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Polapelli Vaishnavi

M.V.Sc. Scholar, Department of
Veterinary Surgery and
Radiology, College of Veterinary
Science and AH, DSVCKV,
Anjora, Durg, Chhattisgarh,
India

SK Tiwari

Department of Veterinary
Surgery and Radiology, College
of Veterinary Science and AH,
DSVCKV, Anjora, Durg,
Chhattisgarh, India

MO Kalim

Department of Veterinary
Surgery and Radiology, College
of Veterinary Science and AH,
DSVCKV, Anjora, Durg,
Chhattisgarh, India

Bhuvan M

Department of Veterinary
Surgery and Radiology, College
of Veterinary Science and AH,
DSVCKV, Anjora, Durg,
Chhattisgarh, India

Alok Jaiswal

Department of Veterinary
Surgery and Radiology, College
of Veterinary Science and AH,
DSVCKV, Anjora, Durg,
Chhattisgarh, India

Netrika Painkra

Department of Veterinary
Surgery and Radiology, College
of Veterinary Science and AH,
DSVCKV, Anjora, Durg,
Chhattisgarh, India

Corresponding Author:

Polapelli Vaishnavi

M.V.Sc. Scholar, Department of
Veterinary Surgery and
Radiology, College of Veterinary
Science and AH, DSVCKV,
Anjora, Durg, Chhattisgarh,
India

Advances and emerging techniques in canine cataract surgery: A review

Polapelli Vaishnavi, SK Tiwari, MO Kalim, Bhuvan M, Alok Jaiswal and Netrika Painkra

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Abstract

Canine cataract is a leading cause of reversible blindness in dogs and poses a significant challenge in veterinary ophthalmology due to its impact on vision and quality of life. Over the past decade, advances in surgical techniques, instrumentation, and perioperative care have markedly improved clinical outcomes and visual rehabilitation. Phacoemulsification remains the gold standard for cataract extraction, with modern systems offering optimized fluidics, reduced ultrasonic energy, and micro-incision techniques that minimize intraocular trauma and facilitate precise lens removal. Femtosecond laser cataract surgery automates precise capsulorhexis and lens fragmentation to improve surgical accuracy and safety. Progress in intraocular lens (IOL) technology, including foldable acrylic lenses, square-edge optics, and enhanced biocompatibility, has reduced postoperative inflammation and posterior capsular opacification, improving visual outcomes. Preoperative diagnostics, such as ocular ultrasonography, slit lamp biomicroscopy and electroretinography, support accurate case selection by assessing retinal integrity and identifying concurrent ocular pathologies. Optimized postoperative protocols involving anti-inflammatory therapy, intraocular pressure control, and antimicrobial prophylaxis have reduced complications, including uveitis and glaucoma. Collectively, these integrated advancements have increased surgical success rates, improved visual restoration, and enhanced the quality of life for affected dogs. Ongoing research continues to refine surgical techniques, develop innovative biomaterials, and incorporate emerging technologies to further advance clinical outcomes in veterinary ophthalmology.

Keywords: Canine cataract, veterinary ophthalmology, phacoemulsification, intraocular lens

Introduction

Cataract formation represents the leading cause of blindness in domestic canines (*Canis lupus familiaris*) globally, carrying profound implications for animal welfare, the human-animal bond, and veterinary ophthalmic practice (Ledbetter *et al.*, 2018) [27]. Defined pathophysiologically as any opacity of the crystalline lens or its capsule, regardless of its size, position, or clinical severity, cataracts disrupt the exquisite transparency required for optimal light transmission and visual transduction (Ofri, 2013). In dogs, the condition presents with high phenotypic heterogeneity, stemming from a dense web of genetic predispositions, metabolic aberrations, toxic insults, traumatic injuries, and senescent alterations (Basher and Roberts, 1995; Peters *et al.*, 2021). Left unmanaged, canine cataracts show a high propensity for progressive maturation, frequently culminating in secondary intraocular pathologies such as lens-induced uveitis (LIU), secondary glaucoma, retinal detachment, and permanent phthisis bulbi (Van der Woerd, 2000).

The veterinary field has witnessed a shift from historic, rudimentary techniques designed to merely remove obstructive lens blocks toward micro-endoscopy and advanced phacodynamic procedures that mimic state-of-the-art human techniques (Biros, 2011). Historically, canine cataract extraction was limited by poor instrumentation, an incomplete understanding of canine intraocular physiology, and a lack of effective anti-inflammatory drugs, leading to high complication rates (Magrane, 1969). Early techniques like Intracapsular Cataract Extraction (ICCE) [27, 37, 3, 39, 51, 4, 29]

and standard Extracapsular Cataract Extraction (ECCE) required large corneal or limbal incisions, resulting in severe postoperative astigmatism, high incidence of intraocular hemorrhage, and frequent retinal detachments.

The introduction of ultrasonic phacoemulsification altered veterinary ophthalmology by allowing lens extraction through micro-incisions (Miller *et al.*, 1987;). Over the past two decades, this technique has been refined with modern fluidics, advanced power modulations (such as torsional and transversal ultrasound), and specialized micro-instrumentation, maximizing surgical precision and reducing collateral thermal or mechanical injury to the corneal endothelium. Concurrently, the introduction of Femtosecond Laser-Assisted Cataract Surgery (FLACS) has brought automation and micrometer-level precision to critical steps like the anterior capsulorhexis and nuclear fragmentation (Donaldson *et al.*, 2013).

Achieving visual success goes beyond the removal of the opaque lens; it requires restoring the eye's refractive state through intraocular lens (IOL) implantation. The development of canine-specific foldability acrylic and silicone IOLs has minimized postoperative refractive errors (such as severe hyperopia), significantly improving functional visual outcomes and patient quality of life (Gemensky-Metzler *et al.*, 2011). Despite these technical leaps, the canine eye remains highly reactive to surgical trauma. Postoperative complications like posterior capsular opacification (PCO), capsular phimosis, transient or sustained postoperative ocular hypertension (POHT), and secondary glaucoma continue to present challenges (Smith *et al.*, 2008) [32, 10, 18, 46].

Anatomy of the lens and historical events

Microscopic and Functional Anatomy of the Canine Lens

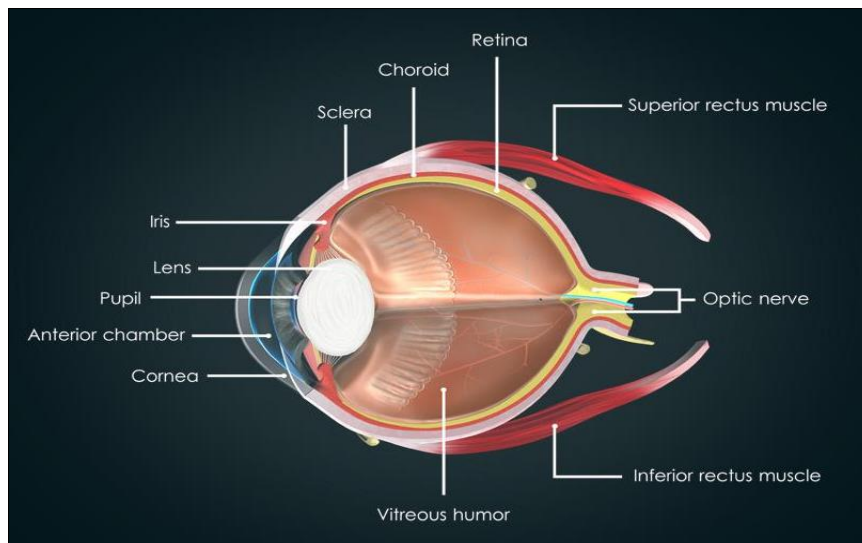


Fig 1: Anatomy of eye

The crystalline lens of the dog is a transparent, biconvex, avascular, and highly dynamic intraocular structure situated within the anterior segment of the globe, directly posterior to the iris and anterior to the vitreous body (Evans and de Lahunta, 2013). It is suspended in this position by a complex suspension apparatus, the ciliary zonules (zonules of Zinn), which arise from the ciliary processes and insert circumferentially onto the equatorial lens capsule (Gelatt, 2007). In dogs, the lens is substantially larger and more spherical relative to the overall volume of the globe than in humans, with an average axial diameter of 7.0 to 7.5 mm and an equatorial diameter of 11.5 to 12.5 mm, weighting approximately 450 to 500 mg depending on the age and breed of the animal (Davidson and Nelms, 1999). This structural difference means that canine cataract surgery demands larger cortical clearance and specialized instrumentation designed to manage higher volume lens materials.

Anatomically, the lens comprises four distinct structural components: the lens capsule, the anterior lens epithelium, the lens cortex, and the lens nucleus (Samuelson, 2013). The lens capsule is a modified, continuous basement membrane secreted by the lens epithelial cells, completely enveloping the lens parenchyma. It is rich in type IV collagen and sulfated glycosaminoglycans, providing high elasticity and tensile strength while acting as a selective semipermeable barrier against macromolecular shifts (Williams *et al.*, 2003). Crucially, the anterior lens capsule is significantly thicker

(ranging from 50 to 70 micrometers in dogs) than the posterior capsule, which can be thin (4 to 8 micrometers) near the posterior pole (Worgul, 1982). This anatomical asymmetry is clinically important, as the thin posterior capsule is vulnerable to accidental rupture during mechanical cortex aspiration or ultrasonic phacoemulsification [12, 14, 8, 42, 56, 55].

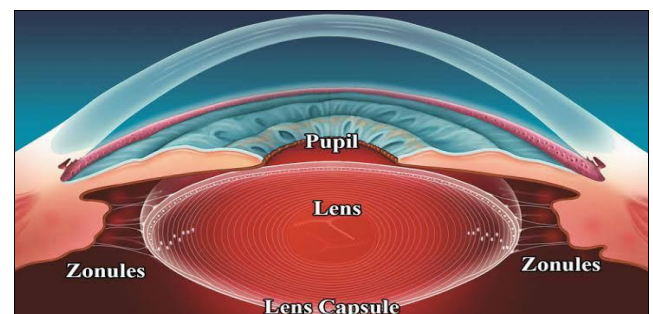


Fig 2: Anatomy of lens

The anterior lens epithelium consists of a single layer of cuboidal, metabolically active cells confined strictly to the internal aspect of the anterior and equatorial capsule (McAvoy *et al.*, 1999). Cells in the central zone maintain homeostatic metabolic transport via sodium-potassium adenosine triphosphatase (Na⁺/K⁺-ATPase) pumps, regulating the hydration state of the entire lens (Gorthy,

1978). As these epithelial cells migrate peripherally toward the equatorial zone often termed the germinative or transitional zone they undergo intense mitotic division, elongate, lose their intracellular organelles (including nuclei, mitochondria, and ribosomes), and differentiate into elongated lens fibers (Mathias *et al.*, 2007). This continuous lifelong production of new lens fibers forces older fibers inward toward the center, leading to progressive compaction and the formation of the dense lens nucleus, while the younger, less compacted peripheral fibers constitute the lens cortex (Augusteyn, 2010). The geometric alignment of these elongating fibers as they meet from opposite poles forms the characteristic Y-shaped sutural lines within the canine lens: an upright 'Y' configuration anteriorly and an inverted 'Y' configuration posteriorly (Samuelson, 2007) [31, 20, 30, 14, 2].

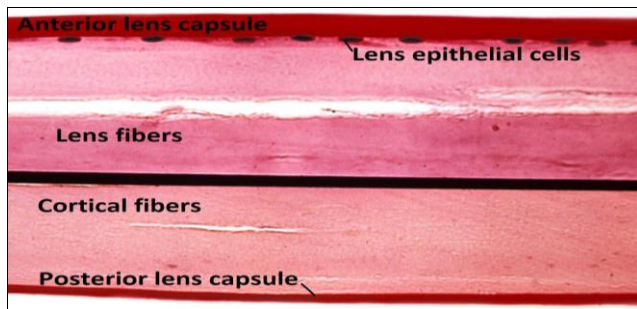


Fig 3: Histological section of lens

Historical evolution of canine cataract surgery

The history of canine cataract surgery reflects a steady progression from simple mechanical displacement to advanced microscopic and laser procedures, following a path similar to developments in human ophthalmology but shaped by the unique biological challenges of the canine eye. The earliest documented surgical intervention for cataracts in veterinary medicine dates back to the late 19th and early 20th centuries, utilizing 'couching' a crude technique where a sharp needle was introduced through the sclera to manually depress the opaque lens into the vitreous cavity (Startup, 1967). While couching restored light passage into the eye, it triggered massive intraocular inflammation, endophthalmitis, or retinal detachment, resulting in low long-term success rates.

By the mid-20th century, Intracapsular Cataract Extraction (ICCE) became the standard method in veterinary surgery (Magrane, 1969). ICCE involved removing the entire lens, including the intact lens capsule, through a large 160- to 180-degree corneal or corneoscleral incision. This was done after mechanically disrupting the ciliary zonules using specialized cryoprobes or alpha-chymotrypsin enzyme solutions (Gelatt, 1973). While ICCE eliminated the risk of postoperative cortical regrowth or posterior capsular opacification, its large wound size caused severe astigmatism, high susceptibility to intraocular infection, and frequent vitreous prolapse into the anterior chamber, which often led to pupillary block glaucoma or retinal tears (Startup, 1969) [47, 29, 13, 48].

To reduce these intraocular risks, Extracapsular Cataract Extraction (ECCE) gained traction in the 1970s and 1980s (Gelatt and Gum, 1984). ECCE required an incision through which a large central opening was made in the anterior capsule (capsulotomy), allowing the dense lens nucleus and surrounding cortex to be expressed or flushed from the eye while preserving the peripheral anterior capsule, equatorial zonules, and posterior capsule (Peiffer *et al.*, 1981). Preserving the posterior capsule provided structural stability to the vitreous face and reduced the incidence of retinal

complications. However, standard ECCE still required a 10- to 12-mm manual incision, which prolonged recovery times and triggered significant postoperative uveitis due to retained lens fragments (Whitley *et al.*, 1993).

The introduction of ultrasonic phacoemulsification, developed for humans by Charles Kelman in 1967 and adapted for dogs in the late 1980s, transformed veterinary ophthalmology (Miller *et al.*, 1987). Phacoemulsification uses a piezo-electric handpiece that oscillates at ultrasonic frequencies (typically 40 kHz) to emulsify the lens nucleus within the eye, allowing the fragments to be aspirated through an incision of 2.5 to 3.2 mm. This minimized corneal endothelial cell loss, reduced breakdown of the blood-aqueous barrier, and accelerated wound healing (Donzel *et al.*, 2017). Today, the integration of Femtosecond Laser-Assisted Cataract Surgery (FLACS) and micro-incision intraocular lenses marks the modern era of veterinary cataract surgery, combining automation with micron-level precision [16, 38, 52, 32, 11].

Classification of cataract

Stagel-Based on opacity and maturation of Canine Cataracts

Canine cataracts are classified based on their stage of development, which directly correlates with structural changes, clinical appearance, and the urgency of surgical intervention. Veterinary ophthalmology recognizes four primary developmental stages: incipient, immature, mature, and hypermature (Davidson and Nelms, 1999; Ofri, 2013).

Incipient Cataracts: This earliest stage is characterized by small, focal opacities that involve less than 10-15% of the total lens volume (Gelatt and Gelatt, 2011). Incipient cataracts do not cause noticeable visual deficits and leave the tapetal reflex intact during distant direct ophthalmoscopy. They are often located along the suture lines or within the peripheral cortex and may remain static or progress slowly (Davidson and Nelms, 1999)



Fig 4: Incipient Cataract

Immature Cataracts: Immature cataracts involve a larger portion of the lens, ranging from 15% to 95% of its volume. Portions of the lens remain transparent, allowing a partial tapetal reflex to be visualized through the clearer areas (Whitley *et al.*, 1993). Vision is moderately to severely impaired depending on the central or peripheral location of the opacities. At this stage, intraocular water absorption can cause the lens to swell, a condition known as an intumescent cataract. This swelling can shallow the anterior chamber and increase the risk of pupillary block (Gelatt, 2007). Immature cataracts are generally considered ideal candidates for surgical phacoemulsification due to their softer nuclear consistency and lower risk of severe lens-induced uveitis (Wilkie and Colitz, 2005) [8, 37, 15, 8, 52, 53].



Fig 5: Immature Cataract

Mature Cataracts: At this stage, the lens is completely opaque, involving 100% of the lens cortex and nucleus. The tapetal reflex is entirely obstructed, and the patient experiences complete functional blindness, retaining only basic light perception (Ofri, 2013). Ophthalmoscopically, the lens appears as a solid, uniform white or pearlescent structure, and the Y-sutures may be prominent. Mature cataracts are often accompanied by subclinical lens-induced uveitis (LIU) caused by the leakage of soluble lens proteins through the intact capsule (Van der Woerd, 2000) [37, 51].



Fig 6: Mature Cataract

Hyper mature Cataracts: If left unmanaged, a mature cataract progresses to hypermaturity, characterized by wrinkling of the anterior capsule and resorption of liquified cortical proteins. This volume loss causes the lens to shrink, often deepening the anterior chamber. Ophthalmoscopically, sparkles or glistening spots may appear within the lens matrix due to cholesterol crystals. In advanced cases, known as Morgagnian cataracts, the cortex completely liquefies, allowing the dense, brownish nucleus to sink to the bottom of the capsular bag (Gelatt and Gelatt, 2011). Hypermature cataracts carry a high risk of severe lens-induced uveitis, secondary glaucoma, and zonular instability, which can complicate surgical removal (Leasure *et al.*, 2014) [15, 26].



Fig 7: Hyper mature Cataract

Anatomical Location-Based Classification

Cataracts are also classified by their specific anatomical location within the lens structure: capsular, subcapsular,

cortical, nuclear, or equatorial (Davidson and Nelms, 1999). Capsular cataracts involve the lens capsule itself and are often congenital or secondary to penetrating trauma. Subcapsular cataracts form directly beneath the capsule, with posterior subcapsular cataracts frequently linked to chronic radiation exposure, toxic insults, or systemic corticosteroid therapy in other species, though they occur secondary to chronic uveitis in dogs. Cortical cataracts involve the lens fibers surrounding the nucleus and often present as wedge-shaped opacities or cortical spokes. Nuclear cataracts are located in the older, central core of the lens; they are typically firm and require higher ultrasonic energy during phacoemulsification. Equatorial cataracts form near the outer perimeter where new lens fibers originate, often detected early through pharmacologic pupillary dilation [8].

Age of Onset Classification

Based on the patient's age at presentation, cataracts are classified into four temporal groups: congenital, juvenile, adult, and senile (Gelatt, 2007). Congenital cataracts are present at birth and are often associated with other ocular anomalies, such as microphthalmia or persistent pupillary membranes (PPM). Juvenile cataracts develop within the first few years of life (typically 1 to 6 years of age) and are predominantly hereditary in purebred dogs. Adult cataracts manifest between 6 and 9 years of age, while senile cataracts occur in dogs older than 9 years, representing age-related oxidative degeneration of lens proteins [14].

Etiology of cataract

Hereditary (Genetic) Causes

Hereditary cataracts are the most common cause in dogs and occur due to genetic mutations affecting lens proteins (crystallins) or lens fiber development. These cataracts may be congenital or develop later in life.

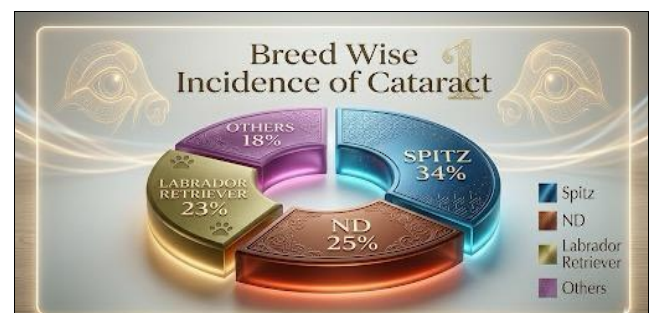


Fig 8: Breed predisposition of cataract

Several breeds show strong genetic predisposition, including

- Cocker Spaniel
- Poodle
- Labrador Retriever
- Golden Retriever
- Siberian Husky
- Boston Terrier

The inheritance pattern is often autosomal recessive, although dominant and polygenic traits have also been reported. Genetic mutations lead to abnormal protein aggregation, resulting in loss of lens transparency. These cataracts are often bilateral and progressive, though the rate of progression varies between breeds.

Diabetic (Metabolic) Cataracts

Diabetes mellitus is one of the most important causes of cataract formation in dogs. Unlike in humans, cataracts in diabetic dogs develop rapidly.

Pathogenesis

- Elevated blood glucose levels increase glucose concentration in the aqueous humor.
- Excess glucose enters the lens and is converted into sorbitol by the enzyme aldose reductase.
- Sorbitol accumulates because it diffuses slowly out of the lens.
- This causes osmotic imbalance, leading to water influx, lens fiber swelling, rupture, and opacification.
- Diabetic cataracts are typically:
 - Bilateral
 - Rapidly progressive (within days to weeks)
 - Often lead to complete blindness if untreated

Age-related (Senile) Changes

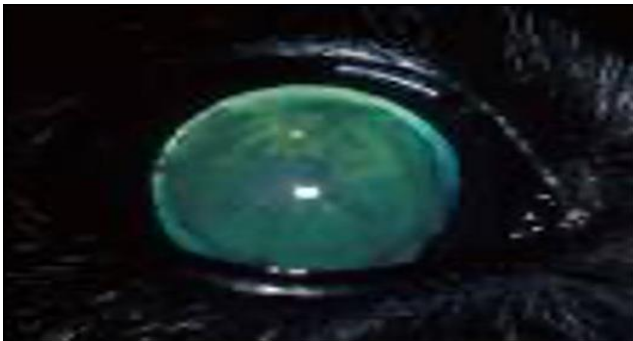


Fig 9: Senile cataract

With aging, biochemical and structural changes occur in the lens, predisposing it to cataract formation.

Mechanisms include

- Oxidative damage due to free radicals
- Decreased antioxidant activity (e.g., glutathione depletion)
- Protein denaturation and aggregation
- Reduced lens elasticity and permeability

These cataracts develop slowly and are usually bilateral. It is important to differentiate them from nuclear sclerosis, which is a normal aging change that does not significantly impair vision.

Traumatic Causes

Trauma is a significant cause of cataracts, particularly in young or active dogs.

Types of Trauma

- Penetrating injuries: rupture of lens capsule leading to immediate cataract formation
- Blunt trauma: concussive forces disrupt lens fibers
- Damage to the lens capsule allows aqueous humor to enter, causing:
 - Rapid hydration of lens fibers
 - Protein denaturation
 - Inflammatory response (lens-induced uveitis)

Traumatic cataracts are usually unilateral and may vary in severity depending on the extent of injury.

Nutritional Deficiencies

Nutritional cataracts are primarily seen in puppies and developing dogs.

Key factors include:

- Deficiency of amino acids
- Lack of antioxidants (vitamins A, C, E)
- Improper milk replacers in neonates

Poor nutrition interferes with normal lens development and metabolism, resulting in structural abnormalities and opacities. These cataracts may be prevented with proper dietary management.

Toxic and Drug-induced Cataracts

Certain drugs and toxins can induce cataract formation, although this is relatively uncommon.

Examples include

- Corticosteroids (long-term use)
- Some antiparasitic or chemotherapeutic agents
- Environmental toxins

Mechanism

- Interference with lens metabolism
- Oxidative damage
- Protein denaturation

The extent of cataract formation depends on dose, duration, and individual susceptibility.

Radiation-induced Cataracts

Exposure to ionizing radiation can damage lens epithelial cells, leading to cataract formation.

Pathogenesis

- Radiation disrupts mitotic activity of lens epithelial cells
- Abnormal differentiation of lens fibers
- Progressive opacification over time

This is usually seen in dogs undergoing radiation therapy for ocular or periocular tumors.

Secondary Cataracts (Associated with Ocular Diseases)

Cataracts frequently develop secondary to other intraocular conditions.

a) Uveitis

Inflammation of the uveal tract alters the intraocular environment:

- Increased inflammatory mediators
- Disruption of lens nutrition
- Capsule damage

This leads to progressive cataract formation.

b) Glaucoma

Elevated intraocular pressure:

- Impairs lens metabolism
- Causes ischemic damage

c) Retinal Degeneration

Conditions like progressive retinal atrophy (PRA) are often associated with secondary cataracts due to altered metabolic interactions within the eye.

d) Lens Luxation

Displacement of the lens disrupts its nutrition and structure, leading to cataract formation.

Congenital and Developmental Abnormalities

Cataracts may arise due to abnormal lens development during gestation.

Causes include

- Genetic defects
- Intrauterine infections
- Toxic exposure during pregnancy

These cataracts are often present at birth and may be associated with other ocular abnormalities such as microphakia or persistent pupillary membranes.

Oxidative Stress and Free Radical Damage

Oxidative stress plays a central role in many types of cataracts.

Mechanism

- Reactive oxygen species (ROS) damage lens proteins and lipids
- Oxidation leads to protein aggregation
- Loss of transparency occurs

The lens normally contains antioxidants like glutathione, but depletion with age or disease predisposes to cataract formation.

Pathophysiology of cataract

The transparency of the crystalline lens depends on the preservation of its highly specialized microstructural architecture and metabolic homeostasis (Worgul, 1982). At the molecular level, lens transparency is maintained by the structural arrangement of water-soluble proteins known as crystallins (alpha, beta, and gamma crystallins), which account for over 90% of the total protein content within lens fibers. These crystallins are packed in a short-range spatial

order that minimizes light scattering (Mathias *et al.*, 2007). Because mature lens fibers cannot synthesize new proteins or degrade damaged ones, any physical or chemical insult that disrupts this molecular arrangement triggers a cascade leading to protein aggregation, phase separation, and clinically visible opacification (McAvoy *et al.*, 1999).

Oxidative stress is a primary pathway in cataracts, particularly senile and post-inflammatory forms (Gorthy, 1978). The lens is regularly exposed to reactive oxygen species (ROS) from metabolic activities and ultraviolet radiation. Under healthy conditions, endogenous antioxidant enzymes such as glutathione (GSH), superoxide dismutase (SOD), and catalase neutralize these free radicals (Mathias *et al.*, 2007). With age or chronic intraocular inflammation, this antioxidant defense system can become depleted, leading to a decline in GSH levels within the lens core. The resulting accumulation of ROS causes oxidative modification of crystallin proteins, leading to the oxidation of sulfhydryl groups, cross-linking, and the formation of high-molecular-weight insoluble aggregates that scatter light (Augusteyn, 2010).

In diabetic canine cataracts, the pathophysiological pathway is largely metabolic and driven by osmotic changes (Basher and Roberts, 1995). The canine lens relies on glucose absorbed from the aqueous humor, which is primarily metabolized via the anaerobic glycolytic pathway regulated by the enzyme hexokinase. In conditions of sustained systemic hyperglycemia, the hexokinase pathway becomes saturated, shifting excess intracellular glucose into the sorbitol pathway (Gerritsen *et al.*, 2019). The enzyme aldose reductase reduces this excess glucose into sorbitol, a sugar alcohol. Because the lens cell membrane is relatively impermeable to sorbitol and lacks sufficient levels of sorbitol dehydrogenase to convert it into fructose, sorbitol accumulates within the lens fibers (Keller and Linke, 2016).

This accumulation creates a hyperosmotic gradient that draws water from the aqueous humor into the lens capsule, causing cell swelling, rupture of the lens fiber membranes, and the formation of prominent fluid vacuoles (Gerritsen *et al.*, 2019). Clinically, this manifests as rapid cortical swelling (intumescence) and the appearance of cortical water clefts along the sutural lines. The rapid mechanical stress from this swelling can tear the lens capsule, releasing soluble lens proteins into the anterior chamber and triggering severe lens-induced uveitis [56, 30, 31, 20, 30, 2, 3, 19, 23].

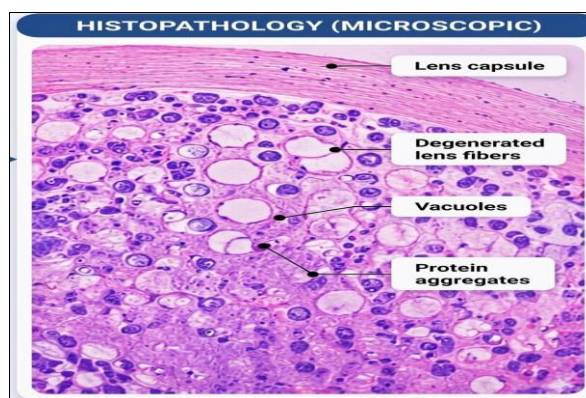


Fig 10: Histopathological section of lens which affected with cataract

The inflammatory response to these leaked lens proteins is divided into two distinct clinical syndromes: phacolytic uveitis and phacoclastic uveitis (Leasure *et al.*, 2014). Phacolytic uveitis is a low-grade, chronic inflammatory

reaction caused by the slow leakage of soluble crystallin proteins through an intact but compromised capsule in mature or hypermature cataracts. Macrophages phagocytize these foreign lens proteins, which can accumulate in the trabecular

meshwork, obstructing aqueous outflow and leading to secondary open-angle glaucoma. In contrast, phacoclastic uveitis is a severe, acute, granulomatous intraocular inflammation triggered by a physical rupture of the lens capsule, often due to penetrating trauma or rapid intumescent swelling. This sudden exposure of large volumes of lens protein recruits neutrophils, macrophages, and T-lymphocytes, which can form fibrovascular membranes, posterior synechiae, and pupillary occlusions, often requiring emergency surgical extraction (Sigle and Nasisse, 2012) [26, 43].

Diagnosis

A comprehensive preoperative diagnostic evaluation is critical in canine cataract surgery to confirm visual potential, characterize lens morphology, and identify coexisting ocular pathologies that could compromise the surgical outcome. The assessment begins with a thorough neuro-ophthalmic examination, evaluating the palpebral reflex, menace response, pupillary light reflexes (PLR), and dazzle reflex. While a mature cataract will block the menace response due to mechanical obstruction of vision, the direct and consensual PLRs and the dazzle reflex must remain brisk and complete. A sluggish or absent PLR or dazzle reflex raises concern for concurrent retinal degeneration or advanced glaucoma, which warrant further investigation.

Slit-lamp biomicroscopy is used to assess the anterior segment with high magnification and focal illumination. This technique allows the clinician to localize lens opacities, evaluate the integrity of the anterior capsule, and detect signs of anterior uveitis, such as aqueous flare, keratic precipitates, or posterior synechiae (Gelatt and Gelatt, 2011). It also helps identify signs of zonular instability, such as iridodonesis (tremoring of the iris) or phacodonesis (tremoring of the lens), which can indicate zonular breakdown and alter the surgical approach. Applanation or rebound tonometry is performed concurrently to measure intraocular pressure (IOP); a low IOP (<10 mmHg) suggests active lens-induced uveitis, while an elevated IOP (>25 mmHg) indicates secondary glaucoma, requiring aggressive medical management before surgery (Klein *et al.*, 2022) [15, 24].

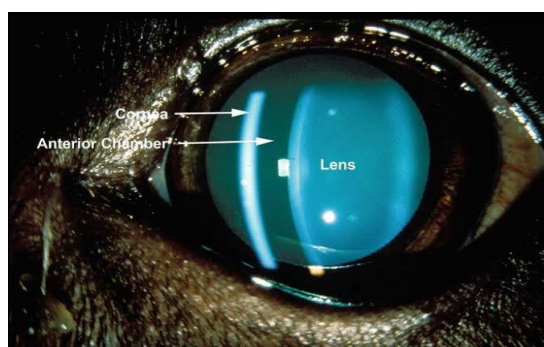


Fig 11: Slit lamp bio microscopy of canine eye

When a mature or hypermature cataract completely blocks direct ophthalmoscopic view of the posterior segment, ocular ultrasonography (B-scan) becomes necessary (Boroffka *et al.*, 2006). Utilizing a high-frequency (10-12 MHz) transducer applied to the cornea or sclera, B-scan ultrasonography provides a two-dimensional cross-sectional view of the posterior chamber, vitreous cavity, and retina. This imaging is vital for ruling out posterior segment pathologies that would preclude a successful visual outcome, such as retinal detachment, vitreous liquefaction (synchysis), vitreous hemorrhage, or persistent hyperplastic primary vitreous (PHPV) (Pumphrey, 2005) [15, 40].

Electroretinography (ERG) is an essential diagnostic test used to objectively evaluate the functional integrity of the photoreceptor cells within the retina (Peters *et al.*, 2021). Performed under a validated dark-adaptation and light-adaptation protocol, the ERG records the electrical potentials generated by the retina in response to light flashes. A normal ERG waveform consists of a negative-going 'a-wave' (representing rod and cone photoreceptor activity) followed by a positive-going 'b-wave' (representing inner nuclear layer bipolar cells). A flat or diminished ERG response indicates retinal dysfunction, such as Progressive Retinal Atrophy (PRA) or Sudden Acquired Retinal Degeneration Syndrome (SARDS). Since these conditions result in permanent blindness, cataract surgery would be contraindicated in their presence (Whitley *et al.*, 1993) [39, 52].



Fig 12&13: Electro retinography & Ultrasonography of eye

Advancements in surgical management

Preoperative Medication and Management

Preoperative stabilization is essential in canine cataract surgery to address lens-induced uveitis (LIU), which occurs when lens proteins leak through the capsule and trigger an inflammatory response. This inflammatory process can compromise surgical outcomes if not adequately controlled prior to intervention (Maggs *et al.*, 2018). Standard protocols require a minimum of 10-14 days of topical medications prior to the procedure to suppress inflammation and stabilize intraocular conditions. This typically includes topical

corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) to restore and maintain the blood-aqueous barrier (Slatter, 2001). Furthermore, systemic health management is critical; in diabetic patients, glycemic control must be optimized, often requiring diagnostic tests such as fructosamine estimation or serial blood glucose curves to ensure safe anesthetic management. Additionally, all systemic infections including dental disease, dermatological infections, and otitis should be resolved at least one month before surgery to minimize the risk of postoperative complications such as endophthalmitis (Gelatt *et al.*, 2013) [28, 45, 17].

General Anesthesia and Pharmacological Protocols

Canine cataract surgery is performed under deep general anesthesia, requiring precise pharmacological management to maintain a stable, centrally positioned globe and controlled intraocular pressure (IOP). Modern anesthetic protocols have significantly improved surgical success and safety, allowing procedures to be performed with minimal physiological disturbance (Tranquilli *et al.*, 2013).

Pre-anesthetic evaluation is mandatory and includes complete blood count (CBC), serum biochemistry, and urinalysis to assess systemic health, particularly renal and hepatic function, which are critical for drug metabolism and excretion (Grubb *et al.*, 2020). In older or diabetic patients, additional screening may be required to reduce anesthetic risk. Continuous intraoperative monitoring of vital parameters such as arterial blood pressure, capnography, and pulse oximetry is essential, as fluctuations in systemic blood pressure can lead to alterations in IOP and compromise surgical outcomes. Proper positioning, typically dorsal recumbency, ensures optimal surgical access and stability during the procedure [50, 22].

Local Anesthesia: Peribulbar Injection Technique

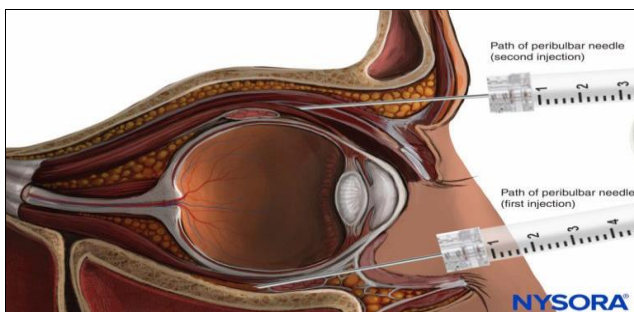


Fig 14: Peribulbar anesthesia in dog

Regional anesthesia is frequently used as an adjunct to general anesthesia to enhance analgesia and reduce the requirement for inhalational anesthetics. The peribulbar block is widely preferred due to its safety profile, as the anesthetic is deposited outside the extraocular muscle cone, minimizing the risk of globe perforation and optic nerve injury associated with retrobulbar techniques (Accola *et al.*, 2006).

The injection technique commonly involves a two-point approach, with one injection administered through the lower eyelid at the junction of the lateral third and medial two-thirds, advancing approximately 20-25 mm toward the equator of the globe (Skarda and Tranquilli, 2007). A combination of 2% lidocaine and 0.5% bupivacaine is frequently used to provide both rapid onset and prolonged duration of anesthesia. Alkalinization of lidocaine with sodium bicarbonate or warming the solution has been shown to reduce injection pain and improve onset time by enhancing tissue diffusion (Stevens *et al.*, 2013) [1, 44, 49].

Specialized Ophthalmic Surgical Instruments

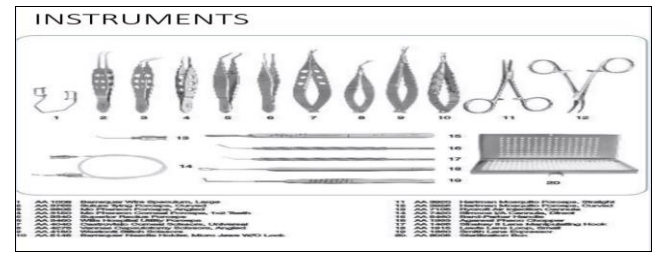


Fig 15: Ophthalmic instruments used for cataract surgery

The evolution of micro-incisional cataract surgery, particularly phacoemulsification, has necessitated the development of highly specialized ophthalmic instruments. The phacoemulsification handpiece is the central instrument, utilizing piezoelectric crystals to convert electrical energy into ultrasonic vibrations, typically operating at frequencies between 35,000 and 45,000 Hz to emulsify the lens nucleus.

Precision instruments such as capsulorhexis forceps are used to perform continuous curvilinear capsulorhexis (CCC), a critical step in maintaining capsular integrity and facilitating intraocular lens (IOL) implantation (Gelatt *et al.*, 2013). Corneal incisions are created using keratomes, usually ranging from 2.5 mm to 3.0 mm, allowing entry into the anterior chamber with minimal tissue trauma.

Additional instruments include IOL injectors or cartridge systems designed to insert foldable acrylic lenses through small incisions, thereby preserving corneal integrity and reducing postoperative astigmatism. Nuclear fragmentation techniques such as “divide and conquer” and “phaco chop” are facilitated by specialized choppers, enhancing surgical efficiency and reducing ultrasound energy exposure to intraocular tissues (Nagahara, 1993) [17, 33].

Intracapsular Cataract Extraction (ICCE)

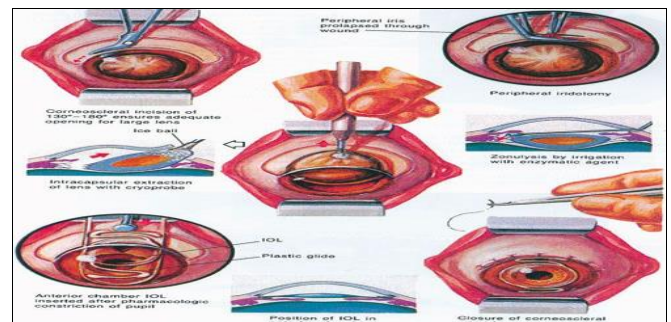


Fig 16: Intra capsular cataract extraction procedure

Intracapsular Cataract Extraction (ICCE) involves the complete removal of the entire lens body along with its intact surrounding capsule (Magrane, 1969). In contemporary veterinary ophthalmology, ICCE is rarely indicated as a primary elective procedure for uncomplicated cataracts due to its invasiveness and high complication rates. However, it remains an option for managing severe lens subluxation or complete luxation, where the ciliary zonules are extensively torn or degenerated, and preservation of the capsular bag is impossible.

The surgical protocol for ICCE requires a large 160- to 180-degree corneal or corneoscleral incision near the limbus to accommodate the full equatorial diameter of the lens. Following the incision, the remaining ciliary zonules are mechanically ruptured using an alpha-chymotrypsin enzyme

solution or a cryoprobe applied directly to the anterior lens capsule. The frozen cryoprobe creates a firm bond with the lens, allowing the intact lens structure to be extracted from the eye (Startup, 1969). The large wound requires multiple interrupted or continuous sutures (typically 8-0 or 9-0 nylon), which can induce significant postoperative corneal astigmatism and breakdown of the blood-aqueous barrier.

This large incision also carries risks of intraocular hemorrhage, vitreous prolapse into the anterior chamber, and postoperative ocular hypertension (Wilkie and Colitz, 2005) [29, 48, 53].

Extracapsular Cataract Extraction (ECCE)

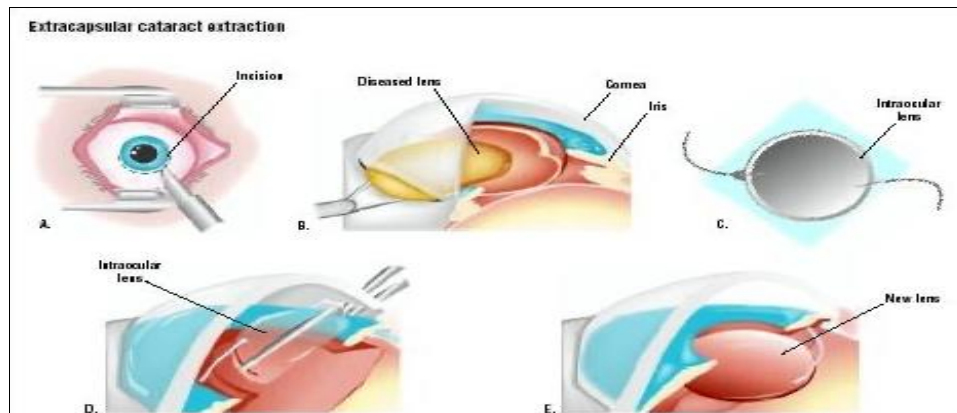


Fig 17: Extra capsular cataract extraction procedure

Extracapsular Cataract Extraction (ECCE) involves removing the dense lens nucleus and cortical parenchyma while leaving the posterior capsule, equatorial zonules, and peripheral anterior capsule intact (Peiffer *et al.*, 1981) [38]. ECCE was a major advancement over ICCE because preserving the posterior capsule maintains a physical barrier between the vitreous cavity and the anterior segment, reducing the risk of vitreous prolapse and subsequent retinal tears (Nasissse, 1989) [34].

The technique requires a manual incision of approximately 10 to 12 mm at the corneoscleral limbus. A large opening is made in the anterior capsule (capsulotomy) using a cystotome needle or micro-scissors, and the firm lens nucleus is is

expressed through the incision using specialized muscle hooks or loops. The remaining cortical material is cleared using manual or automated irrigation and aspiration tips. While ECCE safely manages dense, advanced cataracts, its larger incision compared to phacoemulsification causes higher postoperative inflammation, prolonged healing times, and a higher risk of astigmatism (Gelatt and Gum, 1984). Today, manual ECCE is reserved for cases with very dense, calcified nuclei that cannot be safely emulsified with ultrasound, or for clinical settings lacking advanced phacoemulsification equipment [16].

Phacoemulsification: The Modern Gold Standard

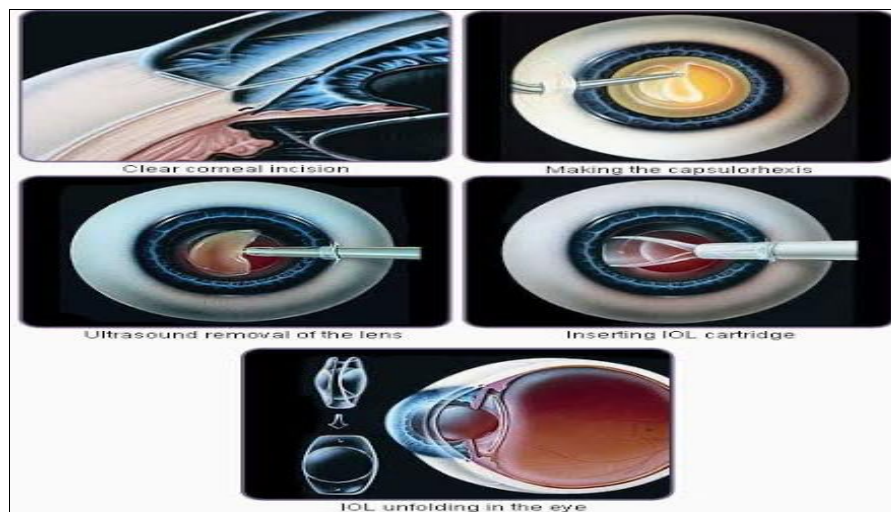


Fig 18: Phacoemulsification procedure

Ultrasonic phacoemulsification is the gold standard for canine cataract extraction, offering excellent visual outcomes through minimally invasive micro-incisions (Miller *et al.*, 1987). The procedure relies on a complex interaction of fluidics and power modulation, known as phacodynamics, to emulsify and remove the lens while protecting intraocular structures. The surgical technique involves several sequential, precise steps [38, 34, 16].

Incision and Viscoelastic Injection

A clear corneal incision, typically 2.5 to 3.2 mm, is created using a calibrated keratome blade. A secondary paracentesis incision (0.8 to 1.2 mm) is made 90 degrees apart to allow the insertion of a secondary instrument (such as a chopper or spatula). The anterior chamber is immediately filled with a cohesive or dispersive ophthalmic viscosurgical device (OVD), such as sodium hyaluronate or hydroxypropyl

methylcellulose, to maintain intraocular volume and shield the corneal endothelial cells from mechanical and ultrasonic trauma (Donzel *et al.*, 2017) ^[11].

Continuous Curvilinear Capsulorhexis (CCC)

Using specialized capsulorhexis forceps or a bent cystotome needle, a continuous, circular opening is made in the central anterior capsule. In canine surgery, achieving a smooth, centered 5.0 to 5.5 mm CCC is critical because the thick, elastic canine capsule resists tearing and is prone to radial 'run-out' extensions that could compromise the entire capsular bag.

Hydrodissection and Hydrodelineation

Balanced Salt Solution (BSS) is injected beneath the remaining anterior capsular rim to separate the lens cortex from the capsule (hydrodissection), followed by injection into the deeper layers to isolate the nuclear core (hydrodelineation). This allows the lens core to rotate freely within the capsular bag, reducing mechanical stress on the ciliary zonules during emulsification.

Nuclear Emulsification Techniques: The phacoemulsification handpiece is introduced, utilizing piezoelectric crystals that vibrate at ultrasonic frequencies to fragment the nucleus. Advanced techniques like 'Divide and Conquer' or 'Stop and Chop' are used to manage the dense canine lens. In 'Divide and Conquer,' the ultrasound tip sculpts deep orthogonal grooves into the nucleus, which is then split into quadrants using a secondary spatula. In 'Stop and Chop,' a mechanical copper cracks the nucleus into smaller segments after an initial groove is made, reducing the total ultrasonic energy delivered into the eye (Biros, 2011). Modern systems use torsional or transversal power modulations, moving the tip in a sweeping lateral direction rather than a longitudinal stroke, which minimizes repulsion of lens fragments and reduces thermal energy generation.

Cortical Aspiration: After nuclear removal, the remaining soft cortical material is systematically cleared using automated irrigation and aspiration (I&A) co-axial tips, leaving a clean, transparent capsular bag ready for intraocular lens implantation (Wilkie and Colitz, 2009) ^[54].

Femtosecond Laser-Assisted Cataract Surgery (FLACS)

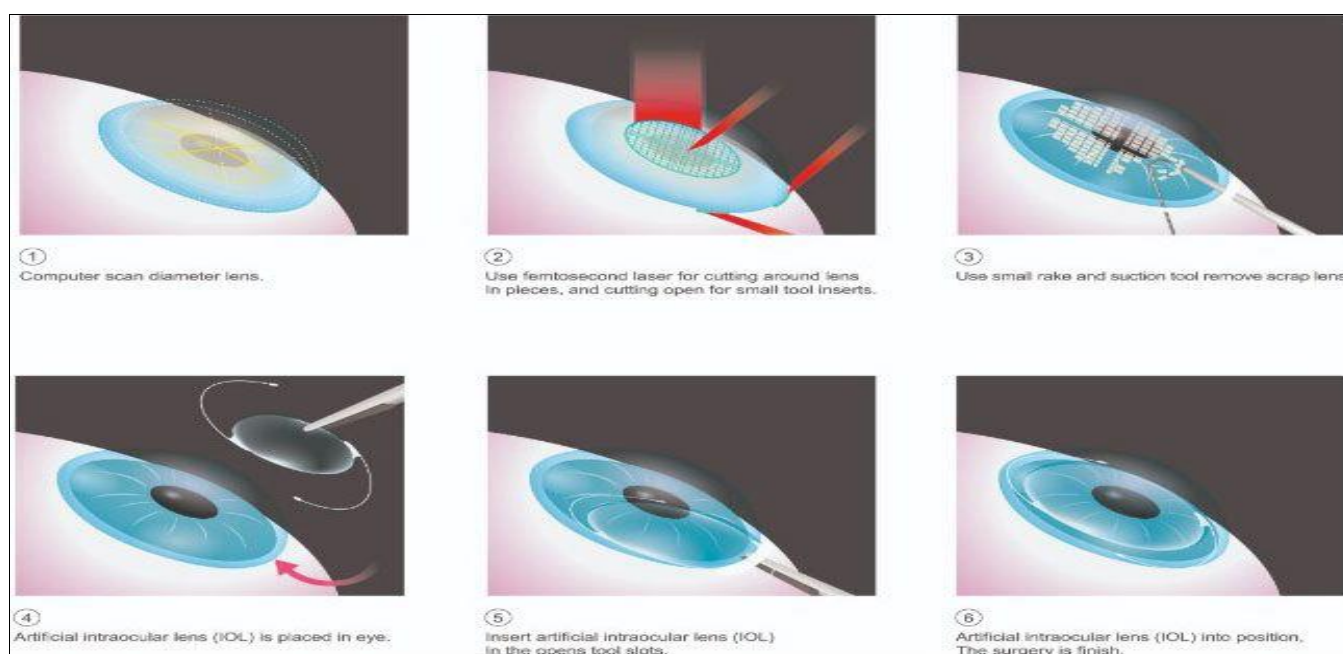


Fig 19: Femtosecond laser assisted cataract surgery procedure

Femtosecond Laser-Assisted Cataract Surgery (FLACS) represents an advanced technological development in veterinary ophthalmology, bringing automation and precision to critical surgical steps (Donaldson *et al.*, 2013). Operating at an infrared wavelength of 1053 nm with pulse durations measured in femtoseconds, the laser induces photodisruption focused laser energy creates micro-plasma bubbles that expand and cleave tissue with minimal collateral thermal damage to surrounding ocular structures ^[10].

In a FLACS protocol, the patient's eye is docked to the laser system using a liquid-optics interface. High-resolution, integrated anterior segment optical coherence tomography (AS-OCT) captures real-time cross-sectional images of the eye, allowing the surgeon to program precise treatment parameters. The femtosecond laser then automates three main steps: the clear corneal incisions, the anterior capsulorhexis, and the nuclear fragmentation. It creates a perfectly circular, centered anterior capsulorhexis of a pre-specified diameter,

reducing the risk of manual tearing or radial extensions in elastic canine capsules. Additionally, pre-fragmenting the nucleus into small cubes reduces the total ultrasonic energy and fluid volume required during subsequent phacoemulsification, helping protect the corneal endothelium. However, the high capital cost of equipment, specialized docking requirements, and longer setup times currently limit FLACS to advanced veterinary referral centers (Chang *et al.*, 2016) ^[6].

Canine intraocular lens (IOL)

Achieving visual success goes beyond the removal of the opaque lens; it requires restoring the eye's refractive state through intraocular lens (IOL) implantation (Nasissse *et al.*, 1990). The normal canine eye is emmetropic, with a total refractive power of approximately 40 to 42 diopters (D), provided by both the cornea and the crystalline lens. When the crystalline lens is removed without replacement, the eye is

left aphakic, causing a severe refractive deficit of +14 to +15 diopters of hyperopia. While aphakic dogs retain some crude, blurred vision, they struggle with image resolution, depth perception, and near-field focus, which can affect their performance and quality of life (Grahn *et al.*, 2004)^[35, 21].



Fig 20: Canine Intra ocular lens

To restore emmetropia, canine-specific intraocular lenses have been engineered with an optical refractive power tailored to the dog, typically ranging from +41.0 D to +43.0 D (Gemensky-Metzler *et al.*, 2011). These lenses feature a large optical diameter (6.5 to 7.0 mm) and an overall haptic diameter (12.0 to 14.0 mm) designed to fit the spacious canine capsular bag. Modern designs use hydrophobic or hydrophilic acrylic materials, which are biocompatible and flexible, allowing the IOL to be rolled and injected through a micro-incision (<3.0 mm) without enlarging the primary wound. The haptic configurations, such as modified C-loop or plate designs, are engineered to exert controlled centripetal pressure within the capsular bag, ensuring long-term centration and stability. Implanting an IOL also provides physical support to the posterior capsule, reducing the incidence of capsule wrinkling and posterior capsular opacification (PCO) (Smith *et al.*, 2008)^[18, 46].

Postoperative care and prognosis

Postoperative care is critical to the success of canine cataract surgery, as the canine eye exhibits a reactive inflammatory response to surgical trauma (Wilkie and Colitz, 2005). The immediate postoperative period requires aggressive anti-inflammatory and antimicrobial therapy to control the

breakdown of the blood-aqueous barrier and prevent secondary complications. Topical therapy forms the mainstay of treatment, utilizing potent ophthalmic corticosteroids (such as 1% prednisolone acetate or 0.1% dexamethasone) administered 4 to 6 times daily, tapered gradually over 6 to 12 weeks based on clinical assessment. This is supplemented with topical non-steroidal anti-inflammatory drugs (NSAIDs), such as 0.1% diclofenac or 0.09% bromfenac, which inhibit cyclooxygenase pathways and provide synergistic control of intraocular inflammation (Gelatt and Gelatt, 2011). Prophylactic broad-spectrum topical antibiotics (e.g., ofloxacin or tobramycin) are administered 4 times daily for the first two weeks to prevent opportunistic bacterial endophthalmitis^[53, 15].

Systemic therapy is used concurrently to manage postoperative inflammation and pain. Oral non-steroidal anti-inflammatory drugs (e.g., carprofen at 2.2 mg/kg BID or meloxicam at 0.1 mg/kg SID) are prescribed for 7 to 14 days. In cases with pre-existing or severe intraocular inflammation, a short, tapering course of oral prednisone (0.5 to 1.0 mg/kg SID) may be indicated, provided there are no systemic contraindications like uncontrolled diabetes mellitus. Patient compliance is ensured through the continuous use of an Elizabethan collar to prevent self-trauma, along with strict cage rest and leash-walks only, avoiding any strenuous activity that could cause ocular hypertension or IOL displacement.

The long-term prognosis for functional visual restoration following uncomplicated phacoemulsification with IOL implantation is favorable, with success rates ranging from 85% to 92% at 12 months post-surgery (Klein *et al.*, 2022). Long-term visual retention relies on regular follow-up evaluations typically performed at 1 day, 1 week, 3 weeks, 6 weeks, 3 months, and biannually thereafter to monitor for delayed complications like secondary glaucoma or retinal detachment.

Complications

Despite advancements in technique, canine cataract surgery carries risk due to the reactive nature of the canine uveal tract and its susceptibility to complications, which can occur intraoperatively or postoperatively (Sigle and Nasissse, 2012).

Complication	Timing & Pathophysiology	Clinical Management Strategy
Postoperative Ocular Hypertension (POHT)	Acute (2-24 hours post-op); caused by retained viscoelastic material blocking the trabecular meshwork.	Topical carbonic anhydrase inhibitors (dorzolamide) and beta-blockers (timolol); systemic mannitol in refractory cases.
Secondary Glaucoma	Chronic; caused by extensive synechiae formation or fibrovascular occlusion of the iridocorneal angle.	Long-term topical prostaglandin analogs (latanoprost), dorzolamide/timolol, or surgical goniosynoviotomy.
Posterior Capsular Opacification (PCO)	Delayed (months to years); caused by proliferation and myofibroblastic migration of equatorial lens epithelial cells.	Nd:YAG laser capsulotomy to clear the visual axis; prevented by using square-edged IOL designs.
Posterior Capsular Rupture (PCR)	Intraoperative; mechanical tear of the thin posterior capsule during sculpting or cortical aspiration.	Immediate conversion to manual aspiration, anterior vitrectomy if vitreous prolapses, and possible sulcus IOL placement.

Fig 21: Post operative complications of canine cataract



Fig 22: Post operative complications of canine cataract i.e. hyphema, uveitis, glaucoma, lens luxation

Conclusion

The evolution of canine cataract surgery from rudimentary couching and large-incision manual extractions to modern micro-incision phacoemulsification represents a monumental advancement in veterinary ophthalmology. Modern surgical management, particularly through the use of ultrasonic phacoemulsification and Femtosecond Laser-Assisted Cataract Surgery (FLACS), has significantly enhanced surgical precision while reducing collateral damage to intraocular structures. These technological leaps, combined with the development of canine-specific foldable intraocular lenses (IOLs), allow for the restoration of emmetropia and a dramatic improvement in the functional quality of life for affected patients.

Success in these procedures is not solely dependent on surgical technique but relies heavily on a comprehensive perioperative approach. Rigorous preoperative stabilization of lens-induced uveitis and meticulous pharmacological management during general anesthesia are fundamental to ensuring a stable surgical environment. Furthermore, the integration of regional anesthesia, such as the peribulbar block, has proven effective in providing superior analgesia and reducing systemic anesthetic requirements.

While complications such as postoperative ocular hypertension and secondary glaucoma remain challenges due to the reactive nature of the canine eye, advanced post-operative care and long-term monitoring have elevated success rates to between 85% and 92%. Ultimately, the synergy of specialized micro-instrumentation, advanced imaging like B-scan ultrasonography, and refined surgical maneuvers ensures that cataract surgery remains a highly effective and predictable solution for visual restoration in domestic canines.

Conflict of Interest

Not available

Financial Support

Not available

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