

Ministry of Defence

Synopsis of Causation

Refractive Error

Author: Dr Paul Johnstone, Ninewells Hospital and Medical School, Dundee
Validator: Dr David Gartry, Moorfields Eye Hospital, London

September 2008

Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

The Ministry of Defence accepts full responsibility for the contents of this synopsis, and for any claims for loss, damage or injury arising from the use of this synopsis by the Ministry of Defence.

1. Definition

- 1.1. Refractive error is said to exist when the eye fails to bring parallel light (distant objects) to focus on the retina. There are 3 types of refractive error: myopia, hypermetropia and astigmatism.
- 1.2. In the myopic (short-sighted) eye, distant objects are brought to focus in front of the retina. This may be because the eyeball is too long (axial myopia) or the refractive elements of the eye too powerful (refractive myopia).
- 1.3. In the hypermetropic (long-sighted) eye, distant objects are brought to focus behind the retina. This may be because the eyeball is too short (axial hypermetropia) or the refractive elements of the eye are inadequate (refractive hypermetropia).
- 1.4. In astigmatism, the refractive power of the eye varies depending on which meridian light enters the eye. If these meridians lie at 90° to each other then regular astigmatism is said to exist. If the meridians lie at 90° to each other but not necessarily in the horizontal or vertical meridians, this is termed 'oblique astigmatism'. If the meridians do not lie at 90° to each other then this is termed 'irregular astigmatism' and is difficult to correct with lenses.
- 1.5. Emmetropia is the normal state i.e. absence of refractive error. Light is brought to a clear focus on the retina without any accommodative (focussing) effort.
- 1.6. Presbyopia is difficulty bringing near objects into focus due to the normal, steady, age-related decline in accommodative power of the lens.

Diagram illustrating types of refractive error

Figure 1 Emmetropia

Figure 2 Myopia

Figure 3 Hypermetropia

Figure 4 Astigmatism

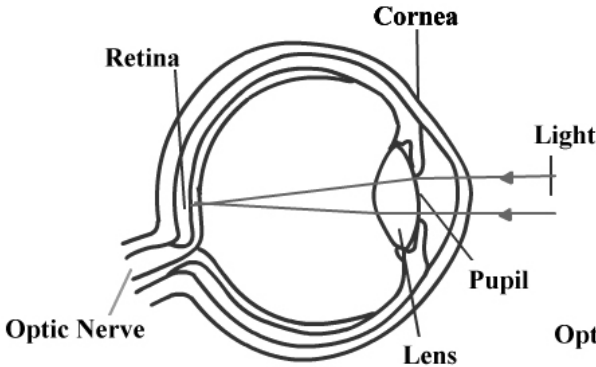


Figure 1

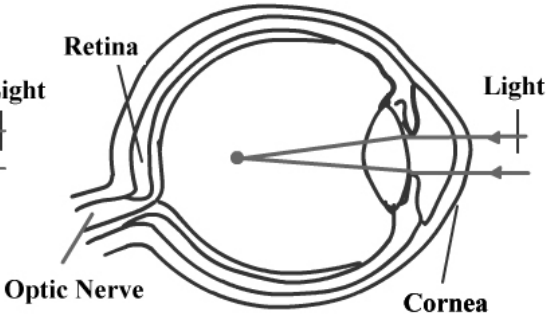


Figure 2

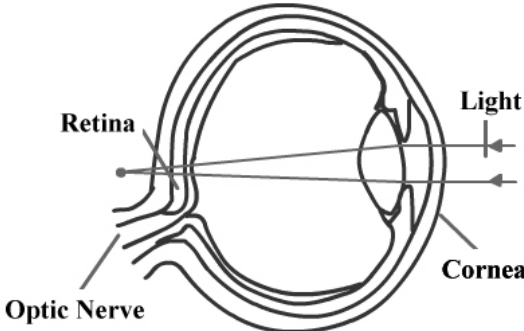


Figure 3

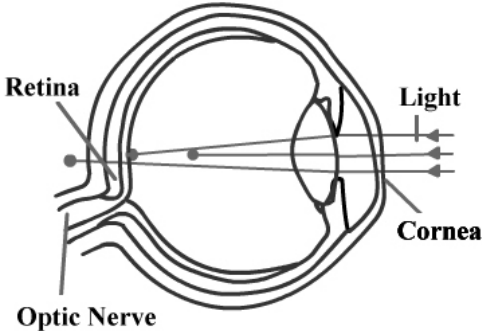


Figure 4

2. Clinical Features

- 2.1. The prevalence of refractive errors depends on the definition of the presence of refractive error. For example, persons with refractive error of +0.25 may be considered either hypermetropic or clinically emmetropic because this level of refractive error has no significant impact on function, nor does it necessitate the need for spectacles.
- 2.2. Different studies have used slightly different inclusion criteria for the presence of myopia and hypermetropia. These studies have been performed in different geographic populations and various age groups. In adults over 40, Hyams et al¹ found prevalence rates of 18% myopia, 57% emmetropia and 24% hypermetropia. A large study that included adults over 40 in the US, Western Europe and Australia² found prevalence rates of 5.8-9.9% hypermetropia and 16.4-25.4% myopia. Adults in Norway³ were found to have prevalence rates of 13-17% hypermetropia and 30-35% for myopia. These large multi-centre prevalence studies offer an overview of prevalence but have limitations because of the slightly different inclusion criteria. Although some studies have described a change in the prevalence of refractive error the studies performed to date suffer from the limitations described in this paragraph.
- 2.3. Hypermetropia may be detected at pre-school screening.
- 2.4. Hypermetropia may present as a squint. Convergent squints may be a result of hypermetropia. This is due to the excess convergence associated with the extra accommodative effort employed by the eye in an attempt to produce a focussed image.
- 2.5. In the presence of squint or refractive error it is important to exclude the presence of amblyopia. This is a condition where lack of visual maturation occurs, typically in one eye, due to the preferential use of the better-seeing eye. If this is not treated appropriately in early childhood, permanently reduced vision will result.
- 2.6. Myopia typically occurs in childhood between the ages of 8 and 14. It usually affects both eyes similarly. It may be discovered on screening programmes or by teachers noticing school pupils unable to visualise items on the board. Children and teenagers may complain of difficulty seeing distant objects.
- 2.7. Astigmatism may cause difficulties in distance and near vision although small clinically irrelevant amounts of astigmatism are very common. It is often found in combination with myopia or hypermetropia. Patients may experience blurred vision or appreciate aberrations of the visual image such as abnormal elongation, distortion or monocular double vision. Patients are probably more likely to be symptomatic if there is an acquired aetiology or the abnormality is asymmetrical.
- 2.8. Diagnosis of refractive error is confirmed by performing a refraction examination. This quantifies the level of refractive error by identifying the power and type of lenses required to correct the defect.
- 2.9. Asthenopia is a term used to embrace symptoms related to refractive error. This includes blurred vision, tired eyes, eye-strain, periocular discomfort or even headache.

- 2.10. Headache is a possible clinical feature of refractive error. Headache associated with refractive errors (HARE) is described in the International Headache Society's (IHS) classification system.⁴ The diagnostic criteria for HARE are:
- 2.10.1. Recurrent mild headache, frontal and in the eyes themselves, fulfilling the following criteria:
- Headache and eye pain first develop in close temporal relation to the refractive error, are absent on awakening and aggravated by prolonged visual tasks at the distance or angle where vision is impaired
 - Headache and eye pain resolve within 7 days, and do not recur, after full correction of the refractive error
- 2.10.2. Uncorrected or miscorrected refractive error (e.g. hypermetropia, astigmatism, presbyopia, wearing of incorrect glasses).
- 2.11. Some authors feel refractive error as a cause of headache may be overstated. One study concluded that headache associated with refractive error (HARE), as defined by The International Headache Society (IHS) criteria, can occur in individuals with refractive error but is rare.⁵ They found no difference in overall headache rates between subjects with refractive error and controls. In the subject group, 6% had headaches that fitted the IHS criteria for HARE. Headache associated with refractive error appeared to be linked with hypermetropia, more so than any other refractive error.
- 2.12. The relationship between uncorrected refractive error and migraine was cited by authors in the early 1900s. More recent studies and reviews have found no significant difference between migraine sufferers and control groups with regard to refractive error.⁶⁻⁸

3. Aetiology

Myopia. Myopia has been studied more extensively than other refractive errors to ascertain the relative importance of genetic and environmental factors.

3.1. **Genetic factors.** There is strong evidence that genetic factors play a part in the aetiology of myopia. It has been observed that identical twins show close agreement in their refractions whereas non-identical twins show little more agreement than control pairs of siblings.⁹ Children of myopic parents tend to have longer eyes, even before developing myopia,¹⁰ and the gene for myopia has been identified in particular families.^{11,12} Studies in young adult populations in the United Kingdom and Denmark compared genetic and environmental factors, concluding there was high heritability of ocular refraction and environmental impact was not significant.^{13,14}

3.2. **Environmental factors.** Other studies have highlighted the association of myopia with environmental factors.

3.2.1. Although these studies have highlighted associations between environmental factors such as near work and myopia, causality cannot necessarily be inferred. Several epidemiological studies have identified higher rates of myopia and progression amongst university students and length of time studying.^{15,16} Occupations requiring intense close work (microscopists, textilers) have also been associated with the development and progression of myopia.¹⁷

3.2.2. Many animal studies have identified induction of myopia secondary to altering the environment of the developing eye. Deprivation of visual stimulation induces myopia in primates.¹⁸ Eyes of children with unilateral visual deprivation, from concurrent pathology, also have been shown to become myopic.¹⁹ The inference from these studies is that there is a local feedback mechanism involving the unfocussed image that controls ocular growth in early development.

3.2.3. Studies on the use of visual display units (VDU) have failed to identify the use of VDU as a cause of onset or progression of myopia.^{20,21} A small transient myopic shift, insufficient to reduce distance acuity, may occur immediately following VDU use but the significance of this is unknown.²² Dry eyes appear to be the major contributor to asthenopic symptoms related to computer use.

3.2.4. Improper lighting conditions of computer workstations may contribute to ocular discomfort. Symptoms of eye strain, blurred vision and particularly glare may be a problem. Measures such as removing intense fluorescent lights, attention to light positioning and computer screen anti-glare filters may improve visual comfort.²²

3.3. **Hypermetropia.**

3.3.1. **Genetic factors.** Like myopia, genetic factors appear to be the major determinant of hypermetropia, although far fewer studies have been published. Hammond et al.²³ demonstrated hypermetropia to be genetically determined to a degree very similar to myopia.

3.3.2. **Ageing.** In adulthood, with the natural onset of presbyopia, there may be some unmasking of hypermetropia that was previously overcome by accommodation. This is not necessarily a result of some external factor.

3.3.3. **Other conditions.** Concurrent conditions that effectively shorten the distance between the cornea and posterior pole may induce hypermetropia. For example, external globe compression from an orbital tumour,²⁴ serous elevation of the retina,²⁵ lid lesions²⁶ that flatten the cornea and reduce its effective power (lid lesions more commonly induce astigmatism due to the uneven distribution of pressure caused by the lesion – typically a cyst.)

3.3.4. **Metabolic changes.** Changes that alter the osmolality of the lens such as hypoglycaemia in diabetes, may induce a fluctuation in refractive error.

3.4. **Astigmatism.**

3.4.1. **Genetic factors.** These play a role in the aetiology of astigmatism, but this may be far less than in hypermetropia or myopia.^{23,27} There are, however, indications that a dominantly inherited form of astigmatism may exist.²⁸

3.4.2. **Acquired astigmatism.** This may result when the curvature of the cornea is altered. This may be due to dysplasia, e.g. keratoconus, or abnormal growth of tissue on the cornea e.g. pterygium. Infection of the cornea, either bacterial or viral (typically herpetic) may induce astigmatism²⁹ during the infection, or from resultant scarring. Scarring may also result from lacerating or penetrating trauma^{30,31} or chemical/thermal injury. Ocular surgery involving the cornea, e.g. cataract surgery,³² corneal grafting³³ or excimer laser,³⁴ may all directly cause astigmatism. Other forms of ocular surgery such as retinal surgery³⁵ or squint surgery³⁶ may also induce astigmatism.

3.4.3. **External factors.** Compression by external lid or orbital lesions, as for hypermetropia, are recognised causes of astigmatism,²⁶ this is often reversible with removal of the compressive lesion.

4. Prognosis

- 4.1. Refractive changes in adults over the age of 40, over a 10 year period, are small (<0.5D[dioptre]) and dependant on age (mild hypermetropia in 40s, mild myopia in 70s.)³⁷ This may also be dependent on the presence of cataract, which may induce a myopic shift in refraction.
- 4.2. Myopia may progress relatively quickly in children and young adults (0.5D per year.)³⁸
- 4.3. The mainstay of treatment is correction of the refractive error with spectacles or contact lenses. Recent advances in excimer laser and accumulation of medium term data on results³⁹ (LASIK, PRK, LASEK) have increased the popularity of its use particularly for low/moderate myopia. Irregular astigmatism is unable to be corrected by lenses and the use of excimer laser for this purpose is possible but remains complex.
- 4.4. Surgical options are also available. Suture manipulation or relaxing incisions have been used successfully for astigmatism secondary to trauma.³¹ Corneal grafting for patients with intractable keratoconus and relentless progression of astigmatism has a relatively good prognosis. Clear lens extraction and phakic intraocular lenses (implantable contact lenses) for high myopia have been used in specific cases^{40,41} but have serious potential complications.

5. Summary

- 5.1. Refractive error is said to exist when the eye fails to bring parallel light (distant objects) to focus on the retina. There are three types of refractive error: myopia, hypermetropia and astigmatism.
- 5.2. Refractive errors are common affecting approximately a third of the general population.
- 5.3. There is good evidence to support a genetic aetiology for myopia and hypermetropia. The role of environmental factors such as near work is less certain. Ocular trauma is an important cause of acquired astigmatism that is often difficult to treat.
- 5.4. As an alternative to spectacle and contact lenses, treatment modalities in the form of excimer laser are increasing in popularity.

6. Related Synopses

Myopia.

7. Glossary

accommodation	The ability of the lens to assume a more globular shape in order to increase the focussing power of the lens.
asthenopia	Group of symptoms that describe ocular discomfort due to refractive error e.g. eyestrain, “tired eyes” and blurred vision.
astigmatism	Refractive error that prevents a focussed image on the retina due to the eye refracting light in different meridians unequally.
cataract	Opacification of the lens.
cornea	Clear, dome shaped structure at the front of the eye responsible for focussing light.
diopetre	Unit by which the strength of lenses is measured.
dysplasia	Disorganisation of tissue structure.
emmetropia	The absence of any refractive error. Light brought to a focus directly on to the retina without accommodative effort. Hence <i>emmetropic</i> .
excimer laser	A method of sculpting the corneal curvature using a variety of techniques e.g. LASIK, PRK, LASEK.
hypermetropia (hyperopia)	Long-sightedness. Error of refraction where the image is focussed behind the retina.
keratoconus	Progressive corneal ectasia where the cornea assumes an irregular conical shape.
lens	Bi-convex structure in the eye that focuses light towards the retina.
migraine	Complex syndrome characterised by unilateral headache and visual or other sensory disturbances.

myopia	Short-sightedness. Error of refraction where image is focussed in front of the retina.
osmolality	Balanced water content.
presbyopia	Difficulty bringing near objects into focus due to the normal, steady, age-related decline in accommodative power of the lens.
pterygium	Triangular sheet of fibrovascular tissue that invades the cornea.
retina	Membranous structure covering the posterior aspect of the eye that converts the visual image into neural impulses before transmission to the brain.

8. References

1. Hyams SW, Pokotilo E, Shkurko G. Prevalence of refractive errors in adults over 40: a survey of 8102 eyes. *B J Ophthalmol* 1997;61(6):428-432.
2. The Eye Diseases Prevalence Research Group. The prevalence of refractive errors among adults in the United States, Western Europe, and Australia. *Arch Ophthalmol* 2004;122(4):495-505.
3. Midelfart A, Kinye B, Midelfart S, Lydersen S. Prevalence of refractive errors in young and middle-aged adults in Norway. *Acta Ophthalmol Scandina* 2002;80(5):501-505.
4. International Headache Society. The international classification of headache disorders, 2nd edition. *Cephalalgia* 2004;24 (suppl. 1):1-160.
5. Gil-Gouveia R, Martins IP. Headaches associated with refractive errors: myth or reality? *Headache* 2002;42(4):256-62.
6. Wilmut EB. Migraine. *B J Physiol Opt* 1956;13:93-7.
7. Vincent AJ, Spierings ECH, Messinger HB. A controlled study of visual symptoms and eyestrain factors in chronic headache. *Headache* 1989;29:523-7.
8. Evans BJW, Patel R, Wilkins AJ. Optometric function in visually sensitive migraine before and after treatment with tinted spectacles. *Ophthal Physiol Opt* 2002;22:130-142.
9. Sorsby, A, Sheridan, M, Leary, GA. Refraction and its components in twins London: Her Majesty's Stationery Office Medical Research Council Special Report Series.1962;(303):1-43.
10. Zadnik K, Satariano WA, Mutti DO, Sholtz RI, Adams AJ. The effect of parental history of myopia on children's eye size. *JAMA* 1994;271:1323-7.
11. Young TL, Ronan SM, Drahozal LA. Evidence that a locus for familial high myopia maps to chromosome 18p. *Am J Human Genet* 1998;63:109-19.
12. Young TL, Ronan SM, Alvear AB. A second locus for familial high myopia maps to chromosome 12q. *Am J Human Genet* 1998;63:1419-1424.
13. Guggenheim JA, Hill C, Yam T-F. Myopia, genetics and ambient lighting at night in a UK sample. *Br J Ophthalmol* 2003;87:580-582.
14. Lyhne N, Sjolie AK, Kyvik KO, Green A. The importance of genes and environment for ocular refraction and its determiners: a population based study among 20-45 year old twins. *Br J Ophthalmol* 2001;85:1470-6.
15. Kinye B, Midelfast A, Jacobsen G, et al. The influence of near work on development of myopia among university students. a three year longitudinal study among engineering students in Norway. *Acta Ophthalmol Scand* 2000;78:26-9.

16. Zylbermann R, Landau D, Berson D. The influence of study habits on myopia in jewish teenagers. *J Paediat Ophthalmol Strabismus* 1993;30:319-22.
17. Simensen B, Thorud LO. Adult-onset myopia and occupation. *Acta Ophthalmol Scand* 1994;72:469-72.
18. Raviola E, Wiesel TN. An Animal Model of Myopia. *N Engl J Med* 1985;312:1609-15.
19. Rasooly R, BenEzra D. Congenital and traumatic cataract: the effect on ocular axial length. *Arch Ophthalmol* 1988;106:1066-8.
20. Rechichi C, Scullica L. Trends regarding myopia in video terminal operators. *Acta Ophthalmol Scand* 1996;74(5):493-6.
21. Cole BL, Maddocks JD, Sharpe K. Effects of VDUs on the eyes; report of a 6-year epidemiological study. *Optom Vis Sci* 1996;73(8):512-528.
22. Blehm C, Vishnu S, Khattak A, Mitra S, Yee RW. Computer vision syndrome: a review. *Surv Ophthalmol* 2005;50(3):253-62.
23. Hammond CJ, Snieder H, Gilbert CE, Spector TD. Genes and environment in refractive error: the twin eye study. *Invest Ophthalmol Vis Sci* 2001;42:1232-6.
24. Friberg TR, Grove AS. Choroidal folds and refractive errors associated with orbital tumours. An Analysis. *Arch Ophthalmol* 1983;101(4):598-603.
25. Keller JT, Polse KA. Central serous retinopathy with transitory monocular hypermetropia- a case report. *Am J Optom Arch Am Acad Optom* 1972;49(9):793-6.
26. Santa Cruz CS, Culotta T, Cohen EJ, Rapuano CJ. Chalazion-induced hyperopia as a cause of decreased vision. *Ophthalmic Surg Lasers* 1997;28(8):683-4.
27. Teikari JM, O'Donnell J. Astigmatism in 72 twin pairs. *Cornea* 1989;8(4):263-6.
28. Clementi M, Angi M, Forbosco P, Di Gianantonio E, Tenconi R. Inheritance of astigmatism : evidence for a major autosomal dominant locus. *Am J Hum Gen* 1998;63(3):825-30.
29. Beigi B, Algawi K, Foley-Nolan A, O'Keefe M. Herpes simplex keratitis in children. *B J Ophthalmol* 1994;78(6):458-60.
30. Baykara M, Dogru M, Ozcetin H, Erturk H. Primary repair and intraocular lens implantation after perforating eye injury. *J Cataract Refract Surg* 2002;28(10):1832-5.
31. Jain S, Azar DT, Pineda R. Management of astigmatism after corneal trauma. *Int Ophthalmol Clin* 2002;42(3):47-55.
32. Green WT, Muir MG. Corneal complications of cataract surgery. *Curr Opin Ophthalmol* 1994;5(4):98-104.

33. Hammoudi DS, Segev F, Abdolell M, Rootman D. Outcome of penetrating keratoplasty performed by cornea fellows compared with that of an experienced staff surgeon. *Cornea* 2005;24(4):410-6.
34. Melki SA, Azar DT. LASIK complications: aetiology, management, and prevention. *Surv Ophthalmol* 2001;46(2):95-116.
35. Randleman JB, Hewitt SM, Stulting RD. Refractive changes after posterior segment surgery. *Ophthalmol Clin North Am* 2004;17(4):521-6.
36. Bagheri A, Farahi A, Guyton DL. Astigmatism induced by simultaneous recession of both horizontal rectus muscles. *J AAPOS* 2003;7(1):42-6.
37. Lee KE, Klein BE, Klein R, Wong TY. Changes in refraction over 10 years in an adult population: the Beaver Dam eye study. *Invest Ophthalmol Vis Sci* 2002;43(8):2566-71.
38. Kleinstei RN, Jones LA, Hullett S, Kwon S, Lee RJ, Friedman NE et al: Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error Study Group. Refractive error and ethnicity in children. *Arch Ophthalmol* 2003;121(8):1141-7.
39. Rajan MS, Jaycock P, O'Brart D, Nystrom HH, Marshall J. A long-term study of photorefractive keratectomy, 12 year follow-up. *Ophthalmology* 2004;111(10):1813-24.
40. Fernandez-Vega L, Alfonso JF, Villacampa T. Clear lens extraction for the correction of high myopia. *Ophthalmology* 2003;110(12):2349-54.
41. O'Brien TP, Awwad ST. Phakic intraocular lenses and refractory lensectomy for myopia. *Curr Opin Ophthalmol* 2002;13(4):264-70.

