Fragmented Coronoid Process (FCP)

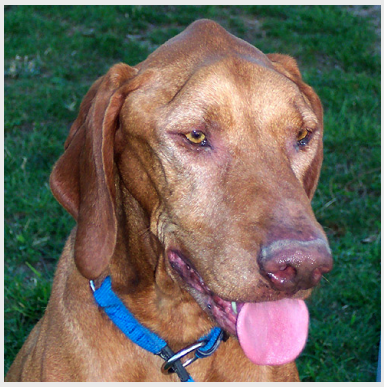
Elbow dysplasia (abnormal development of the elbow) describes several different conditions which affect the elbow. Fragmented coronoid process (FCP) is the most common type of elbow dysplasia. Environmental factors such as diet and activity levels may affect the expression of this condition. Genetic predisposition is believed to be important. A recently published survey of orthopaedic conditions did not list Vizslas as susceptible to FCP (LaFond). See also: Osteochondrosis.

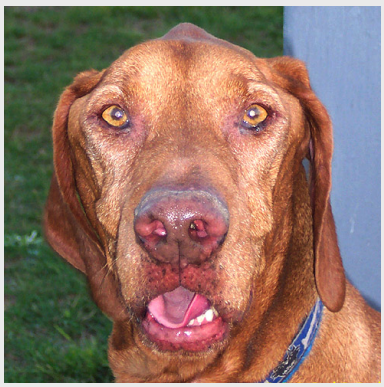
Goniodysgenesis / primary glaucoma

Currently the British Veterinary Association/Kennel Club/International Sheep Dog Society (BVA/KC/ISDS) eye scheme has listed goniodysgenesis/ glaucoma as suspected as being hereditary in Hungarian Vizslas and several other breeds of dog. Glaucoma is the result of increased fluid pressure within the eye. It can be the result of a number of causes, and can be inherited. In goniodysgenesis the normal fluid movement in the eye is disrupted by an abnormality of the tissue responsible for drainage of fluid through the irido-corneal angle. This drainage disruption can cause glaucoma. The CoE of the Hungarian Vizsla Society and of the Hungarian Vizsla Club in the United Kingdom encourage members to have their breeding stock tested.

Haemangiosarcoma, cutaneous

Cutaneous haemangiosarcoma is a cancer arising from the cells lining blood vessels. Goldschmidt found Vizslas to be predisposed to developing these tumours (odds ratio = 5.2). See also: odds ratio.





Q: Does this Vizsla have?

a) "affected" head

b) cranial muscular atrophy

c) skull defect

A: None of the above.

Histopathology performed on the affected muscle diagnosed atrophic myositis.

See: "Masticatory Muscle Myositis", under "Conditions which also occur in Vizslas".

Head deformity

See: Head/Skull problem.

Head/skull problem

Note: The conditions listed below; "affected" head, cranial muscular atrophy, head deformity and skull defects have all been cited by authors of Vizsla books or articles, or by Vizsla Clubs, as genetic conditions affecting Vizslas. They all share in common a possible presenting sign of atrophy (wasting) of the muscles of the head. Despite this similarity, there is much difference in the descriptions of the conditions; some authors describing no alterations in the skull bones, whilst others report the conditions to be primarily a problem of either diminished, or alternately, excess bone growth, with the muscular wasting occurring secondary to this problem.

"Affected" head

When the Vizsla was new to Australia, "affected" head was reported as occurring. The condition was reported in very young animals, or it became apparent in mature dogs. Wasting of the muscles occurred, and X-rays of an affected Vizsla found the bones of the head appeared normal. It was noted the appearance of these dogs bore similarity to dogs with atrophic myositis, which is now considered a form of Masticatory Muscle Myositis. (See also: Masticatory Muscle Myositis, under "Conditions which also occur in Vizslas".) Though not proven, it was generally believed the condition was inherited as a recessive trait (Harris).

Cranial Muscular Atrophy (CMA) and Head Deformity

The Vizsla Club of America Code of Ethics (CoE) states people should breed from dogs free of cranial muscular atrophy (CMA). CMA is not a term found in the veterinary textbooks, and veterinarians consulted assumed one was referring to Masticatory Myositis (see: Masticatory Muscle Myositis under "Conditions which also occur in Vizslas"). The Vizsla Club of America lists CMA and masticatory myositis as separate muscle problems in its health survey (Vizsla Club of America health survey). The only references to CMA which were found, were in the CoE of the Vizsla Club of America, and in the various regional Vizsla clubs in America which have adopted their parent body CoE.

In enquiring about this condition, it was stated this occurs from birth. This contradicts the term "atrophy", as by definition, this implies the muscle was normal, but has now wasted. "Hypoplasia" describes

impaired development. However it would appear the term is even more confusing: an article written by Rod Hitchmough, entitled "Head deformities in the Vizsla" was printed in the November/December 1988 Vizsla News (Vizsla Club of America newsletter), and reprinted in the Vizsla News in July 1993. This article pertains to the condition that is understood to be CMA, as specified in the Vizsla Club of America CoE. The reprint is prefaced with; "*It refers to a condition in which some of the bones of the skull do not grow, and the cranial muscles atrophy, leaving the skull to become more and more misshapen as time passes*." This preface is saying CMA is primarily a bone problem, with muscle atrophy occurring secondarily to this. Yet the term "cranial muscular atrophy" doesn't imply bone involvement, but purely a muscular problem, which is reinforced by the Vizsla Club of America listing CMA under 'muscle problems' its health survey. The article itself does not address what the condition is, or at what age it can be expected to be observed. Hitchmough supports the view expressed by Boggs, and by Australian and New Zealand sources that the condition is attributable to a simple recessive gene. If this were the case, breeding two affected dogs together could only produce affected offspring. If any such matings had been undertaken, their occurrence, and their results, were unknown (Hitchmough). Boggs recounts that Hitchmough had the head of an affected dog radiographed, finding the problem to be one of reduced muscle mass, and not of bone (Boggs 2nd rev), as had been done in Australia (Harris). Given the discrepancy between this information and the preface to Hitchmough's article on head deformities, it would appear the results of the x-rays which Hitchmough had performed were not communicated widely, or were not done so until after the article was published.

To further confuse the question of whether CMA as referred to by the Vizsla Club of America, is believed to be a problem of muscular atrophy, or of abnormal bone development with secondary muscle wasting, their health survey lists "head deformity" under 'bone problems' (Vizsla Club of America health survey). The only disorder affecting growth of the bones of the head that is described in orthopaedic texts (eg Whittick) is craniomandibular osteopathy (see: craniomandibular osteopathy). This most commonly affects the bones of the lower jaw, and is a problem of excess growth, as opposed to the lack of growth described above.

Apart from the confusing use of the term, "cranial muscular atrophy", and that there is no disease known by this name in the veterinary texts, what the Vizsla Club of America does not address is that atrophy of the muscles of the head can occur due to a number of conditions, such as masticatory myositis, injury to the nerve supplying the muscle, or to idiopathic trigeminal neuritis. Atrophy of muscle does not occur without an underlying cause, but is the visible manifestation of another disease process. For example a dog may limp because it has jarred a toe, cut a pad, pulled a ligament, because it has hip dysplasia, etc – "limping" is a symptom of an underlying problem, not the cause. Similarly, atrophy of muscles, except where due to lack of use, is a sign of an underlying problem of either the nerve or muscle. This is not to say there is no underlying hereditary component in Vizslas which results in atrophy of the muscles associated with eating. However, the other known conditions may erroneously be being lumped under the term 'CMA' without due investigation.

Skull defects

Two American authors of Vizsla books have listed conditions affecting the bones of the head as hereditary problems of Vizslas. Coffman mentions "skull defects", however no description is given. As skull is the bone and not the muscle, one assumes this is either the condition referred to as "cranial muscular atrophy" in the CoE of the Vizsla Club of America (see above), or is the condition referred to initially by Boggs (Boggs, rev). Boggs describes affected dogs to have enlarged bones above the eyes, and for the bones to project above the level of the skull. The disorder was reported to occur in some lines, and believed to be inherited as a recessive characteristic (Boggs, rev). [Note this is in contrast to the description included in the preface to the article on head deformities describing CMA, where there is a lack of bone growth.]

In his latest edition, Boggs revises his observations; "*On closer inspection it is found that the temporal and masseter muscles are atrophied causing these bony prominences to become exaggerated. Evident by the time the dog reaches six months of age, the top of the head has a ridge that is not flattened by the presence of muscle*." Incidence of this disorder was increasing in certain lines, and Boggs believed it to be either a recessive trait, or a dominant gene with incomplete penetrance. Boggs reports that in the early 1970s incidence of this skull defect in Vizslas in Australia and New Zealand was high, but with importation of new dogs, fewer cases were observed. Boggs also reports that test breedings established the condition to be inherited as a simple recessive trait. In contrast to this revision, the chapter illustrating conformation of the Vizsla includes a badly formed head which Boggs attributes to enlarged frontal bones, and not to atrophy of the muscles (Boggs 2nd rev).

Masticatory Muscle Myositis is not listed as a condition to which Vizslas are prone, but the descriptions of these head conditions affecting Vizslas, resemble the muscular atrophy seen in the condition. Without biopsies of affected muscles, it is not known if any dogs believed to have 'affected' head, CMA or 'skull defects' may actually have masticatory myositis or neuritis (see text box for example). Masticatory Muscle Myositis and trigeminal neuritis are discussed later, under "Conditions which also occur in Vizslas".

Tail defects

Some Vizslas have been born with crooked tails, or have had a short, or stubbed tail (brachyury). The deformity is believed to be hereditary, though this has not been proven (Gottlieb 2nd). Vizslas with stubbed tails should not be bred (Boggs, rev). The condition is less common than hip dysplasia (White).

Vizsla rash

See: skin rash of puppies.

von Willebrand Disease (vWD)

vWD is the most common inherited bleeding disorder of dogs. It results from a lack of von Willebrand's factor, either because insufficient is produced, or what is made is dysfunctional or unstable. The mode of inheritance of vWD is not the same for all affected breeds of dog, and for some breeds, a DNA test is available for diagnosis. vWD is reported as occurring in Vizslas, but the breed is not one where prevalence is high (Ackerman, White). The Vizsla Club of America's CoE requires breeders do not breed from dogs affected with vWD. In Australia, all the ELISA testing for von Willebrand's antigen is performed at one laboratory, and no Vizslas have been found to be affected (University of Melbourne).

XX Sex reversal

This uncommon condition has been reported in only 16 breeds of dog, the Vizsla being one of them. In this condition, although the dogs lack a Y chromosome, testis tissue develops. The Vizsla in which this condition was reported was a true hermaphrodite. In American Cocker Spaniels XX sex reversal has been found to be inherited as an autosomal recessive trait, and the condition is familial in at least six other breeds (Meyers-Wallen et al, Meyers-Wallen).

Conditions which also occur in Vizslas:

Note: no texts considered these conditions to be one where Vizslas were predisposed. Information about incidence of cranial cruciate ligament injury, and otitis in the breed are presented because they were available. Masticatory myositis and trigeminal neuritis are included given their similarity to other previously discussed conditions which are listed as being hereditary in Vizslas.

Cranial cruciate ligament rupture

Vizslas were not considered a breed at increased risk of rupture of the cranial cruciate ligament (CCL). In a referral population of 1,150 Vizslas, 22 were diagnosed with CCL rupture (around 2 in every 100 Vizslas.) The data was based on records over almost 20 years from 23 veterinary medicine teaching hospitals in North America (Whitehair).

Masticatory muscle myositis (eosinophilic myositis, atrophic myositis)

Hungarian Vizsla with masticatory muscle myositis

This inflammatory muscle disease affects the muscles of the head involved with mastication (chewing) – the temporalis, masseter and pterygoid muscles – which operate to open and close the jaw. The conditions 'eosinophilic myositis', 'atrophic masticatory myositis' and 'cranial myodegeneration' are now generally believed to be phases of the same disease process (Day, Podell, Taylor, Whittick). Braund (Braund (b)) discusses 'masticatory myositis' separately from 'atrophic masticatory myopathy/myositis' as he considers atrophic myositis *"may be a stage of masticatory myositis or it might represent neurogenic atrophy secondary to idiopathic trigeminal neuritis."* Masticatory muscle myositis (MMM) can occur as an acute or a chronic condition. In the acute disease, swelling of the muscles is observed. Chronic MMM is more commonly recognised, and otherwise normal healthy dogs present with atrophy (wasting) of the muscles at the top of the head. MMM can be seen in dogs of any breed, with some authors reporting a higher incidence in large breed dogs, with Day reporting the chronic atrophic type of MMM being more commonly seen in long-nosed breeds. The condition can affect dogs of any age, with some authors reporting increased incidence in young dogs. What causes MMM to occur is not known, but it is an autoimmune disease, as the body's immune system attacks the masticatory muscles. These muscles have a different embryologic origin and they contain a different type of muscle fibre than other muscles of the body. In this disease, antibodies against these particular different fibres are formed, and the other muscles of the dog are not usually affected (Braund (b), Day, Podell, Shelton, Taylor, Whittick).

Idiopathic trigeminal neuritis (or neuropathy) may mimic MMM. In this condition inflammatory processes involve the nerve (Braund (b), Day, Podell, Shelton, Taylor, Whittick). In several instances, tissue from