

Lens disorders in dogs and cats

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Conditions affecting the lens are common in dogs and can also occur in cats. The aim of this article is to understand the functional anatomy of the lens, review the types of lens problems that we see in daily practice, outline diagnostic techniques, and give treatment recommendations.

EMBRYOLOGY, ANATOMY AND PHYSIOLOGY OF THE LENS

The lens is formed in the developing embryo from surface ectoderm, as a section called the lens placode invaginates into the developing optic vesicle. Lens fibres start to continually grow from the equator, extending both anteriorly and posteriorly, and these fibres meet in Y-shaped suture lines (often these are faintly visible). The anterior suture line is in the shape of a Y, while the posterior suture line appears as an inverted Y. These lens fibres continue to be produced throughout life, and are deposited exterior to previous layers, which has been likened to the layers of an onion. Thus, the lens is continuously growing throughout life, with new exterior layers compressing the older central nucleus.

The lens is a transparent, avascular, biconvex disc. It has a central nucleus, which is surrounded by the cortex and contained within an acellular capsule. It has a single cell type, the lens epithelial cell. It is held in position by the support of zonules, which extend from the equator of the lens to the ciliary body. It is positioned in the patellar fossa, on the anterior face of the vitreous. In the dog, it is approximately 7mm thick, 10mm in equatorial diameter, and has a volume of 0.5ml.

The main function of the lens is to refract light, focusing images onto the retina for sharp vision. It is also involved in accommodation, the process by which the eye can change its optical power by altering shape and position, in order to maintain focus on the retina as distance varies. Dogs and cats have a poor accommodative ability compared with people and birds, however.

The lens is the tissue with the highest protein content in the body – it is 65% water and 35% protein. The post-natal lens is devoid of a blood supply, and depends on the surrounding aqueous and vitreous humours to supply oxygen, glucose and nutrients, and to remove waste products. Glucose is metabolised to produce energy using several different metabolic pathways, but mainly through anaerobic glycolysis in the Embden-Meyerhof pathway. Glutathione is a very important anti-oxidant in the lens. The transparency of the lens is dependant on several factors. These include the fact that it is avascular, devoid of pigment and dehydrated (65% water). It has low cellularity, with low cytoplasm density (lack of cell organelles/nuclei). The mainly soluble protein fibres are arranged in a highly organised lattice pattern. Loss of

transparency results in cataract formation. This can arise through several processes, including oxidative damage, proteinase activity, high glucose levels of diabetes mellitus (in dogs), and either high or low levels of calcium.

EXAMINATION OF THE LENS

Clinical examination of the lens requires dilation of the pupil. The pupillary light response (PLR) is checked prior to dilation. One drop of a short-acting mydriatic tropicamide is applied. It takes 20 minutes for the pupils to dilate adequately. However, cases with lens instability or with glaucoma should not have their pupils dilated.

Vision is assessed with the menace response – it is absent with mature cataracts. Visual tracking and maze testing might also be utilised. The PLR is assessed and is expected to be normal. The dazzle reflex is expected to be normal, if retinal function is normal. The cataract is assessed for position, extent and density, ideally with slit-lamp biomicroscopy. Signs of uveitis might be observed when there is a mature cataract, as a low level of lens-induced uveitis is very common. This may be appreciated though signs of conjunctival hyperemia, slight miosis and a darkened iris.

Distant direct ophthalmoscopy (Figure 1) is a useful technique that will highlight any differences in pupil size, diagnose the presence of a cataract, help to distinguish a cataract from nuclear sclerosis, and see aphakic crescent if it is present. A cataract is opaque and it is not possible to see through it, so it is seen as a dark shape over the tapetal reflection. In contrast, with nuclear sclerosis, there is a normal tapetal reflection throughout the pupil.



Figure 1: Technique of distant direct ophthalmoscopy.

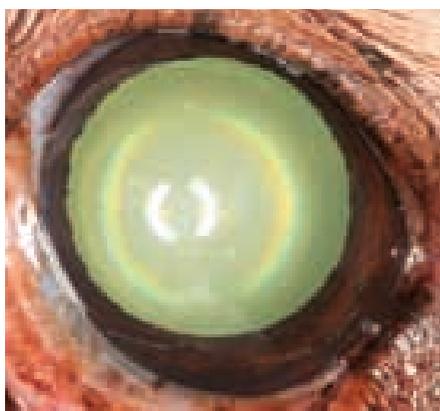


Figure 2: Nuclear sclerosis. The lens nucleus is clearly defined as a ring, but the tapetal reflection can be viewed through the entire lens.



Figure 3: Cataract and lens sub-luxation - a bright crescent-shaped yellow tapetal reflection is visible, representing an aphakic crescent.



Figure 4: Coloboma of the lens from one to eight o'clock - the lens equator is missing and ciliary processes are visible. There are multiple ocular defects, with a developing cataract.



Figure 5: There are multiple spots of pigment on the central anterior lens capsule. There is also a developing mature cataract.

However, there is an obvious thin concentric ring outlining the lens nucleus (Figure 2). An aphakic crescent occurs when the lens slips out of position and there is a crescent shape of brighter tapetal reflection between the edge of the lens and the border of the pupil (Figure 3).

The fundus is examined when it is visible (for example, with immature cataracts). Signs of retinal degeneration will be present if there is Progressive Retinal Atrophy, with tapetal hyper-reflectivity and blood vessel attenuation. Retinal detachment can occur as a result of intraocular pathology such as cataract, uveitis or glaucoma.

The fellow eye is always examined to assess for earlier signs of lens opacity. In the case of lens luxation, signs of iridodonesis, phacodonesis, aphakic crescent and liquified vitreous at the pupil margins are looked for.

The intraocular pressure should be measured, when tonometry equipment is available (and ideally prior to pupil dilation). It is expected to be low with cataracts (below 15mmHg) because of the mild lens-induced uveitis. However, cataracts can lead to intraocular changes that predispose towards glaucoma. In the case of acute lens luxation, a raised intraocular pressure (over 25mmHg) requires prompt intervention. Primary lens luxation often leads to high intraocular pressure due to physical disruption of aqueous flow, secondary uveitis and vitreous clogging the drainage angle. Gonioscopy of the fellow eye might, if there is doubt as to whether the lens luxation is a primary or secondary problem - primary glaucoma with goniodysgenesis can lead to secondary lens luxation. B-mode ultrasonography is useful, especially when the lens is opaque and the fundus cannot be observed. It can

pick up lens capsule rupture, vitreal degeneration, retinal detachment and hyaloid vascular remnants.

Electroretinography is very useful in cases where the cataracts might be secondary to retinal degeneration, e.g. Progressive Retinal Atrophy. Cataract surgery is not performed when there is poor or no retinal function. Laboratory tests are indicated, especially blood glucose measurement and urinalysis to assess for diabetes.

CONGENITAL ANOMALIES OF THE LENS

Congenital lens anomalies are not very common. Because of the timing of lens development at an important stage of embryogenesis of the eye, it is not uncommon for congenital lens anomalies to co-exist with other ocular abnormalities, such as microphthalmia. These defects may be caused by hereditary factors, or else by exogenous factors such as viral or toxic intra-uterine insults.

Aphakia: Absence of the lens - this is very uncommon and would only occur in a very malformed eye such as a cystic eye.

Microphakia: Small lens, usually associated with multiple ocular defects.

Spherophakia: Spherical shape to the lens.

Coloboma: Notching of the lens equator, where a region of lens has not been formed (Figure 4).

Lenticonus: A conical protrusion of the lens capsule at either the anterior or posterior pole.

Embryonic vascular abnormalities: Persistent hyperplastic primary vitreous / persistent hyperplastic tunica vasculosa lentis (PHPV / PHTVL) are remnants of the foetal vascular system which can present as multiple pigmented spots



Figure 6: Central nuclear cataract. There are three small uveal cysts at the iris pupillary margin medially.



Figure 7: A circular refractile opacity is seen within the lens, and this nuclear sclerosis gives the appearance of an opacity.

on the anterior lens capsule (Figure 5), or in more severe cases can lead to cataracts and vitreal abnormalities. PHTVL/PHPV are known to be inherited in the Doberman and Staffordshire Bull Terrier.

Cataract: inherited in the Miniature Schnauzer but possible in any breed, these are essentially nuclear in position and typically do not progress (Figure 6).

ACQUIRED ANOMALIES OF THE LENS

Nuclear Sclerosis

Nuclear sclerosis is a normal aging process of the lens that occurs in all dogs aged seven years and older. Over time, the older lens fibres in the nucleus are compressed by the continual enlarging of the lens as new lens fibres are laid down externally. The hardened nucleus alters the optical properties and causes light scattering, and this is visible as a grey/white milky appearance which the owner observes with diffuse illumination such as room lighting (Figure 7). On examination, there is a clear view of the tapetal reflection by retro-illumination through nuclear sclerosis, but a cataract blocks the passage of light (Figure 8). Thus, the two conditions can be differentiated most easily by distant direct ophthalmoscopy in a darkened room. Nuclear sclerosis is not thought to significantly impact on vision in dogs and cats, and therefore no treatment is necessary.

TEMPORARY DEVELOPMENTAL CATARACTS

Opacities at the extremities of the Y suture lines can sometimes be seen in young puppies (Figure 9). These are temporary cataracts and they usually disappear by 12 weeks of age. They do not require treatment.



Figure 8: Bilateral nuclear sclerosis: a ring is visible within the lens, but the orange/yellow tapetal reflection can be seen through it. There are also two small dark spots - these represent focal posterior polar cataracts and their presence blocks the tapetal reflection behind them.



Figure 9: Three white lens opacities can be seen at the twelve, five and nine o'clock positions. These are temporary suture tip opacities in a ten-week-old dog.



Figure 10: Y-shaped anterior suture line cataract.



Figure 11: Floriform cataract associated with the posterior sutures.



Figure 12: Immature cataract - the fundus is still visible.



Figure 15: Nuclear sclerosis ring, posterior polar cataract, peripheral vacuoles which imply progression of the cataract - this cataract is secondary to inherited Progressive Retinal Atrophy.

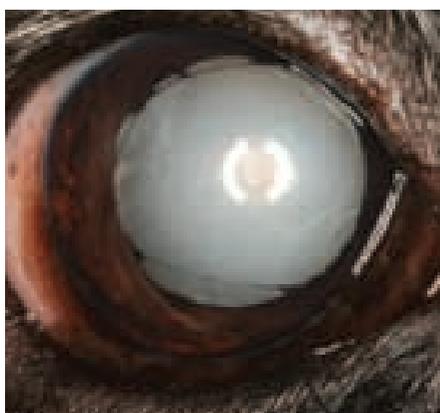


Figure 13: Mature senile cataract. The fundus cannot be examined. Iris atrophy is also present.



Figure 16: Intumescent mature diabetic cataract with clefts visible at the anterior sutures in a Y-shape.



Figure 14: Hyper-mature cataracts with secondary lens-induced uveitis.



Figure 17: Rupture of the anterior lens capsule of a chronic diabetic cataract with uveitis, fibrin and hyphaema.

CATARACT

A cataract is any opacity of the lens (Figures 10-15). They can affect one or both eyes. They can be classified in a number of ways. The most common descriptions are by cause (hereditary, diabetic, traumatic, senile, secondary to Progressive Retinal Atrophy); stage (incipient, immature, mature, hypermature); location (anterior, nuclear, cortical, sub-capsular, equatorial etc); appearance (spoke, triangular, perverulent etc), or age of onset (congenital, juvenile, senile).

Cataracts are very common in diabetic dogs, with 80% developing bilateral intumescent cataracts within 470 days (16 months) of diagnosis. Diabetic cataracts develop rapidly, and are the most common complication of diabetes in dogs. Saturation of the normal glucose metabolism mechanisms within the lens occurs, resulting in the production of the sugar sorbitol. This larger sugar cannot diffuse across the lens capsule and, therefore, builds up

within the lens, resulting in an osmotic gradient that allows water from the aqueous to enter the lens. This disrupts the architecture of the lens due to swelling and rupture of the lens fibres, and results in an irreversible structural change. Diabetic cataracts are described as intumescent, as the lens is swollen (Figure 16). They result in blindness and lens-induced uveitis, and can lead to other complications including lens capsule rupture (Figure 17), lens luxation, retinal detachment and glaucoma.

TREATMENT AND PROGNOSIS

Treatment depends on the extent of the cataract. Small cataracts that are not impairing vision are monitored. Some types of cataracts never change throughout the lifetime of a dog, but there is a potential for progression. Pedigree dogs with hereditary cataracts should not be used for breeding.

Mature and hypermature cataracts cause a low level of



Figure 18: Phacoemulsification surgery to remove the lens cortex and nucleus.



Figure 19: After cataract surgery, the intraocular lens implant is visible behind the iris in the remaining lens capsule. There is a clear tapetal reflection.



Figure 20: The lens is dislocated and is in the anterior chamber.



Figure 21: Posterior lens luxation with a green aphakic crescent, the smooth lens equator is seen.



Figure 22: Lens sub-luxation with visible stretching ciliary zonules, and the lens equator is visible. There is some anterior presentation of vitreous in front of the ventral aspect of the pupil.



Figure 23: Chronic uveitis with conjunctival hyperaemia, corneal oedema, darkening of the iris (it was blue prior to inflammation), and the dorsal lens equator can be seen due to secondary lens luxation.

lens-induced uveitis. This is treated with topical steroids or topical NSAIDs, in order to reduce the likelihood of more serious complications such as glaucoma. Diabetic dogs are normally treated with a topical NSAIDs rather than steroids, to avoid undesirable systemic effects. Cataracts that are causing visual impairment may be amenable to cataract extraction, using phacoemulsification. This highly specialised procedure involves cataract extraction through a small (<3mm) peripheral corneal incision, and the introduction of an instrument with breaks up the lens through high-frequency ultrasonic vibrations and aspirates it (Figure 18). A synthetic intraocular lens is then usually inserted in order to provide sharp focus after surgery (Figure 19). The procedure involves important aftercare including frequent eye drops and re-examination appointments. The prognosis after cataract surgery is very good, and success

rates are approximately 90%. Long-term monitoring is advised to maximize the chances of a good outcome. It is advisable to refer dogs with cataracts early, as there is a much lower success rate with advanced hyper-mature cataracts, and surgery might not always be possible at the later stages.

LENS LUXATION

Lens luxation is the displacement of the lens from its normal position, anteriorly into the anterior chamber (Figure 20) or posteriorly into the vitreous chamber (Figure 21). Lens sub-luxation is partial displacement at the early stages of the disease (Figure 22). Dislocation of the lens may be due to primary inherited defect in the ciliary zonules in certain breeds, including many terrier breeds, the Border Collie, Shar Pei and some spaniel breeds. Primary lens luxation is a bilateral problem, but typically

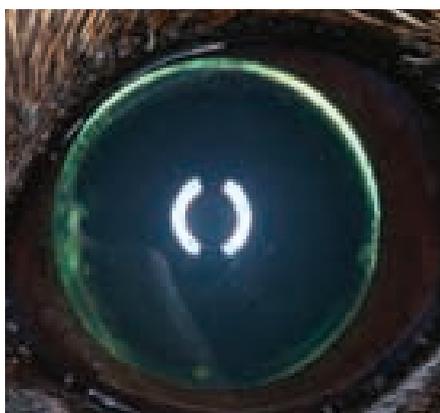


Figure 24: Lens sub-luxation; the lens equator can be seen, there is a bright but narrow aphakic crescent, and some white strands of vitreous are visible at the medial and lateral pupil border.



Figure 25: Sharp corneal penetration injury resulted in hyphaema and lens capsule rupture. This has healed with iris pigment on the anterior lens capsule, focal cataract and corneal scarring.



Figure 26: Chronic uveitis has led to a mature cataract and iris pigment rests on the anterior lens capsule.



Figure 27: Chronic uveitis led to iris pigmentation, cataract and anterior lens luxation.

the pathology does not progress symmetrically in both eyes and on initial presentation, one eye appears to be affected. The condition normally occurs in three-to-six-year-old dogs. Secondary lens luxation can also occur (Figure 23). This condition is usually caused by blunt trauma, chronic glaucoma with the enlarged eye stretching the lens zonules, hypermature cataract or physical displacement from an intraocular tumor.

Presenting signs include acute onset of ocular pain, with blepharospasm, epiphora and a closed eye. There is usually episcleral congestion especially if the entrapped lens results in raised intraocular pressure. There may be corneal edema, either due to raised intraocular pressure, or due to damage to the corneal endothelium if the lens enters the anterior chamber. In the latter case, the corneal oedema is focal and typically located at the centre of the ventral aspect of the cornea. The lens will be visualised sitting in the anterior chamber if it is anteriorly luxated, or the anterior chamber might appear deep due to posterior lens luxation. There may be an aphakic crescent, where the edge of the lens is visible due to the change in position. Examination of the contralateral eye may reveal anterior presentation of vitreous around the pupil margins (looking like small tufts of cotton wool, Figure 24). The lens or iris might 'wobble' due to a lack of support, termed phacodonesis and iridodonesis respectively. Secondary lens luxation does not usually have such an acute presentation. There is a slower onset of symptoms and there may be an enlarged globe with a chronic glaucoma, hyphaema with trauma, or a visible mass with an intraocular tumor.

DIAGNOSIS

The first challenge for lens luxation cases is to determine whether the condition is primary or secondary, as this has implications for treatment and prognosis of both eyes. This involves considering history and signalment, ocular signs, intraocular pressure measurement, and carefully examining the fellow eye for early signs of lens sub-luxation.

TREATMENT

A dog presenting with a sudden onset lens luxation is ideally referred to an ophthalmologist as an emergency. The affected eye needs urgent treatment, and the fellow eye needs to be examined to determine if it is at risk. As assessment is made as to whether there is still potential for vision – the presence of a dazzle reflex and consensual PLR without a raised intraocular pressure are positive prognostic signs. Cases of primary lens luxation are ideally treated with surgical removal of the lens with an intracapsular lens extraction. Treatment of the other eye will be required, but the treatment given depends on the opinions of the ophthalmologist, and the owner's wishes and finances. It is inevitable that the other lens will luxate in the case of primary lens luxation. Treatment may be medical, for example longterm topical prostaglandin analogues, or prophylactic surgical removal of the lens with phacoemulsification.

Secondary lens luxation is treated differently. If there is no potential for vision, it would be more common to enucleate the affected eye. The fellow eye will not be at risk if the primary cause was trauma, unilateral cataract or tumor. However if primary glaucoma is present, this will



Figure 28: Bilateral immature cataracts that were congenital.

need to be addressed with topical prophylactic glaucoma medications e.g. topical carbonic anhydrase inhibitors. The prognosis for vision is good with prompt surgical intervention but surgery always carries a risk of complications such as glaucoma or retinal detachment in the future. If the eye is blind at presentation, enucleation or evisceration with intraocular prosthesis is usually recommended. The second eye is always affected in cases of primary lens luxation so the prognosis is guarded, and prophylactic treatment and long-term monitoring is required.

LENS DISORDERS IN CATS

Conditions of the feline lens are much less common than in dogs. The two main abnormalities seen are cataracts and lens luxation.

Cataracts in cats are most often the consequence of ocular trauma (Figure 25), chronic uveitis (Figure 26), lens luxation (Figure 27) and glaucoma. Congenital (Figure 28) and inherited cataracts have been reported, but are uncommon. Diabetes mellitus doesn't lead to cataracts in cats, like it does in dogs. This is because the feline aging lens has low levels of aldose reductase activity, unlike in dogs of the same age. The most common cause of feline lens opacity is chronic anterior uveitis. These typically start in the peripheral cortex, and may be associated with the lens capsule at sites of posterior synechiae and inflammatory membranes. They are slowly progressive and can lead to total cataract.

When presented with a cat with a cataract, it is important to examine the globe to determine if the underlying cause can be determined, such as trauma or uveitis. Clinical signs of uveitis include conjunctival hyperaemia, keratic precipitates, aqueous flare, fibrin in the anterior chamber, iris changes including hyperaemia, pupil distortion and nodules, and a low intraocular pressure. The other eye should be examined to determine if there are any lens opacities or signs of uveitis. Treatment involves controlling the underlying cause. Sight-impairing cataracts might be removed with phacoemulsification surgery, in a similar fashion to dogs.

Lens luxation is most commonly a result of chronic anterior uveitis (Figure 29). The anterior chamber in the cat is quite deep, so the presence of the lens can sometimes not interrupt the outflow of aqueous. However in some



Figure 29: Anterior lens luxation - the cataractous lens occupies the majority of the anterior chamber.

circumstances, the presence of the lens in an abnormal location along with other changes to the drainage angle leads to an increase in intraocular pressure and glaucoma. Chronic glaucoma might be another consequence of chronic anterior uveitis, which can lead to an enlarged

Condition	Breeds affected
Congenital Hereditary Cataract	Miniature Schnauzer
Persistent Hyperplastic Primary Vitreous	Dobermann
	Staffordshire Bull Terrier
Hereditary Cataract	Alaskan Malamute
	Australian Shepherd
	Belgian Shepherd Dog (all varieties)
	Bichon Frise
	Boston Terrier
	Cavalier King Charles Spaniel
	German Shepherd Dog
	Giant Schnauzer
	Irish Red and White Setter
	Large Munsterlander
	Leonberger
	Miniature Schnauzer
Primary Lens Luxation	Norwegian Buhund
	Old English Sheepdog
	Poodle (Standard)
	Retriever (Chesapeake Bay)
	Retriever (Golden)
	Retriever (Labrador)
	Siberian Husky
	Spaniel (American Cocker)
	Spaniel (Welsh Springer)
	Staffordshire Bull Terrier
	Border Collie
	Bull Terrier (Miniature)
Fox Terrier (Smooth)	
Fox Terrier (Wire)	
Lancashire Heelers	
Parson Russell Terrier	
Sealyham Terrier	
Tibetan Terrier	

Table 1: Hereditary conditions affecting the lens assessed through the BVA/KC/ISDS Eye Scheme

Condition	Breeds affected
Hereditary Cataract	Australian Shepherd
	Boston Terrier (early onset)
	French Bulldog
	Staffordshire Bull Terrier (early onset)
Primary Lens Luxation	Australian Cattle Dog
	Chinese Crested Dog
	Jack Russell Terrier
	Jagdterrier
	Lancashire Heeler
	Miniature Bull Terrier
	Parson Russell Terrier
	Patterdale Terrier
	Rat Terrier
	Sealyham Terrier
	Tenterfield Terrier
	Tibetan Terrier
	Toy Fox Terrier
	Volpino Italiano
Welsh Terrier	
Yorkshire Terrier	
Wire-haired Fox Terrier	

Table 2: Hereditary conditions affecting the lens with DNA tests available at the Animal Health Trust www.aht.org.uk

globe (buphthalmos). The distended globe can result in tearing of the lens zonules holding the lens in position,

also leading to lens luxation.

A cat that presents with lens luxation should be examined to determine whether uveitis is present. It is important to always examine the other eye to determine if there is a bilateral condition presenting at different stages of the disease. A work-up for feline uveitis is required if this is present. The intraocular pressure should be measured also, as glaucoma is a painful and blinding condition that can be the cause or result of lens luxation.

Treatment involves controlling the underlying inflammatory process, and surgical removal of the lens with a lensectomy (intracapsular lens extraction). Prognosis depends on many factors including chronicity and whether or not glaucoma is present, but in many cases is favourable.

REDUCING HEREDITARY CATARACTS AND LENS LUXATION

Eye health schemes are active in assessing purebred breeding dogs for cataracts, in order to reduce the incidence by removing affected dogs from the breeding programme (Table 1). In Ireland, this service is available from Dr Terry Grimes, UCD, Ian Millar, Earlswood Vets Belfast, and Natasha Mitchell, Eye Vet Limerick.

A DNA test can be carried out on certain breeds at the Animal Health Trust, UK (Table 2). Genetics is a rapidly developing field, with continuing research allowing for new tests to be offered regularly.

Reader Questions and Answers

1. HOW COMMON ARE CATARACTS IN DIABETIC DOGS?

- (a) They are inevitable
- (b) They occur in 20% of diabetic dogs within one year of diagnosis
- (c) They occur in 80% of diabetic dogs within sixteen months of diagnosis
- (d) They are rare because of low aldose reductase activity in older dogs

2. HOW COMMON ARE CATARACTS IN DIABETIC CATS?

- (a) They are inevitable
- (b) They occur in 20% of diabetic cats within one year of diagnosis
- (c) They occur in 80% of diabetic cats within sixteen months of diagnosis
- (d) They are rare because of low aldose reductase activity in older cats

3. POTENTIAL CONSEQUENCES WITH CATARACTS INCLUDE

- (a) Lens-induced uveitis
- (b) Glaucoma
- (c) Retinal detachment
- (d) All of the above

4. LENS LUXATION IN CATS IS MOST COMMONLY ASSOCIATED WITH:

- (a) Chronic uveitis
- (b) Hereditary primary lens luxation
- (c) Diabetes
- (d) Intraocular tumour

ANSWERS: 1. C, 2. D, 3. D, 4. A