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Myopia, posture and the visual environment

W Neil Charman

Faculty of Life Sciences, University of Manchester, Manchester, UK

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Correspondence: Neil Charman
E-mail address: neil.charman@manchester.ac.uk

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Abstract

Evidence for a possible role for the peripheral retina in the control of refractive development is discussed, together with Howland's suggestion (Paper presented at the 13th International Myopia Conference, Tubingen, Germany, July 26–29, 2010) that signals to generate appropriate growth might be derived from ocular oblique astigmatism. The dependence of this, or similar peripheral mechanisms, on exposure to a uniform field of near-zero dioptric vergence is emphasized: this is required to ensure a consistent relationship between the astigmatic image fields and the retina. This condition is satisfied by typical outdoor environments. In contrast, indoor environments are likely to be unfavourable to peripherally-based emmetropization, since dioptric stimuli may vary widely across the visual field. This is particularly the case when short working distances or markedly asymmetric head postures with respect to the visual task are adopted.

Introduction

For more than a century, those interested in refractive development have wrestled with the question of why some individuals become myopic.[1–4] The search for an answer has become more urgent with the finding that the prevalence of myopia has risen markedly over the last few decades in many parts of the world.[5–9] While genetic factors undoubtedly play a major role,[8,10–12] these cannot account for the recent steep increase in myopia prevalence.

Attention was focused initially on axial refraction. Animal experiments showed that this could be modified by lenses, occlusion, diffusers and other methods, implying that visual experience after birth influenced the eye's final refractive state[13–17] and that an active emmetropization mechanism must exist, as proposed originally by Straub (cited by Sorsby[18]). The effectiveness of this mechanism and its endpoint are, presumably, affected by genetics, environment, lifestyle and, perhaps, other factors such as diet and disease.[19,20] While nearwork has long been accepted as being in some way involved in myopization,[1–4] it appears that periods of outdoor activity may offer protection against myopic change.[21–23] More recently, following early work by Hoogerheide *et al.*,[24] it has been suggested that refraction and imagery in the peripheral retina might be of importance.[25–30] Interestingly, early workers emphasized the importance of posture,[1,3,31] although few later papers have been devoted to this topic.[32,33]

It is argued here that consideration of the spatial distribution of the dioptric stimuli offered by outdoor and indoor visual environments offers a possible approach to the integration of these ideas into a broader understanding of the origins of at least some types of myopia. The question of the classical distribution of refractive error is considered first and then the possible role of the peripheral retina in emmetropization, Next consideration is given to the impact of the different visual environments on the proposed emmetropization process, and lastly to the effect of posture.

What do we mean by emmetropia?

As is well known, the classical distribution of refractive errors in unselected populations is not normal but shows an excess of values in the range about 0 to +2 D (*Figure 1a*), even though the values of individual ocular parameters are normally distributed. Tonic accommodation levels are typically around 1 D (*Figure 1b*). Exercise

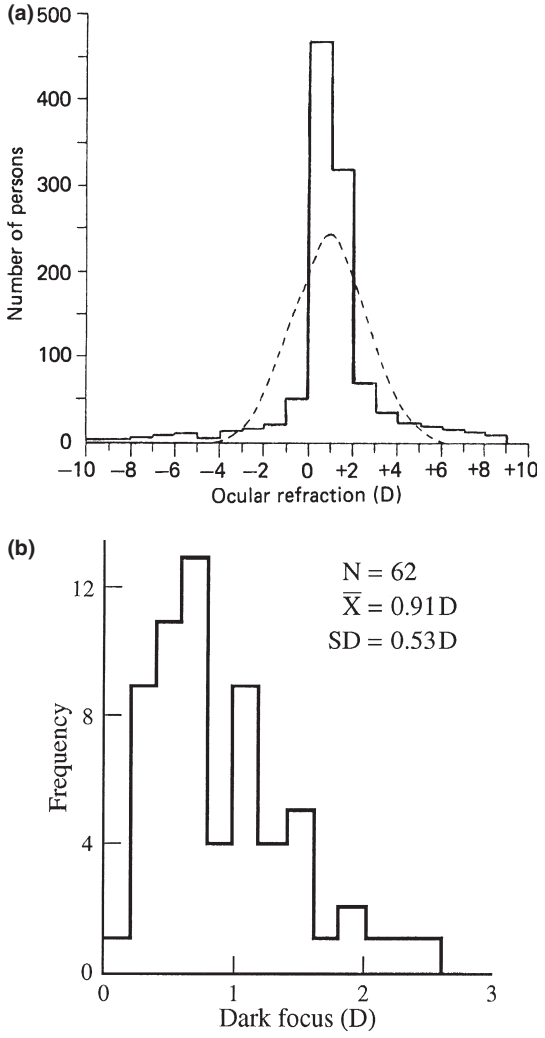

Figure 1. (a) Typical example of the distribution of refractive error in an unselected population (based on 1033 young men, after Sorsby *et al.*[34]) The dotted curve represents the corresponding normal distribution. (b) Distribution of tonic accommodation (dark focus) in 62 young adults (after McBrien & Millodot[35]).

of this accommodation would therefore shift the peak of the refractive distribution to around 0 D. It therefore appears that any emmetropization mechanism responsible for normal refractive development must be capable of producing the required distance focus with an accuracy of the order of ±1 D. The refraction of both eyes usually develops in a very similar manner, with roughly 75% of the population having anisometropia ≤0.25 D.[36] The emmetropization process appears only to be capable of operating over a limited range of initial refractive error. Hirsch[37] suggested that, at the age of 6 years, those who were myopic, even by a small amount, would generally have increased myopia by the age of around 14 years, while those who refractions were +1.5 D or more would either maintain or increase their hyperopia (see also Zadnik *et al.*[38]).

Emmetropization: a role for the peripheral retina?

Although other emmetropization mechanisms based on axial focus have been proposed,[39–41] only a recent hypothesis involving the peripheral retina will be considered here. As noted earlier, interest in the influence of peripheral imagery derives from the work of Hoogerheide *et al.*[24]. In a longitudinal study of young airline pilots, they found that those suffering a progressive myopic shift in axial refraction initially tended to be relatively hyperopic in the peripheral retina. In contrast, refraction remained stable in those pilots with peripheral relative myopia. Animal experiments show that if negative lenses are used to place the image behind the retina, the developing eye grows to bring the image into focus, i.e. it becomes myopic.[13–17] It has therefore been suggested[25–30] that, since in the young pilots with peripheral hyperopia the peripheral image lay behind the retina, a local retinal signal was generated which resulted in an appropriate local increase in growth to bring the image into focus. Constraints on eyeball shape meant that this tended to also cause an increase in axial length, resulting in an axial myopic shift. Although the study on pilots involved young adults, the assumption is that a qualitatively similar mechanism might apply in children. Recent experiments with monkeys using peripheral form deprivation or annular lenses to produce relative hyperopia in the peripheral field confirm that peripheral imagery can influence axial length, even though the central field remains unobstructed.[42–44] Further studies show that emmetropization can still occur in the absence of an intact fovea.[44,45] Additional support for the concept that focus in the peripheral retina is important for emmetropization comes from clinical evidence in humans, which indicates that myopia is more common among children suffering from pathologies which affect peripheral vision.[20]

Is the peripheral retina sensitive to defocus? Cone and ganglion densities diminish rapidly with field angle, so that peripheral resolution is low and insensitive to defocus.[46] However, this is not the case for detection sensitivity or a variety of other visual functions, where focus has to be held between fairly narrow limits if peak performance is to be maintained. *Figure 2* compares total depth-of-focus, defined here as the full dioptric range over which detection threshold remains above 75% of its peak value, at different retinal locations. The square symbols are taken from Wang *et al.*'s data[46] for the detection of radial and tangentially-oriented gratings by single subject, and the two circular points are derived from mean contrast thresholds for two subjects and 0.2° diameter

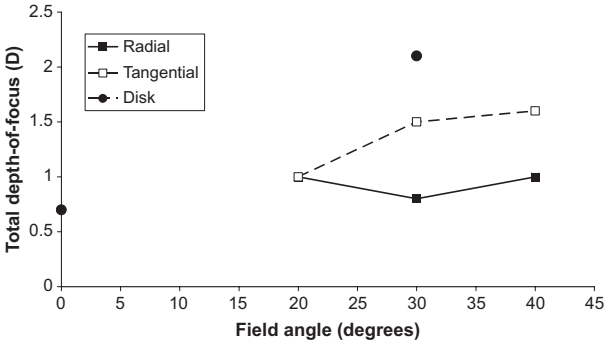

Figure 2. Total depth-of-focus as a function of field angle along the horizontal meridian. The open and filled squares are based on data for the detection of radial and tangential gratings respectively, for a single subject.[46] The filled circles are based on the contrast sensitivity for a 0.2° white disk.[47] Total depth-of-focus is taken as being the dioptric range over which performance is ≥75% of its peak value.

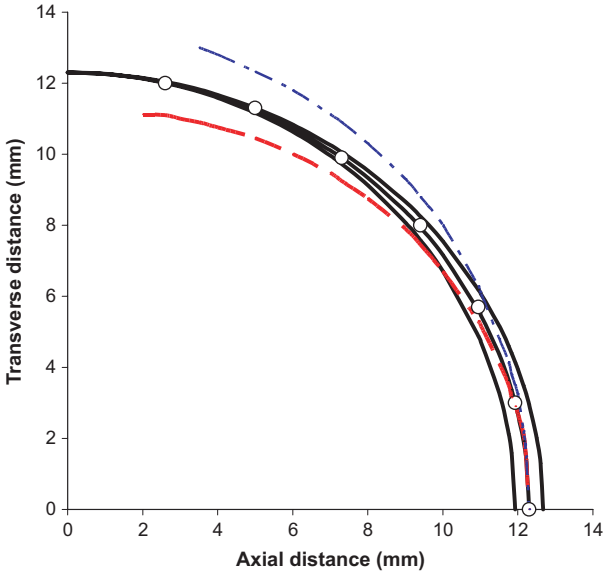

Figure 3. Relationship between astigmatic image surfaces and arcs of the nasal retina (black lines) for different axial lengths (see text). The red and blue dashed lines represent the approximate form of the tangential and sagittal image surfaces respectively. The filled circles represent the points at which the chief rays from the centre of the aperture stop (with its centre axially positioned at −8.3 mm) cut the spherical retina: the corresponding field positions are 0, 10, 20, 30, 40 50 and 60°.

white disks against a white background.[47] It is obvious that errors of focus greater than about ±1 D are likely to cause detectable drops in detection performance over a substantial area of retina. Although the judgements involved in the detection tasks of *Figure 2* involve higher level processing, it seems reasonable to expect that focus shifts of this magnitude must affect neural outputs at the retinal level and that, at least in principle, these might influence refractive development.

An obvious problem is how the peripheral retina derives a defocus signal that can control growth. Howland[48] has recently sketched an ingenious solution. He points out that, like all optical systems, the eye suffers from oblique astigmatism. In dioptric terms, this varies approximately as the square of the field angle and amounts to about 6 DC at a field angle of 60°.[49] Thus, rather than there being a unique image surface at each point in the periphery, there are two surfaces, corresponding to the radial (sagittal) and tangential focal line images of each object point: the tangential surface lies anterior to the sagittal surface. Although for convenience it is often said that eyes have relative peripheral hyperopia or myopia, these descriptions refer to the spherical equivalents of the refractions rather than implying the existence of a unique image surface.

Howland[48] suggests that, under normal conditions, the emmetropization mechanism makes use of the positions of the two astigmatic image surfaces relative to the retina to control eye growth. He notes that there is good evidence for the existence in the peripheral retina of neurons tuned to the orientation of image structure.[50] He therefore hypothesises that retinal circuitry exists which allows local comparison of the outputs of those neurons tuned to edges or other structures which are oriented radially (i.e. parallel to the field meridian) and those which respond to tangential structure (i.e. perpendicular to the field meridian).

Figure 3 illustrates this situation. Arcs of the horizontal nasal retina are shown for three eyes, one of which has a spherical retina, the others which are axially too long or too short by amounts which correspond to about one dioptre of myopia and hyperopia respectively: all the eyes are assumed to have the same transverse semi-diameter. The posterior portion of the eyeball is therefore part of a prolate ellipsoid for the 'long', myopic eye and of an oblate ellipsoid for the 'short', hyperopic eye. The simplifying assumption has been made that the anterior optics of the eye and the associated image surfaces remain constant. The two astigmatic image surfaces are drawn to correspond to the typical levels of oblique astigmatism given by Atchison and Smith,[49] and the open circles indicate the approximate points at which the chief rays cut the spherical retina at 10° intervals of field angle, based on Lotmar's[51] wide-angle eye model: note that the chief rays are not perpendicular to the peripheral retina. In terms of Howland's hypothesis it can be seen that, over a range of field angles, if the eye is too short (hyperopic) the tangential image surface lies closer to the retina and the output of the corresponding neurons will be higher.

Ocular growth will then accelerate. If, on the other hand, the eyeball is too long, the sagittal image surface will be nearer the retina and the outputs of the 'sagittal' neurons will exceed those of the 'tangential' population, leading to slowed ocular growth. Only when the two image surfaces bracket the retina in an approximately symmetrical way will the outputs of the two populations of neurons be in balance, stabilizing growth to maintain an emmetropic eye. This implies that the mean spherical error of emmetropes should be close to zero across the visual field, as observed in practice.[52]

What is not clear in Howland's suggested mechanism is the weighting that might be associated with different areas of the retina in the control of refractive development. For a constant increment in field angle, the annular area of retina increases linearly with the field angle, implying that the more peripheral areas of the retina might be of greater importance. On the other hand, if the anterior eyeball is constant in form (admittedly an oversimplification) and only the posterior part of the eye varies in its growth, the spatial position of more extreme periphery of the retina is unaffected by the refraction and hence cannot influence axial length. As can be seen from *Figure 3*, modelling[51–53] suggests that this insensitivity might occur at field angles ≥50°. On the other hand, amounts of oblique astigmatism are small for field angles ≤15°. Roughly speaking, then, the hypothetical mechanism would be expected to be most effective for field angles between about 15 and 50°, over which oblique astigmatism in the horizontal meridian varies from about 1 to 5 DC, with perhaps the greatest weighting occurring around the centre of this range. For comparison, in their experiments on monkeys, Smith *et al.*[42–44] found abnormal refractive development when, even though there was an intact central visual field up to around 40° in diameter, the periphery was form-deprived or hyperopically-defocused.

There is a further interesting aspect that follows from Howland's basic hypothesis. Hoogerheide *et al.*'s[24] findings with young pilots suggested that, among emmetropes, those with initial peripheral hyperopia tended to become myopic. If we again make the simplifying assumption that all individuals had the same anterior optics and hence the same astigmatic image surfaces, the existence of relative peripheral hyperopia implies that these eyes possessed eyeballs which were initially relatively more prolate in shape, than those of eyes with relative peripheral myopia.[54–57] Presumably even though growth of the adult eye is normally largely complete, the hyperopic peripheral field can still stimulate some further growth but a myopic peripheral field, corresponding to more oblate eyeball, cannot 'slow' growth which has already occurred.

Influence of the visual environment

If Howland's hypothesis, or some variant of it, is really the explanation of emmetropization then it is reasonable to ask why some individuals start to become myopic in childhood or later and why the prevalence of myopia has increased. It is here that the visual environment, and its changes in recent years in some parts of the world, may be of importance.

One assumption inherent in Howland's emmetropization model, although not discussed in his original presentation, is that the vergences of dioptric stimuli approximate to zero across the entire visual field. This is necessary to ensure that there is a consistent relationship between the astigmatic image surfaces and the retina. However if, say, the lower visual field was relatively near, with a dioptric vergence of −2 D, while the upper was at 0 D, the image surfaces in the 'emmetropic' case of *Figure 3* would both lie at the equivalent of 2 D behind the upper retina, accelerating growth in that region. Maintenance of this state might therefore be expected to lead to an asymmetric eyeball. The value of outdoor activity as an inhibitor of refractive change[21–23] might therefore derive from the fact that outdoor environments closely approximate to Howland's requirement for a visual field whose vergence does not change significantly with position and is always near zero.

In contrast to the outdoor environment, the values of indoor dioptric stimuli usually vary widely across the visual field (*Figure 4*), potentially ranging from zero to the vergence of the closest object present. As the eyes fixate and accommodate on different objects within the indoor scene, no consistent relationship is likely to exist between the retina and the image surfaces in the periphery. Thus the possibility for effective functioning of any emmetropization mechanism which relies on the integration of systematically-varying defocus signals across the peripheral retina is minimal.

Presumably exposure to a distance environment for relatively short daily periods might be enough to set the normal growth mechanism for emmetropia[21–23] even though a child might spend much of the day in indoor activities, in much the same way that, in animal experiments, relatively brief periods of normal vision negate the effects of form deprivation.[57] The recent longitudinal data of the CLEERE study group[23] show that, for at least 3 years before and 3 years after onset of myopia, hours per week spent in outdoor activities by children who became myopic were significantly less (by around 1–2 h) than for those who remained emmetropic: in contrast, there was little difference between the hours spent on other activities before myopia onset.

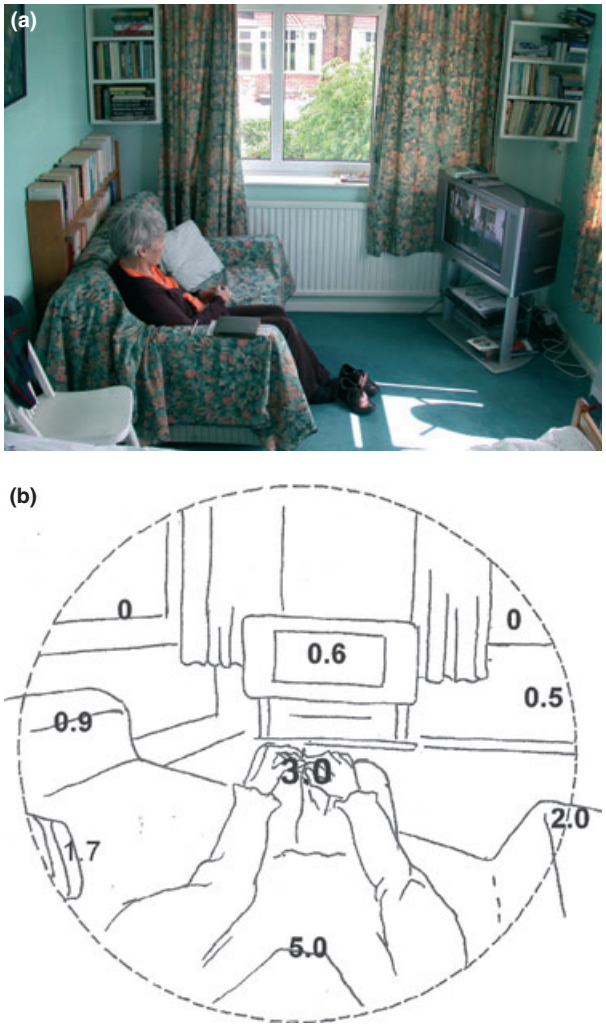

Figure 4. (a) Domestic interior (b) Approximate corresponding vergences of dioptric stimuli within a field of 50° radius, from the point of view of the seated subject when threading a needle. Note the wide range of vergences present and their irregular spatial distribution.

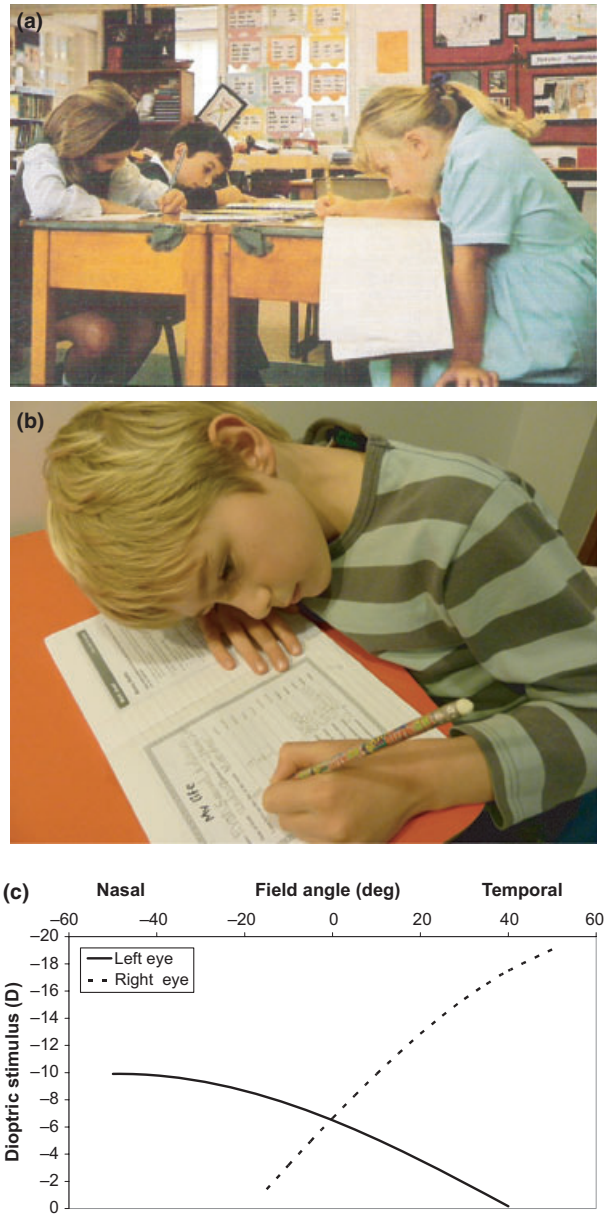

Figure 5. (a) A group of children undertaking a school writing task. Note the differing postures and relatively short working distances. (Photograph by courtesy of *Optometry Today*) (b) A schoolboy writing and (c) approximate corresponding variation of dioptric stimuli along the nasal-to-temporal meridian of the field for each eye of the child in (b). For simplicity it has been assumed the desk is wide.

Effect of posture

For the individual, the range of dioptric stimuli experienced will obviously depend upon both the environment itself and the head position and orientation within that environment. Short working distances are likely to increase the stimulus range. Although formal studies suggest that children adopt reasonably long working distances for reading and writing (about 0.25 m for 6–10 year-olds[58]), this is not always the case (*Figure 5a,b*), particularly when the tasks are of long duration and the children take up their preferred relaxed postures. Working distances may be very short and pronounced head turn may mean that the spatial distributions of dioptric stimuli available to each eye may differ markedly. For example, in *Figure 5b* a boy (left-handed) views his work with his head resting almost horizontally on his right hand and pronounced head turn with respect to the page and desk surface. The approximate values of the dioptric stimuli along the nasal-to-temporal meridian of the field of each eye are given in *Figure 5c*. Even with symmetrical convergence, it can be seen that, because of the obliquity

of the lines of sight with respect to the child's workbook and desk, wide variations in vergence occur along this field meridian. Dioptric stimuli are higher for the right eye because it is closer to the desk surface. Variations in stimulus vergence also occur along all other field meridians. Assuming that accommodation to the fixation point is maintained, much of the peripheral field must be badly out-of-focus. Under these circumstances, in which most of the peripheral field is markedly out-of-focus, it seems unlikely that Howland's suggested emmetropization mechanism (or any other mechanism based on peripheral imagery) could function effectively. Hence a type of form-deprivation myopia would result.

Discussion

The suggested overall approach, depending upon peripheral retinal imagery, appears to provide a plausible framework for understanding aspects of emmetropization and some of the possible factors in modern educational and other environments which might be involved in the greater prevalence of myopia. However, it glosses over many important problems such as the causes and influence of nasal/temporal,[52] vertical/horizontal[59,60] and other asymmetries, the possible influence of axial astigmatism, the apparently more irregular eye shape in myopia,[61] the interactions between genetic susceptibility and optics and so on. It may, for example, be that emmetropization effects in different meridians are influenced by the lack of rotational symmetry about the fovea in the retinal structure,[62] decentrations or tilts of components[63] or by the growth constraints imposed by the orbit or extraocular muscles.[52] The discrepancy between the optical and visual axes may also play some role.[64]

Perhaps the most interesting feature of these general ideas is the detrimental role of the non-uniform spatial distribution of dioptric stimuli across the visual field which is typically found in indoor environments, particularly when short working distances and head tilts occur. Note again that the problem is not necessarily the occurrence of high accommodation demands as such that is the problem, but rather the range of dioptric stimuli across the field. It would be predicted that those children who habitually adopt short working distances and asymmetric postures with substantial head tilt for writing, reading or playing computer games might be at greater risk of developing myopia. The implication is that children should be encouraged to sit back from their work and avoid such asymmetric postures. This would to some extent represent a return to the 'visual hygiene' recommendations of Donders[1] and other early workers, although their recommendations were primarily based on the desire to avoid substantial accommodation and convergence as well as forward head tilt, which was believed to produce 'ocular congestion' and globe distension.

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