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The role of neural and optical factors in limiting visual resolution in myopia

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Abstract

The myopic growth process has the potential to modify both the optical and neural performance of the eye. We provide three simple models, based on different types of retinal stretching, to predict changes in neural resolution resulting from axial length increases in myopia. These predictions are compared to visual acuity (VA) measures in 34 subjects with refractive errors ranging from plano to -14 D. Our results show a reduction in VA with increasing myopia but not in a manner predicted by our models. We discuss the relative contribution of optical and neural factors to the reduction in visual resolution in myopia. © 1998 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Myopia results from a mismatch between the eye's optical power and its axial length. Most human [1–5] and animal [6,7] myopes have elongated vitreous chambers indicating that myopia is primarily axial in origin. The precise nature of the structural changes in the posterior chamber that leads to myopia seems to vary and may be the result of stretching limited to the equatorial region [8], global expansion of the vitreous chamber [9], posterior pole elongation [10] or some combination of these factors. Myopia can also be pathological, a condition characterised by the presence of a posterior staphyloma. There is some evidence that the retina in axially myopic eyes may simply be a normal retina stretched to cover the increased area of the expanded myopic vitreous chamber [11,12]. Although most studies have attributed myopia to structural changes in eye size, recent studies have shown that myopic eyes are also optically

different from emmetropic eyes in that they exhibit increased monochromatic aberrations [13–15].

Since optical aberrations [16] and the density of retinal neurons [17] can both limit visual acuity (VA) in emmetropes, it would be reasonable to expect reduced VA in myopia as a result of increased optical aberrations, reduced retinal sampling due to retinal stretching, or some combination of these two factors. Experimental studies measuring the effect of spectacle lens (SL) corrected myopic refractive error on visual performance have proved inconclusive and are complicated by the effects of spectacle minification resulting from the correction of myopia. Fiorentini and Maffei [18] reported a decrease in contrast sensitivity in SL corrected myopes when compared to emmetropes. Collins and Carney [19] demonstrated reduced contrast sensitivity over the whole spatial frequency range in a high myope group in comparison to a low myope group following SL correction. Similarly, Applegate [13] found reduced high contrast VA with increasing myopic refractive error. The exception to these reports is a study by Thorn et al. [20] which found no high spatial frequency variation in the contrast sensitivity function in a group of high myopes when compared to a group of

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emmetropes. Interestingly, Collins and Carney [19] went on to report that after correcting their subjects with contact lenses (CLs) only the highly myopic subjects exhibited a loss in contrast sensitivity in comparison to emmetropes. Furthermore, these losses were found only at the highest spatial frequency measured (24 cycles per degree, c deg^{-1}). It is important to note that despite the fact that axial myopes corrected with CLs will have larger retinal images than emmetropes, no reports have provided evidence that CL corrected axial myopes have better VA than emmetropes.

Reduced VA and contrast sensitivity (CS) in high myopes are difficult to interpret since the SLs used to correct the myopia act as optical minifiers, and thus the reduced visual performance may reflect optical minification and not increased optical aberrations or reduced retinal sampling densities. Knapp's Law (Knapp, 1869 cited in [12]) predicts that if axial myopes are corrected with SLs placed at the anterior focal point of the eye the retinal image size will be the same as that found in the emmetropic eye. Taking this into account we would predict the axial myopic eye corrected at the cornea, with a CL or direct corneal reshaping by radial keratotomy, will have a retinal image that is magnified when compared to the image size in emmetropic or spectacle corrected axial myopic eyes and thus might be expected to exhibit increased VA when compared to emmetropes.

The purpose of the present study was to examine VA in a large sample of confirmed axial myopes with both SL and CL corrections in an attempt to ascertain the contributions of magnification, retinal stretching and optical aberrations. Our data confirm that, when magnification effects are factored out, VA does decline with increasing myopia, CL correction does not lead to increased VA in axial myopes when compared to emmetropes but does when compared to myopes corrected with SL, and both retinal and optical factors may contribute to the decline in VA with increasing myopia.

2. Methods

2.1. Subjects

Young visually normal subjects ($n = 34$) participated in the study and had refractive errors ranging from plano to -14 D. Axial length and corneal radii were measured on all subjects using an A-Scan Ultrasonograph and an Allergan Humphrey Auto-Keratometer. All subjects had < 0.75 D of astigmatism and were optimally corrected with the appropriate spherocylindrical correction with either CLs or spectacles. The spectacle prescription was measured at a back vertex distance of 15 mm. Soft CLs were used in all of the

subjects with fitting carried out at least 30 min prior to VA measurement. Any residual astigmatic error (< 0.75 D) was corrected with a trial SL.

2.2. Visual acuity measurements

VA was measured following both SL and CL correction using two different log MAR Bailey–Lovie high-contrast letter charts, each employing an interpolated scoring technique. This method improves the accuracy of VA measurement and thus aids the distinction between subjects with similar VAs [21]. The Bailey–Lovie charts were maintained throughout the study at a luminance of $\approx 160 \text{ c dm}^{-2}$.

3. Results

3.1. Optical and anatomical modelling

3.1.1. The relationship between myopia and axial length

Throughout this study we based our modelling on the assumption that all myopia is axial. To confirm the validity of this assumption, we constructed a model using paraxial optical theory [22] to predict the increase in axial length with increasing myopia. The model was constructed using the formula:

$$a = \frac{-n'Rh}{D(Rh + D)} \quad (1)$$

where Rh is the amount of myopic refractive error (dioptries), $D = 60$ dioptries, $n' = 1.336$ and a is the increase in axial length from the emmetropic schematic eye value (m).

From Eq. (1) the modelled increase in axial length from the emmetropic schematic eye was found to be non-linear. The model predicts that larger increases in axial length are required to provide a refractive change of one dioptre at higher degrees of myopia. We compared the axial length measures collected on our subject group with our predictive model (Fig. 1a). Comparison between the actual and predicted axial length values shows a good correlation ($r = 0.84$). Fig. 1(b) shows no significant relationship ($r = 0.09$) between corneal radius and refractive error which suggests the changes in refractive error are not corneal. Multiple regression analysis, using refractive error as the dependent variable and axial length and corneal radius as independent variables, revealed that axial length accounts for 71.6% of the variance with the combination of corneal radius and axial elongation only resulting in 73.1% of the variance. Therefore only 1.5% of the myopia in the subject group is attributable to corneal radius changes. This compares well to the findings of previous studies [3–5,23] and from our figures it would seem reasonable to assume that the myopia found in most of our

subjects is the result of axial elongation. Model axial length predictions are continued into the hyperopic refractive errors purely as a continuation of the model. We have no data to support our predictions in this group.

3.1.2. The effect of magnification on visual acuity

The next stage of the study assessed the effects of SL and CL correction on VA using a simple paraxial magnification optics model. In this model we have assumed that the myopia is axial and that the distance from the back surface of the CL to the entrance pupil of the eye is 3.68 mm (simplified schematic eye, [24]). The magnification of the eye is altered by the correction mode and is predicted by the lens magnification formula:

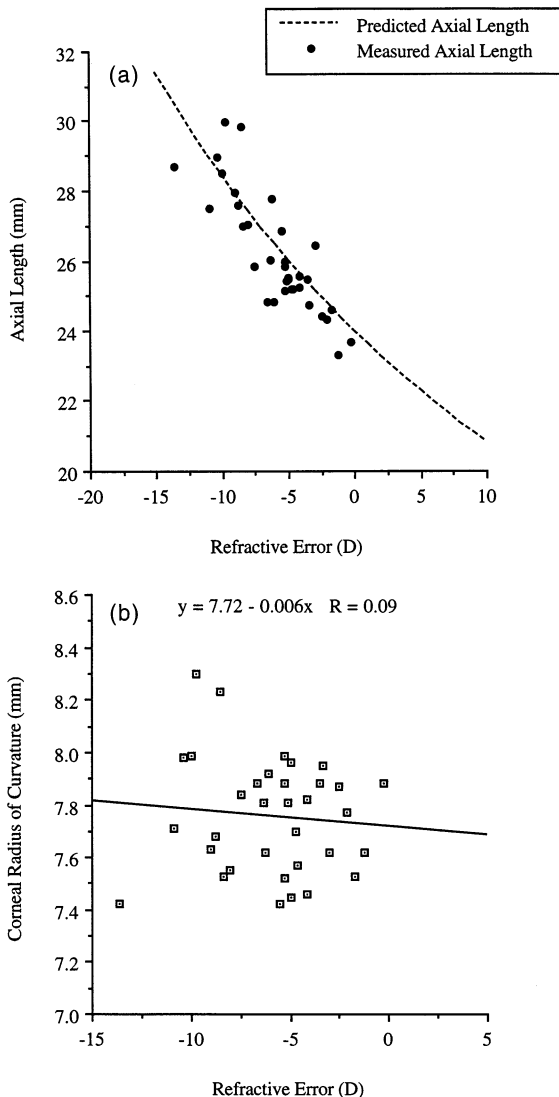


Fig. 1. (a) A plot of axial length against refractive error. The dashed line represents the increase in axial length with refractive error predicted from a schematic eye model. (b) A plot of the corneal radius data against refractive error ($n = 34$).

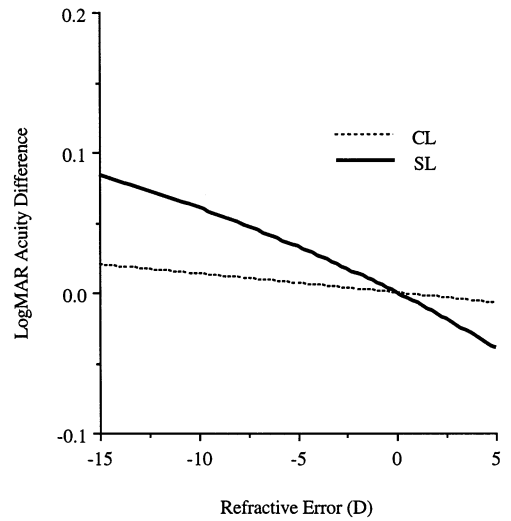


Fig. 2. A predictive optical model shows the effects of magnification on log MAR VA resulting from SL and CL correction in axial myopia. The model predicts that both correction modes will produce a minified retinal image with SL correction producing the greater minification. Our calculations assumed that the distance from the back surface of the CL to the entrance pupil of the eye was 3.68 mm and that the SL back vertex distance was 15 mm.

$$\text{Lens magnification} = \left(\frac{1}{1 - t/nF_1} \right) \left(\frac{1}{1 - hFv} \right) \quad (2)$$

where h is the distance from the correcting lens back surface to the entrance pupil of the eye in metres, Fv is the back vertex power of the correcting lens, t is the lens thickness in metres, n is the refractive index of the correcting lens and F_1 is the surface curvature of the correcting lens. The first factor in the equation is termed the ‘lens shape factor’ and the second is the ‘power factor’. If we assume the correcting lens is thin, t will equal zero and the lens shape factor will be equal to unity.

Using Eq. (2) we calculated the lens magnification for both CL and SL correction and converted the values to log MAR VA scale. In log MAR terms each line on the chart relates to a change in magnification of 25.89%. The converted values are illustrated in Fig. 2 and reveal both correction modes will minify the image resulting in log MAR VA declining with increasing myopia. These calculations relate to the eye in its uncorrected state and on the log MAR scale each line change is equal to a 0.1 log unit change in acuity. The other interesting feature in terms of our study is that CL correction will produce a retinal image that is magnified when compared to SL correction (Fig. 2). This relative retinal magnification produced by moving the correction from the SL to CL plane is equal to the lens magnification value in CL correction divided by the lens magnification value in SL correction.

To our knowledge no studies have directly tested the hypothesis that a simple paraxial model can predict the image magnification produced by changing from SL to

CL correction. However, some indirect evidence is provided by Bradley et al. [12] when they found aniseikonia following CL correction to change in line with predicted retinal image size differences when compared to SL correction in the same subject. Applegate and Howland [25] have also cited magnification differences between spectacle and corneal correction as the reason for VA increases following refractive surgery.

Taking into account the differences in magnification occurring as a result of lens correction we will now look at the magnification effects relative to the emmetropic eye. As mentioned in Section 1, Knapp's Law (Knapp, 1869 cited in [12]) predicts retinal image size in the SL corrected axial myope will equal the retinal image size in the uncorrected emmetropic eye. From this and our model prediction that a CL corrected myope would produce a magnified retinal image relative to SL correction, it would seem reasonable to predict that the retinal image size in the CL corrected axial myope would actually increase in comparison to the emmetropic eye. In log MAR VA terms this would result in CL corrected axial myopes having improved VA in comparison to both SL corrected axial myopes and emmetropes if we assume there is no change in neural performance resulting from myopic growth.

In attempting to test this hypothesis we have to consider the variability of log MAR VA measures. Using test–retest measures of uncorrected vision, Lovie-Kitchin [21] demonstrated that a difference in Bailey–Lovie log MAR VA of 0.16 logunits could be confidently accepted as a real change in acuity. However, this value was based on test–retest variability in a group of uncorrected subjects. Assessment of this data set shows that a great deal of the variability found by Lovie-Kitchin ([21], see Fig. 1b) was at reduced VA levels. In this study we wanted to assess the variability of Bailey–Lovie measures at higher levels of VA as all of our subjects would be fully corrected. No other studies have investigated the variability of fully corrected subjects and to gain a more accurate indication of the variability we would expect in our corrected subjects we re-analysed the data of Lovie-Kitchin [21]. Our analysis only considered the subjects with acuity measures of 0.0 logunits (6/6) or better. This subgroup consisted of 33 subjects and the mean difference in log MAR VA was found to be 0 with a S.D. of 0.039 logunits. This relates to a significant change in corrected VA being of an order of magnitude of 0.078 logunits ($2 \times$ S.D.) or \approx four letters on a Bailey–Lovie chart. Even using this lower variance value, our predictions in Fig. 2 would suggest that any significant differences in VA resulting from magnification differences between CL and SL wearers will only be noted at higher levels of myopia.

3.1.3. Predicted effects of vitreous chamber growth in axial myopia

Myopic growth has the potential to modify visual performance by altering receptor and neural density in the retina [26]. Changes in retinal sampling density will lead to changes in the maximum resolvable spatial frequency [17,27], and hence may have a direct impact on VA. We have modelled the changes in retinal stretching and hence sampling density, and predicted the effect on visual resolution resulting from three types of myopic growth patterns: (1) equatorial stretching, (2) global expansion and (3) posterior pole expansion. Our model is based on the simplified emmetropic schematic eye [24] and the neural cut-off frequency in c deg^{-1} which aliasing occurs. The neural cut-off is termed the Nyquist limit (Vn):

$$Vn = \sqrt{3}s^{-1} \quad (3)$$

where s is the centre–centre spacing of foveal cones in degrees. Williams [27] calculated the Nyquist limit to be 56 c deg^{-1} ($\sim 193 \text{ c mm}^{-1}$) using the assumptions that: (1) the closest spacing of human foveal cones was $3 \mu\text{m}$; (2) 0.29 mm on the retina corresponds to 1 deg . These two assumptions are valid for the emmetropic eye but are subject to changes resulting from myopic growth. The three models we present here demonstrate the potential effect of myopic growth on the Nyquist limit and show how the effect is dependent on the type of stretching involved (Fig. 3). The emmetropic model eye was made up of a posterior sphere of diameter 22.37 mm with an additional anterior chamber depth of 1.8 mm . The anterior chamber depth was kept constant in every model.

The equatorial stretching model produces axial elongation by stretching/growth of the equatorial region of the globe. This produces no anatomical changes at the posterior pole and has no effect on retinal sampling in terms of samples mm^{-1} . However, the Nyquist limit will increase because the angular projection of a given extent of retina decreases with increasing axial length. Overall global expansion of the vitreous chamber (model 2) produces vitreous chamber elongation by uniform expansion across the entire sphere. In this model the linear centre–centre spacing will increase and therefore lower the Nyquist limit. However, this reduction will be counteracted, to some extent, by the increased size of one degree on the retina in relation to the predicted axial length change. The third model of axial elongation assumed that stretching took place at the posterior pole in a radially expanding manner from a radius positioned 5.59 mm in front of the fovea of our simplified schematic eye. There is no evidence in the literature to support any specific selective expansion model, but we felt that stretching from a point halfway between the centre of the emmetropic eye and the posterior retina would be representative of these types

of selective stretching. The three models are illustrated diagrammatically (Fig. 3a) along with the quantitative predictions for the effect of myopic growth on the Nyquist limit (Fig. 3b). The dashed lines in Fig. 3(a)

indicate the area of the retina where neural stretching takes place.

In normal human subjects the neural Nyquist limit is above the optical cut-off for foveal vision and below it in the periphery [28]. From our model predictions, depending on the type of myopic stretching at the retina, it is possible that the neural resolution limit for the fovea will decrease, perhaps below the optical cut-off, such that the foveal resolution limit of high myopes might be retinally and not optically limited. For example if we incorporate an optical cut-off of 45 c deg^{-1} into our model [29] and this cut-off remained constant at all levels of myopia (i.e. optical performance remains constant), then the neural resolution limit for the posterior pole model would fall below the optical cut-off at $\approx -5 \text{ D}$ (Fig. 3b). Interestingly this would lead to visual performance being limited neurally instead of optically in myopia $> -5 \text{ D}$. Of course this value is dependent on the unlikely assumption that the optical cut-off remains constant at all levels of myopia. The reported increased aberrations [13–15] have the potential to reduce the optical cut-off in higher myopes, making the transition from optically-limited to retinally-limited resolution occur at higher levels of myopia. In the case of global expansion we would predict that resolution only becomes sampling-limited with more than 15 D of myopia and in the equatorial stretching model increased Nyquist limits found with increasing myopia will result in resolution always being limited optically.

3.2. Experimental results

SL and CL VAs were measured for each subject and have been plotted against refractive error in Fig. 4(a). In both correction modes the VA decreased (MAR increased) with increasing myopia. A linear fit was applied to each data set. In the SL data the intercept was at -0.185 logunits and the correlation co-efficient (r^2) was 0.61 ($r = 0.78$). The slope of the data was 0.021 which approximately translates to a one letter reduction in log MAR VA per dioptre of increase in myopia. In the CL data the intercept was at -0.153 logunits and the correlation coefficient was 0.25 ($r = 0.50$). The slope was 0.011 which translates approximately to a one letter reduction in log MAR VA per two dioptres of myopia. From these values and the variance values calculated from the Lovie-Kitchin [21] data we would predict that a real reduction in VA would be confidently predicted at $>4 \text{ D}$ of myopia in SL and 8 D of myopia in CL correction. Also Fig. 4(a) gives the impression that our almost emmetropic subject (-0.25 D of myopia) has lower log MAR VA than the myopic subjects with up to -6 D of refractive error. We feel this result is probably due to our near emmetropic subject having lower log MAR VA than most emmetropes. This conclusion is based on the data of

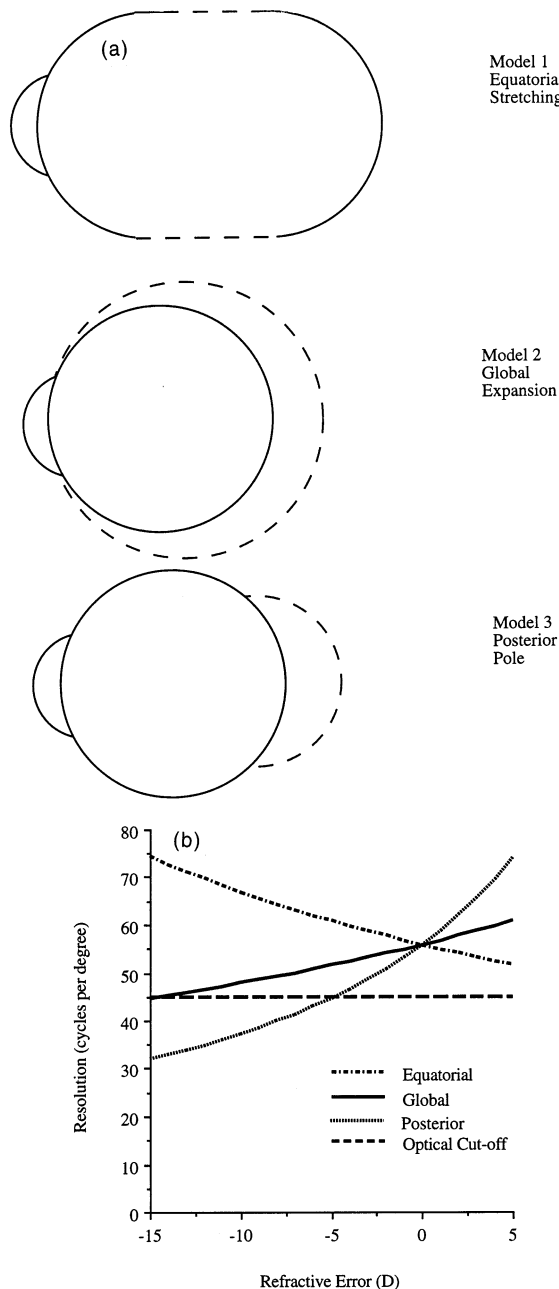


Fig. 3. (a) Diagrammatical representation of our three myopic growth models. The dashed lines represent the areas where neural stretching has occurred. The three figures are not scaled accurately and serve only as an indication of the type of growth taking place. (b) The graph represents the predicted changes in visual resolution resulting from three types of myopic growth in c deg^{-1} . In addition an optical cut-off of 45 c deg^{-1} is incorporated into the graph. Using the assumption that the optical cut-off remains constant the graph shows the posterior pole model neural resolution limit will fall below the optical cut-off at $\approx -5 \text{ D}$.

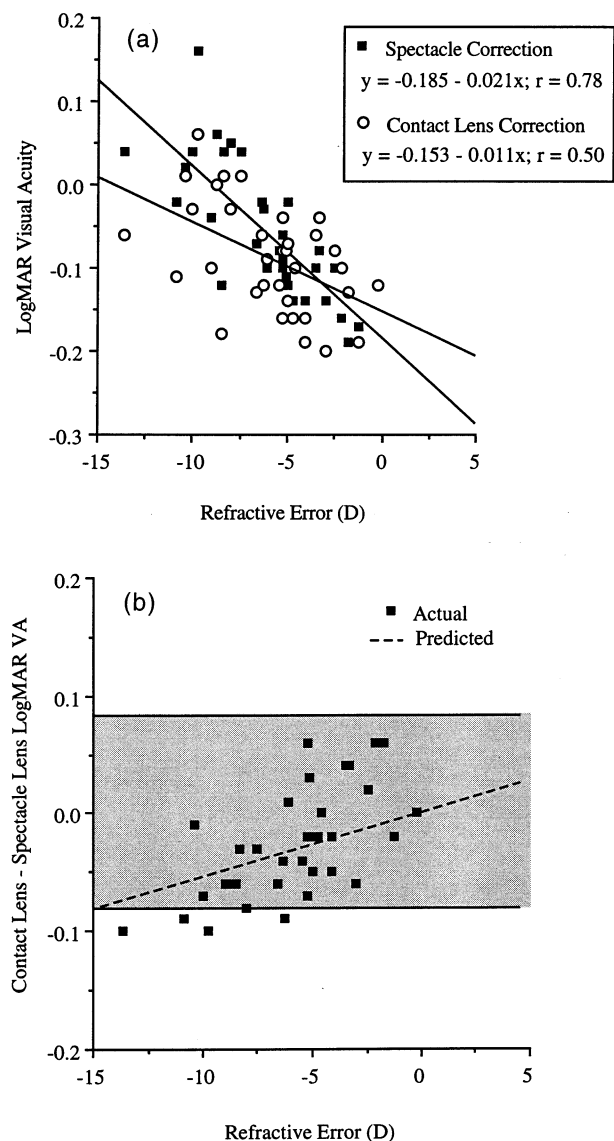


Fig. 4. (a) Comparison of CL and SL log MAR VA measures showing VA decreased (MAR increased) with increasing myopia. These findings indicate that our predictions from Knapp's Law and our magnification model are incorrect and validates the assumption that changes in the anatomical or optical performance are producing a reduction in VA in myopia. (b) The VA difference between CL and SL is compared to the difference predicted from our magnification model in Fig. 2. A good correlation existed between the difference in CL and SL and refractive error ($y = 0.032 + 0.01x$, $r = 0.65$). The dashed line is relative magnification difference between the two correction modes. In general terms VA was found to be better in CL correction than SL correction. The difference between the two groups is quite small and was not statistically different from the magnification model (paired t -test, $p = 0.367$). The shaded area represents the variance of the log MAR measures for subjects with VA of 0.0 logunit or better (re-analysed from the data of [21]). Data points within this shaded area cannot be considered as real changes in VA.

Lovie-Kitchin [21] who recorded unaided log MAR measures as high as -0.30 log units. As these measures were unaided we can assume the subjects were either emmetropic or had low degrees of hyperopia. This

suggests that emmetropes can have higher log MAR VA than the value found in our near emmetropic subject.

To assess the influence of magnification on the data we plotted the VA difference between CL and SL and compared the values to the VA difference we would predict from our magnification model (Fig. 4b). Both the model and the data indicate a similar trend with the difference in VA between the two forms of refractive correction increasing with increasing myopia. The VA difference between the two groups was not statistically different from that predicted by our relative retinal magnification model ($p = 0.367$). The data was subject to large variability and despite the predicted magnification of the CL image in comparison to the SL image some subjects performed better with SLs than CLs. In terms of our magnification model the general trend of the data would suggest that CL VA was superior to SL VA but the difference between the two measures was too small to measure accurately using log MAR charts. Any differences between the two correction modes were generally found to lie within the 0.08 logunits test-retest variance of the data ($\pm 2 \times$ S.D. of the re-analysed Lovie-Kitchin data) and therefore could not be considered true differences in log MAR VA. The test-retest variance value is represented by the shaded area in Fig. 4(b). The figure illustrates that four subjects with higher degrees of refractive error exhibited a real increase in VA as a result of CL correction. This suggests that moving the correction from the spectacle plane to the cornea can have a positive effect on VA [25] but this improvement can only be measured with confidence at higher levels of myopia. One interesting feature of this data is that the subjects who exhibit a real change in VA produce a greater effect than would be predicted by image size differences. This greater than predicted difference in some subjects may result purely from the inherent variability of the log MAR chart, however, we can not discount the possibility that this larger than predicted difference could be the result of aberrational differences between CL and SL correction in some individuals.

Our findings in Fig. 4(a) indicate that VA is reduced in higher levels of myopia in both CL and SL correction. This result is interesting in terms of our earlier predictions (based on the magnification model and Knapps Law) which show the retinal image size will be the same in SL corrected axial myope as in an uncorrected emmetrope and will be increased for a CL corrected axial myope. Theoretically, if no optical or neural changes occurred in the CL corrected axial myopic eye the increase in linear retinal image size should translate to an increase in the angular log MAR VA. In similar terms the SL corrected myope would be expected to have the same log MAR VA as the uncorrected emmetrope. The fact that both CL and SL

correction has resulted in reduced VA therefore suggests that changes in the anatomical or optical performance are producing a reduction in VA in myopia.

Having shown that either anatomical or optical changes (or a combination of the two factors) have contributed to a reduced VA in myopic eyes we will now examine what has changed. To compare the log MAR VA data with our anatomical predictions we converted both sets of values into c mm^{-1} in the retinal domain using simple paraxial optics theory. This seemed reasonable as our findings in Fig. 4(b) indicate conversion into c mm^{-1} will result in the two data sets essentially becoming duplicate measures of visual performance in each subject. To confirm this we compared the CL and SL values following conversion to c mm^{-1} (Fig. 5). A good correlation was found between the two data sets although there was some variability which was illustrated by the $y = x$ line in Fig. 5 ($y = 17.23 + 0.82x$; $r = 0.88$; $p = 0.126$). This variability was likely to result from the variance found between log MAR measures or aberrational differences between the two measurement modes. Both sets of converted values were plotted against refractive error in Fig. 6. Analysis of variance revealed a significant reduction in the visual performance with increasing myopic refractive error in both the SL correction data ($r = 0.81$; $p < 0.001$) and the CL correction data ($r = 0.70$; $p < 0.001$).

4. Discussion

Our biometric data confirm earlier reports [3–5] showing that most cases of significant myopia are due predominantly to axial elongation of the posterior chamber with changes in optical power of the eye only

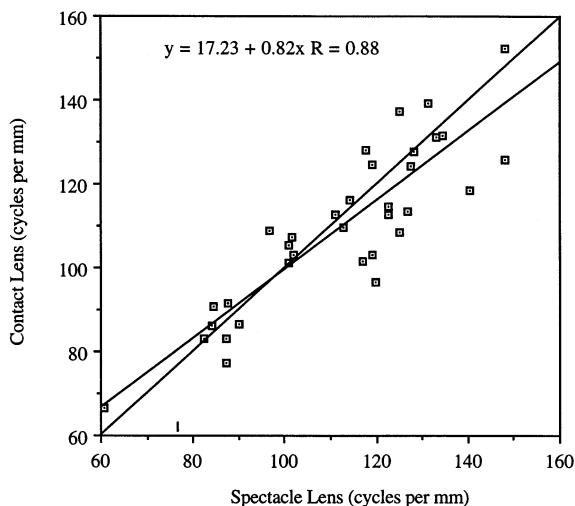


Fig. 5. Comparison of the SL and CL VA values following conversion to c mm^{-1} . A good correlation was found between the two data sets.

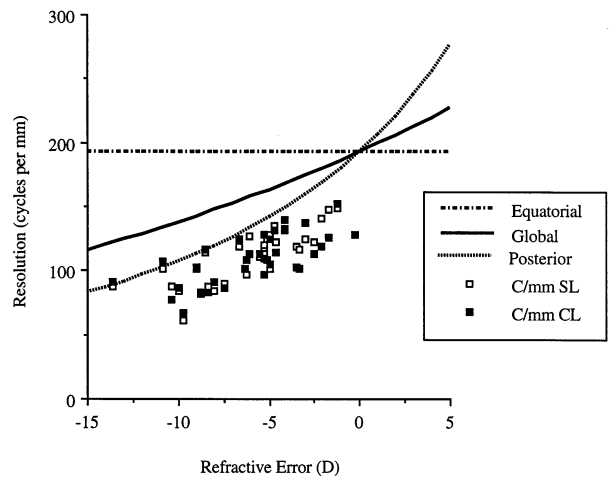


Fig. 6. Graph showing the visual resolution of each subject for both the SL data and CL data (in c mm^{-1}) plotted against refractive error. The model predictions of Fig. 3 following conversion to c mm^{-1} are also included. The graph shows the posterior pole model to most accurately predict the reduction in visual resolution with increased myopia. However most subjects were found to have lower visual resolution than the model prediction.

making a small contribution (Fig. 1). We also found, as others had [18,19], that VA decreased (MAR increased) with increasing myopia with both SL and CL corrections (Fig. 4a). Optical modelling in the methods section would predict that VA in axial myopia would actually improve in CL correction and would remain constant in SL correction. However, a real reduction in VA was found at higher degrees of myopia in some of our subjects and the general trend of the data was a reduction in VA in both correction modes. This result could not be accounted for by our optical modelling and suggests that either degraded retinal image quality or retinal stretching or some combination of the two are responsible for the reduction in VA. Comparison of our VA data in c mm^{-1} to predictions from three simple models of vitreous chamber growth patterns failed to demonstrate that decreased resolution was caused purely by decreased retinal sampling (Fig. 6).

Two possible explanations could account for the failure of our anatomical models to predict the decrease in VA observed experimentally in most of our subjects. One explanation is that all three retinal sampling models are wrong. Our posterior pole model most accurately reflects the reduction in visual resolution found with increasing myopia but in most subjects the model seems to overestimate the visual resolution. This may be the result of assumptions used in the construction of the model. One such assumption was the value of 5.59 mm from the posterior retina used as the radius around which posterior pole stretching occurs in a circular fashion. There is no evidence to suggest that this value is correct and if posterior pole stretching occurred from a point closer to the retina than 5.59 mm the model

would decline at a greater rate with increasing myopia. In contrast, the resolution value would decline at a lower rate if the value used in the model is > 5.59 mm. Our model is also dependent on the emmetropic Nyquist limit value used. We used a value of 193 c mm^{-1} (based on the calculations of [27]) as our emmetropic Nyquist limit and based all our modelling calculations around this value. The use of a lower emmetropic Nyquist value would result in an improved model prediction of the data. A further limitation of our model is the assumption that all myopia is axial in origin. The data in Fig. 1 shows that despite the large percentage of myopia resulting from axial elongation there is some corneal contribution which is more obvious in certain individual subjects. The introduction of a corneal component will modify our model but is unlikely to increase the accuracy of the predictions as corneal involvement will result in axial elongation (and therefore neural stretching) being reduced which in turn will produce an increase in the posterior pole model resolution limits.

The other possibility is that the decrease in VA is due to a decrease in the optical quality of the retinal image as a result of the ocular growth process. There are now several studies that show that optical aberrations, and in particular spherical aberrations increase with increasing myopia [13–15]. Our study therefore supports the idea that myopia includes changes in the anatomy of both the vitreous chamber, resulting in myopia, and the anterior eye, resulting in decreased retinal image quality.

The above conclusion must be considered tentative as some limitations exist in the study that have not been accounted for in our analysis. Considerable differences in optical aberrations are thought to exist between the SLs and soft CLs [30]. These aberrations are primarily longitudinal spherical aberration, coma and defocus due to field curvature and astigmatism. The magnitudes of the aberrations are dependent on individual corneal asphericity that has recently been found to flatten less rapidly in higher degrees of myopia [23]. We did not account for aberration changes between the two correction modes in our analysis as overall aberration levels will vary considerably among individuals and are difficult to predict. Interestingly, the differences in VA between SLs and CLs demonstrated that some high myopes VA increased more than the magnification model predicted with CLs. This higher value probably results from magnification factors and the variability of log MAR VA measures alone but the possibility of a CL related reduction in optical aberrations in high myopia cannot be discounted. The greater than predicted increase in VA between correction modes found in this study was not demonstrated in the results of Collins and Carney [19]. In their data switching correction modes from CL to SL was well predicted by

magnification changes in their highly myopic subject group at high spatial frequencies.

Another limitation of the study is the assumption that letter MAR will correspond to grating resolution limits throughout our anatomical and optical modeling. Letters differ from gratings in that they contain a broad range of spatial frequencies and components of multiple orientations [31]. Letter acuity is also more sensitive than grating acuity to changes in resolution resulting from blur [29,32], amblyopia [33] and foveal eccentricity [34]. Despite this our assumption seems reasonable as the resolution limits for gratings and letters at the fovea in a range of fully corrected subjects seem to correspond [32]. However as we demonstrate in this study retinal stretching in myopic eyes has the potential to produce changes in neural sampling which in turn could lead to differences being found between grating and letter acuity.

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