Third Edition

Ophthalmology

Shyamanga Borooah
Peng Yong Sim
Mark Wright
Baljean Dhillon

Foreword by Professor Andrew Dick

pocket tutor



JP

Contents

Foreword *v*
Preface *vii*
Dedication *xii*
Acknowledgements *xiii*

Chapter 1 First principles
1.1 Development of the eye 1
1.2 Anatomy and physiology of the eye 3
1.3 Optics 18

Chapter 2 Ophthalmology in practice
2.1 History taking 21
2.2 Examination 24
2.3 Investigations 42
2.4 Diagnosis 43
2.5 Treatment 45

Chapter 3 Diagnostic algorithms 55

Chapter 4 Refractive apparatus
4.1 Anatomy and physiology 67
4.2 Clinical scenario 68
4.3 Hypermetropia 70
4.4 Myopia 73
4.5 Astigmatism 75
4.6 Presbyopia 77

Chapter 5 Orbit
5.1 Anatomy 81
5.2 Clinical scenario 82
5.3 Preseptal cellulitis and orbital cellulitis 85
5.4 Thyroid orbitopathy 87

Chapter 6 Eyelid and lacrimal apparatus
6.1 Anatomy and physiology 93
6.2 Clinical scenarios 94
6.3 Entropion 99

6.4 Ectropion 101
6.5 Facial nerve palsy 104
6.6 Ptosis 107
6.7 Blepharitis 110
6.8 Chalazion 112
6.9 Eyelid malignancies 114
6.10 Acquired nasolacrimal duct obstruction 118

Chapter 7 Conjunctiva
7.1 Anatomy and physiology 123
7.2 Clinical scenario 123
7.3 Infective conjunctivitis 126
7.4 Allergic conjunctivitis 130
7.5 Subconjunctival haemorrhage 133

Chapter 8 Cornea and sclera
8.1 Anatomy and histology 137
8.2 Clinical scenario 138
8.3 Dry eye/tear film instability 140
8.4 Infectious keratitis 144
8.5 Scleritis and episcleritis 148

Chapter 9 Diseases of the lens
9.1 Anatomy and physiology 153
9.2 Clinical scenario 153
9.3 Cataracts 156
9.4 Cataract surgery 159

Chapter 10 Uvea
10.1 Anatomy 165
10.2 Clinical scenario 165
10.3 Anterior uveitis (iritis) 167
10.4 Posterior uveitis and intermediate uveitis 171

Chapter 11 Retinal disease
11.1 Anatomy and physiology 177
11.2 Clinical scenarios 178
11.3 Diabetic retinopathy 183
11.4 Hypertensive retinopathy 188
11.5 Retinal vein occlusion 190
11.6 Retinal artery occlusion and amaurosis fugax 193
11.7 Age-related macular degeneration 196

11.8 Retinal tears and detachment 199
11.9 Retinitis pigmentosa 202
11.10 Adult retinal tumours 205

Chapter 12 Optic nerve and visual pathways
12.1 Anatomy and physiology 211
12.2 Clinical scenarios 211
12.3 Angle closure glaucoma 220
12.4 Open angle glaucoma 224
12.5 Secondary glaucomas 231
12.6 Optic neuritis 231
12.7 Optic atrophy 238
12.8 Anterior ischaemic optic neuropathy 241
12.9 Papilloedema 247
12.10 Visual pathway defects 250

Chapter 13 Ocular motility and pupils
13.1 Terminology of ocular motility 255
13.2 Anatomy and physiology 256
13.3 Clinical scenarios 258
13.4 Third (oculomotor) nerve palsy 263
13.5 Fourth (trochlear) nerve palsy 266
13.6 Sixth (abducens) nerve palsy 269
13.7 Internuclear ophthalmoplegia 273
13.8 Non-paralytic strabismus 275
13.9 Nystagmus 277
13.10 Pupil disorders 279

Chapter 14 Paediatric ophthalmology
14.1 Embryology 283
14.2 Clinical scenario 283
14.3 Amblyopia 285
14.4 Retinopathy of prematurity 287
14.5 Retinoblastoma 290

Chapter 15 Trauma
15.1 Clinical scenario 293
15.2 Chemical injury 295
15.3 Corneal foreign body 299
15.4 Penetrating trauma 301
15.5 Blunt orbital trauma 304

Index *309*

chapter 4

Refractive apparatus

Refractive errors are one of the most common causes of blindness in the world. Most are easily treated by spectacle correction. Increasingly, contact lens wear, laser refractive surgery, and refractive lens exchange are used for correction of refractive error.

4.1 Anatomy and physiology

Refraction is a manifestation of the refracting components of the eye (the cornea, aqueous humour, lens, and vitreous humour), their refractive indices and the length of the eye (**Figure 4.1**). Refractive errors occur when the eye is unable to focus incoming light rays onto the retina.

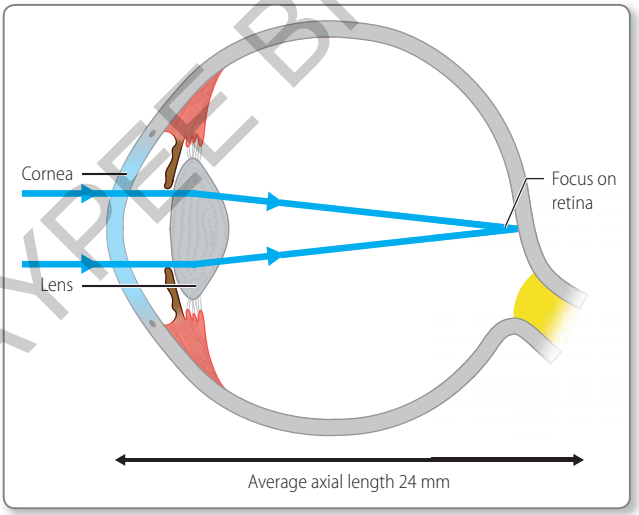

Figure 4.1 Correct focusing of light in the eye. The main refracting components of the eye are the cornea and lens.

JAYPEE BROTHERS

4.2 Clinical scenario

Gradually blurring vision

Presentation

A 55-year-old man presents to the outpatient department of his local hospital complaining of gradually increasing blurred vision.

Diagnostic approach

Clarify whether the patient has true blurring with reduced visual acuity or whether he has double vision.

Diagnostic clues in the history The patient states that the loss of vision is gradual and bilateral. Bilateral blurred vision will either be caused by systemic changes or a single defect affecting the visual pathway beyond the chiasm. Rarely, a disease affecting one eye will also simultaneously affect the other eye (e.g. retinal detachment or retinal vein occlusion).

The gradual onset helps rule out certain conditions such as acute vascular disorders. Next ask when the reduced vision occurs: is it at all the time or only in certain situations? Ask whether the reduced vision affects part or all of the visual field. If only part of the visual field is affected, this points towards a defect in the visual pathway beyond the chiasm (see **Table 4.1**).

Further history

The patient states that his vision is actually reduced with no double vision. This seems to occur only when reading and has recently also been worsening when working at his computer and affects all of his vision. Distance vision is unaffected. He has no other symptoms.

Previous medical history The patient reports that he has been diagnosed with hypertension.

Previous ocular history There is no previous ocular history of note. The patient does not wear glasses and he has not attended an optometrist for some time.

Medications The patient takes bendroflumethiazide for hypertension.

JAYPEE BROTHERS

Cause	Disease
Refractive	Myopia: difficulty with distance vision Hypermetropia: greater difficulty with near vision Astigmatism: distance and near blurry
Corneal	Corneal degeneration: corneal opacity and occasionally recurrent erosions and foreign body sensation
Lens	Cataract: glare Presbyopia: difficulty with near vision but not distance if refraction correct
Retinal	Retinal degeneration (e.g. retinitis pigmentosa): impaired dark adaptation, partial field loss
Post-chiasmal	Tumour: other neurological signs, partial field loss

Table 4.1 Differential diagnoses for gradual bilateral loss of vision

Social history The patient is an accountant and a non-smoker.

Diagnostic approach

The fact that the vision loss covers most of the field of vision reduces the chance of a post-chiasmal disorder (i.e. a defect caused by interruption of pathways after the chiasm). The clue in the history is the intermittent nature of visual blurring. The patient states that the deficit only occurs when viewing near objects. This means that a global reduction in vision (e.g. due to cataracts) is eliminated, leaving an accommodation defect such as presbyopia, a defect in accommodation with age, as a possible cause.

Examination

The results of a general examination were normal. **Table 4.2** shows the ophthalmic examination results.

Diagnostic approach

The examination seems normal except for the disparity between near and distance. The patient states that he has not been to the optician in some time, which means that his refractive correction has not been checked recently. When accommodation reduces with age, this suggests presbyopia.

JAYPEE BROTHERS

Right eye		Left eye
6/6 N24	**Visual acuity** **Unaided distance** **Unaided near**	6/6 N24
Normal direct and consensual response	**Pupil**	Normal direct and consensual response
Clear	**Cornea**	Clear
Clear	**Anterior chamber**	Clear
Clear	**Lens**	Clear
Clear	**Vitreous**	Clear
Clear view of fundus	**Fundus**	Clear view of fundus
Normal	**Disc**	Normal

Table 4.2 Ophthalmic examination: results for a patient with gradually blurring vision

4.3 Hypermetropia

Hypermetropia, or far sightedness, is a refractive error in which parallel light rays entering the eye are focused beyond the retina (**Figure 4.2**). It is denoted by the (+) symbol, meaning that positive dioptric powered lenses (converging lenses) are required to correct the error.

Epidemiology

Hypermetropia is more common in children, partly because they have a shorter eye. At birth, on average a child is +2 dioptres in power. This reduces with time and, as a child's eyes grow longer, they become more emmetropic. Afro-Caribbean populations have an increased prevalence of hypermetropia, whereas East Asian populations have a reduced prevalence.

Clinical features

The clinical features of hypermetropia include:

- Blurry vision

JAYPEE BROTHERS

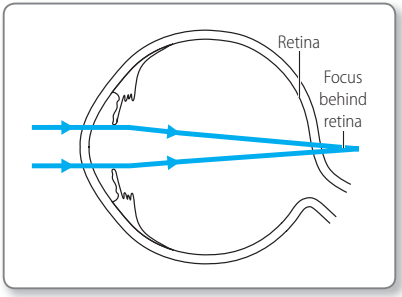

Figure 4.2 Convergence of incoming light behind the retina in hypermetropia.

- Headache, as the eye constantly accommodates to try to maintain retinal focus

Clinically, hypermetropia can be divided into physiological, pathological, and functional causes (**Table 4.3**). **Table 4.4** shows the classification of hypermetropia.

Clinical insight

The average eye axial length in a newborn is 17 mm; it reaches 24 mm in adulthood.

Risks

The risks of hypermetropia include acute angle closure glaucoma as hypermetropes generally have shorter eyes and, therefore, a more occludable drainage angle (see Chapter 12).

Hypermetropia also increases the risk of a convergent squint and, in children, there is a risk of amblyopia, which is developmental suppression of vision (see Chapter 14).

Investigations

In children, retinoscopy with a cycloplegic agent (e.g. cyclopentolate), which blocks accommodation, allows measurement of the refractive power.

Normal retinoscopy and subjective correction can be performed in adults. Review ocular motility to rule out any squint and perform a full eye examination.

Clinical insight

A pinhole corrects visual acuity by up to three lines on the Snellen chart and is useful to estimate full corrected vision if a patient's glasses are not available.

JAYPEE BROTHERS

Type of hypermetropia	Causes	Clinical features
Physiological	Variation of normal Hereditary Flatter cornea Thinner lens Shorter eye	Blurry vision Headache or pain using eyes Squint Amblyopia (Section 14.3)
Pathological	Developmental abnormality of cornea and lens Acquired damage (e.g. trauma)	Generalised systemic abnormalities History of trauma
Functional	Presbyopia (Section 4.6) resulting in lack of accommodation	Older than 40 years Blurry vision and headaches with near work

Table 4.3 Clinical classification of hypermetropia

Description	Power (dioptres)
Low	< +2.25
Moderate	+2.25 to +5.00
High	> +5.00

Table 4.4 Classification of severity of hypermetropia

Differential diagnosis

The differential diagnoses include:

- Other causes of reduced vision (see **Figure 3.2**)
- In adults, rarely, a raised retina (e.g. fluid beneath the macula)

Management

The decision on whether to treat hypermetropia and how it is to be treated depend on a number of factors. These include the patient's age, symptomatology, power of the other eye, whether amblyopia is present, and whether there is an associated squint.

Low hypermetropia does not need to be treated if the patient has no symptoms and there is no other abnormality

JAYPEE BROTHERS

with both eyes. Otherwise, treatment should be given initially with full refractive correction. This takes many forms, including glasses, contact lenses, and corrective laser surgery.

Prognosis

In the majority of children, their eyes become less hypermetropic with age, normalising by the age of 5–10 years. Children with amblyopia or squint are less likely to achieve full binocular vision.

Clinical insight

Refractive correction does not reduce the risk of associated problems (glaucoma, retinal detachment, and macular degeneration).

Adult physiological hypermetropia is easily treated with refractive correction. Pathological hypermetropia has a worse final visual prognosis owing to anatomical disruption to ocular structures.

4.4 Myopia

Myopia, or short sightedness, results when parallel rays of light entering the eye have a focal point short of the retina (**Figure 4.3**). This is caused by a combination of the refractive components and the length of the eye. It is denoted by the (-) symbol, meaning that a divergent or negative lens is required to correct the condition.

Epidemiology

Myopia is one of the most common causes of treatable blindness in the world. The prevalence of myopia varies from region

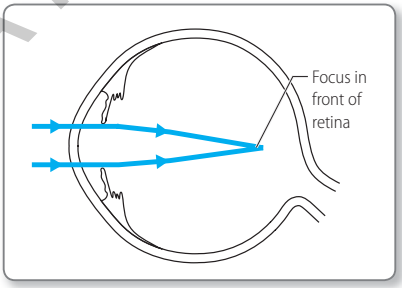

Figure 4.3 Convergence of incoming light to a focal point in front of the retina in myopia.

JAYPEE BROTHERS

to region. In East Asian countries, the prevalence may be as high as 80%; in Western Europe, it is 40%; and in some African countries, it is as low as 10%. Myopia is rare in children, although it is more commonly found in those born prematurely. Prevalence increases with age as the infant's eye grows longer.

Causes

Myopia has a large hereditary component. Several genes have now been identified that cause high myopia. There is increasing evidence that environmental factors (e.g. a higher level of education and increased indoor time) play a part in risk for myopia.

Pathogenesis

During early childhood, the eye gradually becomes more myopic in a process known as emmetropisation. This results from a balance between the eye growing longer and the cornea flattening. In some cases, this process continues beyond emmetropia – normal vision – leading to the development of myopia. In a small minority, the development of myopia progresses throughout life. The exact mechanism is unknown, but genetics are thought to play a large part in setting the range of myopia, with childhood visual activity adjusting the level of final myopia.

Other causes of myopia include pathology such as changes to the refractive components of the eye, which can be congenital or acquired. Congenital causes include genetic or developmental conditions such as Down syndrome. Acquired causes include cataract, which results from increased lens refractive power and, therefore, shortening of the focal length. In addition, trauma to the cornea or lens can result in myopia by alteration to the optical components of the eye.

Clinical features

The main clinical feature of myopia is blurry vision, especially for distance. **Table 4.5** shows the classification of severity of myopia.

JAYPEE BROTHERS

Description	Power (dioptres)
Low	< –3.25
Moderate	–3.25 to –6.00
High	> –6.00

Table 4.5 Classification of severity of myopia

Risks

Patients with myopia have an increased risk of retinal detachment and macular degeneration. Reduced trabecular meshwork aqueous flow and weakness of the optic nerve head in myopia increase the risk of open angle glaucoma (see Chapter 12).

Investigations

Obtain a full history and examination. Assess visual acuity to rule out any sinister causes of myopia, such as trauma, and give special consideration to asymmetric myopia. Asymmetric myopia in a young child may be a sign of congenital glaucoma.

Management

Assess patient factors when deciding to treat myopia. Treatment is not necessary if patients are asymptomatic (e.g. in low myopia) except in amblyopia (see Chapter 14); however, the decision should take account of considerations such as driving and occupation (aviation, military, police, and driving heavy goods vehicles).

Spectacle correction is still the most common form of correction.

Prognosis

The prognosis is generally good for moderate and low myopia. However, less than –8D, the risk of retinal detachment (see Chapter 11) and macular degeneration increases markedly.

4.5 Astigmatism

The focus of incoming light rays into the eye is mainly affected by the cornea and the lens. Most commonly, the cornea is round

JAYPEE BROTHERS

like a football. However, in astigmatism, the cornea is shaped more like an oval rugby ball, and light rays entering the eye at different points of the cornea are focused differently depending on where they enter the cornea (**Figure 4.4**). This results in a blurred retinal image. A cylindrical lens is required to correct astigmatic errors.

Epidemiology

Approximately one-third of people have some form of astigmatism.

Causes

Astigmatism is most commonly a result of irregular corneal curvature. A rare form of abnormal corneal curvature is a condition called keratoconus which results from an ectasia of the cornea. The lens occasionally causes astigmatism as a result of irregularities in its surface. Trauma or surgery can both result in astigmatism by causing irregular shaping of the cornea or misaligning the lens.

Pathogenesis

Astigmatism is mainly hereditary. The exceptions are the acquired causes affecting corneal curvature.

Clinical features

The clinical features of astigmatism include blurred vision and headache.

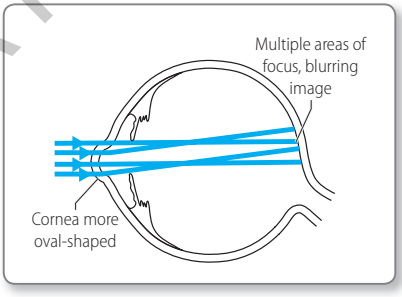

Figure 4.4 Focusing parallel light rays entering the eye in astigmatism.

JAYPEE BROTHERS

Investigations

Perform a full ophthalmic investigation. Assess visual acuity with a pinhole to give an estimate of the final corrected vision if the patient's glasses are not available. The corneal curvature should be examined by keratometry, which is a measure of the refractive power of the cornea, and by corneal topography, which is a measure of the steepness of various areas of the cornea.

Management

The most common form of correction is with cylindrical optical spectacle correction. Soft contact lenses are useful but are costlier because they have to be specially made to enable them to stay in the correct position for corneal curvature. Hard contact lenses alter the shape of the cornea, thus correcting mild astigmatism. Corneal laser refractive surgery can correct mild-to-moderate astigmatism.

Prognosis

The prognosis is generally very good for the correction of astigmatism.

4.6 Presbyopia

Presbyopia is the reduction in accommodative ability with age.

Epidemiology

Loss of accommodation, the change in the eye's focus for near objects, is remarkably consistent throughout all populations and between the sexes. The amount of possible accommodation gradually declines with time, reaching almost zero by the age of 50 years (**Figure 4.5**).

Pathogenesis

Presbyopia is part of the normal ageing of the lens and its capsule, ciliary body, and zonular fibres. With time, the lens and capsule become less elastic and hence less able to become thicker to assist in accommodation. The elastic ability almost halves between the ages of 20 and 40 years. The ciliary body

JAYPEE BROTHERS

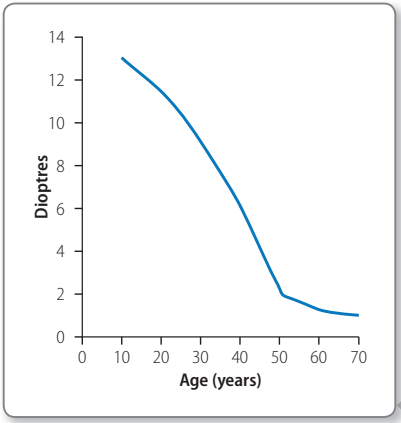

Figure 4.5 The decline in accommodation with age.

has a smaller effect and, in later years, is less able to contract and hence enable accommodation. The change in the zonular fibre attachment on the lens capsule means that accommodation may be more difficult.

Clinical features

The clinical features of presbyopia include:

- Age older than 40 years
- Difficulty with near work
- Tiredness and/or strain especially when reading

Clinical insight

Some myopes may never be affected by presbyopia as reading distance will focus light perfectly on the back of the eye without refractive correction.

Diagnostic criteria

Presbyopia is diagnosed when patients have normal distance vision but reduced near vision. The near vision can be corrected by the addition of convergent (+) lenses.

Investigations

Accommodation is measured objectively by using a ruler. Letters can be placed on the end of the ruler and the ruler

JAYPEE BROTHERS

gradually moved towards the patient. When the patient indicates that the letters are blurred, the distance between the patient and the ruler should be noted. The near-point (or RAF) rule is a device used to measure the near points of accommodation and convergence.

Differential diagnosis

Occasionally, a failure of accommodation mimics presbyopia. This is differentiated by other ocular signs, such as a dilated pupil, as in Adie's pupil (tonic dilated pupil), and third nerve (oculomotor nerve) palsy. A history of sudden onset can also indicate failure of accommodation.

Management

Correct symptoms with convergent (+) lensed spectacles. If there are no other refractive abnormalities, reading spectacles are appropriate. If there is associated myopia or hypermetropia, use bifocal or varifocal lenses. Although there is no treatment to cure or prevent presbyopia, developments in accommodating intraocular lens technology enable recovery of some accommodative ability.

Prognosis

Presbyopia is easily treated with spectacle correction.

JAYPEE BROTHERS

pocket tutor

Third Edition

Ophthalmology

pocket tutor Ophthalmology is the ideal pocket-sized companion for medical students, junior doctors, primary care practitioners and optometrists seeking to improve their practical knowledge of eye disorders.

- ✓ In practice, chapter outlines examination techniques, investigations and management options
- ✓ Updated diagnostic algorithms provide diagnostic pathways for common presentations
- ✓ Clinical scenarios and concise disease descriptions that teach clinical decision making and summarise the disorders seen in practice

New to this edition: addition of higher quality clinical photos and figures, updated chapters with the latest evidence, and new links to narrated videos to help hone clinical examination and diagnostic skills.

pocket tutor is the best-selling series that reinforces knowledge fast: in practice, in tutorials, and before examinations. Reminders of key underlying principles, insightful clinical scenarios, and an accessible format mean you can trust **pocket tutor** to provide you with the right answer, whenever you need it.

JP medical publishers

ISBN 978-1-78779-164-0

www.jpmedpub.com