

PROCEEDINGS

**22th annual meeting of the
INTERNATIONAL ELBOW WORKING GROUP**



Elbow Dysplasia Film Reading Session

**Saturday September 8th, 2007
Munich, Germany**

WELCOME ADDRESS

Dear participant,

The Board of the International Elbow Working Group (IEWG) welcomes you in München, at the Elbow Dysplasia Scrutineers seminar. The IEWG is an affiliate of the World Small Animal Veterinary Association, the world wide association of companion animal veterinarians, and has close contacts with the Board of the Federation Cynologique International, the world wide organisation of dog-breeder clubs. The IEWG was founded by a group of veterinarians and breeders with concern regarding different forms of hereditary elbow dysplasia in 1989 in the USA. The working group evaluated over the years in a group of professionals with special interest in elbow dysplasias on their etiological, population and molecular-genetic, as well as clinical and radiological point of view.

The FCI and IEWG advocate screening of the breeding stock as well as of their offspring of breeds at risk, and both emphasize the importance of a uniform screening method in the different countries.

The IEWG has its annual meeting often in conjunction with the world congress of the WSAVA, but has decided to organise in 2007 a course for ED scrutineers instead, as it did in 2005. At that first training course for scrutineers and other interested veterinarians participants of 22 different countries were present.

At this years course, participants will be informed about the aetiology of the different forms of ED, the systematic approach of screening according to IEWG, and the radiological techniques and added value of different views, followed by an exercise in evaluating radiographs according to the IEWG-protocol, with eventually a general discussion of the cases. This programme will be completed with examples how different European countries organised their ED-screening programmes and which results has been reached so far for different breeds.

The IEWG is very grateful to the Board of the European Society of Veterinary Orthopaedics and Traumatology (ESVOT) and the hosting Clinic for Companion Animal Surgery of the Ludwig-Maximilians-University with the head Prof. Dr. U. Matis, for their hospitality allowing IEWG to give again its work shop in this most elegant environment with the efficient organised ESVOT-congress and its smooth infrastructure, grace to the ESVOT-congress committee and the employees of Congress & Seminar Management (CSM).

The IEWG is happy to welcome you at this interactive workshop on radiological screening for elbow dysplasia in a uniform way according to the guidelines of the IEWG, as has been accepted by the FCI and in use in different countries already.

I welcome, also on behalf of the two other IEWG-board members Dr How and Prof Tellhelm, the input and personal experiences of the participants and invite them to take radiographs with them for discussion. We thank Prof. Flückiger and others for their kind cooperation to give an interesting seminar and a fruitful course.

Prof. Dr. H.A.W. Hazewinkel
Chairman IEWG

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Animal Health

PROGRAMME IEWG ELBOW DYSPLASIA FILM READING SESSION

Morning session

- 9.00 – 9.10 opening and welcome
- 9.10 – 9.45 Elbow dysplasia, definition and known aethologies Prof. Dr. H.A.W. Hazewinkel
- 9.45 – 10.45 Scoring for primary and secondary lesions Prof. Dr. M. Flückiger
- 10.45 – 11.15 coffee break
- 11.15. – 12.00 Screening according to IEWG Dr. B.Tellhelm
- 12.00 – 13.30 Lunch break

Afternoon session

- 13.30 – 14.30 Film reading wet lab: Representative radiographs will be studied and scored in small groups
- 14.30 – 14.45 Tea break
- 14.45 – 17.00 General discussion of projected copies together with all participants. A final arthrosis score, a score of primary lesions and possibly other scorings will be discussed and explained; results can be notated in the personal printed copy of the radiographs, serving as a guideline.

List of speakers

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Elbow dysplasia, definition and known aetiologies

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Introduction

Elbow dysplasias (ED) occur frequently in 4-6 months old dogs of medium and large body size, during the period of high growth velocity. Since developmental skeletal diseases, either due to genetic disease or due to nutritional influences or trauma, are frequently seen in this category of companion animals all three can be held responsible for the occurrence of ED. It is known that each form of ED will lead to osteoarthritis (OA) with possibly severe consequences for the well-being of the animal and its owner. Therefore great effort has been undertaken by different research groups to elucidate the etiology of ED to come to guidelines how to prevent their occurrence. First the definitions will be given of the primary entities of ED as well as of the different grades of osteoarthritis (OA). Second, the different aspects involved in these heritable elbow diseases will be reviewed.

Definitions

Originally the term “elbow dysplasia” or “*dysplasia articulationis cubiti*” covered generalized osteoarthritis of the elbow joint with an ununited anconeal process (UAP) (Corley et al, 1968). Now UAP is just one of the different entities which are covered by the term “ED”, which is here defined as the group of elbow dysplasias including 1) ununited anconeal process (UAP), 2) fragmented medial coronoid process (FCP), 3) osteochondrosis (OC) or osteochondritis dissecans (OCD), and 4) incongruity (INC) of the elbow joint. These four entities have in common that they all occur in the elbow joint (although OCD occurs also in other joints), that they are all seen in young growing dogs (although they can be overlooked) of medium and large size, that they can cause lameness (but not in all cases and not for the first time only in young dogs), and that they will cause osteoarthritis (but that can vary per individual dog and perhaps even per breed).

Primary lesions

These lesions can be graded as absent (ED grade 0), suspected-present (ED grade II) or present (ED grade III). We distinguish the following primary lesions:

1. UAP: Separation in the cartilaginous bridge between the secondary ossification centre of the anconeal process and the olecranon, resulting in (can cause a) partially or completely detached anconeal process, referred to as ununited anconeal process (UAP)
2. FCP or MCPD (= medial coronoid process disease): Fissuring of the medial coronoid process of the ulna with partial to complete separation (fragmentation) of the medial coronoid process from the ulna; primary a subchondral bone lesions with secondary cartilage changes (Guthrie et al, 1992), although also *chondromalacia* at the medial coronoid process is considered part of this entity.
3. OC: Local thickening of growing epiphyseal cartilage with delayed endochondral ossification, which may develop into OCD with a single or fragmented detached cartilage flap.

“Kissing lesion”: An abrasion of the articular cartilage, sometimes extending into the subchondral bone (radiologically often slightly more lateral than the OC-lesion), and here caused by a fragmented coronoid process (Morgan et al, 2000). This finding is graded as a OCD-like lesion.

4. Elbow incongruity (EI, INC): The subchondral bone of the trochlear notch of the ulna and of the radial head are not parallel to the opposing humeral subchondral bone. There are different forms of EI:

The radius longer than the ulna with a narrowing of the joint space between the tip of the anconeal process and the humeral condyle, a distally gradual widening of the joint space between the ulnar semilunar notch and the humeral condyle and the radial head proximal of the coronoid process of the ulna

The longer ulna with a wider joint space between the proximal radius and the humeral condyle and the step between the more proximally located distal edge of the ulnar trochlear notch (i.e., the lateral coronoid process) and the radial head (and displacement of the distal humerus cranially). This can also be considered as an underdeveloped or too small trochlear notch.

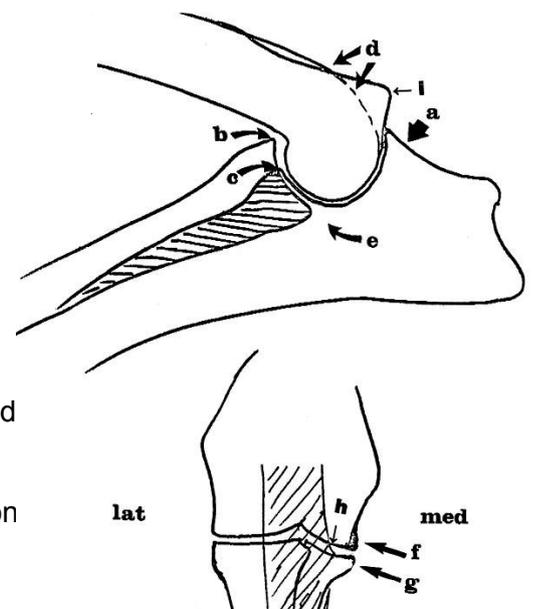
The alignment between the subchondral bone of the trochlear notch and the radial head is more elliptical than the circular contour of the humeral condyles described by Wind (1986) Developmental elbow luxation with lateral displacement of the (often hypoplastic) radial head with a comparative overgrowth of the radius (as seen in chondro-dysplasia in non-chondrodystrophic breeds)

5. Osteoarthritis is radiologically characterized by new bone formation at the edges of the joint. In addition, enthesophytes (i.e. new bone formation at the sites of attachments of tendons, ligaments, and joint capsule, resulting from abnormal tension placed on the soft tissue attachments near the joint margins) can be formed.

Regardless of the primary cause, the pattern of OA is similar. The different locations where osteophytes and enthesophytes are visible in case of OA are given in Fig. 1.

Fig. 1 Locations for grading of elbow OA

- a. the proximal surface of the anconeal process
- b. the cranial aspect of the radial head
- c. the cranial edge of the medial coronoid process
- d. the caudal surface of the lateral condylar ridge
- e. sclerosis of the ulnar notch, at the base of the coronoid
- f. on the medial surface of the medial epicondyle
- g. at the medial edge of the medial coronoid process
- h. indentation of the subchondral bone: OCD (-like) lesion



Grading of Elbow Osteoarthritis (OA)

Borderline OA (B) can be defined as increased radiographic density (sclerosis) in the ulna caudal to the trochlear notch. In addition, minimal changes at the dorsal border of the anconeal process which is considered as a normal edge and grouped under border line. This can be scored separately or as Grade 1 (see table 1)

Grading definitions:

Grade 0 OA: no signs of osteophytosis or osteosclerosis

Grade I OA: When at any of the locations listed a – i. osteophytes are present of < 2 mm, or presence of osteosclerosis

Grade II OA: When at any of the locations listed a-i osteophytes are present of 2-5 mm.

Grade III OA: When at any of the locations listed a-i osteophytes are present of ≥5 mm.

Table 1 Scoring for ED making use only of the OA-score

Scoring for Elbow Dysplasia		Radiographic Findings
0	Normal elbow joint	Normal elbow joint, No evidence of incongruency, sclerosis or arthrosis
1	Mild arthrosis	Sclerosis of ulnar trochlear notch and/or Step > 2 mm between radius and ulna and/or Presence of osteophytes < 2 mm high
2*	Moderate arthrosis	Presence of osteophyte 2 - 5 mm high
3*	Severe arthrosis	Presence of osteophytes > 5 mm high

(B) "Borderline" between score 0 and 1 is allotted to undetermined cases reflected as minimal joint remodelling (in some countries).

In several countries the presence of a primary lesion such as UAP, FCP, OCD, or INC of > 2 mm, automatically results in a ED score 3; the *suspicion* of primary lesions results in a ED score 2 (see table 2).

6. Sclerosis.

An alteration in normal bone architecture, i.e., a decrease in normal bone porosity, is depicted on a ML view of the elbow joint as an increase on bony opacity with loss of trabecular markings (a white area), in the trochlear notch just caudal to the lateral coronoid process. Osteosclerosis is considered as one of the first signs of ED in young dogs, especially when the primary cause can not be identified as in some cases of FCP. This area can be compared with a control radiograph of the non-affected elbow in case of unilateral ED. However, since FCP often occurs bilaterally, the use of the opposite elbow joint will not be of help. In a survey with 17 Labrador retrievers (6-16 months of age) with FCP and 17 without FCP as diagnosed by arthroscopy, radiographic density was objectivated and expressed as pixels: an extremely significant correlation between pixel intensity of the projection of the lateral coronoid process revealed in dogs with FCP (Burton et al, 2007). Microscopically, this area is characterised by reduced intertrabeculae

spaces (Wolschijn et al, 2004), either due to mechanical overloading or influence of MMPs, enzymes which play a role in osteoarthritis.

Imaging techniques

Radiographs play a major role in the diagnosis of ED, both in a clinical setting, to determine the phenotype in case of (DNA-)research as well as in screening the population. "More views will give more insight" counts also true in case of radiological investigation, especially in case the primary lesion is of importance to know. According to a large study in 447 Bernese Mountain Dogs by Lang et al (1998), 12% of them had a primary ED without OA yet. Therefore, screening for ED in Bernese Mountain dogs should include at least two perpendicular views. This seems especially true in breeds where OCD is anticipated to be the primary cause of ED (table 3).

In case the secondary signs only are of importance, a limited number of views can be sufficient. In addition to the ML and AP views, other views have been developed including ML view with 15 degree supination (exorotation) of the antebrachium (Voorhout et al, 1987), distomedial-proximolateral oblique view (Haudiquet et al, 2002). A FCP can be detected more accurately using computerized tomography (Tromblee et al, 2007), possibly ultrasonography (Knox et al, 2003), and certainly magnetic resonance imaging (MRI) (Snaps et al, 1997). The latter revealed the highest accuracy (95.5%), sensitivity (100%), and negative-predictive value (100%) for mineralised FCPs and on average 10% less for non-displaced, non-mineralized FCPs (Snaps et al, 1997), whereas CT revealed a accuracy of 86.7%, a sensitivity of 88.2% and a negative predictive value of 84.6% (Carpenter et al, 1993). Linear tomography and arthrography did not add much to findings of plain radiology (Carpenter et al, 1993). In case of occult cause of lameness, bone scintigraphy can point to the area of bone pathology including FCP with high sensitivity without invasive technique (Schwartz et al, 2004)

Table 2. The 'Osteoarthritis (OA) score' schedule plus the 'Primary cause' schedule help to come to the 'final ED score according to the IEWG'.

Osteoarthrose

OA	0	I	II	III
Localisation (fig.1) \ mm	0	<2	2-5	>5
a				
b				
d				
f				
g				
Architecture				
c	-		+	
e	-	+		
OA score				

Primary cause

ED	0	2	3
	-	±	+
UAP			
FCP			
OCD(-like) [h]			
INC			

Final ED score according to IEWG

O	NORMAL
	INC > 2 mm Osteophytes < 2 mm Sclerosis
1	Osteophytes 2-5 mm Suspect (±) of primary cause
2	Osteophytes 2-5 mm Suspect (±) of primary cause
3	Osteophytes > 5 mm Positive for primary cause

Etiologies

Genetic influences

Purebred dog populations represent genetically closed populations, in which high selection intensities and subsequent high levels of consanguinity are common. When only few of the members of the breed (e.g. mainly the champions) are used for reproduction, then a genetic bottle-neck is created unintentionally but surely, reducing the genetic heterogeneity. It is to be expected that this selection procedure, which is common place in many breeds, may lead to increasing incidence of genetic diseases when the selected breeding stock by chance carried the genetic risk factor for ED or any other genetic disease. When the genetic risk factor has a dominant inheritance pattern leading to clinical signs before breeding age, then the dog and its parent(s) can be discarded from reproduction, like in chondrodysplasia (Carrig et al, 1988). But in case the genetic risk factor has a recessive or polygenetic inheritance pattern, has a variable pattern in penetration, or is based on a genetic diseases with a high influence of environmental aspects then, especially when manifest at older age, the entity has all chances to spread around in the population before being recognised. This is especially so, when there is a lack of adequate disease registration. In such diseases, the spread of the disease allele can be considerable before these diseases are recognised as genetic diseases within certain breeds (Ubbink 1998, Patterson et al, 1989).

Although ED occurs in well described breeds (Table 3), not each form of ED is seen in all of them:

Incongruity of the elbow joint (INC) can be caused by a variety of causes as clearly reviewed by Samoy et al (2006) and Gemmil et al (2007). Some incongruity of the joint is considered as normal, with the radius bearing 51-52% of the weight (Mason et al, 2005). INC due to an ulnar overgrowth is seen in 80% of the Bernese Mountain dogs with osteoarthritis in the elbow joint. In a survey of a large group of Bernese Mountain dogs this type of incongruity was seen in all cases with elbow lameness in conjunction with a fragmented coronoid process (Ubbink et al, 1999). Population analyses revealed that the disease was introduced right after WW II by a limited number of founding fathers into the breed.

Fragmented coronoid process of the medial aspect of the ulna is seen in many breeds and in large percentages, up to 50% of the screened population (Svenson et al, 1997, Ubbink et al, 1999). The heritability estimates (h^2) are between 0.24-0.43 for Bernese Mountain Dogs, 0.77 for Labradors and 0.45 for Golden Retrievers (Guthrie and Pidduck, 1990). For Retrievers, these figures are for osteochondritis dissecans of the medial humeral condyle (OCD) plus FCP, and thus found to be polygenetic in addition to multifactorial (Padgett et al, 1995), although there is enough evidence to conclude that FCP and OCD are two different, independently inherited entities (Padgett et al, 1995, Janutta et al, 2006). For FCP and OCD alone, h^2 has not been calculated yet.

The united anconeal process (UAP). It is seen in chondrodystrophic breeds (like Bassets) and as part of elbow incongruity in certain breeds (German Shepherd, St Bernard) as well as due to nutrition or traumatic induced radius curvus syndrome (Hazewinkel, 1998). Although often not easy to recognise, in dogs with an UAP a FCP can coincide as was reported by Meyer-Lindenberg et al (2006) in 16% of the cases of UAP. In a survey in 520 German Shepherd dogs in France, FCP was diagnosed in 11.3% of the cases, INC in 16.3% and UAP only in 1.1% of the dogs (possibly due to pre-screening), whereas combinations were seen in 42.2% of the joints with ED (Remy et al, 2004).

Table 3 Breed susceptibility for 3 entities included in Elbow Dysplasia

Fragmented coronoid process	OCD	UAP
Bernese Mountain Dog (140)	Newfoundland (261)	Bernese Mountain Dog (50)
Irish Wolfhound (93)	Rottweiler (174)	Rottweiler (27)
St Bernard (53)	Labrador (109)	Mastiff (20)
Mastiff (48)	Great Dane (87)	Newfoundland (14)
German Shepherd (44)	Golden Retriever (42)	Labrador (9)
Bullmastiff (39)		German Shepherd (8)
Rottweiler (36)		Golden R. (5)
Labrador (20)		

odds ratios given in brackets (LaFond et al, 2002)

When different entities of ED are not differentiated, ED has been proven to be polygenetic (Kirberger et al, 1998). It has been shown in Swedish breeding programmes that based on massive screening for ED and open registration of the results, the prevalence of OA due to ED decreases due to a decrease in incidence of the primary causes of OA (Swenson et al, 1997). Cluster analysis of one breed, using computerized programmes containing all pedigrees of the investigated population, reveals histograms representing a group of related dogs with 1/8th of the genome in common. We evaluated Labrador Retrievers, Bernese Mountain dogs and Golden Retrievers in groups, non-selectively chosen from the Dutch population and representative for that population. All dogs were screened radiologically for FCP on 4 radiographs according to the technique described by the IEWG on their web page. Certain related groups revealed members positive for FCP in 27- 50% of the investigated dogs. These positive dogs are spread over the country so the environment (i.e., housing, rural vs town area) are different in many cases. In Labradors the affected groups were quite related and less related with non-affected groups, whereas in Bernese Mountain dogs all groups of 1/8th-related dogs were affected with ED and all groups were connected which each other at the 8th generation (born just after 1945); in Golden Retrievers ED was seen frequently in related groups and these groups were related with each other, although to a lesser grade as was seen in Labradors (Dijkshoorn et al, 2005). Although this method does not show the inheritance pattern, it is proven to be an effective method for persuading kennel clubs to take measurements like the obligation of screening before breeding.

DNA-analysis focussed on collagen candidate genes in Labradors with FCP, did not reveal any indication of the involvement of these candidate genes in this skeletal disease. Genome wide scan, using 300 polymorphic markers was expected to be more promising with the possible abnormal locus at the first and thirteenth chromosome, although it did not indicate affected alleles yet (Salg et al. 2006). Linkage analysis (i.e., determining the region of the genome containing the disease locus) limits the studies of candidate genes. Fine mapping might lead to the affected allele. Since FCP is unique in the canine species, it is unlikely that alleles mapped in other species may be of help. High density mapping and association studies may stimulate the molecular genetic studies in elbow dysplasia. Eventually, it is to be expected that DNA-analysis of the population will detect dogs with the affected gene(s) which did not express the disease (due to optimal environmental circumstances) or are heterozygous for the disease. DNA-analysis of the potential breeding stock will fore come a lot of frustration for breeders who now experience affected offspring of phenotypically normal parent dogs and thus a slow decrease of the incidence of these hereditary diseases in next generations.

Environmental influences

The expression of hereditary diseases can be influenced by environmental factors; like in hip dysplasia in German Shepherds where the heritability estimates were $h^2=0.3$, demonstrating the high influence of environmental factors (Distl et al, 1991). The h^2 for ED in Rottweilers (which may include all possible entities of ED, see Table I) is 0.31 ± 0.04 with detectable improvement in case of a breeding programme (Mäki et al, 2000). Not too much is known of heritability estimates for FCP and other EDs (see 'genetic influences'), but the h^2 seems to be higher, i.e., environmental influences are lower in ED than in HD. The penetrance of FCP in Labradors is higher in males than in females, at least 2:1 in Labradors (Salg et al, 2006) or in mixed populations (Carpenter et al, 1993). From the heritability estimates of <0.5 , it can be concluded that other factors may play a significant role in the manifestation of ED. From different studies it became clear that breeding with ED-negative dogs (based on radiological screening) will decrease the incidence of ED considerably when compared with breeding of positive x positive, or positive x negative, or negative x unknown (Svenson et al 1997). ED in Labradors (and in Golden Retrievers to a lesser extent) is seen more frequently in male than in female dogs, although it can be expected that environmental factors do not differ to such a degree between genders. We calculated that, in case ED follows the hereditary pattern of a variability in expression of a major, dominant gene, the penetration of FCP in male Labradors is 70% and in female dogs is only 28%. In other words, in a Labrador with the disease allele for FCP, this disease is expressed in 70% of the males and only in 28% of the females, thus a phenotypical negative female has a higher chance to pass the affected genes to the next generation than a negative male Labrador, even when screened carefully. For breeders this is important information, since it warrants not only the screening of the breeding stock, but also of related animals (i.e. littermates) and offspring, which might tell more about the genetic make-up of a particular dog than the radiograph of its own elbow joints.

Nutritional influences

From a variety of studies it became clear that nutrition has a major impact on skeletal development. Food with a high calcium content has proven in field studies (Slater et al, 1992, Kallfelz & Dzanic, 1989) as well as in standardised laboratory circumstances (Schoenmakers et al 1999, Schoenmakers et al 2000) that high mineral intake will cause disturbances in endochondral ossification. This makes the skeleton more vulnerable to mechanical influences like overweight as well as to OCD lesions and possibly chondromalacia. More recently, it became clear that vitamin D intake increased to a level that it will not lead to hypervitaminosis D (with calcification of soft tissues), will also disturb endochondral ossification by direct influence and not by increasing intestinal calcium absorption (Tryfonidou et al, 2002). High food intake and thereby excessive calcium and vitamin D intake does also lead to osteochondrosis (Lavelle 1989, Hedhammar et al, 1974). High calcium or high vitamin D intake will cause retained cartilage in growth plates and thus a disturbance of growth in length of the fast growing growth plates, in particular of the distal ulna and distal radius. Disturbance of growth in length may lead to radius curvus syndrome or short radius syndrome, respectively. Elbow incongruity in case of radius curvus syndrome may coincide with UAP, whereas incongruity with a shortened radius may coincide with FCP.

Secondary ossification centres (like the anconeal process) ossify via the process of endochondral ossification, whereas the coronoid process develops exclusively by appositional ossification (Breit et al, 2004). In large breed dogs the completion of ossification takes longer than in small breed dogs, for the coronoid process this is 20 weeks vs. 14 weeks of age, respectively (Breit et al, 2004). Dogs raised on food with a calcium excess have disturbed endochondral ossification and will take more time to complete the ossification process (Voorhout et al, 1985), despite undisturbed increase in body weight, which makes the softer cartilage protuberances more vulnerable to damage.

Nutritional imbalances (like calcium or vitamin D excess) will not cause skeletal disturbances in dogs which are not genetically predisposed to develop these disturbances (Nap et al, 1993).

These findings are of great value for owners of a single dog, who want to prevent ED to develop in their pet by providing an optimal environment to mature. A high quality dog food prepared particularly for puppies of large breeds should be provided, characterized by a lowered calcium content (~1.0% calcium of dry matter base) and a controlled vitamin D content (~500 IU/ kg food). It has been shown that an increased protein level of high quality, typical for the better puppy diets, does not have a negative influence on skeletal development whereas it is of importance for soft tissue growth and immunological defence systems (Nap et al, 1993).

In conclusion, the different entities of ED are caused by genetic and environment influences; one of the latter may be nutrition. However, dietary influences come only to expression when the genetic vulnerability is there, i.e. when the h^2 is between 0 and 1.

Mechanical influences

Traumatic injury of growth plates, especially Salter Harris type V fractures, may disturb growth in length of either the radius or the ulna, and as such may be responsible for the overloading of ulna or radius, respectively. In case the coronoid is mainly cartilaginous or the anconeal process is still separated from the olecranon by a cartilaginous layer (< 5 months of age), this can result in a FCP or UAP, respectively. Only seldom, there can be an indication of a traumatic fracturing of the anconeal process or of a coronoid process in adult dogs, while both age and history will differ from that of ED in young, fast growing dogs. Meyer-Lindenberg et al describe 263 lame dogs with arthroscopically confirmed FCP in total 332 affected joints and in 5 lame dogs a FCP without any radiological signs of OA in dogs >3 years of age; these 2% might have suffered a traumatic fragmentation of the coronoid, or the FCP has caused minimal arthritic changes (Meyer-Lindenberg et al 2002).

Not too much is known yet about the influence of loading on skeletal development in dogs. It is known that unloading will cause both disuse osteoporosis as cartilage degradation, especially in young fast growing individuals. Some of most compelling evidence that supports a causal relationship between cartilage function and form comes from animal experiments in which the joint loading is either increased or decreased above normal levels. Increasing the functional loading of joints through moderate exercise causes an increase in articular cartilage thickness, proteoglycan content, and mechanical stiffness of the tissue, though strenuous exercise can lead to the formation of cartilage lesions (Wong & Carter, 2003). In a large, well controlled study in fowls it became clear that a functional adaptation of joint cartilage to weight bearing occurs during the first months of life and is important for the development of resistance to injury during later life (Brama et al, 1999). Immobilization or other means of joint unloading has led to a thinning and softening of the uncalcified part of articular cartilage, an increase in subchondral vascular eruptions, and a decrease in proteoglycan content. The structural and biochemical changes associated with joint unloading can only partially be reversed when the joint is remobilised. Physiologic joint loading results in functional adaptations that increase the resistance of the cartilage and are beneficial to the overall health of the tissue. The areas of enriched proteoglycan content are logically the areas most resistant to the degenerative changes that beset a joint during osteoarthritis (Wong & Carter, 2003).

Further research is needed to learn more about the optimal weight bearing or training activities of young, fast growing dogs to develop optimal functional adaptation. Some joint incongruity is considered as normal in non-arthritic elbow joints, increasing with increasing body weight (Janach et al, 2006). However, joint incongruity as seen in Bernese Mountain Dogs, with constant overloading of the remaining weight bearing surface, (i.e., the contact area of the humero-ulnar joint), can hold responsible for the fragmentation of the apex of the medial coronoid process. Bernese Mountain dogs with a FCP without INC can be

seen only in 20% of the Bernese with ED. Based on the findings of Gemmill et al (2005), it can be assumed that the radius is too short rather than the ulna too long, although it can not be excluded that the ulnar notch is underdeveloped (i.e., too small) in relation to the humerus. The cause for FCP in Labradors, Golden Retrievers and Rottweilers, characterized by a fragmentation of the coronoid process at the radio-ulnar joint is still unknown, however the anatomical study of Wolschrijn & Weijts (2004) in coronoid processes of Golden Retrievers pups may give an indication. An anisotropic structure of the trabeculae with an orientation in the direction of the proximodistal axis of the ulna is already present at 6 weeks after birth. This primary alignment is perpendicular to the humeroulnar articular surface, matching the direction of the compressive forces applied to the medial coronoid process by the humeral condyle. The secondary alignment appears at 13 weeks after birth and is directed along the cranio-caudal axis of the medial coronoid process, toward the attachment of the annular ligament. Excessive pulling force of this ligament might be responsible for the fragmentation of the coronoid process in Retrievers. Vulnerability of the growing bone at the site of the coronoid process along the split lines is also supported by the work of Künzel and co-workers (2004).

Danielson et al (2006) describe the presence of direct and indirect indications of microdamage in subchondral (trabecular) bone in the medial coronoid area of elbows of dogs with FCP, which coincided with loss of osteocytes and their lacunocanalicular network and thus their nutrition and intercellular communication. The FCP self revealed porosity whereas the surroundings reveal osteosclerosis. Wind (1982) Ubbink et al (1999), Collins et al (2001) and Gemmil et al (2005) are supportive of the findings of Danielson et al (2006) in that they all report on the strong association between joint incongruity and FCP. A decreased radius of the ulnar notch is hold responsible for FCP by Wind (1982) although this is questioned by others (Preston et al, 2000; Maierl et al, 2000)

In any case, it is very unlikely that normal weight bearing even during playing is responsible for the FCP frequently seen in particular breeds. The osteosclerosis can although express an abnormal micro-architecture of the joint caused by shearing forces with fragmentation of bone fragments as a result.

Conclusion

In summary, elbow dysplasias (including UAP, FCP, OCD and INC) could spread among certain dog breeds, due to the (over)use of a limited number of breeding dogs affected with a disease allele which did not come to expression in all cases. Based on the heritability estimates as published in veterinary literature, environment may play a role in cases the genotype comes to expression. Since dietary intake of calcium and vitamin D may cause disturbances in endochondral ossification and thus may play a role in the occurrence of UAP, FCP, OCD and INC, unbalanced diets or excessive food (and thereby mineral) intake should be avoided. Although trauma may play a role in the occurrence of FCP along the split lines in possibly young skeleton with delayed modelling, the preventive or causative influence of physical activity or over-use on elbow joint development in dogs is still largely unknown. When the animal is not genetic at-risk, these environmental factors (like diet and micro-trauma) will not play a significant role in the occurrence of ED.

Screening of elbow joints to decrease the incidence of ED in the breeding stock and its offspring, is advocated by many researchers, kennel clubs, as well as by the FCI, OFA, IEWG and other umbrella organisations (Swenson et al, 1997, Kirberger et al, 1998). The quality of the radiological investigation (radiographs and scrutineers) as well as breed, age and sex of the animal will influence the success of a breeding programme for ED (Mäki et al, 2000). Only supranational and open registration of well defined diseases entities and breeding measures based upon the findings of this screening will decrease the incidence

of elbow dysplasias. In the future DNA-screening techniques may lower the incidence of ED even further.

Standardisation for ED screening within a country, in Europe or preferably within a breed world-wide, will facilitate comparisons of screening-certificates and the quality of the breeding stock. (See for a concept ED-form the last page of these proceedings).

An evaluation of genetic counselling service in one dog breed revealed that breeders tended to rank exterior phenotypic characteristics first and reports from the counselling service on four frequent occurring diseases second. This indicates the need for better communication to the breeders world to inform about the function and advantages of selected breeding and the responsibility of breeders and their associations to improve the quality of life of the dogs they breed (Traas et al, 2006)

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Elbow Dysplasia: Correct Radiographic Technique and Film Interpretation

(Official IEWG - Requirements for the Screening Procedure)

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Radiographic technique

- Minimal age for routine screening is 12 months. Some breed-clubs ask for older animals. Growing dogs with forelimb lameness always need elbow radiographs, regardless of their age!
- Rare Earth screens with a speed of 200 or less are recommended.
- The elbow is placed directly on the cassette, no grid is necessary for the examination (radiographs of very large dogs may profit from using a grid).
- Each elbow is radiographed separately, the beam is collimated, which improves image quality.
- Both elbows are radiographed.
- The mediolateral projection is taken with the elbow in flexed position (45° opening angle).
Focus on concentric superimposition of the medial and lateral part of the humeral condyle. To highlight the MCP a mediolateral 15° oblique view with the elbow extended and 15° supinated is recommended. Good results are achieved with a setting around 60 kV and rather low mAs (mAs product depending on film-screen-system and focus-film-distance).
- A craniocaudal view with 15° limb pronation (and, if possible, 15° beam angulation in proximal direction) is strongly recommended to check for OC/OCD lesions. (In the UK a fully extended mediolateral view is requested instead.)
- Radiographs are permanently marked including the date of the examination, the identity of the dog, the identity of the owner of the dog and the clinic taking the films.

Screening procedure

- Radiographs for elbow disease are screened by qualified persons, preferably board certified radiologists (ACVR, ECVDI) or small animal surgeons (ACVS, ECVS). An open list of qualified persons has been filed at the FCI office by the advisory panel of the scientific committee of the FCI.
- If the radiographic findings are ambiguous, a repeat examination is indicated after 3 months.
- A possibility for appeal prior to public release of the results is provided. Appeal is based on the original radiographs and a set of new radiographs retaken not earlier than 6 months after the initial set. Both elbows are re-radiographed and submitted regardless of the elbow in debate.
- Results of the evaluation are open to researchers, dog owners and breeders.
- Radiographs will be archived at an appropriate location for 10 years.

Film Interpretation

Radiographic findings vary depending on etiology, severity, and duration of ED, and breed. A radiographic diagnosis of ED is based on presence of arthrosis and/or of a primary lesion such as

- malformed or fragmented medial coronoid process (FCP)
- united anconeal process (UAP)
- osteochondrosis of the medial humeral condyle (OCD)
- marked incongruity of the articular surface with step formation and/or subluxation (INC).

Additional findings (of various etiologies and variable relevance) such as periarticular mineralisation (mineralisation/avulsion of flexor tendons at the medial epicondyle), DJD of unknown origin or any other abnormality noted should preferably be reported as well.

Radiographic findings indicative of FCP (Fragmented Medial Coronoid Process)

Mediolateral radiograph

- Increased subchondral bone opacity (sclerosis) at the base of the coronoid processes, (loss of trabecular pattern)
 - Step of > 2 mm between radius and ulna (often in BDM, GSD)
 - Indistinct and/or shortened and/or blurred cranial edge of the medial coronoid process.
- Please note: A fragment is rarely seen!
- Reduced or patchy opacity of the medial coronoid process
 - Osteophyte formation anywhere on the elbow joint such as on the
 - anconeal process dorsally and laterally
 - lateral humeral epicondyle
 - medial humeral epicondyle (check also for flexor mineralisation/avulsion/metaplasia)
 - cranial border of the radius and/or humerus
 - Uneven and/or increased width of the joint space between humerus and radius.

Cranio-caudal radiograph:

- Bony irregularity and/or new bone formation on the medial border of humerus and ulna
- Note: A bony fragment is rarely seen!
- Step between radial and ulnar subchondral bone plate
- Humeroradial joint space medial wider than lateral, particularly in BMD
- Subchondral bone defect in the medial humeral condyle with or without subchondral sclerosis (OC/OCD or kissing lesion). A loose fragment is rarely seen. Beware of artifact: The sagittally running radiolucent line superimposed over the MCP on a slightly pronated projection usually represents the edge of the ulna and not a fissured PCM!

Radiographic findings with OC/OCD (Osteochondrosis, Osteochondritis dissecans)

Typical findings are

- Defect in the articular surface of the medial humeral condyle, best seen on the craniocaudal or (less reliably) mediolateral extended view. A loose bony fragment is rarely visible.
- Small lesions are missed on a mediolateral projection! An craniocaudal view is essential for a correct diagnosis! The defect may also be missed on technically suboptimal films!

Radiographic findings with UAP (Ununited Anconeal Process)

- Irregular radiolucent line between anconeal process and ulna after 18 weeks of age
- Irregular shape, opacity, and outline of the anconeal process
- Progressive DJD depending on duration of process
- UAP may occur in combination with FCMP!

Scoring

Radiographic findings are scored based on the severity of the arthrosis (DJD) and/or presence of a primary lesion using the IEWG (Int. Elbow Working Group) protocol

Scoring for Elbow Dysplasia

Radiographic Findings

Scoring for Elbow Dysplasia		Radiographic Findings
0	Normal elbow joint	Normal elbow joint, No evidence of incongruency, sclerosis or arthrosis
1	Mild arthrosis	Sclerosis of ulnar trochlear notch and/or Step > 2 mm between radius and ulna and/or Presence of osteophytes less than 2 mm high
2 *	Moderate arthrosis	Presence of osteophyte 2 to 5 mm high
3 *	Severe arthrosis	Presence of osteophytes more than 5 mm high

(B) "Borderline" between score 0 and 1 is allotted to undetermined cases reflected as minimal joint remodelling in some countries.

* In several countries the presence of a primary lesion such as UAP, FCP, OCD, or severe INC of more than 2 mm automatically results in a score 3, and the suspicion of such a lesion in a score 2, regardless of the severity of arthrosis. However, in other countries the presence of a primary lesion with no secondary arthrosis formation will result in a score 0!

For USA please refer to www.offa.org

For the UK please refer to

http://www.bva.co.uk/public/chs/ED_Proc._Notes_2007_2007.pdf

References

- <http://www.ncbi.nlm.nih.gov/entrez/query.fcgi> (Enter: Elbow dysplasia dog)
- Morgan JP, Wind A, Davidson A: Hereditary bone and joint diseases in the dog, schlütersche 2000. ISBN 3-87706-548-1. An excellent book!
- www.thekennelclub.org.uk
- www.offa.org

Correct Elbow Positioning and Angulation for ED Scoring

Mediolateral projection

The dorsal edge of the anconeal process is clearly delineated



Craniocaudal 15° pronated projection

The lateral edge of the olecranon and the lateral edge of the humerus are superimposed



Additional projections (optional)

Mediolateral 135° opening angle



Fully flexed and slightly supinated



Fully extended



Pathologies

Early arthrosis formation is usually best seen at the anconeal process and the lateral humeral epicondyle.

The fragment off the medial coronoid process is rarely seen.

Common findings with FCP are sclerosis of its base and osteophytes on the anconeal process:



normal



Sclerosis

Examples of various ED cases

Ununited Anconeal Process (UAP):



Osteochondrosis (OC/OCD)



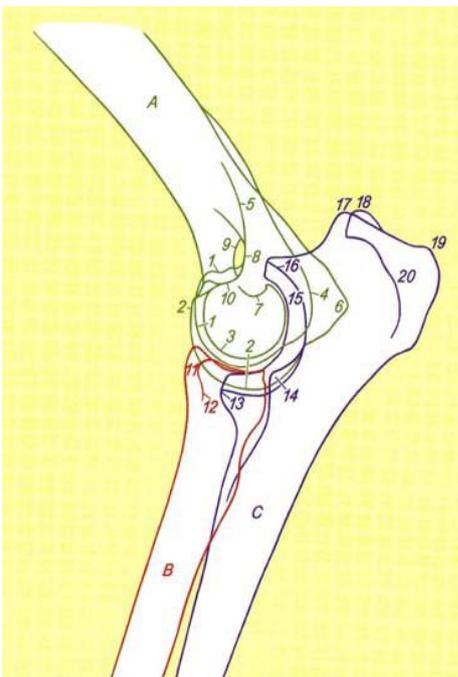
Fragmented medial coronoid process (FCP)



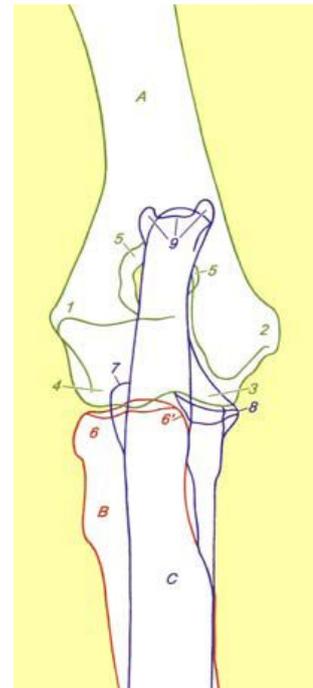
Normal Elbow Joint

(from Waibl et al.: Atlas of Radiographic Anatomy of the Dog, Parey 2003)

Mediolateral view



Cranio-caudal view



Caution: This diagram shows no pronation
Apply 15° pronation for ED scoring!

Legend

A Humerus	B Radius
C Ulna	2 medial humeral condyle
4 lateral epicondyle	6 medial epicondyle
13 medial coronoid process	14 lateral coronoid process
16 anconeal process	3 medial humeral conyle
7 lateral coronoid process	8 medial coronoid process

The IEWG Screening Protocol for Elbowdysplasie

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The diagnosis of elbow dysplasia (ED) in screening of dog breeds is based on the evaluation of radiographs according to the protocol of the International Elbow Working Group (IEWG).

The most recent update of this protocol is available on the IEWG web site.

This protocol defines the radiological findings in “abnormal” elbows : the presence of arthrosis and/or the major forms of primary lesions (FCP, OCD, UAP, Incongruity). Any other abnormal findings should also be reported.

The degree of arthrosis in a joint is graded as either “normal” (Grade 0), mild (Grade 1, osteophytes less than 2 mm high), moderate (Grade 2, osteophytes 2 – 5 mm) and severe (Grade 3, osteophytes higher than 5 mm). The primary lesions have to be mentioned (for details see web site IEWG).

An important decision one has to make is to differentiate between “normal” and “abnormal” joints. Frequently there are elbows with only minimal changes at only one site of the joint (mostly the dorsal border of the anconeal process). In many cases one can even not be sure that these findings are osteophytes representing signs of arthrosis or enthesiophytes at the insertion of the humero-anconeal ligament. These joints are not “normal” and have to be scored as arthrosis grade 1. That may exclude those dogs from breeding. On the other hand we find elbows with osteophytes at several sites of the joint and close to 2 mm in size. Although both joints have to be scored as grade 1, their radiological appearance is very different.

Therefore a “borderline grade” has been introduced by IEWG in 2003, analogous to the screening of CHD. IEWG has set the radiological criteria for a borderline grade :

(B) "Borderline" between score 0 and 1 is allotted to undetermined cases reflected as minimal joint remodelling in some countries.

It is in the decision of the scrutineer to use this grade or not.

Inconcuity (INC) also is a finding that requires more precise definition if it is to be used for the classification of elbows.

The absence of strict grading criteria and the rather big inter-reader differences in cases of steps lower than 2-3 mm made this finding less valuable in cases of screened dogs, from which no clinical informations are available.

According to the IEWG protocol an INC is a primary lesion like FCP, OCD or UAP. That means, that **all** dogs showing this radiological finding have to be excluded from breeding. In my opinion this is not the way to handle ED scoring in a responsible manner.

In the very most elbows with steps higher than 3 mm and an INC caused by clear deformation of the ulna notch the joint shows secondary changes which help to score it in a correct way, and the INC can be used as an additional criteria.

As a suggestion for the discussion : elbows showing no other radiological changes but a step > 2 mm should be scored as grade I.

In genetical studies than the heritability of this criterium can be examined.

In general, the IEWG screening protocol only allows classification in different grades of diseased elbows based on the degree of arthrosis. The term “ED” includes arthrosis and primary lesions. We have no protocol for grading ED comparable to CHD. But this is what most of the breed clubs want to have : a clear classification of ED including the primary

lesions. So in practice in different countries primary lesions were scored in ED 2 or ED 3 respectively.

But using an “ED-grading”, other problems arise. There are many elbows showing radiological signs from which we know, that they are frequently accompanied with FCP, even when a fragment is not visible. Typical criteria for this are a blurred and deformed medial coronoid process (MCP) often together with reduced density, increased density of the ulnar notch caudal to the MCP and short radius.

In Germany those joints are the most frequent reason for complaints against the score of the expert. Many of the x-raying veterinarians are not very familiar with the typical findings in cases of FCP, if there are no obvious osteophytic reactions. But even in cases with osteophytes of more than 2 mm this often will be interpreted as normal. So one focal point of my presentation will be the radiological findings in these cases.

Many scrutineers score elbows that are “suspicious for FCP” in ED 2 even when there are only osteophytes less than 2 mm or no detectable osteophytes.

Dog owners misinterpret the term “suspicious” as “near normal” or as a very questionable change of the joint and will not accept the grading “ED 2”. A better term is MCP disease. On the other hand for me, as an experience out of many clinical cases, the specificity of this sign for the existence of an FCP is very high so that I would like to score them in “ED3”. In any case the primary lesions have to be mentioned beside an ED-grading, because genetical studies have shown a different modus of inheritance.

We also should discuss the procedure in case of an appeal in these special cases. Of course the appeal expert has to score the x-rays. Beside this for a possible final decision a CT examination should be the standard, not MRT. This is now included in the appeal procedure in Germany.

Heritability of ED, scored on the basis of the IEWG protocol is not sufficiently high in some breeds to achieve an adequate response to selection. So breeding value estimation can be an alternative procedure.

But with the “ED grading” elbows/dogs can only be classified in 4 (5 using “borderline”) categories. Depending on the breed 50% to about 80% of the dogs have “normal” elbows and are scored as ED grade 0. On the other hand only few dogs were scored as ED 2 and ED 3. The small number of categories and their asymmetric distribution in the screened population makes it difficult to work with BVE.

So the use of other classification protocols for ED screening in addition to that currently proposed by IEWG may be of advantage. One variation is the use of a “point system” as published by Lang and coworkers last in 1998 (1).

In a study including more than 5000 German Shepherd Dogs it has been demonstrated that this method gives no marked advantage in this breed using it as a basis for selection against ED compared with the ED-grading according to IEWG.

But it provides the possibility to mark the special lesions in detail, which lead to the final score. This is an advantage in molecular genetic studies.

References

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International Elbow Working Group

The International Elbow Working Group [IEWG] was founded in 1989 by a small group of canine elbow experts from the USA and Europe to provide for dissemination of elbow information and to develop a protocol for screening that would be acceptable to the international scientific community and breeders.

The annual meeting is organized for the purpose of exchanging information and reviewing the Protocol. All interested persons are invited to attend the meeting and to participate in its activities.

The IEWG is an affiliate of the WSAVA.

IEWG meetings were held in

1989	Davis
1990	San Francisco
1991	Vienna
1992	Rome
1993	Berlin
1994	Philadelphia
1995	Konstanz
1996	Jeruzalem [cancelled]
1997	Birmingham
1998	Bologna
1999	Orlando
2000	Amsterdam
2001	Vancouver
2002	Granada
2003	Estoril
	Bangkok
2004	Rhodes
2005	Amsterdam
	Mexico
	Munich
2006	Prague

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