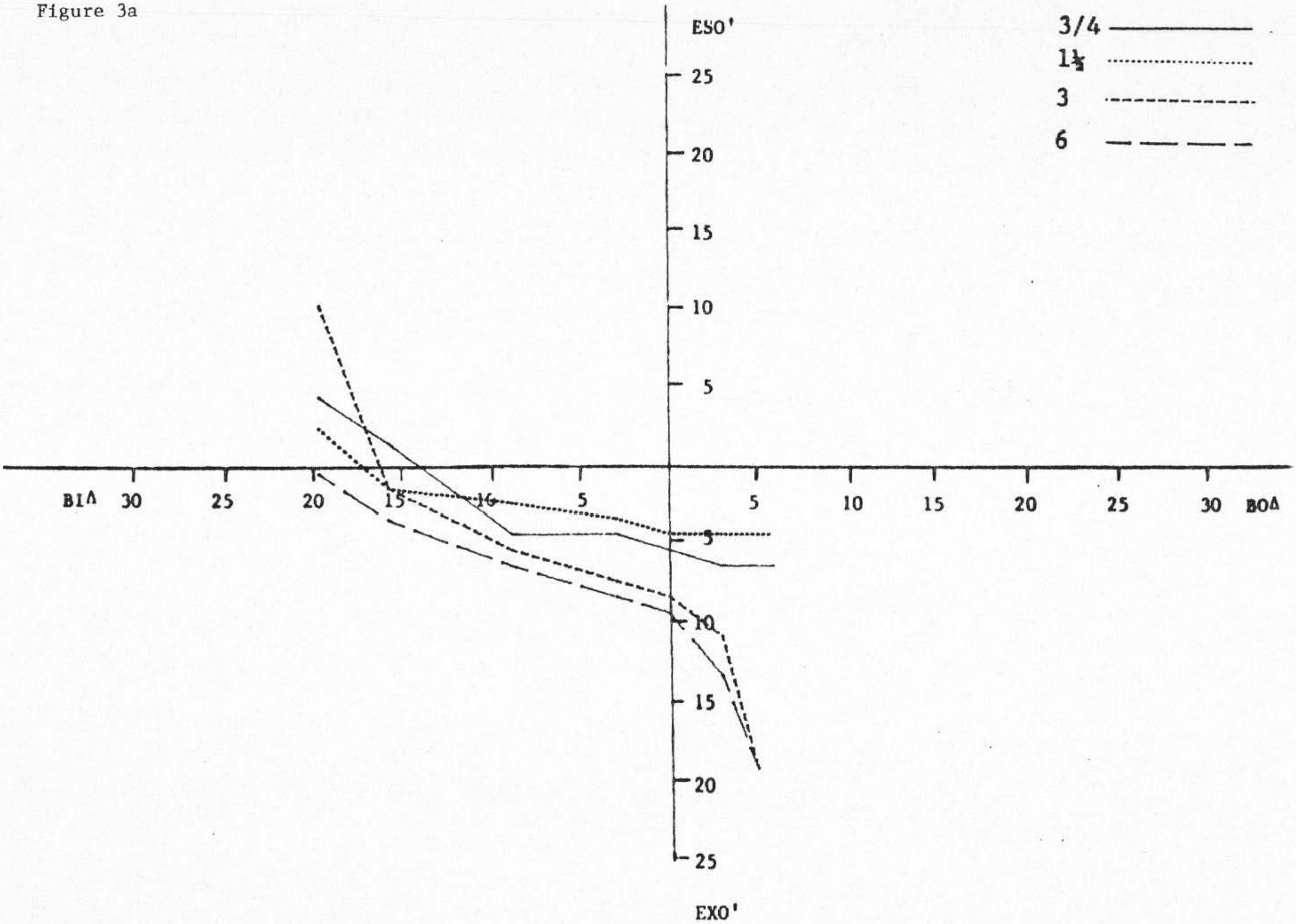
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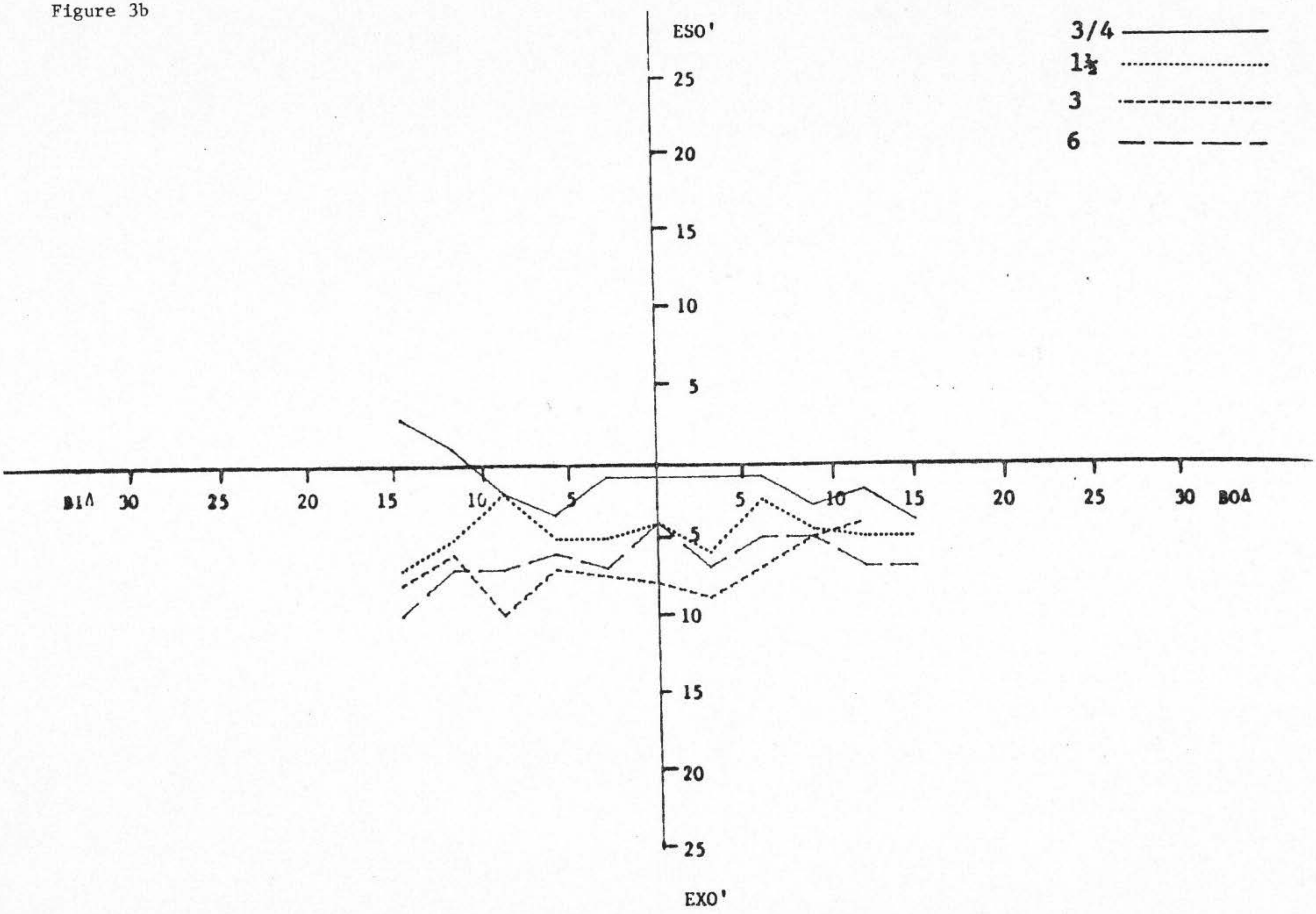
Figure 3b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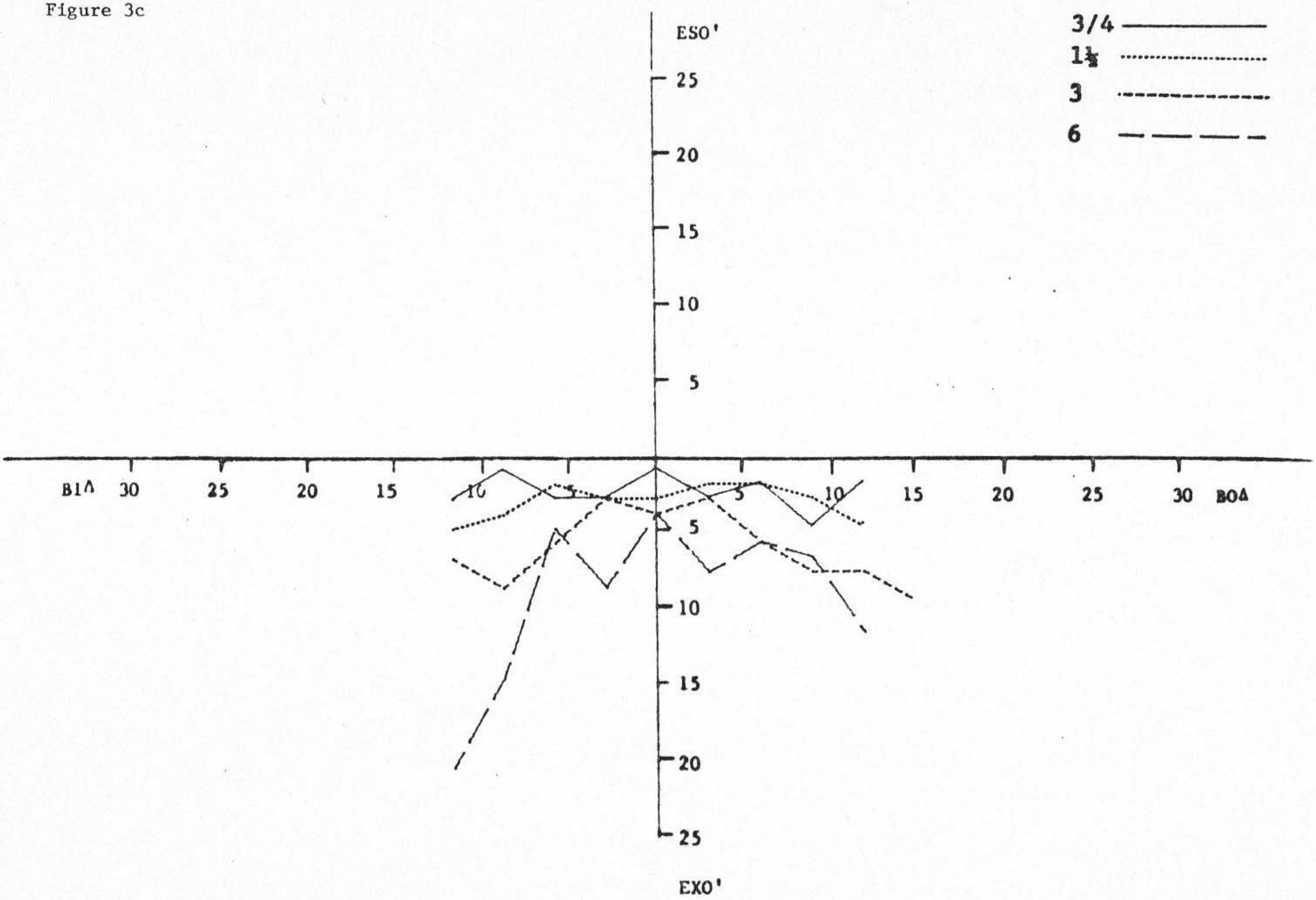

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Figure 4a

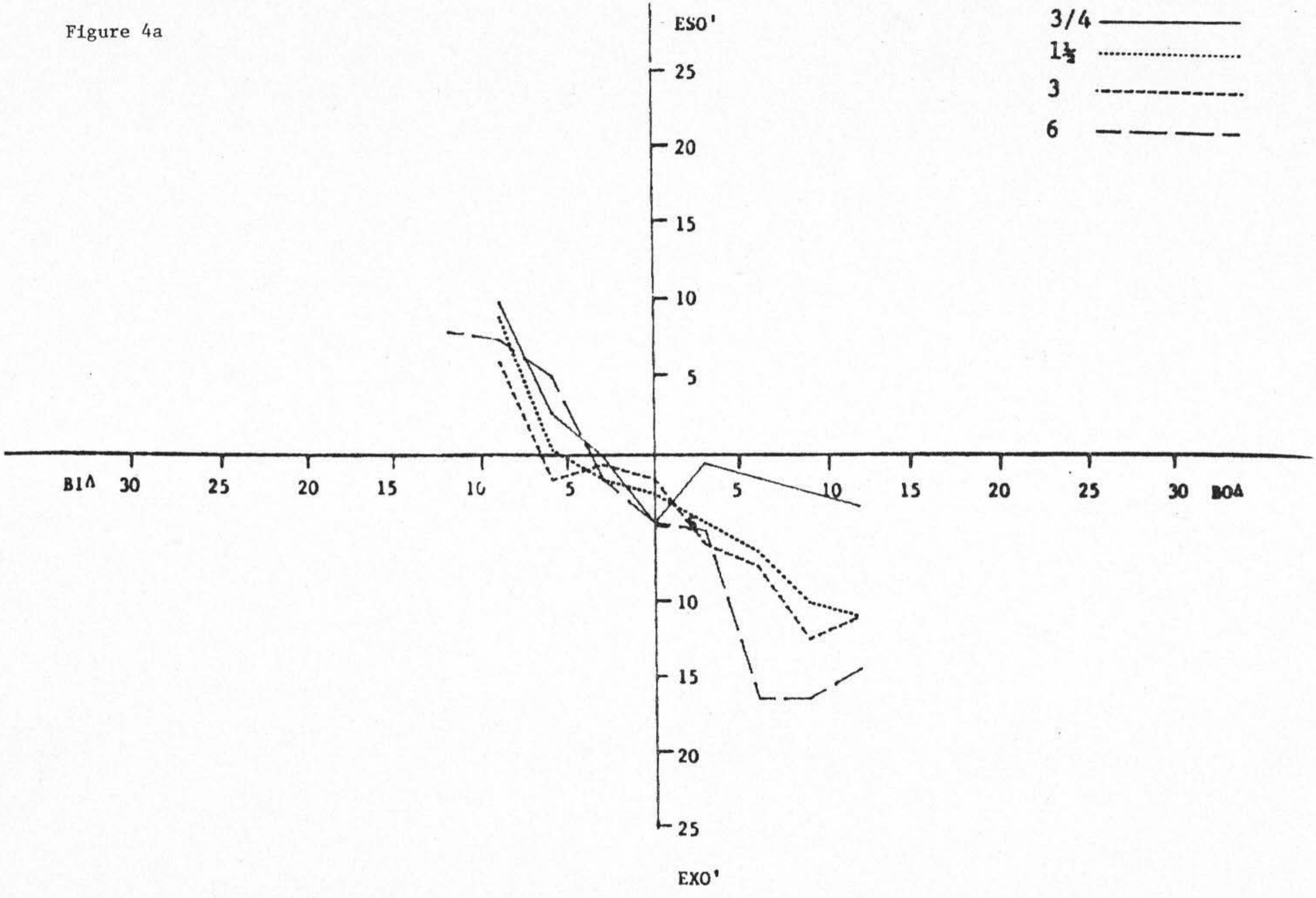
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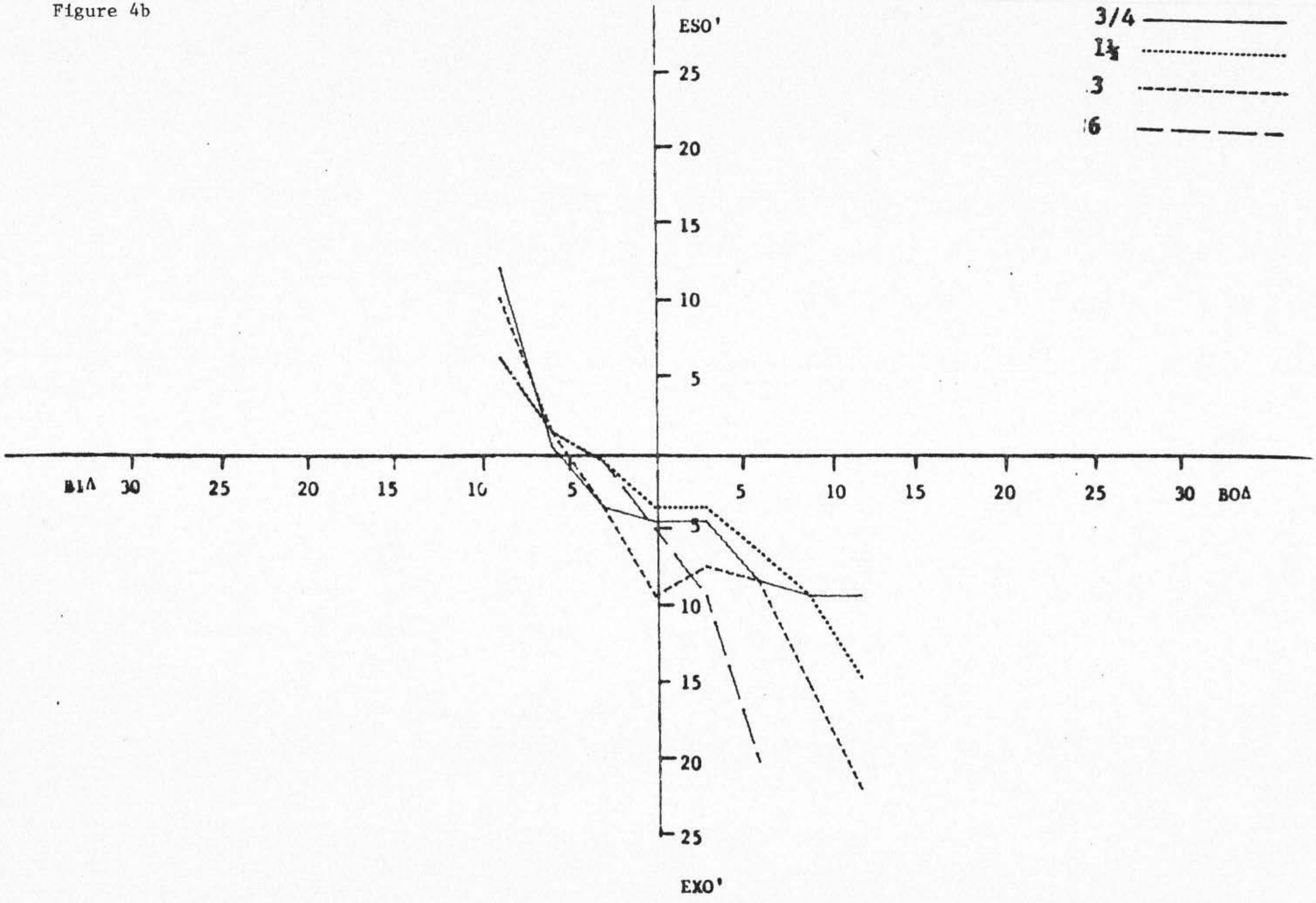
Figure 4b


3/4
1½
3
6
ESO'
25
20
15
10
5
5
10
15
20
25
EXO'
BIΔ
30
25
20
15
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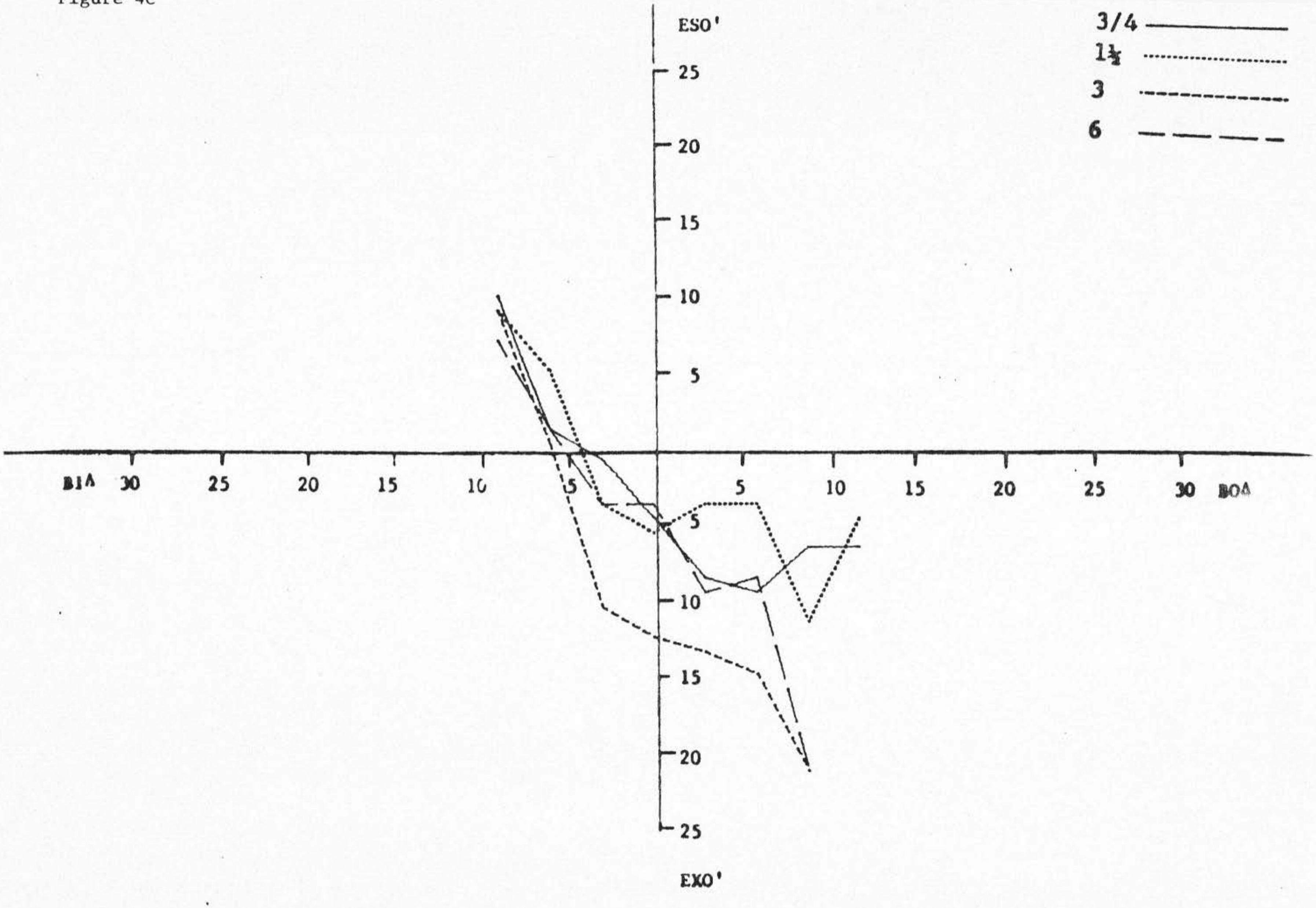
5
5
10
15
20
25
30
BOΔ

Figure 4c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5
10
15
20
25
EXO'
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 5a

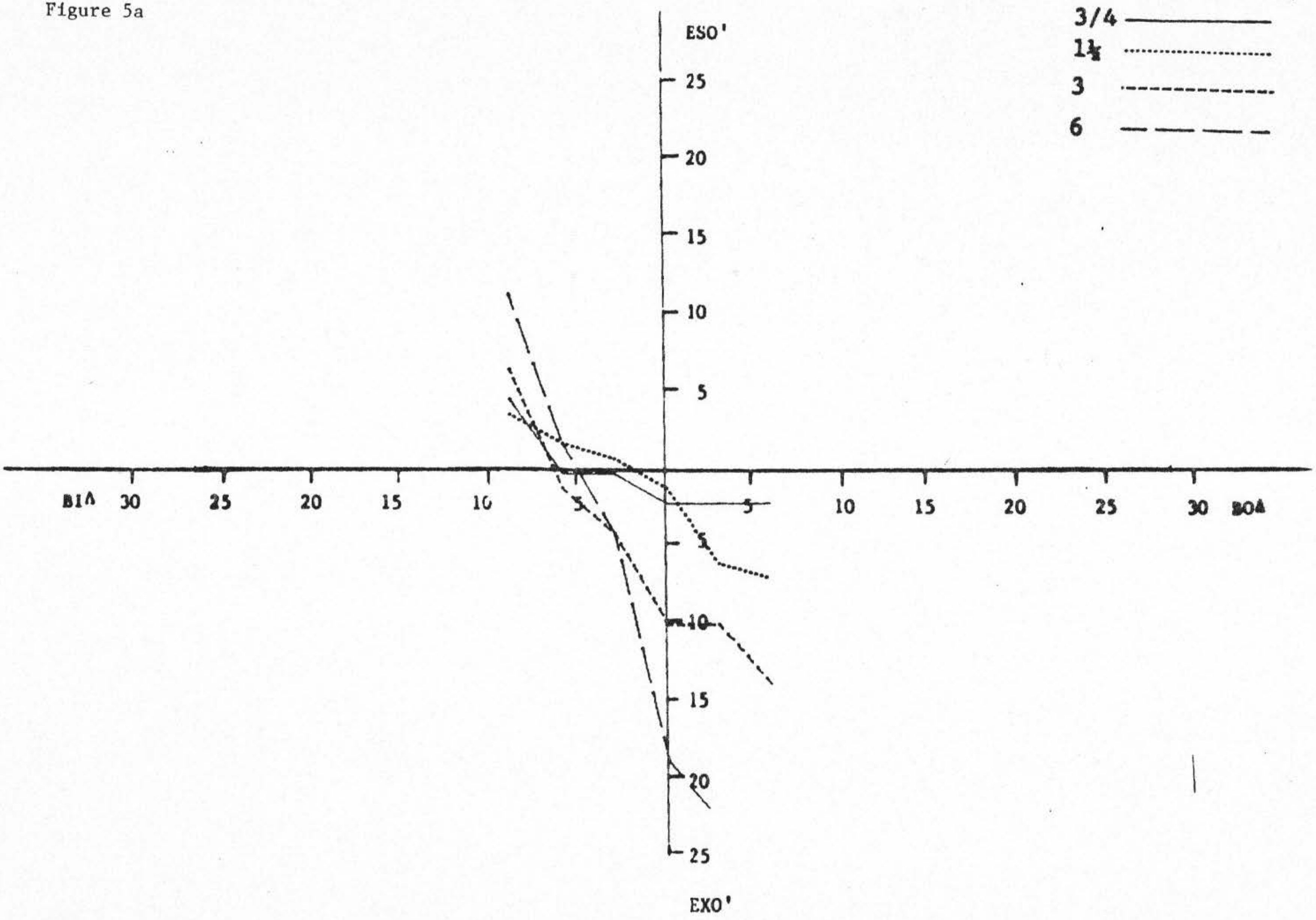
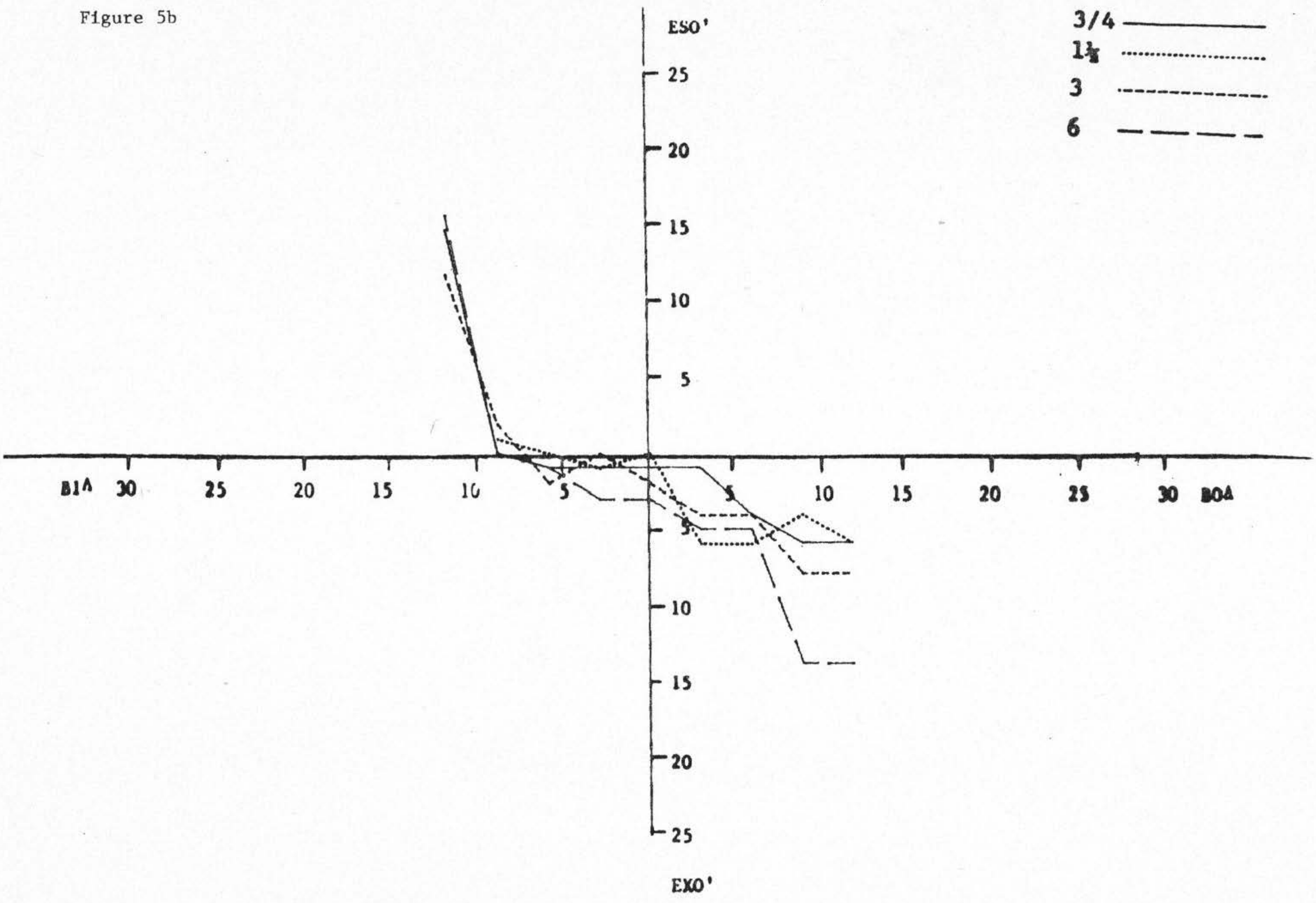

ESO'
EXO'
BIΔ
BOΔ
30
25
20
15
10
5
5
10
15
20
25
30
25
20
15
10
5
5
10
15
20
25
3/4
1½
3
6

Figure 5b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10
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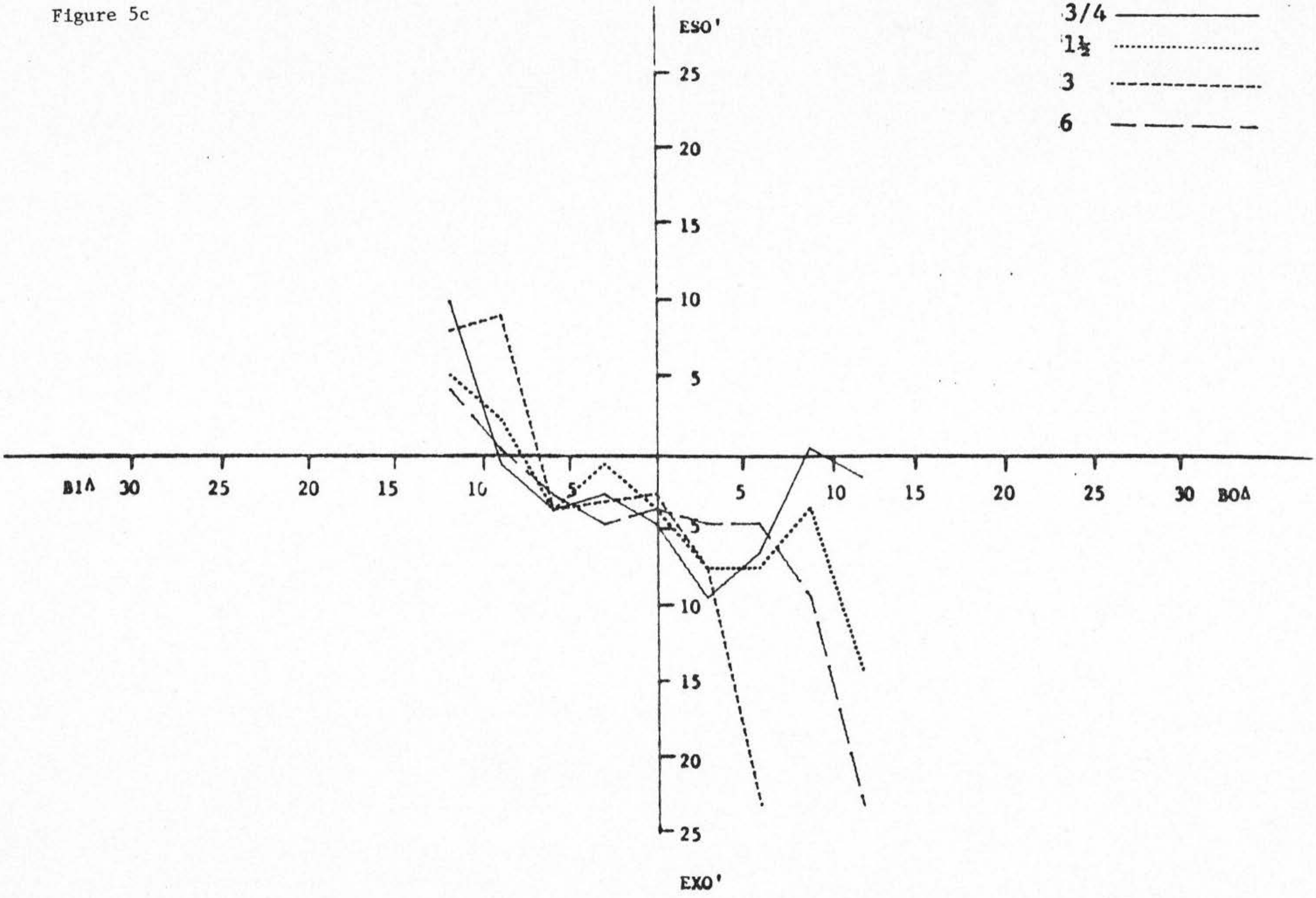
5
10
15
20
25
EXO'
BIA 30
25
20
15
10
5
5
10
15
20
25
30 BOA
3/4
1½
3
6

Figure 5c



ESO'
25
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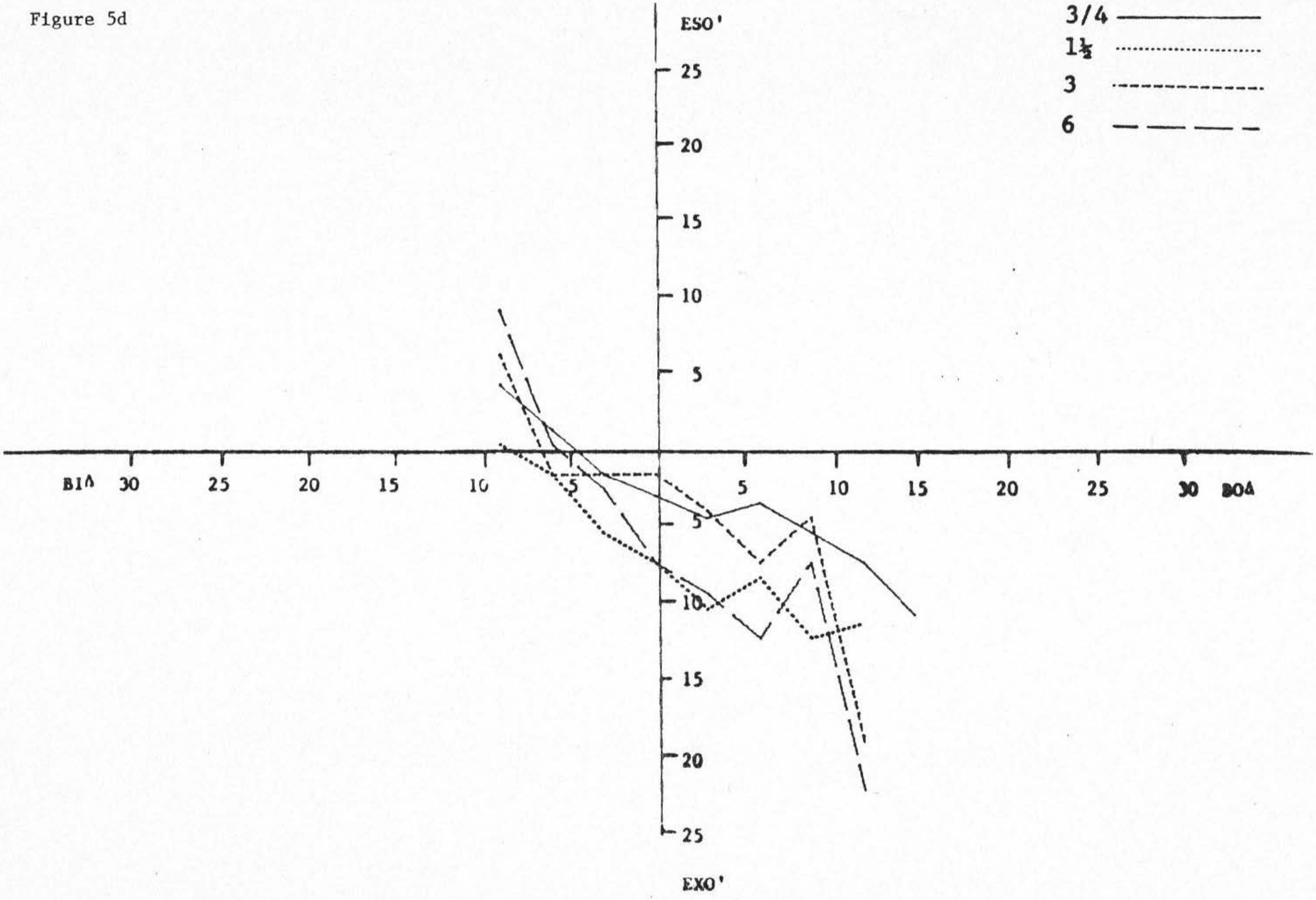
10
5
5
10
15
20
25
EXO'
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 5d



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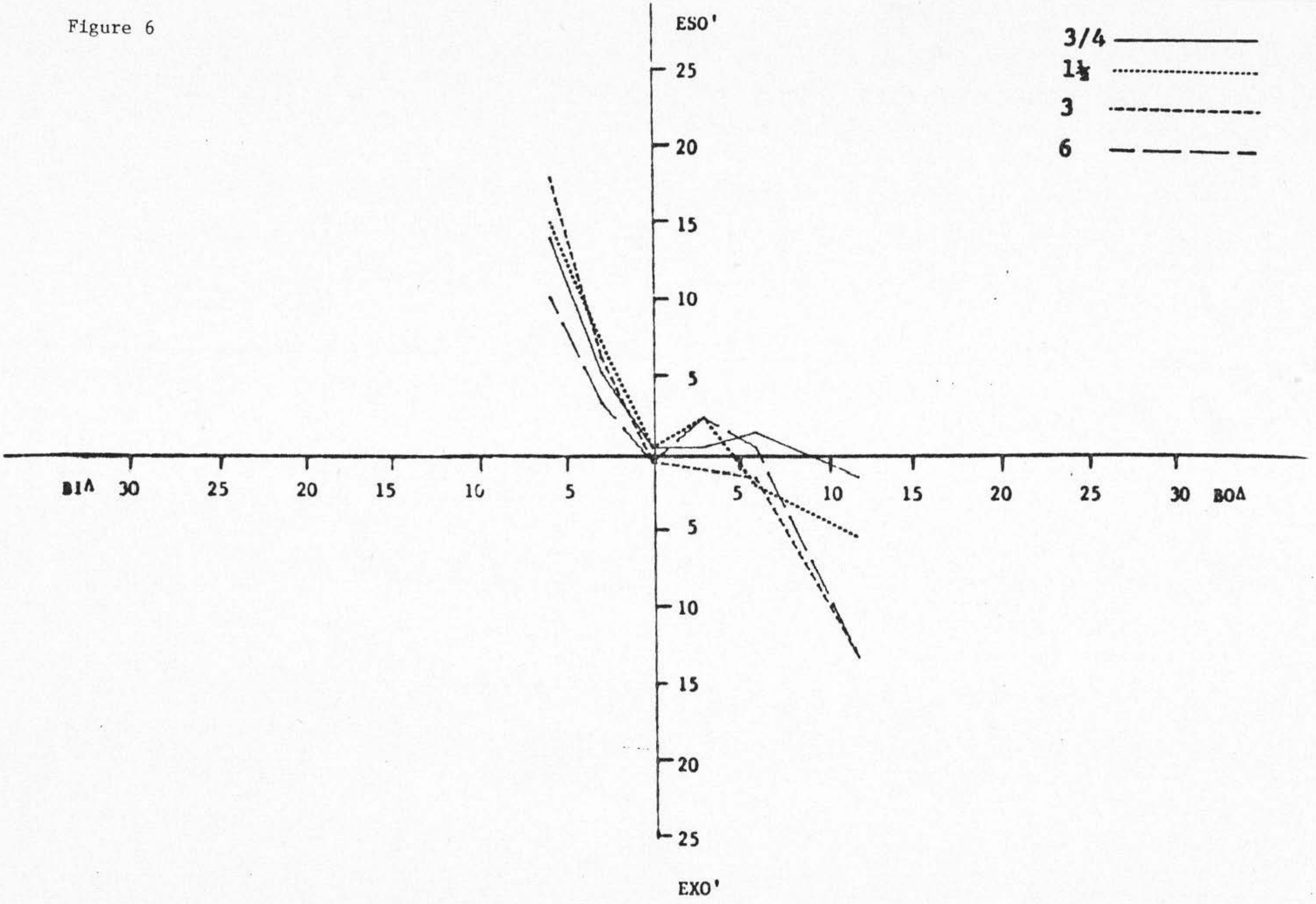
10
5
5
10
15
20
25
EXO'
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 6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20
25
EXO'
BIΔ
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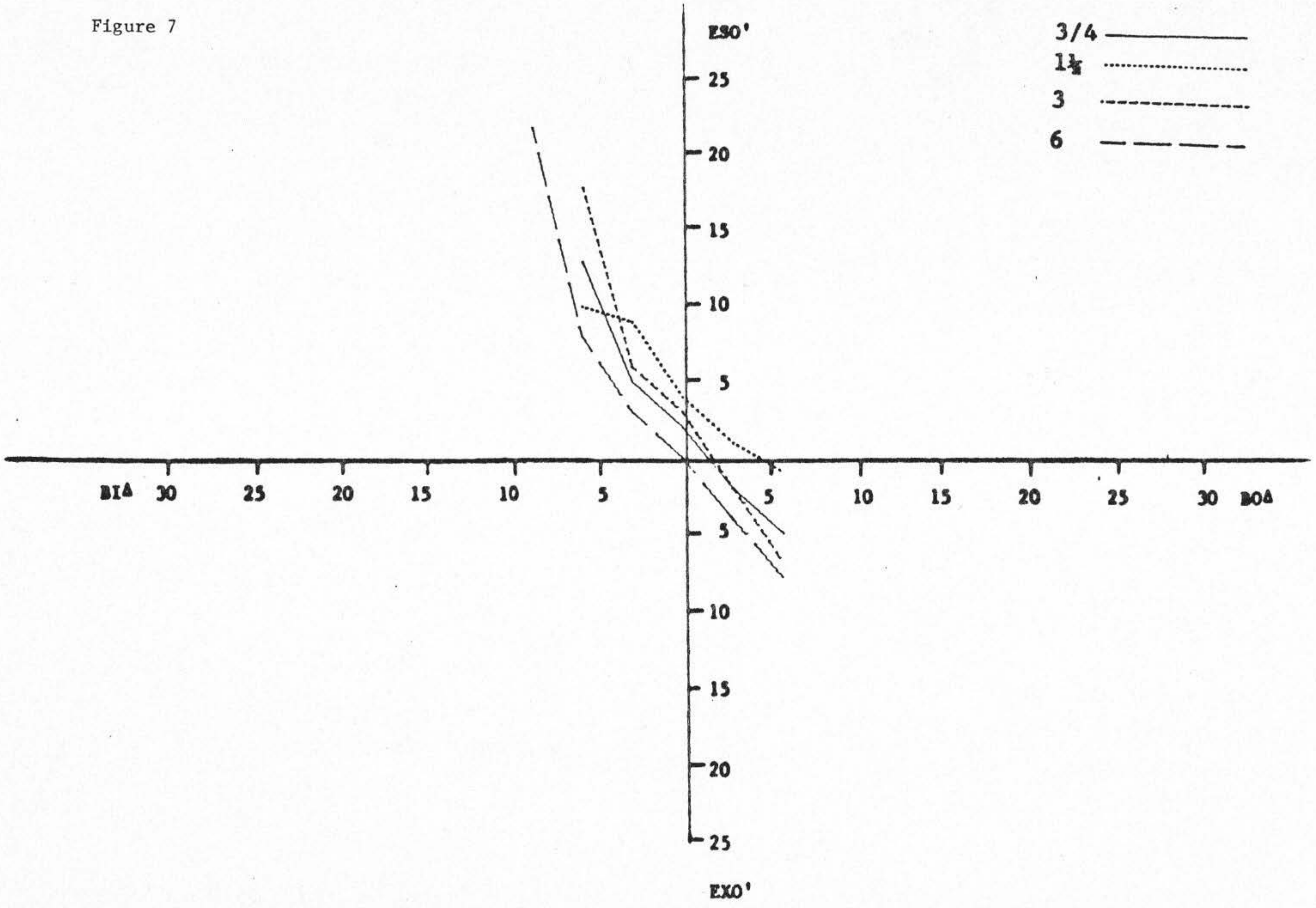
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 7


ESO'
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5
5
10
15
20
25
EXO'
BIΔ
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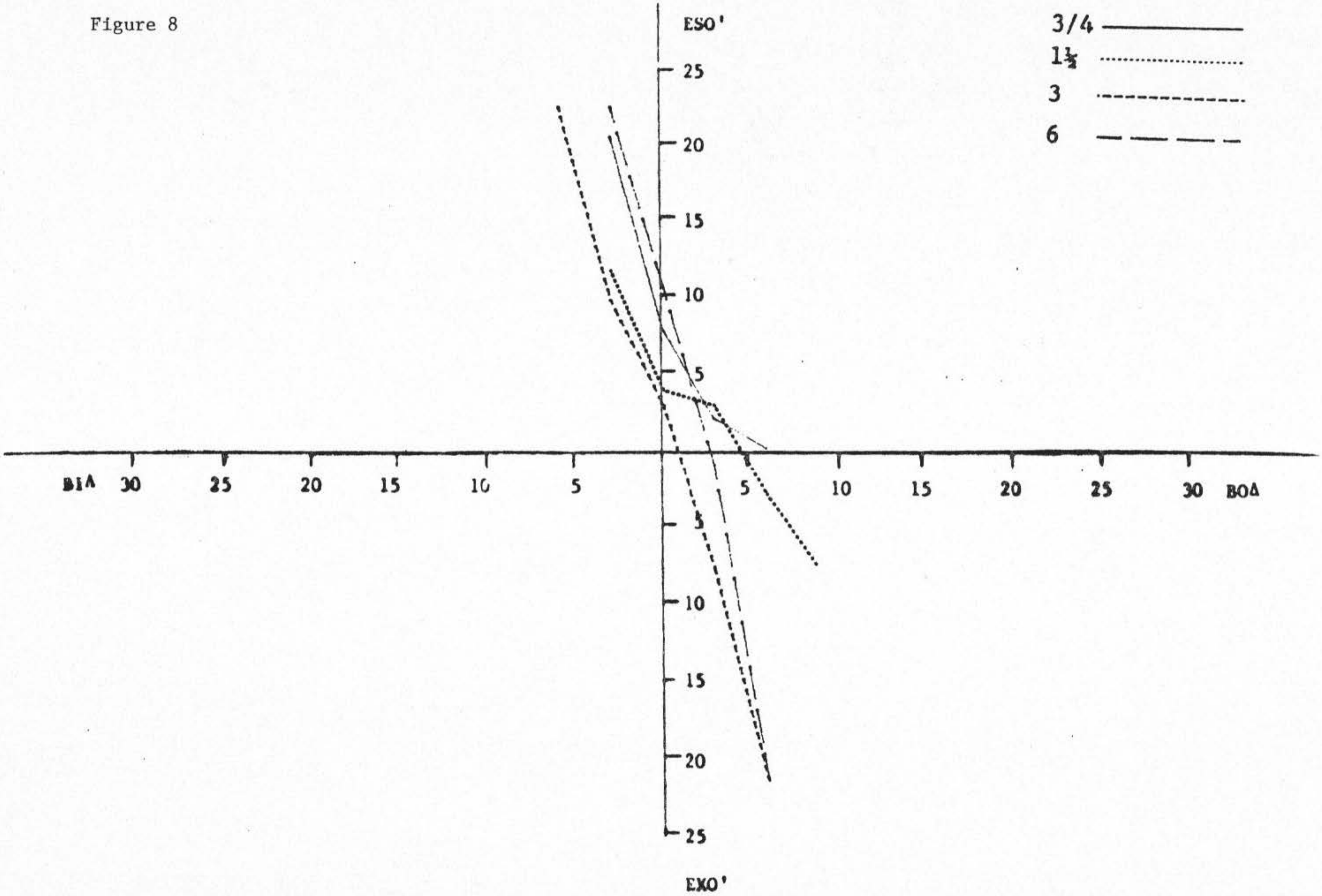
25
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 8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20
25
EXO'
BIΔ
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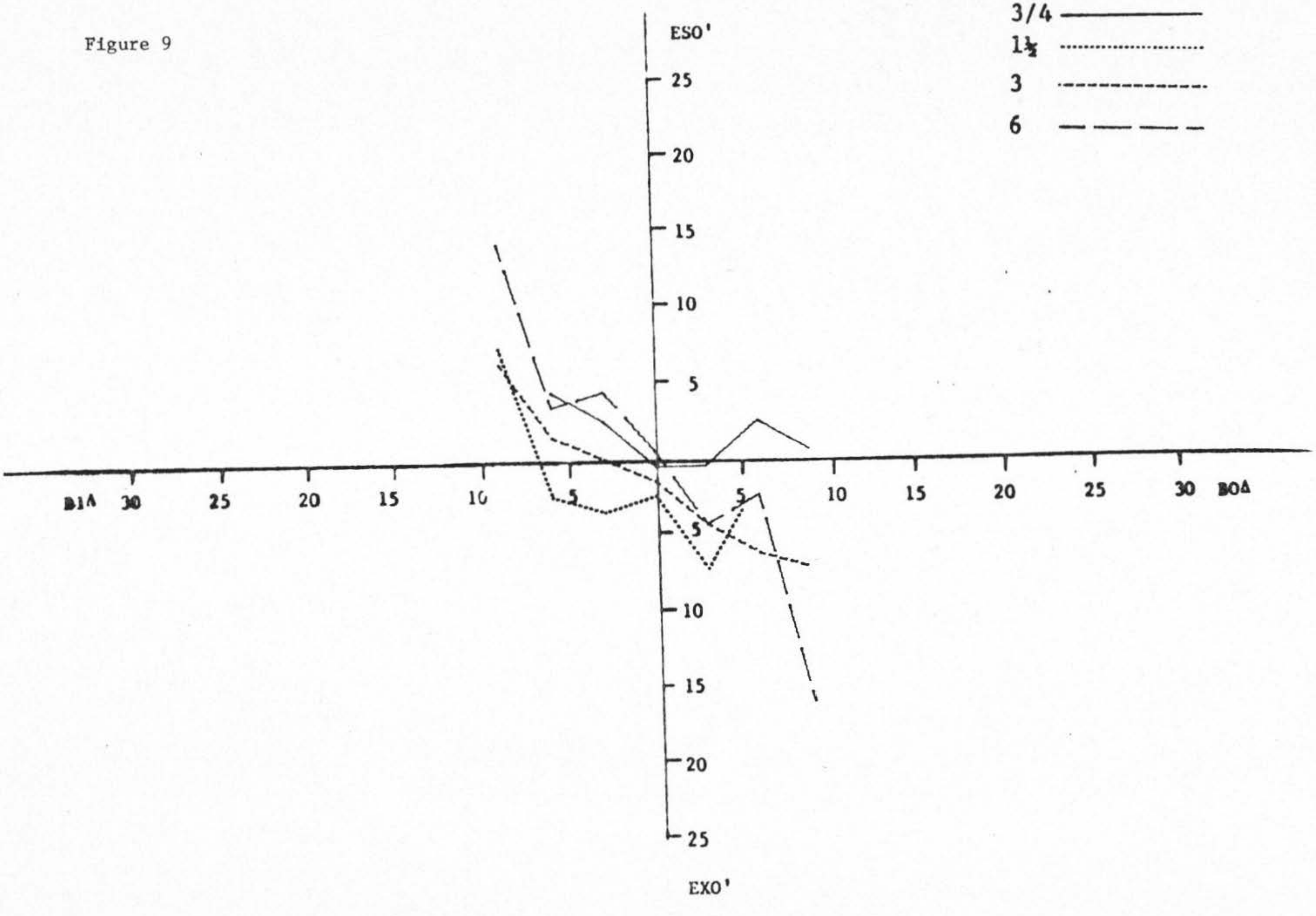
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 9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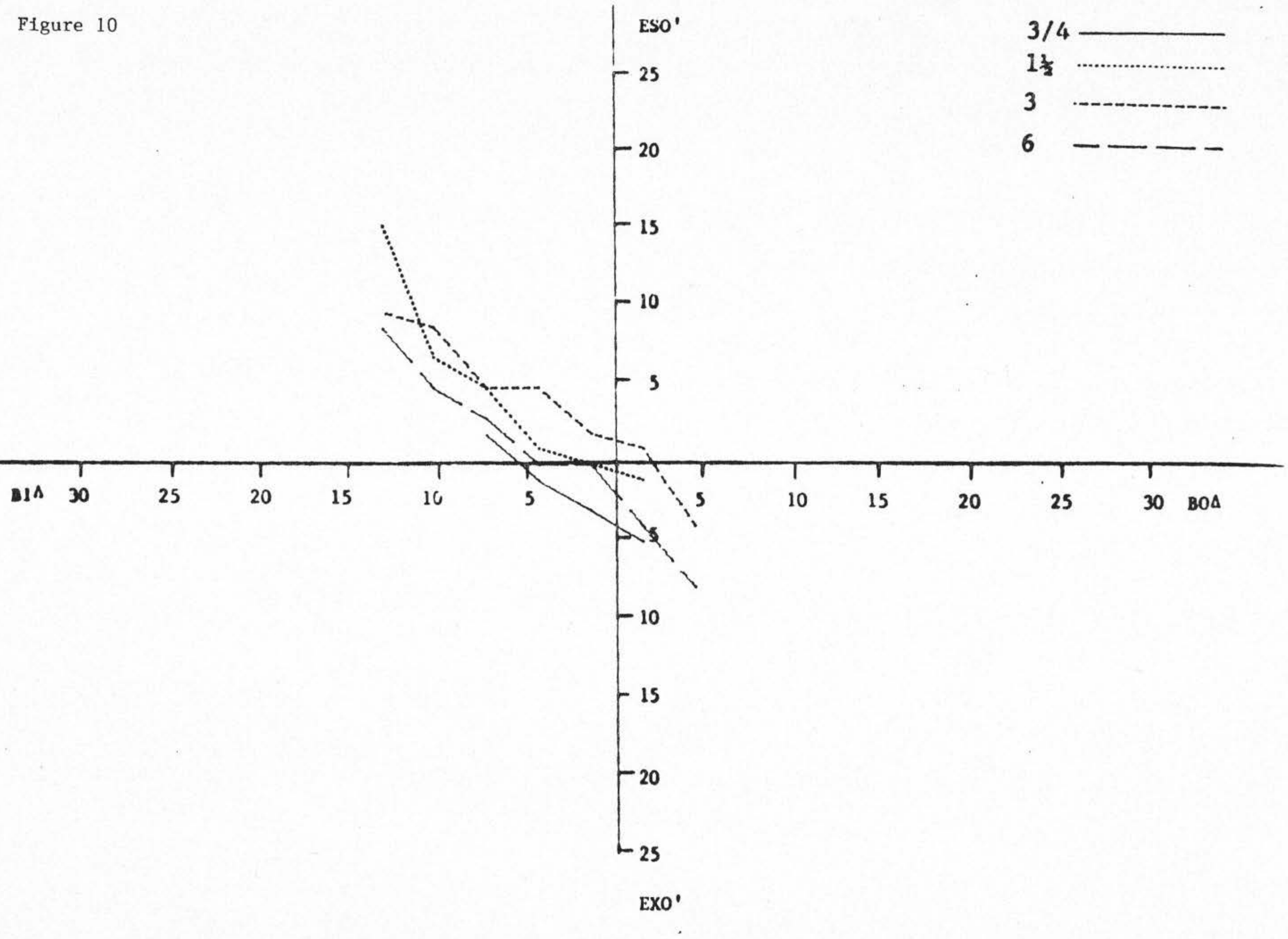
3
6
ESO'
EXO'
25
20
15
10
5
5
10
15
20
25
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ

Figure 10


3/4
1½
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ESO'
25
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15
10
5
10
15
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25
EXO'
BIΔ
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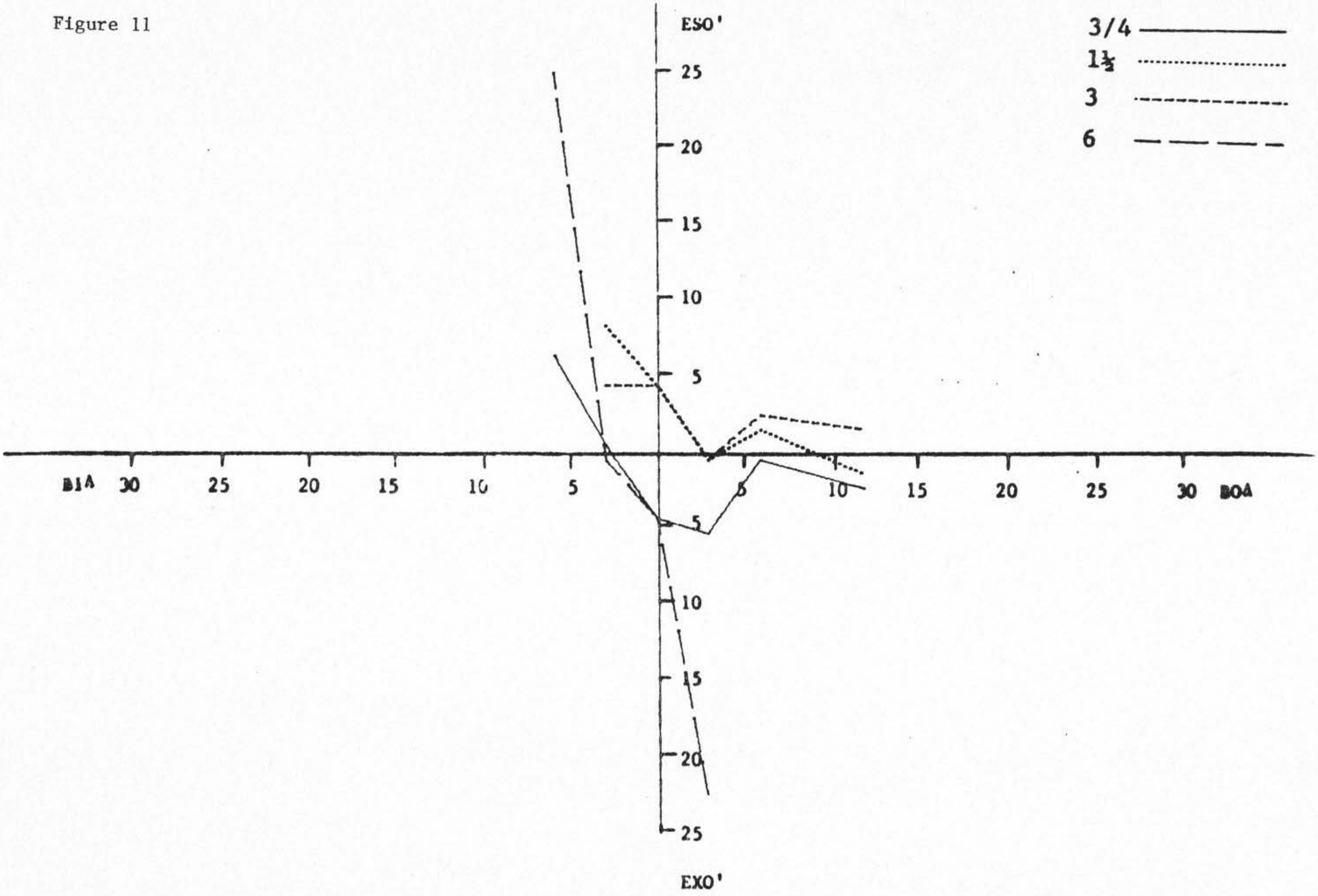
20
15
10
5
5
10
15
20
25
30
BOΔ

Figure 11


ESO'
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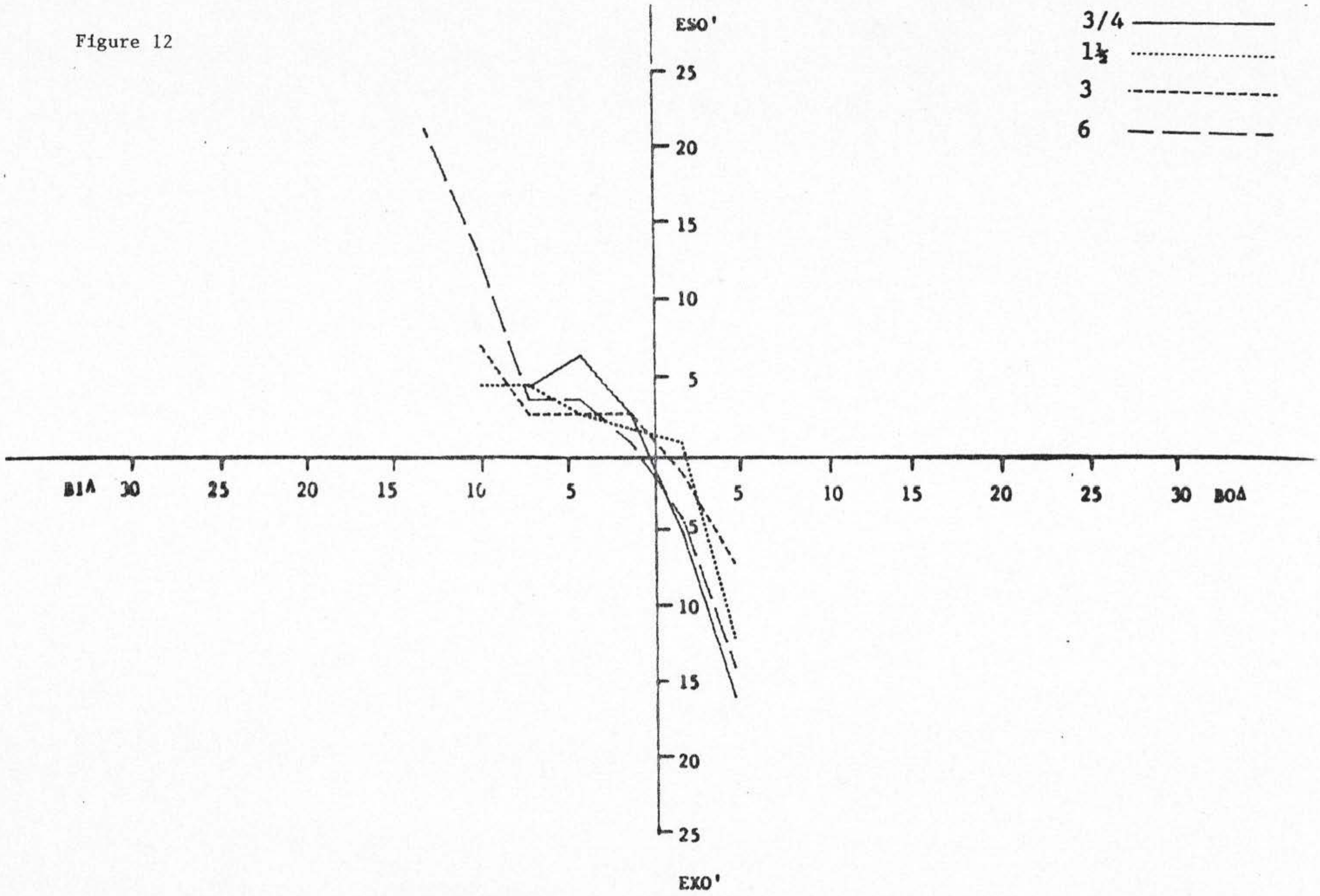
10
15
20
25
EXO'
BIA
30
25
20
15
10
5
5
10
15
20
25
30
BOA
3/4
1½
3
6

Figure 12


3/4
1½
3
6
ESO'
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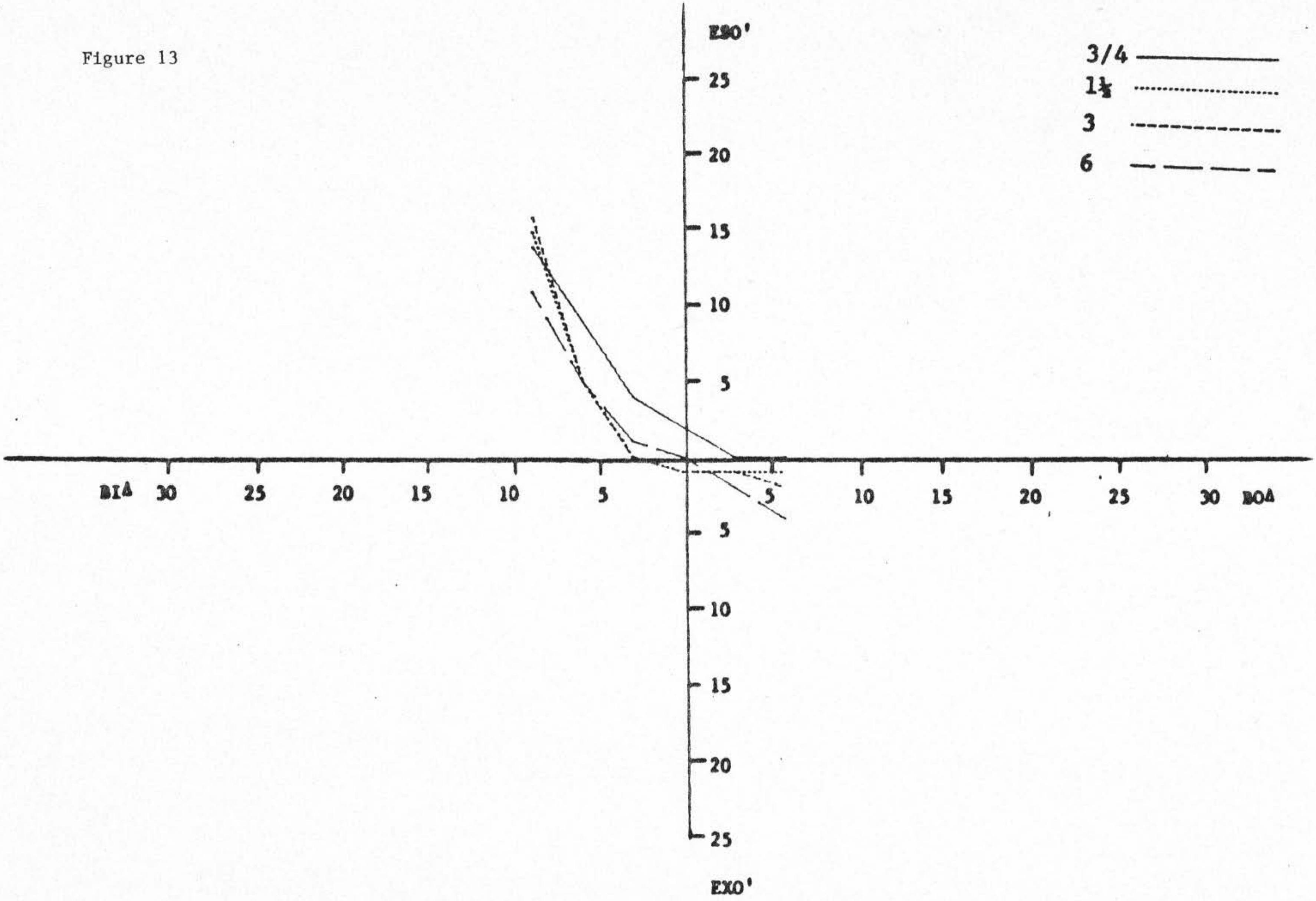
20
15
10
5
5
10
15
20
25
EXO'
BIA
30
25
20
15
10
5
5
10
15
20
25
30
BOA

Figure 13


ESO'
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10
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5
10
15
20
25
EXO'
BI△
30
25
20
15
10
5
5
10
15
20
25
30
BO△
3/4
1½
3
6

Figure 14

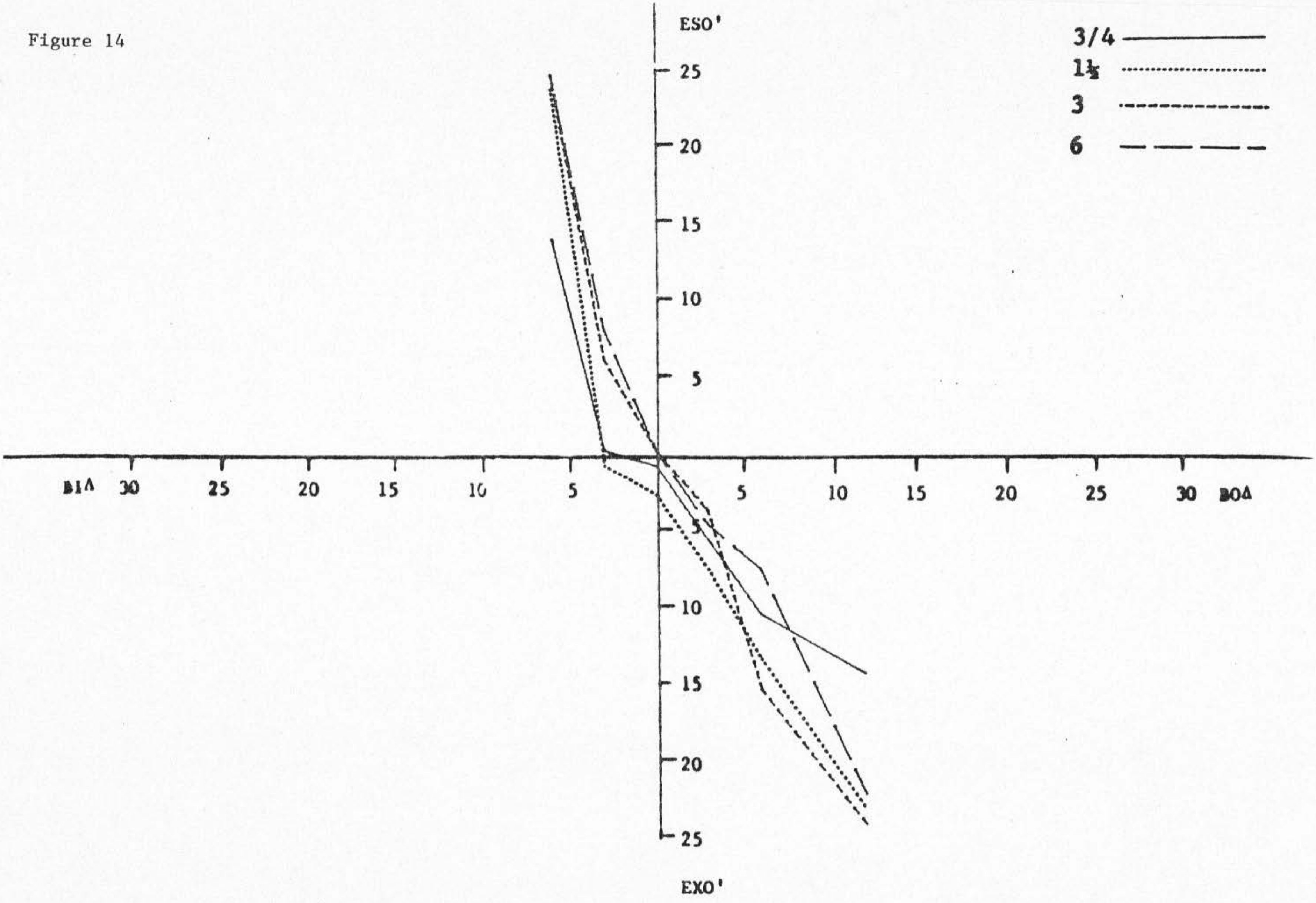
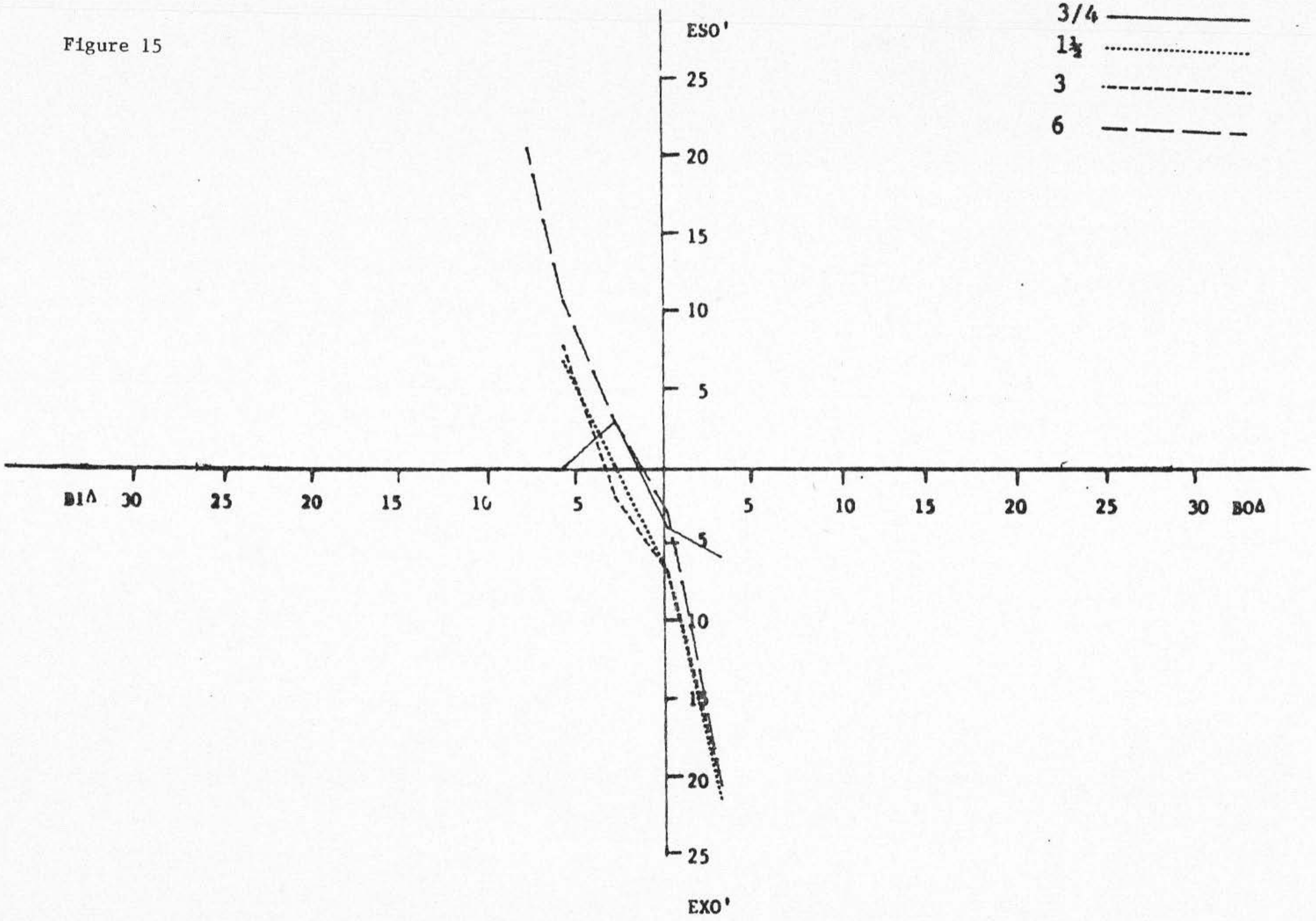

ESO'
25
20
15
10
5
BIΔ 30
25
20
15
10
5
5
10
15
20
25
30 BOΔ
5
10
15
20
25
EXO'
3/4
1½
3
6

Figure 15


ESO'
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20
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10
5
5
10
15
20
25
EXO'
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
3/4
1½
3
6

Figure 16

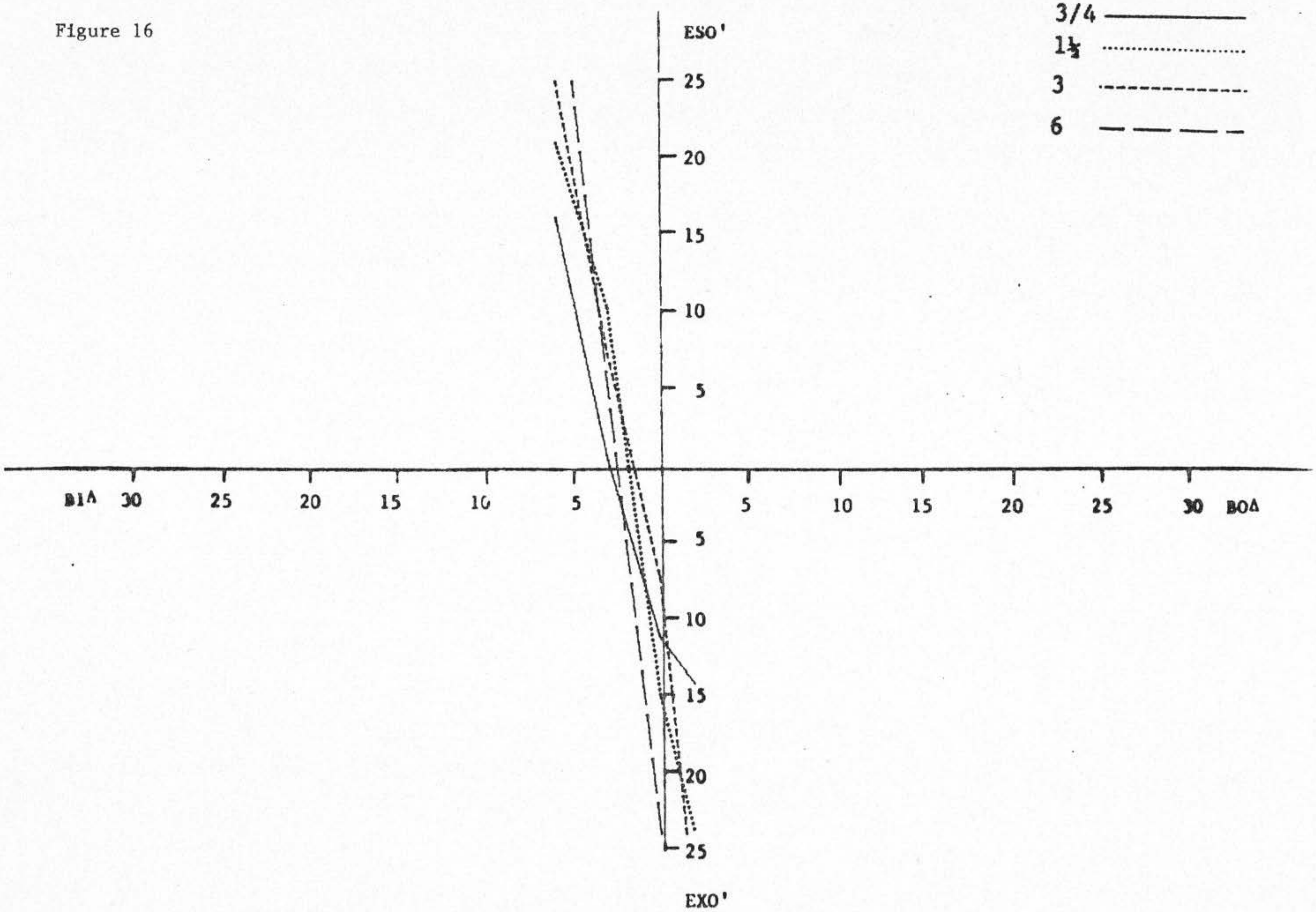
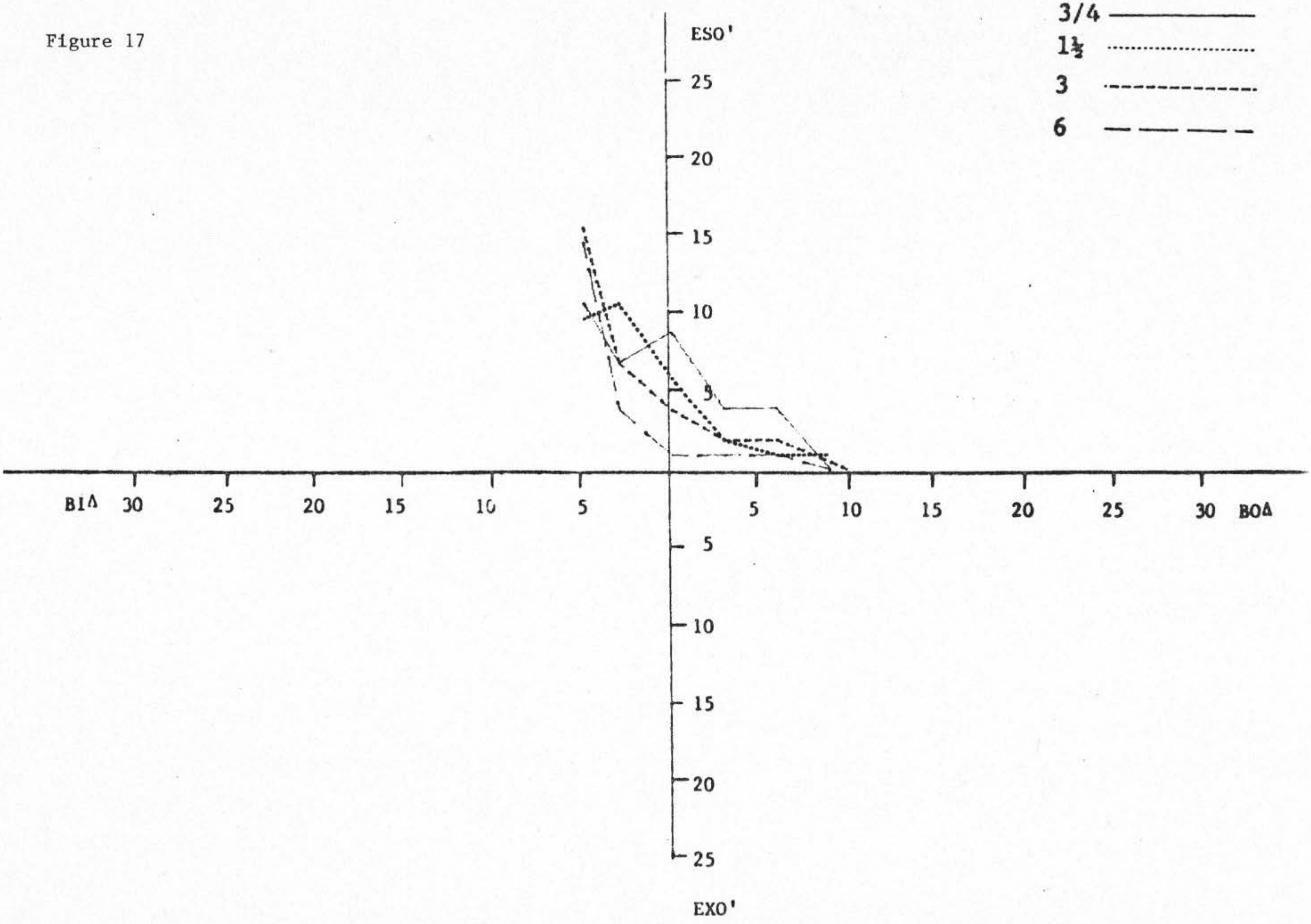

3/4
1½
3
6
ESO'
25
20
15
10
5
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
5
10
15
20
25
EXO'

Figure 17


3/4
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3
6
ESO'
25
20
15
10
5
BIΔ
30
25
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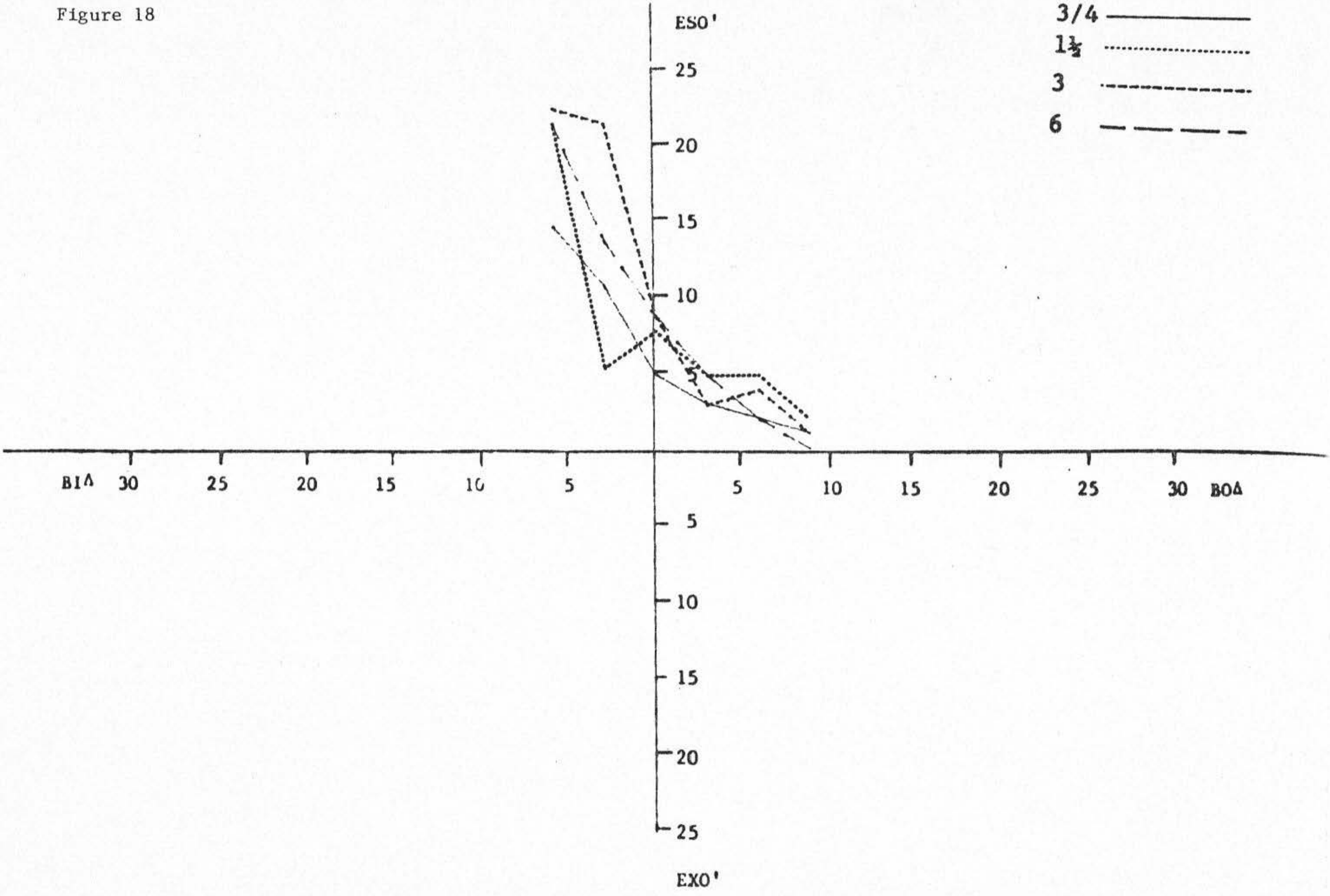
10
5
5
10
15
20
25
30
BOΔ
5
10
15
20
25
EXO'

Figure 18



ESO'
25
20
15
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5
BIΔ 30
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5
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10
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30 BOΔ
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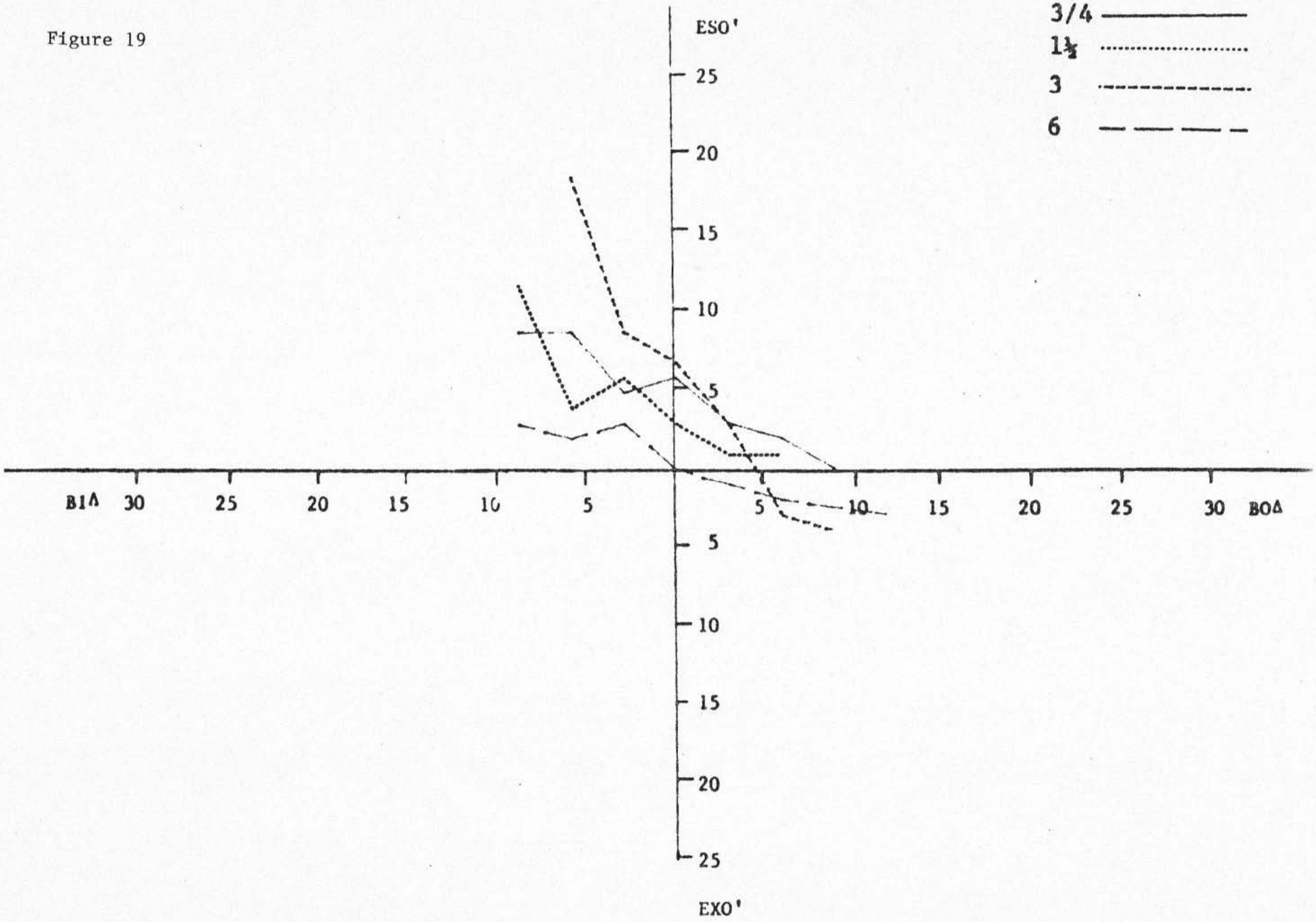
15
20
25
EXO'
3/4
1½
3
6

Figure 19


3/4
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6
ESO'
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5
10
15
20
25
EXO'
BIΔ
30
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20
15
10
5
5
10
15
20
25
30
BOΔ

Figure 20

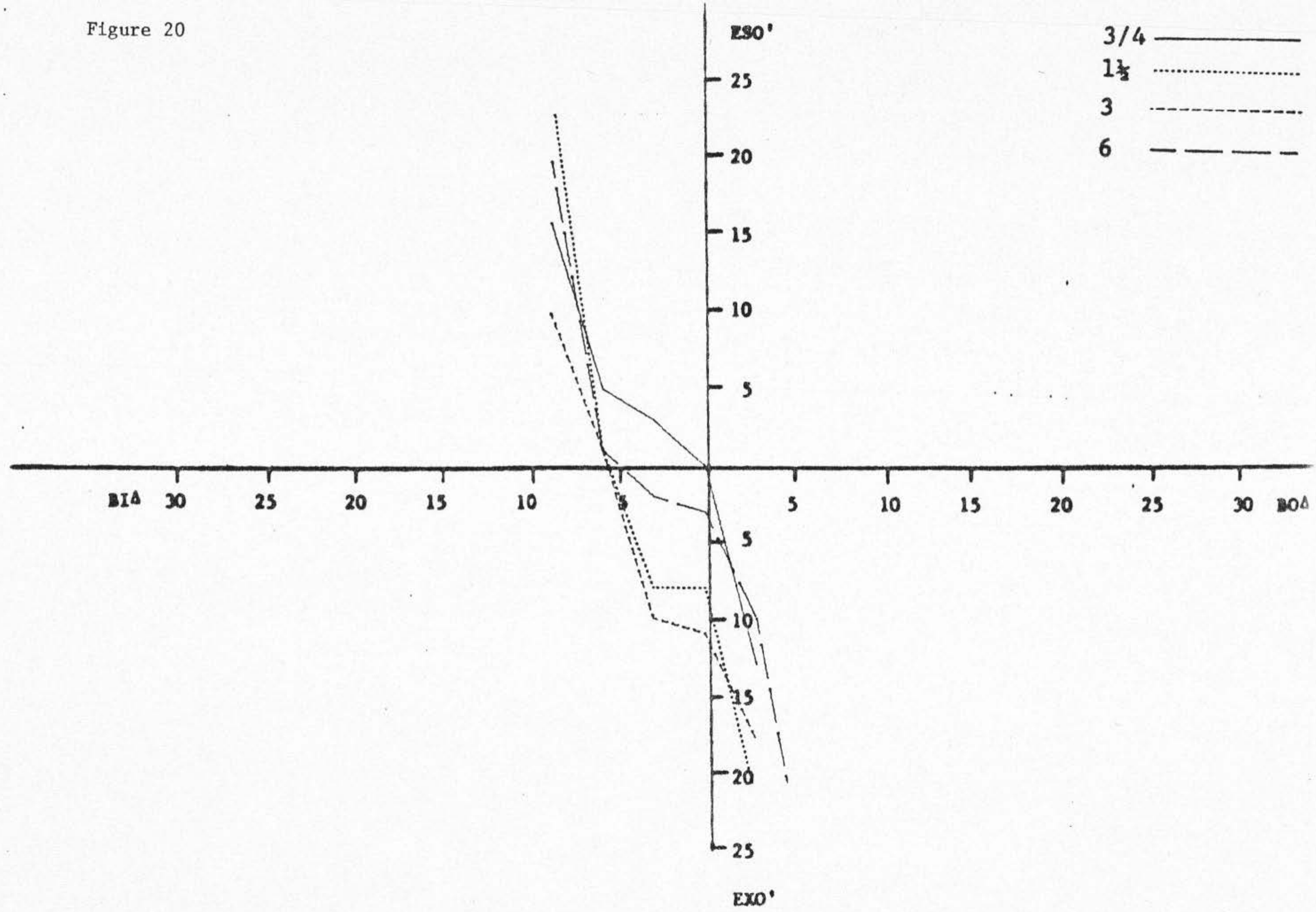
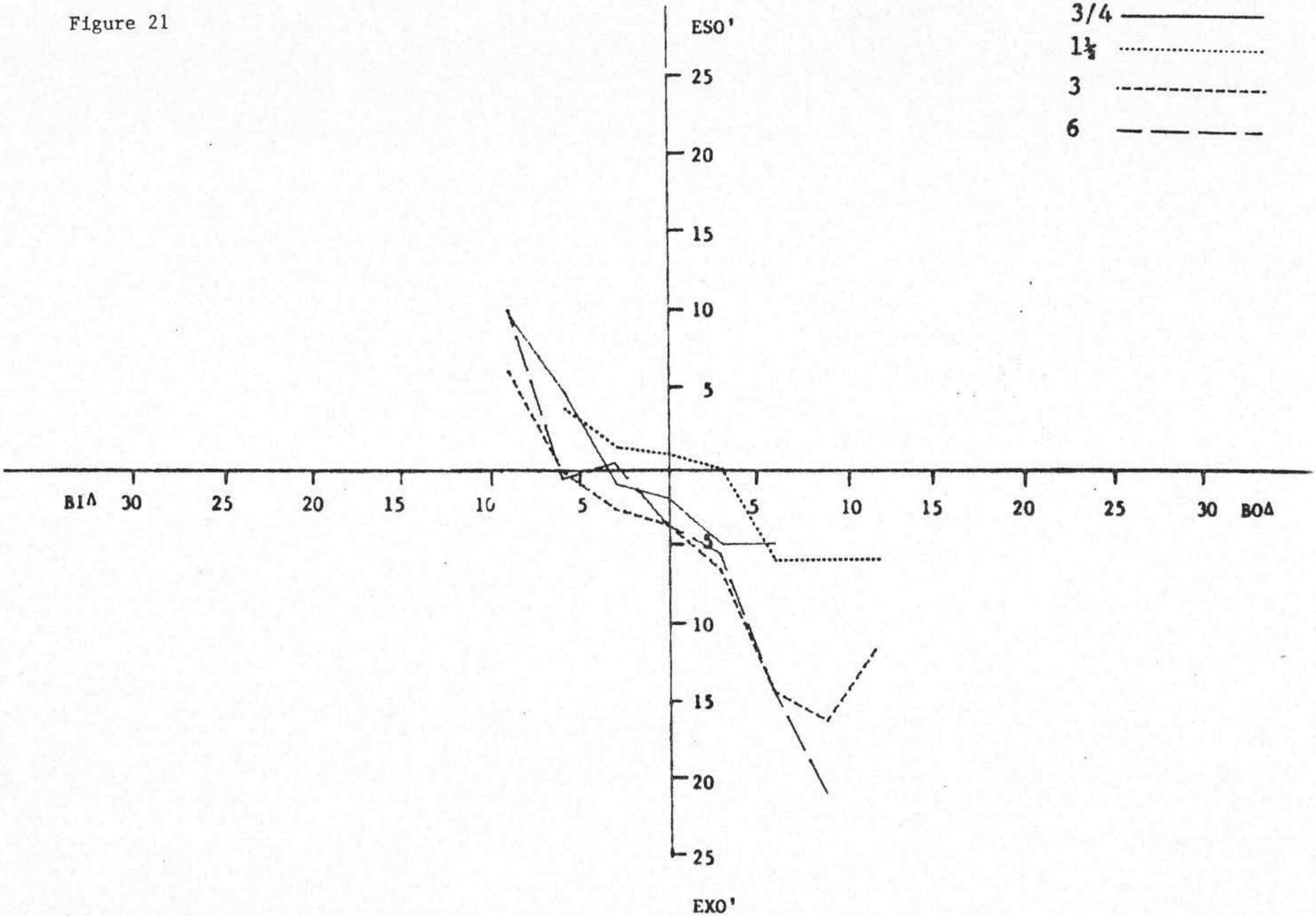

ESO'
25
20
15
10
5
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOΔ
5
10
15
20
25
EXO'
3/4
1½
3
6

Figure 21


3/4
1½
3
6
ESO'
25
20
15
10
5
5
10
15
20
25
EXO'
BIΔ
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25
20
15
10
5
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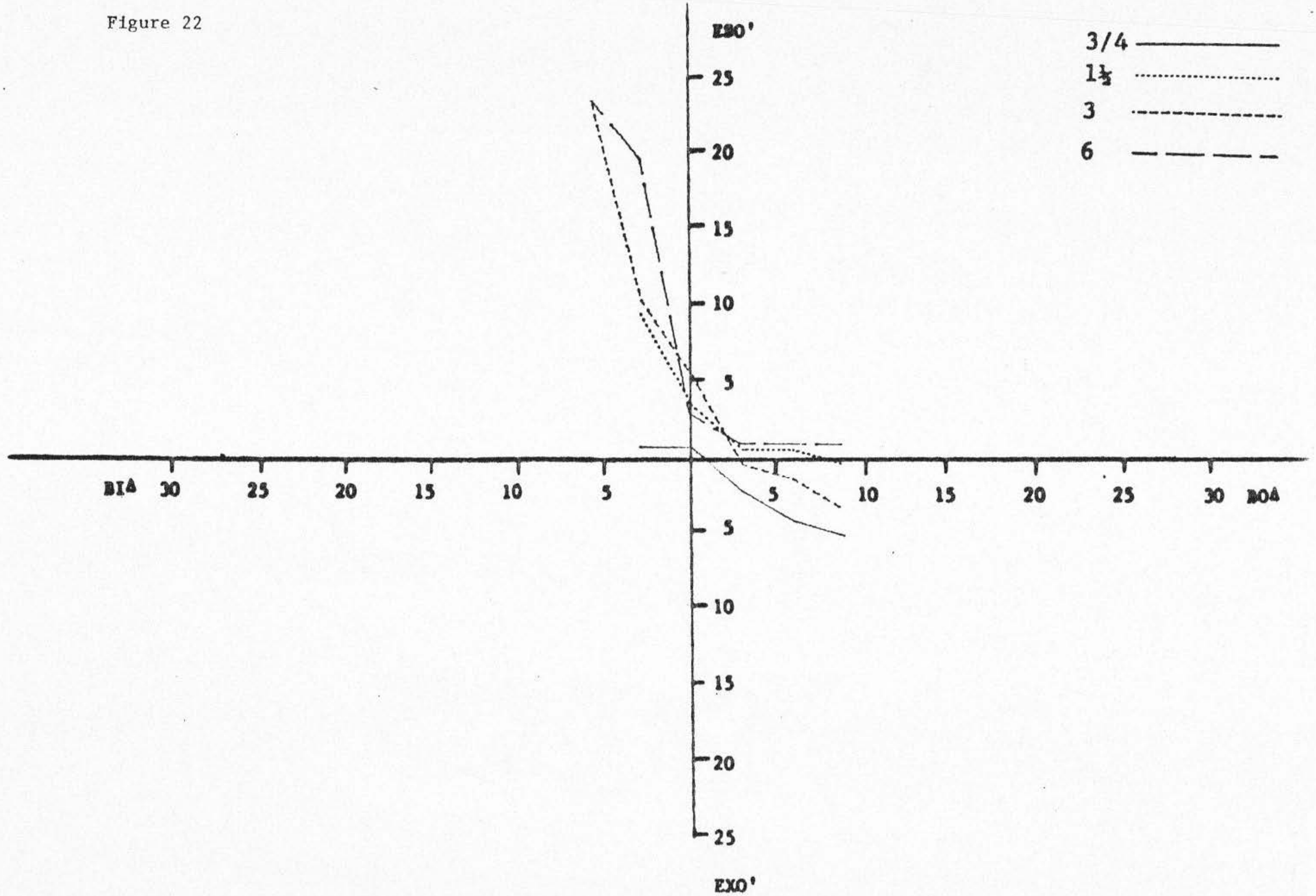
10
15
20
25
30
BOΔ

Figure 22


ESO'
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EXO'
BIΔ
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15
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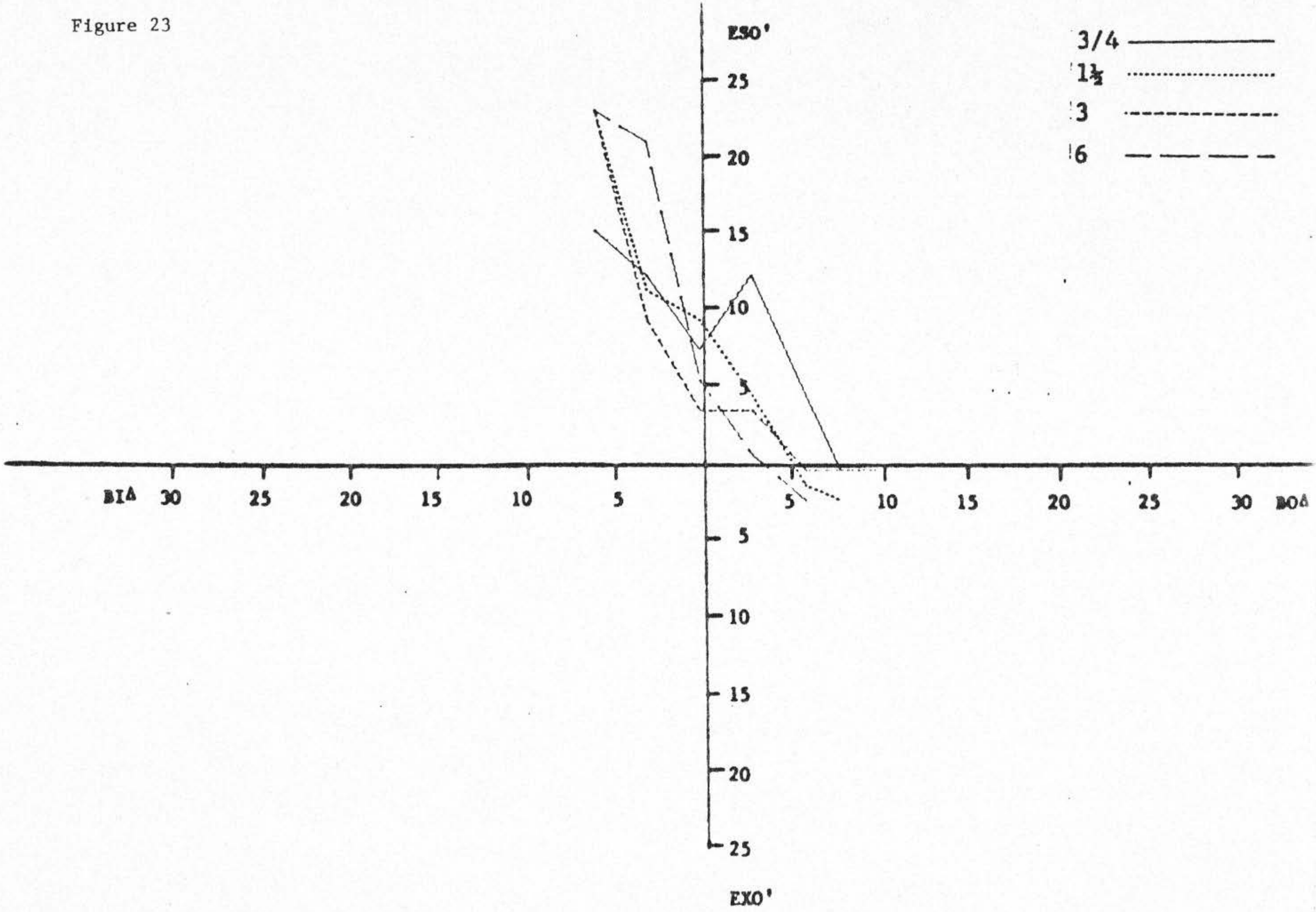
25
30
BOΔ
3/4 ————
1½ ··········
3 ------------
6 — — — —

Figure 23


ESO'
EXO'
BIΔ
BOΔ
30
25
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25
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25
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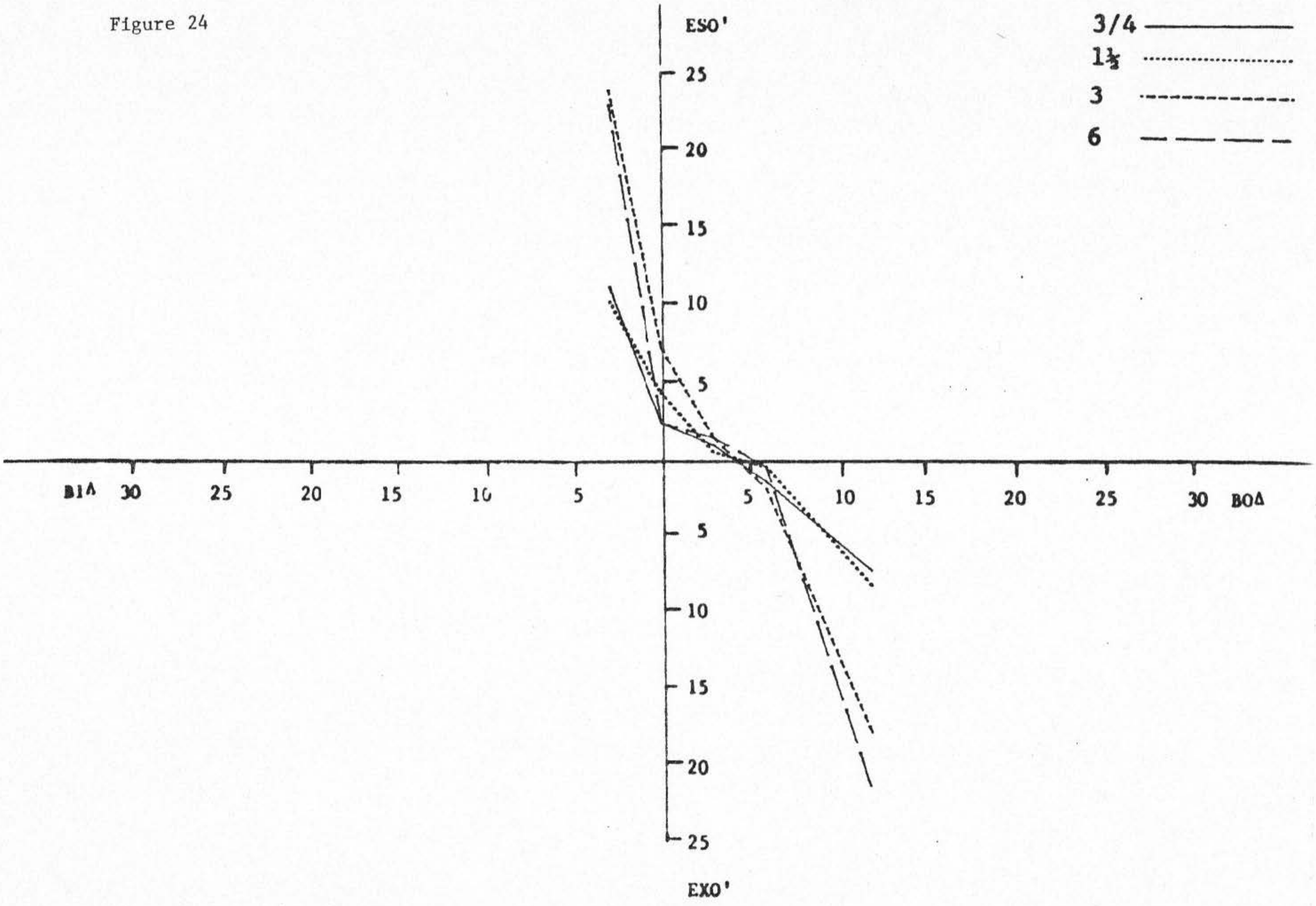
15
10
5
5
10
15
20
25
3/4
1½
3
6

Figure 24


ESO'
3/4
1½
3
6
25
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15
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BIΔ
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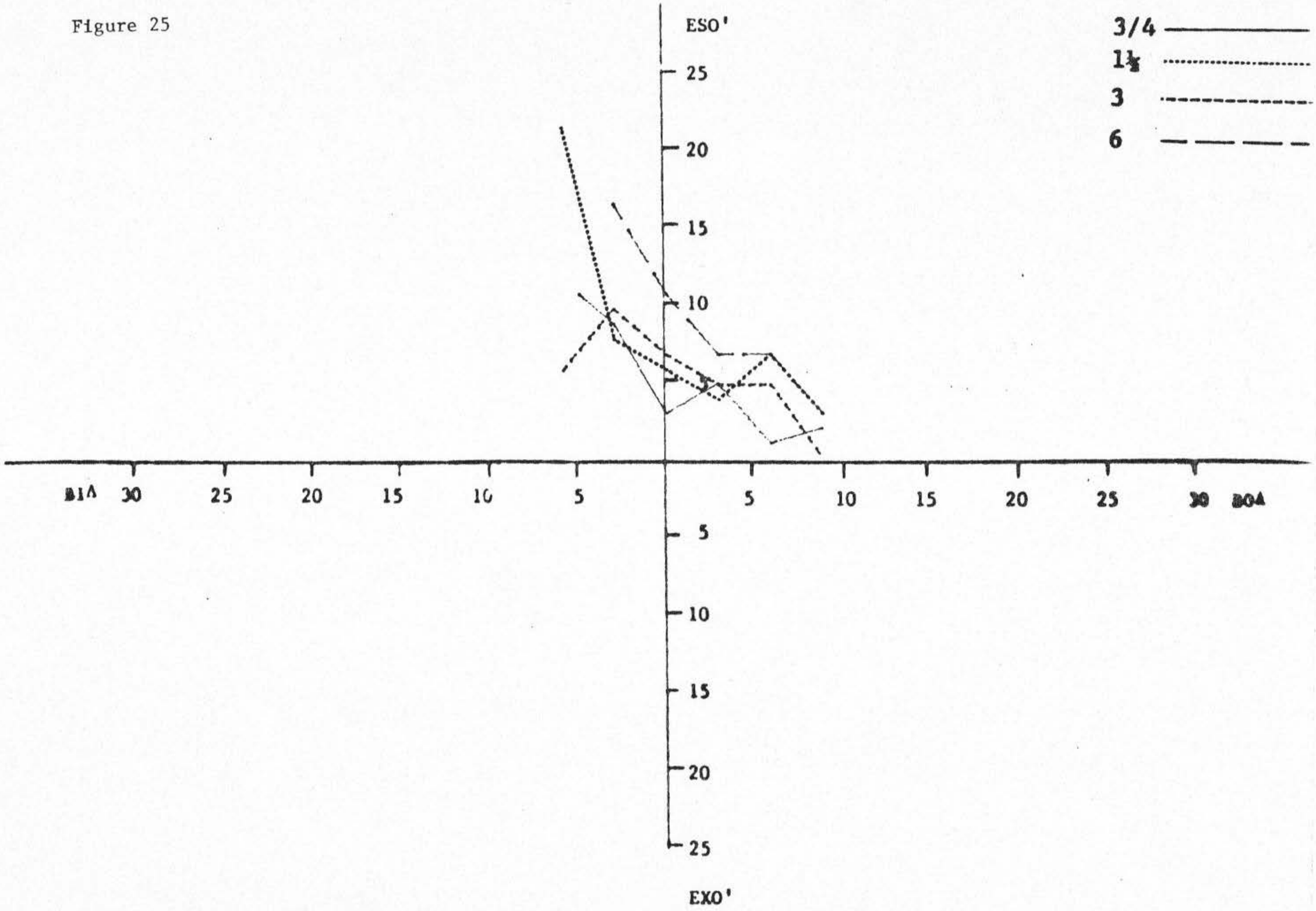
25
30
BOΔ
5
10
15
20
25
EXO'

Figure 25


ESO'
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1½
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BIΔ
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30
BOΔ
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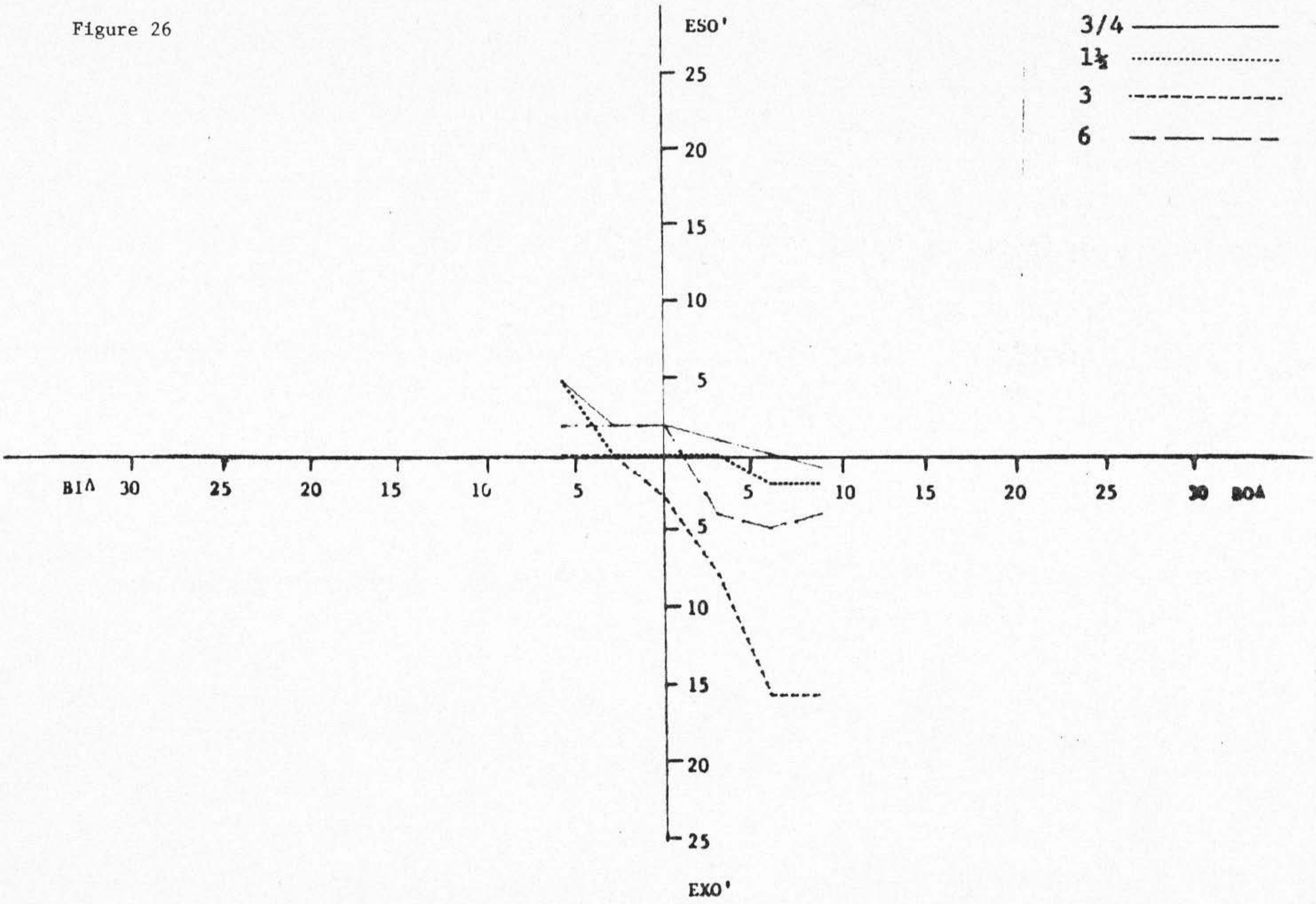
10
15
20
25
EXO'

Figure 26



ESO'
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EXO'
BIΔ
30
25
20
15
10
5
5
10
15
20
25
30
BOA
3/4
1½
3
6

REFERENCES

1. Sheedy, J.E. and J.J. Saladin, Association of Systems with measures of oculomotor deficiencies, Am. J. Optom. Physiol. Opt., 55: 670-676, 1978.

2. Sheedy, J.E., and J.J. Saladin, Phoria, vergence, and fixation disparity in oculomotor problems. Am. J. Optom. Physiol. Opt., 54:474-478, 1977.

3. Ogle, K.N., Researches in Binocular Vision, Philadelphia: W.B. Saunders, 1950.

4. Ogle, K.N., Martens, T.G., Dyer, A.J., Oculomotor Imbalance in Binocular Vision and Fixation Disparity. Philadelphia: Lea and Febiger, 1967.

5. Carter, D.B., Studies of fixation disparity II, Am. J. Optom. Arch. Am. Acad. Optom., 35:590-598, 1958.

6. Stark, L., Video Displays, Work, and Vision. Washington, D.C.: National Academy Press, 1983: 171

7. Krishnan, V.V., Shirachi, D., and Stark, L., Dynamic measures of vergence accommodation, Am. J. Optom. Physiol. Opt., 54: 470-473, 1977.

8. Toates, F.M., Accommodation function of the human eye, Physiological Reviews, 52:828-863, 1972.

9. Toates, F.M., Vergence eye movements, Doc. Ophthal., 37: 153-214, 1974.

10. Semmlow, J.J. and N. Venkiteswaran, Dynamic accommodative components in binocular vision, Vis. Res. 16:403-410, 1976.

11. Schor, C.M., Fixation disparity: a steady state error of disparity - induced vergence, 57:618-631, 1980.

12. Schor, C.M., The relationship between fusional vergence eye movements and fixation disparity, Vision Res., 19:1359-1367, 1979.

13. Jones, A and J.J. Saladin, The stability of the forced vergence fixation disparity curve, submitted to Am. J. Optom. and Physiol. Optics, 1982.

14. Saladin, J.J., and Carr, L., Fusion lock diameter and the forced vergence fixation disparity curve. To be published in Dec. 1983 issue of Am. J. Optom. Physiol. Opt.

15. Hampton, D.R., and Kertesz, A.E., Fusional vergence response to local peripheral stimulation, J. Opt. Soc. Am., 73:7-8, 1983.

16. Semmlow, J. and D. Heerema, The role of accommodative convergence at the limits of fusional vergence, Invest. Ophthal., 18:970-976, 1979.

17. North, R. and D.B. Henson, Adaptation to prism-induced heterophoria in subjects with abnormal binocular vision or asthenopia, Am. J. Optom. and Physiol. Optics, 58:746-752, 1981.