

Ministry of Defence

Synopses of Causation

Cataract

Author: Dr Mike Coulthard, Medical Author, Medical Text, Edinburgh
Validator: Mr M Wagih Aclimandos, Kings College Hospital, Denmark Hill, London

April 2010

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. A cataract is a clouding that develops in the lens of the eye. It may obstruct the passage of light. Cataracts can affect visual acuity or cause glare depending on their density and location.
- 1.2. In order to better understand the development of cataracts it is necessary to consider the normal physiology of the lens.
- 1.3. The human lens, unlike that of other animals continues to grow throughout life. The rate of growth varies. It is most rapid in embryonic and foetal phases. It develops from the [ectodermal](#) layer in the embryo.¹ Throughout life further layers are laid down. The lens is susceptible to changes in generalised [epithelial](#) tissue disorders (e.g. dermatitis and severe eczema) because of this derivation.
- 1.4. At birth the lens is almost spherical. Lens growth slows down after birth but because of differing rates of growth in different planes it assumes the characteristic “lens” shape.²
- 1.5. The lens is covered with an epithelial tissue (the capsule) inside which is the lens substance.
- 1.6. The lens substance is composed of epithelial fibres which develop in concentric layers. The centre is termed the nucleus of the lens. The rest of the lens is termed the cortex. The cortex adjacent to the capsule is termed subcapsular.
- 1.7. The capsule is in contact with the [aqueous humour](#) anteriorly and the [vitreous humour](#) posteriorly. The lens itself is [avascular](#), any electrolyte or water reach it via the aqueous and vitreous humour through the capsular membrane.
- 1.8. Any upset of the delicate balance of transfer of water or electrolytes can result in cataract formation. The site of the cataract often indicates the causation.
- 1.9. Morphology
 - 1.9.1 Cataracts can be classified according to the part of the lens in which they form.
 - 1.9.2 **Capsular cataracts** may be congenital or acquired. Causes include [pseudo-exfoliation syndrome](#), gold treatment, chlorpromazine, trauma and in association with posterior [synechiae](#) in anterior [uveitis](#).
 - 1.9.3 **Subcapsular cataracts** may be anterior or posterior. Anterior occur in acute angle closure [glaucoma](#), [Wilson’s disease](#) and amiodarone treatment. Posterior may be secondary to [vitrectomy](#), other posterior eye disease, [antimitotic](#) therapy or age related. Other causes include [myotonic dystrophy](#), corticosteroids and irradiation.
 - 1.9.4 **Nuclear cataracts** can be age related or caused by diabetes. Some congenital cataracts, (e.g. maternal rubella or galactosaemia) may look like

nuclear sclerosis but are in effect lamellar (an opaque layer in the region of the nucleus).

- 1.9.5 **Cortical cataracts** are age related or congenital. The congenital forms may not compromise vision if localised (anterior or posterior polar). The congenital forms may be white or deep blue in colour.
- 1.9.6 **Lamellar cataracts** are sandwiched between the clear nucleus and the cortex. They are invariably congenital.
- 1.9.7 **Sutural cataracts** are congenital and usually familial. They are common and have no visual importance.
- 1.9.8 **Mature cataracts** are ones where the lens protein is opaque, whereas **an immature cataract** has some remaining transparency. If the cortical lens proteins are liquefied the cataract is termed **hypermature**.
- 1.9.9 A hypermature cataract with the lens floating freely in the capsular sac is termed a **Morgagnian cataract**.

1.10.1 Accommodation

Accommodation is the process of changing the power of the lens in order to get a clear image at various distances. This is achieved by contracting the Ciliary muscle (a circular muscle which surrounds the lens). Changing the size of this 'ring' muscle alters the thickness and hence the power of the lens.

1.10.2 Presbyopia

The ability to vary the power of the lens diminishes with age. Most people reach the stage where they are no longer able to accommodate sufficiently to read between the age of 45 and 55. This is called Presbyopia.

2. Clinical Features

- 2.1. The main symptom is normally a slowly progressive painless decrease in vision. Cataracts usually develop slowly over a number of years. Rarely they can develop quickly over weeks or months. This is often the case in traumatic cataracts or diabetes induced.
- 2.2. Cataracts may be unilateral or bilateral . Most are bilateral but not necessarily symmetrical.
- 2.3. As they develop vision gradually deteriorates. Not all cataracts progress. Some may be static for quite long periods.
- 2.4. Nuclear cataracts increase the [refractivity](#) of the lens and increase [myopia](#). Patients with [presbyopia](#) may notice some improvement in their near vision as the cataract develops. Posterior subcapsular cataracts affect near vision first.
- 2.5. The peak ages for cataract development are in the first decade of life (congenital cataracts) and over the age of 65 years (age related or “senile” cataracts).
- 2.6. Common early symptoms are difficulties with vision in bright light and at night. Drivers often complain of a problem with glare from the lights of oncoming traffic.
- 2.7. These symptoms should always be investigated by a thorough ophthalmological examination by the general practitioner, optician or ophthalmologist.
- 2.8. Cataracts are the leading cause of blindness in the world.

3. Aetiology

- 3.1. The aetiology of cataract formation is multifactorial.
- 3.2. **Congenital causes**³
 - 3.2.1. Drugs (e.g. corticosteroids) in the first trimester of pregnancy.
 - 3.2.2. Intrauterine infection (e.g. rubella, toxoplasmosis) in the first trimester.
 - 3.2.3. Maternal medical conditions (e.g. type 1 diabetes)
 - 3.2.4. Genetic disorders (e.g. galactosaemia, [galactokinase](#) deficiency, [Fabry disease](#), [Niemann-Pick disease type A](#), [congenital ichthyosis](#), [Rothmund-Thomson syndrome](#), [Werner syndrome](#), [Cockayne's syndrome](#) and [Lowe syndrome](#))
 - 3.2.5. Down's syndrome. Cataract formation is the commonest cause of visual impairment in these children.
 - 3.2.6. 33% of congenital cataracts are inherited and not associated with metabolic or systemic disease.
 - 3.2.7. Cataracts from these causes are invariably diagnosed well before adult life.

Acquired causes

- 3.3. **Ageing.** Age related or senile cataract is the commonest form of cataract. The addition of lens layers throughout life (see section 1.3) eventually creates a hard dehydrated lens nucleus (a nuclear cataract). The lens nucleus also tends to become slightly yellow as it ages. Ageing may also produce changes in the outer lens cortical layers, which become opaque (cortical cataract).⁴ Posterior subcapsular cataracts also develop. Cataracts usually start to develop during the fifth decade and are common from the end of the sixth decade. Women have a significantly higher incidence of nuclear and cortical cataracts than men of corresponding age. There is no significant gender difference for posterior subcapsular cataracts.⁵ It is difficult to accurately separate the effects of lifestyle (section 3.8) from those of ageing.

3.4. Disease

- 3.4.1. **Diabetes** is associated with cortical cataract formation. Diabetic patients both type 1 and type 2 develop age-related cataracts at an earlier age than the general population.⁶ Young type 1 diabetics who are poorly controlled occasionally develop "true" diabetic cataracts. In these cases the poor diabetic control results in gross disturbance of the body's fluid balance. High sugar levels cause osmotic overhydration of the lens. Spoke-like cataracts arising from the periphery, can appear very rapidly but may also resolve completely when the diabetic control is restored.

- 3.4.2. **Wilson's disease** is associated with the development of a characteristic “sunflower” cataract. This is a disc shaped coloured opacity in the pupil zone anteriorly, with petal like spokes that extend towards the periphery. Metallic copper is deposited in the anterior lens capsule. This cataract does not reduce visual acuity.⁷ If the Wilson's disease is treated by liver transplant or penicillamine the cataract may disappear.
- 3.4.3. **Severe atopic eczema/dermatitis** may cause cataract formation.⁸ Most are asymptomatic. The incidence of cataracts in a large group of atopic patients was approximately 10%. Two types are reported, posterior subcapsular and anterior subcapsular. The anterior is more common and can develop during adolescence or early adult life. The administration of corticosteroids for the treatment of the severe atopic disease may be partly responsible for the posterior subcapsular cataracts.
- 3.4.4. **Myotonic dystrophy**. 90% of patients develop cataracts. Most appear after the age of 20 years and progress slowly.⁹
- 3.4.5. **Severe prolonged episodes of diarrhoea** (if severe enough to be life threatening). These have been shown to give a three to fourfold risk of cataract formation.¹⁰ These were reported in patients with pre-existing poor nutrition and chronic diarrhoea problems. Episodes of diarrhoea in otherwise healthy young adults have not been associated with this risk.
- 3.5. **Trauma** is the commonest cause of unilateral cataract in young patients.
- 3.5.1. **Contusion and concussion injuries**. Direct blows to the eye cause the cornea to be pushed inwards. When the force is severe the cornea can be forced against the lens and iris. The lens may strike the [retina](#) and [choroid](#). Following the blow the lens then may be thrust forwards by the concussion wave rebounding from the back of the eye. This can cause severe damage. The cataracts form as a result of direct mechanical injury to the lens or due to rupture of the capsule allowing leakage of the aqueous or vitreous humour into the lens. Posterior rupture is the more common. Fluid entry occurs rapidly and a mature cataract forms. These cataracts are known as **concussion cataracts**. If the leak of aqueous or vitreous humour continues it results in complete opacity of the lens.
- 3.5.2. When the force of the blow is very mild, capsular damage is minor and leads to discrete small opacities which remain static.
- 3.5.3. When the force of the blow is moderate it results in a rosette shaped cataract which appears quite quickly (in 1 or 2 days).¹¹ It can appear in the anterior or posterior cortex or both.¹² A [Vossius ring](#) may also appear.
- 3.5.4. Occasionally, after moderate blows, a smaller rosette shaped cataract may develop in the posterior cortex up to 2 years after the injury.
- 3.5.5. **Penetrating eye injuries**. If the lens is ruptured by a penetrating object, the capsule may heal if the rupture is small. If the rupture is of significant size, the lens is overwhelmed by the entry of fluid and progresses rapidly (in 1 or 2 days) to a mature cataract.¹¹

- 3.5.6. During [vitrectomy](#) procedures even if the posterior capsule is not injured directly a transient posterior subcapsular lens opacity may form due to overhydration of a group of lens fibres. These opacities resolve completely. A permanent cataract often develops within 3-5 years following vitrectomy.
- 3.5.7. **Blows to the head.** These cause a similar effect to the less severe form of concussion cataract without capsular damage as described in section 3.5.3 above (a small rosette shaped cataract). A Vossius ring may also form on the anterior capsule.

3.6. Radiation (ionising and non-ionising)

- 3.6.1. **Ionising Radiation.** All electromagnetic radiation can result in a radiation cataract of a typical appearance. Radiation cataracts vary only in the energy required to cause them and the time they take to develop. Cataracts may develop after a single acute exposure to gamma-radiation in a dose of over 4000 millisieverts. The time taken for these to develop varies from 6 months to 50 years.¹³ Most of the forms of radiation cause ionisation of water and generation of free radicals which are powerful reducing agents and damage cell DNA. Such changes inhibit [mitosis](#) and affect cells with a high metabolic rate such as the lens equatorial fibres. This results in the formation of posterior subcapsular lens opacities. Recent studies suggest that the accumulated dose of radiation from repeated chronic exposure which causes damage is substantially lower than previously thought.¹⁴ The dose of radiation that was thought to be safe was 2 Gy (that is 2000 millisieverts in respect of gamma-rays and X-rays) but these studies revealed cataract formation in technicians at half of this dose. N.B. A gray (Gy) is the international unit of absorbed radiation dose (1 Gy = 1 joule/kg) and the sievert (Sv) is the international unit of equivalent radiation dose, which takes account of the circumstance that different types and energies of radiation will produce different amounts of biological damage.
- 3.6.2. **Heat.** Non-ionising radiation such as infrared causes temperature to rise in the iris pigment epithelium. After long periods of exposure, this results in the formation of posterior subcapsular cataracts and exfoliation of the anterior lens capsule.¹⁵ These were seen in glass blowers and furnace workers before the introduction of protective goggles. This type of cataract is recognised as a prescribed industrial disease in occupations involving frequent (5 or 6 days per week) and prolonged (over 5 years) exposure to radiation from red-hot or white-hot material without suitable eye protection.
- 3.6.3. **Radiofrequency waves (microwaves and radar).** The lens can be damaged by radiofrequency waves, due partly to the heating effect and partly to the direct effects of the radiation. Adherence to the National Radiological Protection Board (NRPB) guidelines will remove any risk of cataractogenesis.^{13,16}
- 3.6.4. **Lasers** will cause cataract formation but only with direct exposure of the eye to the beams. Patients receiving repeated laser treatment to the retina are at risk. Patients receiving [photorefractive keratectomy](#) (PRK) also have

a significant risk of cataract formation but this may be because of prolonged postoperative application of corticosteroid eye drops. Operators of laser equipment are not at risk as long as equipment is properly maintained and tested.

- 3.6.5. **Ultraviolet light exposure.** UV light is classified by its wavelength. UVA is 315 – 400nm (nanometres), UVB is 280 -315 nm and UVC 200 – 280 nm. All three damage tissues and are present in sunlight. UVC does not reach the earth's surface. UVB is present in sun and electric arc light. It is absorbed by the lens and can cause damage. UVA, which is the predominant component of the light in sunbeds, is almost all transmitted by the lens and causes little damage.
- 3.6.6. Cataracts are more common in tropical countries but this observation may be due to other causes (e.g. nutrition or dehydration).
- 3.6.7. Cataracts form possibly due to free-radical formation. The role of UV light in human cataractogenesis is now generally accepted.¹⁷ Studies have revealed that men are more susceptible than women. The risks associated with UV exposure appear to be limited to the formation of cortical cataracts, which take many years of exposure to develop. For example, a study of 838 watermen in North America, who were exposed to high intensities of direct and reflected UVB over many years (between 37 and 50 years of continuous exposure), showed a significant increase in formation of cortical cataracts. A number of other studies on smaller numbers of persons exposed to high levels of UVB have similarly reported an increased risk.

Hats with brims and sunglasses protect against this risk.¹⁸ There is no convincing evidence of a link between UV light exposure and the development of either nuclear or subcapsular cataracts.¹⁹

3.7. Other physical agents

- 3.7.1. **Electric shock.** This can occur as a result of an industrial or domestic accident or lightning. Cataracts may form with voltages as low as 220 volts. The current travels along the path of least resistance from the head, through the eyes along the optic nerve into the brain, to the nerves and finally to the ground. If the site of the shock is equidistant from both eyes then cataracts develop in both eyes. If the shock is to one side the cataract develops on that side.
- 3.7.2. Following the shock, ring-shaped vacuoles appear in the midperiphery of the lens. These coalesce and gradually disappear leaving white dust like opacities in the epithelial region. Later, greyish white streaks appear along the lens fibres and radiate towards the midperiphery. Finally these form an anterior subcapsular opacity.²⁰
- 3.7.3. **Ultrasound.** Ultrasonic vibration can induce cataract formation by producing heat and by the vibration of the tissue interfaces. The damage is dependent on power levels and the frequency. No ill effects have been associated with the power levels used in medical practice. Domestic and

industrial use is also safe as long as the equipment is properly maintained and tested. Ultrasonic equipment is used in cleaning, welding, machining and material testing equipment.

3.8. **Lifestyle factors** leading to increased risk of cataract development are:

- 3.8.1. **Smoking** is associated with nuclear cataract formation. The mechanism is thought to be due to carbamylation (a chemical reaction) of lens proteins by the cyanide in cigarette smoke.²¹
- 3.8.2. **Nutrition.** Poor nutrition is associated with a low antioxidant status. This increases the risk of free-radical damage which leads to cataract formation.
- 3.8.3. **Alcohol.** High alcohol intake is associated with an increased risk of cataract development at an earlier age.²² It is associated with the development of nuclear, cortical and subcapsular cataracts.

3.9. **Iatrogenic.** Numerous drugs are associated with cataract development.

- 3.9.1. **Corticosteroids** either systemic or topical are associated with development of posterior subcapsular cataracts which are discoid and slowly increase in size.²³ Their borders are sharp but have a blue haze. The precise relationship between dose, duration of therapy and development of cataract is unclear. Children appear to be more susceptible than adults. Systemic administration of a dose of 10 mg or less per day for up to 1 year is thought to be safe but there appears to be a genetic susceptibility in children which has also to be taken into consideration. In adults the dose of 10 mg per day or less for up to 1 year is not associated with increased cataract development. If a higher dose is needed a “pulsed” regime or intermittent reducing dose regime is advised. In adults the genetic susceptibility is not so clear due to the problem of eliminating other factors which contribute to cataract formation (e.g. age, nutrition).
- 3.9.2. **Miotics** (e.g. pilocarpine) used in the treatment of open angle [glaucoma](#) cause anterior subcapsular cataracts. The mechanism is possibly due to altered ion transport in the lens.
- 3.9.3. **Antimalarial drugs.** Mepacrine and chloroquine but not hydroxchloroquine may cause a white flaky posterior subcapsular lens opacity. This is associated with use for over 1 year. Long term users should have annual eye checks.²⁴
- 3.9.4. **Phenothiazines** (e.g. chlorpromazine) may cause the development of anterior polar cataracts. A daily dose of 300mg appears to be the minimum required to produce changes. Chlorpromazine is a photosensitising agent and produces free radicals which may be the mechanism of the lens damage.
- 3.9.5. **Antimitotic drugs** such as busulphan may cause posterior subcapsular cataracts.
- 3.9.6. **Gold therapy** in rheumatoid arthritis may lead to anterior capsule cataracts

due to the deposition of gold in the anterior capsule. 50% of patients being treated for over 3 years develop these changes.

- 3.9.7. **Amiodarone** is now commonly in use in cardiac disease for the treatment of severe cardiac rhythm disorders. It causes anterior subcapsular opacities which do not usually compromise vision.²⁵
- 3.9.8. Other less commonly used drugs which are cataractogenic are thallium, dinitrophenol, and eye drops with mercurials as preservatives.

3.10. Secondary to ocular disease

- 3.10.1. **Uveitis** (iridocyclitis) is the commonest cause of secondary cataract formation. The inflammation commonly results in the formation of posterior **synechiae** and subcapsular or posterior cortical lens opacities. These opacities may however be due to treatment of the disease with corticosteroids.
- 3.10.2. **Scleritis** is also associated with cataract formation. This again may be related to treatment of the disease with corticosteroids.
- 3.10.3. **Glaucoma**. Open angle glaucoma in itself is not associated with cataract formation. The use of **miotics** or other antiglaucoma drugs may result in cataract formation. See section 3.9.2.
- 3.10.4. **Retinitis pigmentosa**. All retinal pigment degenerations are associated with cataract formation, usually posterior subcapsular. They occur earlier in the recessive and **X – linked** forms of the disease than in the dominant form. The mechanism is unclear. It is likely that it is due to toxic products released by the degeneration of the retina or possibly due to the absence of a factor synthesised by the retina which is necessary for normal lens growth.
- 3.10.5. **Gyrate atrophy** causes subcapsular cataracts.
- 3.10.6. **Pseudo-exfoliation syndrome** causes anterior capsular and nuclear cataracts.
- 3.10.7. **Degenerative myopia** is associated with various forms of cataract i.e. posterior cortical, subcapsular and nuclear.
- 3.10.8. **Retinal detachment and retinal surgery** frequently result in a posterior subcapsular cataract especially if silicone injection or gas tamponade are used.
- 3.10.9. **Vitreoretinal surgery** may cause an anterior subcapsular cataract. A transient subcapsular cataract occasionally occurs following vitrectomy as described in section 3.5.7.
- 3.10.10. **Tumours of the ciliary body** are sometimes associated with cortical or lamellar cataracts in the affected quadrant.

- 3.10.11. **Infective cataracts.** In children and adults herpes zoster, herpes simplex and toxoplasma can cause cataract formation indirectly as a result of [uveitis](#). Sometimes however infective agents (bacterial, fungal or viral) may attack the lens directly and cause abscess formation and cataracts (e.g. following surgery).
- 3.10.12. **Infective agents** can cause congenital cataracts. In maternal rubella the virus cannot cross the lens capsule until the infant is about 6 weeks of age. Lens opacities which may be uni- or bilateral are not usually present at birth. They appear several weeks or months later. The cataracts are usually nuclear and have a dense pearly appearance.²⁶

4. Prognosis

- 4.1 A small number of cataracts have no effect on vision or are transient and unimportant. Most cataracts however steadily worsen and are only treatable by surgery.
- 4.2 Surgery is offered at a stage where the visual acuity has deteriorated to a level that is not acceptable to the patient. Surgery is 95% effective in restoring vision to a level acceptable to the patient. The lens is usually removed and replaced with an acrylic lens implant. If not the eye is rendered [aphakic](#) and powerful spectacle lenses have to be worn to correct vision.
- 4.3 The 5% of cases that do not achieve a satisfactory result are due either to other ocular problems (usually retinal changes) present at the time of surgery or rarely to complications of the surgery itself.
- 4.4 The presence of retinal changes may not have been apparent prior to surgery due to difficulty viewing the retina through an opaque lens.
- 4.5 Removing the lens before Presbyopia will result in the loss of accommodation. The insertion of intraocular lens implant provides clear vision at a particular distance but does not restore the ability to accommodate. Additional glasses are therefore necessary unless the patient relies on one eye for reading and the other for distance.

The so-called “accommodating” implants are not truly accommodating. They are a type of multifocal lenses which are not yet widely accepted. Their main disadvantage is that the claimed benefit is on the expense of overall clarity of vision.

- 4.6 Infection is a possible complication of any eye surgery. The incidence of endophthalmitis (sight-threatening inflammation of the internal ocular spaces and adjacent structures) following cataract surgery varies between 1 in 400 and 1 in 1700 cases. The variation may be due to operative technique. The routine use of prophylactic antibiotic ophthalmic preparations following cataract surgery reduces the risk of infection significantly.

5. Summary

- 5.1. A cataract is an opacity in the lens of the eye or its capsule. Most cataracts compromise vision. A multitude of factors causes cataract formation. The common causes are ageing, trauma, coexisting medical conditions, or resulting from another eye disease.
- 5.2. By far the most common type of cataract in adult life is caused by degenerative changes in the lens fibres. These changes are due to the ageing process with genetic factors, lifestyle and systemic disease playing an important role.
- 5.3. Most cataracts eventually compromise vision and require surgery.
- 5.4. Surgery is 95% successful with modern techniques and prophylactic antibiotic use.

6. Related synopses

Eye Infections

Eye Injuries

Glaucoma

7. Glossary

antimitotic	An agent that inhibits rapidly dividing cells.
aphakic	Without a lens.
aqueous humour	A transparent fluid occupying the space between the lens and the cornea of the eye.
avascular	Having few or no blood vessels.
choroid	A vascular membrane which contains large branched pigment cells that lies between the retina and the sclera (tough white outer coat of the eyeball).
ciliary body	An annular structure on the inner surface of the anterior wall of the eye composed largely of the ciliary muscle attached to the lens, responsible for altering the shape and focal length of the lens.
Cockayne syndrome	A syndrome of dwarfism, deafness and poor vision due to cataracts, retinal degeneration and optic atrophy.
congenital ichthyosis	An inherited recessive disorder with changes which cause thickening in skin and nails, atrophic sweat glands and nuclear lens opacities.
ectodermal	Arising from the external primitive germ layer of the embryo.
epithelial	Relating to the epithelium - a membranous cellular tissue that covers a free surface or lines a tube or cavity of the body.
Fabry disease	A disorder of lipid metabolism that is inherited as an X-linked recessive trait (q.v.). Characterised by skin lesions on the lower trunk, severe pain in the extremities, corneal opacities, cataracts, and vascular disease of the kidneys, heart or brain.
galactokinase	An enzyme responsible for breaking down galactose.
glaucoma	A disease of the eye usually associated with a raised pressure within the eyeball.
gyrate atrophy	An inherited disorder that results in night blindness, myopia and peripheral atrophy of the choroid and retina.
iridectomy	Surgical removal of part of the iris of the eye.

Lowe's syndrome	A rare hereditary disease which is inherited as an X-linked recessive trait (q.v.). Characterised by cataracts, glaucoma, nystagmus (involuntary rhythmical movements of the eyes), severe mental retardation and generalised poor muscle tone.
miotic	A drug which causes constriction of the pupil of the eye.
mitosis	The process of cell division.
myopia	Nearsightedness i.e. where the lens focuses the images in front of the retina instead of on it.
myotonic dystrophy	A dominantly inherited disease characterised by muscle wasting and tonic relaxation of skeletal muscles.
Niemann-Pick disease type A	An inherited lipid storage disease due to a deficiency of sphingomyelinase (an enzyme which acts on the lipid present in nerve and muscle tissue).
photorefractive keratectomy (PRK)	Removal of some of the corneal surface with a laser to correct myopia.
presbyopia	A condition in middle and older age where the lens loses its elasticity and is unable to focus near objects on the retina.
pseudo-exfoliation syndrome	Secretion of fibrogranular material throughout the anterior chamber of the eye.
refractivity	The ability of the lens substance to bend light in order to focus the image on the retina.
retina	The sensory membrane which lines most of the large posterior chamber of the eye. It contains the nerve endings which pick up the image focussed on it by the lens and transmit the nerve impulses via the optic nerve to the brain.
retinitis pigmentosa	A group of diseases which feature a progressive degeneration of the retina.
Rothmund-Thomson syndrome	An inherited recessive disorder with skin changes, poor sexual development, saddle shaped nose, abnormal hair growth and cataracts which develop at an early age (from the age of 4 years in some cases) and progress rapidly. Associated with premature senility, diabetes and arrested growth.
scleritis	An inflammation of the sclera (tough white outer coat of the eyeball).

synechia	Adhesion of part of the iris to either the cornea (anterior synechia) or the lens (posterior synechia).
uveitis (iridocyclitis)	Inflammation of the uvea – the middle layer of the eye (consisting of the iris, ciliary body and the choroid).
vitrectomy	Surgical removal of all or part of the vitreous body.
vitreous humour	The jelly like fluid which fills the posterior chamber of the eye.
Vossius ring	An imprinting of the iris pigment when the iris is forced back against the lens during trauma.
Werner syndrome	An inherited recessive disorder causing premature senility, diabetes, poor sexual development and arrested growth.
Wilson's disease	An hereditary recessive disorder of copper metabolism where copper is deposited in the liver and the eye.
X-linked	Genetic diseases where the causative gene is carried on the X (male) chromosome.

8. References

- ¹ Deepak K, Hall AB, Rosenthal AR. Pathophysiology and epidemiology of cataract. In: Yanoff M, Duker JS, Augsburger JJ et al. editors. *Ophthalmology*, 2nd ed. Philadelphia, PA: Mosby; 2004. p.275-9.
- ² Forbes JE, Holden R, Harris M et al. Growth of the human crystalline lens in childhood. *Proceedings of Xth ISER Meeting Stresa, Italy. Exp Eye Res* 1992;55:172.
- ³ Krishnamurthy R, Vanderveen DK. Infantile cataracts. *Int Ophthalmol Clin* 2008 Spring;48(2):175-92.
- ⁴ West SK, Valmadrid CT. Epidemiology of risk factors for age related cataracts. *Surv Ophthalmol* 1995 Jan-Feb;39(4):323-34.
- ⁵ Kanthan GL, Wang JJ, Rochtchina E et al. Ten-year incidence of age-related cataract and cataract surgery in an older Australian population. The Blue Mountains Eye Study. *Ophthalmology* 2008 May;115(5):808-14.
- ⁶ Tumosa N. Eye disease in the older diabetic. *Clin Geriat Med* 2008 Aug;24(3):515-27.
- ⁷ Walshe JM. The eye in Wilson's disease. *Birth Defects Orig Artic Ser* 1976;12(3):187-94.
- ⁸ Roth HL, Kierland RR. The natural history of atopic dermatitis. A 20-year follow-up study. *Arch Dermatol* 1964 Feb;89:209-14.
- ⁹ Burian HM, Burns CA. Ocular changes in myotonic dystrophy. *Am J Ophthalmol* 1967 Jan;63(1):22-34.
- ¹⁰ Harding JJ. *Cataract: biochemistry, epidemiology and pharmacology*. London. Chapman and Hall: 1991.
- ¹¹ Grewal DS, Jain R, Brar GS, Grewal SP. Posterior capsule rupture following closed globe injury: Scheimpflug imaging, pathogenesis and management. *Eur J Ophthalmol* 2008 May;18(3):453-5.
- ¹² Bord SP, Linden J. Trauma to the globe and orbit. *Emerg Med Clin North Am* 2008 Feb;26(1):97-123.
- ¹³ Okladnikova ND, Sumina MV, Pestemikova VS et al. Long-term consequences of external gamma-radiation according to the results of the observation of the personnel of the first atomic power plant in the country. *Klin Med (Mosk)* Jan 2007;85(10):21-6.
- ¹⁴ Chodick G, Bekiroglu N, Hauptmann M et al. Risk of cataract after exposure to low doses of ionizing radiation: a 20-year prospective cohort study among US radiologic technologists. *Am J Epidemiol* 2008 Sep;168 (6):620-31.
- ¹⁵ Lipman RM, Tripathi BJ, Tripathi RC. Cataracts induced by microwave and ionizing radiation. *Surv Ophthalmol* 1988 Nov-Dec;33(3):200-10.

-
- ¹⁶National Radiological Protection Board. Report of an advisory group on non-ionizing radiation. Documents of the NRPB; Vol 3, No 1. London: HMSO; 1992.
- ¹⁷ Hightower KR. A review of the evidence that ultraviolet irradiation is a risk factor in cataractogenesis. *Doc Ophthalmol* 1994-95;88(3-4):205-20.
- ¹⁸ Taylor HR, West SK, Rosenthal FS et al. Effect of ultraviolet light on cataract formation. *N Engl J Med* 1988 Dec;319(22):1429-33.
- ¹⁹ Gallagher RP, Lee TK. Adverse effects of ultraviolet radiation: a brief review. *Prog Biophys Mol Biol* 2006 Sep;92(1):119-31.
- ²⁰ Lee MS, Gunton KB, Fisher D et al. Ocular manifestations of remote lightning strike. *Retina* 2002;22(6) 808-10.
- ²¹ Hiller R, Sperduto RD, Podgor MJ et al. Cigarette smoking and the risk of development of lens opacities. The Framingham studies. *Arch Ophthalmol* 1997 Sep;115(9):1113-8.
- ²² Sarma U, Brunner E, Evans J, Wormald R. Nutrition and the epidemiology of cataract and age-related maculopathy. *Eur J Clin Nutr* 1994;48:1-8.
- ²³ Abraham AG, Condon NG, Gower EW. The new epidemiology of cataract. *Ophthalmol Clin North Am* 2006 Dec;19(4):415-25.
- ²⁴ Cumming RG, Mitchell P. Medications and cataract. The Blue Mountains Eye Study. *Ophthalmology* 1998 Sep;105(9):1751-8.
- ²⁵ Flach AJ, Dolan BJ, Sudduth B et al. Amiodarone induced lens opacities. *Arch Ophthalmol* 1983;101:1554-6.
- ²⁶ Sharan S, Sharma S, Billson FA. Congenital rubella cataract: a timely reminder in the new millennium? *Clin Experiment Ophthalmol* 2006 Jan;34(1):83-4.