



STAFFORDSHIRE BULL TERRIER HEALTH ISSUES

www.sbtcv.org.au

find us on facebook as well

Distichiasis

Sometimes the condition is referred to as a double row of eyelashes, for extra hairs arise from the edge of the eyelid to rub against the corneal surface. The effects are variable and mild irritation to corneal ulceration will be seen. Treatment is extremely difficult and invariably involves surgery to remove the hair roots permanently. Plucking out the offending hairs is useful, but requires the maximum cooperation of the patient!

Of course it is followed by hair regrowth, and many surgical techniques have been invented to remove the roots. Even then success is difficult to achieve, and the dog may have to suffer this condition throughout its life. It is the most common eye defect found in the Stafford in South Africa.

Entropion

Primarily an inherited condition. It is due to an excess of eyelid tissue, or a small eye, or both, the result being that a varying amount of hair-covered eyelid can turn in to rub directly against the cornea or conjunctiva, or both.

It is usually extremely painful, and the damage caused to the cornea can render the eye blind. Most dogs are affected by six months of age and in some the signs of the problem (excessive blinking and a wet face) may be seen within the first month of life.

Occasionally the condition is self-correcting as the puppy grows, but in the vast majority of affect dogs surgery is necessary to turn the eyelid away from the surface of the eye. Usually such surgery is successful, but it is much better that, as with the other inherited eyelid defects, breeders try to avoid producing this condition in their stock.

Ectropion

Primarily an inherited condition, in which the lower eyelid droops away from the eyeball to expose the third eyelid and the conjunctiva. Exposure of the delicate mucous membrane causes conjunctivitis.

Correction is possible by complicated surgery in which the eyelid is lifted and short-

ened. Occasionally further surgery may be necessary to change completely the shape of the eyelids.

Progressive Retinal Atrophy (PRA)

PRA is a term used to describe a number of inherited retinal degenerations involving several breeds. The group is broadly divided into two, generalized PRA and central PRA. In the former, blindness at night time (nyctalopia) is an early indication of the presence of the disease, but eventually the dog is rendered totally blind.

Cataract is a common secondary feature of the disease. In central PRA night blindness is not a feature and though vision is severely affected, the dog may not become totally blind. In both groups of PRA there is degeneration of the photoreceptors, but in the generalized form this degeneration is the inherited defect, whereas in central PRA rod and cone degeneration follows an inherited defect elsewhere in the retina.

Persistent Hyperplastic Primary Vitreous (PHPV)

This is a congenital condition (present from birth) in which there is a developmental defect in the normal regression of some of the intraocular structures of the eye. PHPV can range from being very mild to severe abnormalities which may lead to blindness. The presence of mild abnormalities are usually seen as small brown pigmented dots on the posterior lens capsule. Previously the literature indicated that this was always observed as a bilateral phenomenon but recently it has been stated that affected dogs may show unilateral involvement, although this is





STAFFORDSHIRE BULL TERRIER HEALTH ISSUES

www.sbtcv.org.au

find us on facebook as well

less common.

The present knowledge of the mode of inheritance of this disease is thought to be an autosomal irregular dominant with variable expression. Due to PHPV seldom resulting in secondary cataracts in the Stafford, those that are mildly afflicted will seldom show any form of visual impairment during the course of their lives. Even those that are more severely afflicted, may be capable of adapting by using peripheral to compensate. Stafford breeders should therefore not assume that the problem is absent simply because they have not encountered blatant signs of visual impairment, instead discerning breeders should ensure that all their Staffords are tested through the National Eye Scheme. Courtesy Stafford Mall

Hip Dysplasia

Hip Dysplasia is a terrible genetic disease because of the various degrees of arthritis (also called degenerative joint disease, arthrosis, osteoarthritis) it can eventually produce, leading to pain and debilitation. The very first step in the development of arthritis is articular cartilage (the type of cartilage lining the joint) damage due to the inherited bad biomechanics of an abnormally developed hip joint. Traumatic articular fracture through the joint surface is another way cartilage is damaged. With cartilage damage, lots of degradative enzymes are released into the joint.

These enzymes degrade and decrease the synthesis of important constituent molecules that form hyaline cartilage called proteoglycans. This causes the cartilage to lose its thickness and elasticity, which are important in absorbing mechanical loads placed across the joint during movement. Eventually, more debris and enzymes spill into the joint fluid and destroy molecules called glycosaminoglycan and hyaluronate which are important precursors that form the cartilage proteoglycans.

The joint's lubrication and ability to block inflammatory cells are lost and the debris-tainted joint fluid loses its ability to properly nourish the cartilage through impairment of

nutrient-waste exchange across the joint cartilage cells. The damage then spreads to the synovial membrane lining the joint capsule and more degradative enzymes and inflammatory cells stream into the joint. Full thickness loss of cartilage allows the synovial fluid to contact nerve endings in the subchondral bone, resulting in pain. In an attempt to stabilize the joint to decrease the pain, the animal's body produces new bone at the edges of the joint surface, joint capsule, ligament and muscle attachments (bone spurs). The joint capsule also eventually thickens and the joint's range of motion decreases.

No one can predict when or even if a dysplastic dog will start showing clinical signs of lameness due to pain. There are multiple environmental factors such as caloric intake, level of exercise, and weather that can affect the severity of clinical signs and phenotypic expression (radiographic changes). There is no rhyme or reason to the severity of radiographic changes correlated with the clinical findings. There are a number of dysplastic dogs with severe arthritis that run, jump, and play as if nothing is wrong and some dogs with barely any arthritic radiographic changes that are severely lame.

Courtesy OFA

