The pressure within the eye, the intraocular pressure (IOP), is based on the balance between production and drainage of aqueous humour. A balanced intraocular pressure is required to keep the eye in shape, provide nutrients to intraocular structures and maintain normal function of these structures. Increased intraocular pressure occurs when there is a defective drainage of aqueous humour from the eye. Decreased intraocular pressure is the result of either reduced production, or in case of injury, leakage through a defect in the globe wall.

**Tonometry**

Tonometry is the measurement of IOP. Tonometry is essential in the work-up of animals with ocular conditions. Thus, a reliable tonometer should be standard equipment in all companion animal practices. Recommended types include rebound tonometer with a magnetized probe that bounces off the cornea when the cornea is touched with the probe (TonoVet) and applanation tonometer that measures the counter pressure when the probe touches the cornea (e.g. TonoPen).

*The results of IOP measurements must always be correlated with clinical findings and must be compared with IOP in the fellow eye*

Normal IOP in dogs is 15-25 mm Hg
Normal IOP in cats is 15-35 mm Hg

Older animals have lower IOP than younger
Debilitated animals have lower IOP than healthy animals
IOP values increase if the animal is stressed
IOP values increase if the animal is firmly restrained during measurement
If IOP has been elevated over a period and the eye has become buphthalmic, the eye will not shrink noticeably even if the IOP is lowered

In order to evaluate the effect of treatment and correctly adjust therapy, IOP measurements should be repeated at regular intervals during the treatment period. This is as important in uveitis, as well as for glaucoma or combinations of uveitis and glaucoma.

**Low intraocular pressure – ocular hypotension**

The most common cause of ocular hypotension is intraocular inflammation (uveitis). Acute anterior uveitis (iritis) is a painful condition causing photophobia and blepharospasm. Affected eyes are red, with both episcleral (deep) and conjunctival (superficial) hyperaemia. Small blood vessels may extend across the limbus, the junction between sclera and cornea. The cornea may be blue with loss of transparency, because of oedema. The iris is swollen, the pupil is miotic and more constricted than normal. In acute uveitis the composition of the exudate determines the appearance of the fluid in the anterior chamber where changes can easily be observed.

Fibrinous exudate may cause adherence between the iris and lens (posterior synaechia), resulting in aqueous being trapped behind the iris and subsequently a gradual increase in intraocular pressure. Inflammatory cells and fibrin blocking the iridocorneal angle may also cause secondary glaucoma. Inflammation in the posterior uvea, the choroid, causes less dramatic clinical signs, but the eye appears red with hyperaemia of episcleral blood vessels. Swelling of the choroid may cause fluid to accumulate behind the retina, resulting in retinal detachment and blindness. The retina may also be affected by concurrent inflammation, chorioretinitis.
A thorough work-up is recommended in all animals with uveitis, as uveitis can be associated with systemic diseases. The choice of treatment depends on primary diagnosis and severity of clinical signs. Treatment includes anti-inflammatory treatment/treatment of pain, mydriatics, plus antibiotics when necessary. Occasional secondary ocular hypertension must be diagnosed and adequate drugs instilled. Long-term treatment is often required in uveitis. IOP measurement is an important diagnostic aid and should be monitored throughout the whole period of treatment. Treatment should not be stopped before IOP is back to normal.

High intraocular pressure - ocular hypertension - glaucoma
The most common cause of ocular hypertension is obstruction of outflow. Causes include abnormal development of the iridocorneal angle, resulting in insufficient drainage (primary glaucoma). This is a relatively common and breed-related disease that affects both eyes. Clinical signs develop in middle aged dogs, but not necessarily in both eyes simultaneously. Secondary glaucoma develops as a sequel to other ocular disease, either because of intraocular tumours, posterior synaechia, or by obstruction of the iridocorneal angle and/or the ciliary cleft by inflammatory cells.

Early clinical signs of glaucoma include serous discharge, blepharospasm, hyperaemia of conjunctival, episcleral and scleral vessels, slow or absent pupillary light reflexes and mydriasis. Fundus is normal in early stages, except from the optic disc that appears pale, later cupped (pressed backwards). Clinical signs in more advanced stages include pain, mydriasis, corneal oedema, conjunctival, episcleral and scleral vessel congestion, buphthalmia, retinal degeneration and blindness.

- IOP > 30 mm Hg over some days damages the optic nerve and retina
- IOP > 40 mm Hg is painful and causes enlargement of the globe (buphthalmia)
- IOP > 40-50 mm Hg leads to paralysis of the sphincter muscle of the iris and causes mydriasis

The principles for treatment of primary glaucoma have been to reduce the intraocular pressure, either medically or by surgery. Newer research may indicate that other agents, i.e. neuroprotective agents, may also be helpful in maintaining vision in affected dogs. Nevertheless, even if adequately treated, glaucoma is a malignant disease with a guarded prognosis. In breed-related primary glaucoma close follow-up and treatment of the fellow, normotensive eye is indicated.

IOP measurement is an essential part of work-up and treatment of all ocular conditions.