PROCEEDINGS

15th annual meeting of the

INTERNATIONAL ELBOW WORKING GROUP

Friday Oktober 24th, 2003

Bangkok, Thailand

WELCOME ADDRESS

Dear participants,

The board of the International Elbow Working Group (IEWG) is grateful to the organisers of the 28th World Congress of the Worls Small Animal Veterinary Association (WSAVA) and the Veterinary Practitioner Association of Thailand (VPAT) for the hospitality offered, to organise a pre-congress programme dedicated to elbow dysplasias. The IEWG has been founded in 1989 by a group of veterinarians and dog breeders with a concern about ED. The purpose of the working group is to gather and exchange knowledge and experiences about hereditary elbow diseases regarding aetiology, diagnosis, treatment, and prevention as well as screening on its presence. The latter includes a standardised method of radiological film reading, a standardised method of scoring of the presence of the primary lesion or secondary osteoarthrosis as well as a standardized scoring form. The ED-certificate which has been discussed and improved during different IEWG-meetings, including the most recent meeting earlier this year in Estoril (Portugal) at the FECAVA-conference. The ED-certificate will be presented to the WSAVA-board in Bangkok. It gives insight not only into the identification of dog, owner and screeners, but especially also into the amount and direction of radiological views used for screening and the findings the final score was based upon. All the aspects of concern of the IEWG are directed to diminish the incidence of the developmental disturbances of the elbow joint in growing dogs of numerous breeds, with a great impact on the quality of life for both the patient and its owner. Therefore the WSAVA recognised the IEWG as an affiliated group.
The IEWG can only accomplish this goal with the help of several experts who accepted the invitation to give an update in their field of expertise, by the organisers of the congress of the WSAVA~VPAT in Bangkok providing space in the pre-congress programme and a lecture room to have the meeting, by the moral support of the WSAVA, and by the financial support of its sponsors Iams Pet Food and Pfizer.

We are looking forward to an interesting, scientific meeting together with colleagues of the hemisphere were till so far IEWG members has not met before. Since the IEWG meeting is open to all veterinarians, members and non-members, both present at the WSAVA~VPAT congress as those visiting us at the web page (http://www.vetmed.ucdavis.edu/iewg), we are assured of a large group of interested veterinarians to exchange knowledge. We wish you a fruitful meeting in Bangkok.

Dr. H.A.W. Hazewinkel president	Dr. K.L. How secretary	Dr. B. Tellhelm treasurer

The International Elbow Working Group acknowledges the financial support by

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PROGRAMME IEWG 2003 BANGKOK MEETING

Friday Oktober 24th, 2003

Boardroom 2, Queen Sirikit National Convention Centre, Bangkok Thailand

8.30-9.00	Registration	
9.00-9.15	Welcome	Hazewinkel
9.15-10.15	Hereditary aspects of Elbow Dysplasia	Hedhammar
10.15-10.45	Break	
10.45-11.45	Clinical diagnosis and surgical treatment of Elbow Dysplasia	Hazewinkel
11.45-12.45	Imaging techniques for diagnosing Elbow Dysplasia	Miyabayashi
12.45-14.00	Lunch break	
14.00-15.00	Elbow Dysplasia in the dog, what is it?	Kirberger
15.00-15.45	Nutritional influences on orthopedic conditions in the dog	Nap
15.45-16.15	General discussion and closing remarks	
16.15-17.00	General meeting IEWG [open to all members]	

List of speakers

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The hereditary aspects of canine Elbow Dysplasia

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Over 30 years have elapsed since conditions affecting the elbow was recognised as clinical entities affecting large sized breeds of Dogs to great extent. We have learned quite a lot and agreed on several measures to be taken against it. That includes selection of breeding stock as well as feeding practices. There are still however gap in our knowledge, weaknesses in our approaches and things we miss to handle and advice professionally on how to enhance elbow conformity.

Although we have agreed on the term Elbow Dysplasia, it must be remembered that it is made up by several different entities, eventually but not necessarily, resulting in elbow arthrosis. As the different entities most likely have different genetic background and probably each influenced by many genes the genetic background is more complex than i.e. for hip dysplasia.

The prevalence of entities making up elbow arthrosis in different breeds varies, calling for more breed specific measures. Crude measures suitable to handle elbow arthrosis in Bernese Mountain Dogs and Rotweilers does not necessarily serve the same purpose in the Retrievers. In for example German Shepherd the prevalence of Ununited Proc Anconeus calls for specific measures. The situation in countries with extensive screening of the entire population is in a different situation than those where barely the breeding stock is screened to any greater extent.

Despite an impressive and accelerating rate of publications within the field, there are still knowledge lacking. We also continuously need to re-evaluate guidance and measures taken based on present knowledge. Radiological techniques, genetic analyses as well as breeding and feeding advice must be based on solid scientific grounds.

Evidence of inheritance of various conditions affecting the elbow joint as well as results from succsessfull screening/controll programs are now available.

Besides particular breedispositions for ununited anconeal process (UAP) fragmentation of proc coronoideus (FCP), osteochondrosis of the medial epicondyle of humerus (OCD), incongruity of radius and ulna as well as nonspecific osteoarthrosis of the elbow joint evidence has been gathered for the inheritance of these conditions within various breeds.

Although there is ample evidence that in all the above mentioned conditions the genetic impact is a significant factor, it is important to review previous studies to determine not only conditions that have actually been proven to be inherited, but also to define the population (breed) to which the heritability applies.

Based on the genetics of elbow arthrosis as manifested radigraphically screening programs for elbow dysplasia have been instituted in many countries

Awareness of the heritability have caused Swedish breeders to select for sound elbows in Rottweilers and Bernese Mountain dogs since 1984 and several other breeds since 1990

Based on the above mentioned studies and experiences it ca be concluded:

a) that there is a genetic disposition for ununited anconeal process, fragmented coronoid process and/or osteochondrosis of the medial epicondyle of humerus as well as ostearthrosis of the elbow as manifested radiographically in certain breeds.

b) that selection of breeeding stock with reference to their own elbow status as well as progeny already produced will affect the frequency of ununited anconeal process , fragmented coronoid process and/or osteochondrosis, osteoarthrosis of the elbow

c) that a radiological screening program for elbow arthrosis identifying both normal and affected individuals has proven its value in reducing frequency of elbow arthrosis.

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Clinical diagnosis and surgical treatment of Elbow Dysplasia

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Introduction

Practicing veterinarians are confronted with young dogs suffering from front leg lameness due to developmental diseases in the elbow joints. Screening in different countries demonstrated that large percentages of different breeds including German Shepherd, Labrador, Golden Retriever, Rottweiler, and Bernese Mountain Dog, suffer from different developmental diseases of the elbow joint all resulting in osteoarthrosis (OA) of this joint. These diseases include ununited anconeal process (UAP), fragmented coronoid process (FCP), osteochondritis dissecans (OCD), and elbow incongruity (INC). These entities are grouped together under the name of "elbow dysplasias" (ED) which makes it easier to understand by dog breeders and owners. Here will be discussed the

clinical diagnosis of UAP, FCP, OCD, and INC and their surgical treatment.

ED are recognized by veterinarians and breeders as a serious problem for certain populations. Depending on the specific sub-population and the method of investigation, ED are seen in 46-50% of the Rottweilers, 36-70% of the Bernese Mountain Dogs, 12-14% of the Labradors, 20% of the Golden Retrievers, 30% of the Newfoundlanders, and 18-21% of the German Shepherds (1) but also in Great Danes, St Bernards, Irish Wolfshoud, Great Pyrenees, Bloodhounds, Bouviers, Chow chows and chondrodystrophic breeds (2,3). The clinical signs due to a comparable coronoid lesion in Retrievers are more severe than in Rottweilers. Read et al (1997) reported that 57% of a group of 55 Rottweilers in a prospective study developed radiographic signs of FCP but "only" 15% showed physical signs including joint effusion, pain and crepitation during examination and 10% developed lameness (7). In a follow up study, with a follow-up period ranging from 0.5-8 years (mean 2.7 years) the success rate of surgical treatment of FCP was 78% in a group of 64 Retrievers (with 67.8% males) operated at young age. Only 33% of the conservatively treated dogs with a FCP (i.e., low body weight and controlled activity but no surgery), were not lame (13); in a group of older dogs (>2.5 years) the success rate decreased till 32%. This stresses the importance of an early diagnosis and surgical treatment.

ED can be separated into different disease entities including UAP, FCP, OCD and incongruities of the elbow joint (INC). From recent studies it became clear that OCD and FCP, and also INC and FCP are diseases with a different hereditary background. They should be considered as different diseases, all causing lameness and OA. The success rate of surgical treatment of elbow dysplasia depends on the complete diagnosis before surgery, the correct surgical positioning and on atraumatic surgical approach as well as the careful aftercare by the owner.

Clinical investigation

The clinical investigation starts with registration of the breed and age of the dog (lameness starts at 4-10 months of age) and inspection of the dog in standing position; in almost 50% of the cases the paw of the affected leg is externally rotated and slightly abducted. On palpation, the elbow is effused. Effusion is usually most pronounced in case of UAP than in FCP or OCD. Effusion of the elbow joint is felt at the side of the anconeal muscle. With the dog in lateral recumbency, the range of motion (ROM) of the elbow joint is examined while the thumb is placed on the anconeal muscle to register crepitation; attention is payed when pain is evoked. It should be realised that the anconeal process is part of the humero-ulnar jouint, whereas the coronoid process is part of the radio-ulnar joint. In case of a UAP, there is in particular crepitation and pain sensation at a firm hyperextension of the elbow joint due to contact between anconeal process and humerus as well as the anconeal process at its fracture area. In case of FCP, crepitation and pain reaction can be evoked at prolonged hyperextension, in particular when the radius and ulna are exorotated at the same time (i.e., stress in the radio-uilnar joint). The latter findings can also be present in case of OCD. Diagnosis of ED can be confirmed by radiography.

UAP and FCP occurs bilaterally in 30% and more than 50% of the cases, respectively, and therefore both elbow joints should be investigated, even in case of unilateral lameness. In case there are no radiographic abnormalities visibly, auxiliary techniques (computed tomography, bone scintigraphy, arthroscopy) may be of value.

INC with the ulna longer than the radius is in most cases as such not painful although the range of motion can be diminished. It is more the OA and/or the FCP which can occur as a consequence of this form of INC which causes lameness, crepitation and joint effusion. Radiographs will reveal the abnormal joint alignment, although it is clamed by some that this can also be caused by artefacts, or wrong interpretation by film readers. The INC which goes together with a radius longer than the ulna is mostly seen as part of the radius curvus syndrome, both in chondrodystrophic breeds as in dogs with an abnormal growth in length of the ulna, due to trauma or too high calcium intake. In those case the diagnosis can be made by inspection of the leg (curved radius, valgus position of the paw) and elbow joint (elbow effusion), its passive motion (crepitation, pain on extension) and radiological investigation (incongruity, UAP or pressure of humerus agains anconeal process = distraction cubiti).

Surgical treatment

Ununited anconeal process (UAP)
In chronic cases of UAP with sclerosis at the base of the anconeal process visible on radiographs, it can be decided to remove the anconeal process from the elbow joint. Arthrosis formation will continue to develop but probably slower than when an irritating ununited anconeal process remains in place (9). Probably it will not diminish OA development due to a slight (but clinically unnoticeable) instability of the joint.
Especially in case of a partial separation of the anconeal process due to elbow incongruity, an osteotomy of the ulna (ulnotomy) can be performed to allow the anconeal process to reattach. The spontaneous restoration of elbow congruity after ulnotomy may be expected in dogs under 12 months of age, since at an older age the interosseus muscle becomes to be to firm thus preventing the ulna to shift proximally. Therefore, in young dogs a low osteotomy is performed, in more mature dogs an osteotomy 5 cm distal of the level of the radial head. In case of UAP in chondrodystrophic breeds, ulnectomy with control by an ring fixator can be the method of choice.
In selected cases, where the anconeal process detached recently and still fits at the place of origin, a lag screw (with or without additional im pin) may lead to bony connection between the anconeal process and the olecranon. Since in these cases OA development was minimal at the time of surgery and the normal joint stability was restored, no OA will develop in these joints in the period after surgery.

FCP and/or OCD
The conventional surgical approach of the FCP and/or OCD lesion is at the medial side of the joint. The aponeurosis between pronator teres and flexor carpi radialis (or the more subtle separation between flexor carpi radialis muscle and the humeral head of the deep digital flexor muscle) is separated by blunt dissection. When the apex of the coronoid process is fractured, it is removed and the edges smoothened with a small curet. When the FCP is sandwiched between the ulna and the medial aspect of the radial head, the intact, medial aspect of the coronoid should be removed allowing the removal of this type of FCP. Removal of the medial aspect is performed with a curet or a small (2-5 mm) osteotome, taking great care not to damage humeral condyle or radius cartilage. When fissures are present in the apex of the coronoid, the apex will also be removed. The joint is frequently flushed with saline to improve the surgical view and remove the debris.

INC with or without FCP
Elbow incongruity (INC) due to a short radius is frequently seen in Bernese Mountain Dogs (BMD), but also other breeds (Retrievers, Mastiff Napolitano) may be affected. In dogs with lameness due to FCP we remove the coronoid, in cases with lameness due to FCP ánd severe incongruity, congruity is restored. In dogs under one year of age this can be performed by a partial (approx. 2 cm) ulnectomy. In animals over one year of age we perform the restoration of elbow incongruity with the aid of the external ring fixator after partial ulnectomy, since the interosseus muscle is too rigid to allow for spontaneous correction.

Arthroscopy can be used to diagnose lesions which could not be noticed on radiographs, especially cartilage lesions as in case of FCP and OCD (19). In addition, it can be used to assist in reattaching the anconeal process (Dr Bardet, personal communication) and to remove the OCD flap and curette its lesion, or to remove the FCP (20, 21). It may have the advantage of 1) a magnification of smaller lesions which might be overlooked during conventional surgical approach, 3) visualisation of areas which can not be seen during conventional surgery, and 3) an early recovery, when compared with surgical approach with massive soft tissue disruption. However, based on his large experience in both conventional surgery and arthroscopy, Bardet (see http://www.vetmed.ucdavis.edu/iewg/jfb2002.pdf) could not find a better outcome in a follow-up study with either conventional or arthroscopical surgical treatment (19).

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Imaging techniques for diagnosing Elbow Dysplasia

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In this presentation, we review radiographic techniques involved with the elbow. These include positioning and pertinent anatomy on each specific view. In addition, common areas of abnormalities associated with elbow dysplasia are pointed out. Briefly, other imaging modalities such as computed tomography (CT), magnetic resonance imaging (MRI), and bone scintigraphy are reviewed.

Radiography is a widely available technique. However, good planning will reward you with high quality radiographs that will aid you to make a correct diagnosis. You need to know about a film-screen system you have. The use of a small focal spot also makes a detailed view of the elbow. Often, we need to use sedation for good positioning. Well-positioned films are prerequisites for correct interpretation.

To make a well-contrasted film, a relatively low kVp setting is preferred. I typically use between 50 to 60 kVp. In some film-screen systems, you may not want to use lower than 50 kVp. Check with your radiographic film venders. Since the elbow is thinner than 10 cm, you do not need to use a grid. Because of that, you can use a small focal spot which is usually available under 100 mA. Another important factor is a collimation. A well-collimated view is not only safe for the holders, but also improves the quality of the radiograph due to a reduced amount of scatter radiation. If you can sedate a young and happy dog, you will appreciate how easy it becomes to take radiographs of the elbows of such a dog. Since we are taking the radiographs in young dogs, you may safely use acepromazine. In addition, ketamine and diazepam, narcotics, and propofol are other readily available sedatives.

Radiation safety should be practiced well. Again collimation, sedation, and protective gloves and gown (if you need to hold a dog) are components you need to remember each time you take radiographs.

How many films do we need to take for a good elbow evaluation? It depends on the age of a patient. In most cases, immatured dogs (less than 12 months) would show primary lesions on radiographs. Thus, you may need to take more than 2 views (I take 5 views). In adult dogs, it will become difficult to detect the primary lesion, since secondary degenerative changes mask the lesion. Thus, you may need 2 orthogonal views to diagnose elbow degenerative joint disease (DJD).

Elbow dysplasia is composed of 4 distinctive diseases: osteochondritis dissecans (OCD), medial coronoid disease (fragmented coronoid process: FCP), ununited anconeal process (UAP), and elbow incongruity disease (INC). To detect lesions associated with these diseases, you may take 5 views: mediolateral, supinated mediolateral, flexed mediolateral, craniocaudal, and craniolateral-caudomedial oblique views. Here, we review how to take these views and pertinent anatomy.

A mediolateral view is a bit of a challenge. When you take this view as a table top view (placing a cassette under the elbow), you often have the distal leg to be in a higher level than the elbow. This is most likely due to a dropped position of the shoulder area. I recommend you to place a sponge under the shoulder to raise that area. Secondly, you may flexed the elbow slightly. When the elbow is extended, it usually rotates. On this view, you would like to see the joint congruity. Thus, a good positioning is a must. I use this view to diagnose:

(1) medial coronoid disease, (2) elbow incongruity disease, and (3) ununited anconeal process.
A supinated mediolateral view was recommended, when an outline of the medial coronoid process needs to be scrutinized. The easiest way to have this is to place a small sponge under the olecranon. You may literally supinate the distal leg, if you want. Then, you need to hold the leg, that I do not think that it is necessary. Look for a clear outline of the medial coronoid process.
A flexed mediolateral view is the most difficult view to make. First, you need to bend the elbow and at the same time isolate the elbow. When you bend the elbow, it often bring the elbow under the sternabrae. Thus, it is helpful to slightly rotate the thorax to move the sternabrae away from the elbow. Remember to add 4 to 6 kVp from the mediolateral exposure technique. When you bend the elbow, the muscles become thick, and you need to compensate for that. This view was first widely utilized to detect a ununited anconeal process that was a common condition in working dogs such as German Shephard Dog. You find that we screened for this particular condition. In addition, on this view, the dorsal aspect of the anconeal process is easily seen. This is the area where you will see the early formation of osteophytes. Since elbow dysplasia causes secondary DJD, presence of osteophytes may be enough to diagnose “elbow dysplasia”.
A craniocaudal view is not much used to detect any of elbow dysplasia. However, this may be the only view where we can see the fracture of or ununited distal humeral condyle. To make a true craniocaudal view, you need to place the olecranon slightly medially. It is often helpful to palpate the olecranon after you place the dog in a ventral recumbent position. The humerus and distal leg should be lined up in a straight line. Extend the elbow as much as possible. In addition, a slight angle of x-ray beam is also helpful to outline the distal humeral condyle.
A craniolateral-caudomedial oblique view is made to see the medial humeral condyle without overlapping the olecranon. I call it as a “lazy” craniocaudal view. This is a position where the olecranon is lateral to the midline. This view is often used to see a “fragmented” coronoid process. However, the apparent “fragmented” piece is usually an osteophyte formed along the medial coronoid process. Actually, this is the best view to detect subchondral defects in the medial humeral condyle due to osteochondritis dissecans (OCD).
Let us review the views we use to diagnose each entity of the elbow dysplasia. For OCD, a craniolateral-caudomedial oblique view is scrutinized for subchondral bone defects (lucency).
For medial coronoid disease, good mediolateral and supinated mediolateral views are evaluated for unclear cranial outline. Obliqued craniocaudal views are for detection of osteophytes along the medial coronoid process. On flexed mediolateral views, osteophytes on the anconeal process can be readily detected, indicating presence of DJD.
For ununited anconeal process, flexed mediolateral views are scrutinized for a radiolucent line in the anconeal process. For incongruity disease, a true mediolateral view is used to detect a step between the lateral coronoid process and radial head and apparent incongruity between the humerus and semilunar notch.
With elbow dysplasia, most dogs will develop lameness as a clinical sign. However, some dogs do not show any lameness. Thus, radiographic screening has been recommended. The International Elbow Working Group has developed an evaluation form and submitted it to The World Small Animal Veterinary Association for its approval. In most countries, the cost of screening evaluation is an important factor. Thus, a flexed mediolateral view is often used for submission to screening organizations such as The Orthopedic Foundation for Animals (OFA). Once osteophytes are detected, the elbow is considered to have developed elbow dysplasia. Thus, OFA does not evaluate radiographs of dogs less than 2 years of age for a certificate.
Briefly, we discuss other imaging modalities such as CT, MRI, and bone scintigraphy. Computed tomography is useful to detect non-displaced cartilaginous medial coronoid process. On the radiograph, this lesion results in a truncated appearance of the medial coronoid process. Surgically or arthroscopically, this lesion may not show any gross abnormality. Thus, pre-surgical evaluation by CT is helpful. However, the cost of the CT examination may be a limitation. Magnetic resonance imaging is a great way to see the cartilage and other soft tissues in the elbow. However, there is not many diseases of soft tissue origin associated with elbow dysplasia. Thus, a high cost of this examination is certainly a limitation. Bone scintigraphy is extremely sensitive for any active new bone formation. Actively developing DJD will show a positive lesion. However, radiation control may become an issue in some countries. In addition, specialized equipment is required to obtain images. Again, these limitation may become a problem as a screening tool.
In summary, a good radiographic study can result in detection of primary lesions in young dogs and diagnosis

of DJD in adult dogs. High quality radiographs are a key to the success in reaching a correct diagnosis.

Elbow Dysplasia in the dog – What is it ?

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Introduction

Elbow dysplasia is the abnormal development of the elbow joint. Elbow dysplasia is an all-encompassing term comprehensible to the dog breeding fraternity which has gained popular recognition, particularly in communicating scientific information to the lay public. In scientific publications however, specific aetiologies should be referred to where possible or, if only secondary arthritic changes are seen on routine radiographic views with no evidence of the primary cause, the term elbow arthrosis should be used. As elbow dysplasia is a developmental condition, clinical signs are usually seen from 4-8 months and are followed by elbow arthrosis. Long standing elbow arthrosis due elbow dysplasia may only be diagnosed later in life, often as result of undetected bilateral elbow dysplasia. This is as result of subtle lameness in both elbows only resulting in a mildly peculiar gait which the owner may not have realised was abnormal at the time. Often elbow dysplasia may be present with no clinical evidence and breeders are unaware of the extent of the problem in their breeding stock. Elbow conditions usually included in elbow dysplasia are:

- Fragmented medial coronoid process (FMCP)
- Osteochondrosis and osteochondritis dissecans (OCD) of the medial humeral condyle
- Ununited anconeal process (UAP)
- Elbow incongruity

These conditions, singly or in combination, result in irreversible elbow arthrosis with resultant pain and lameness. Other conditions which are occasionally included under elbow dysplasia are:

- Medial epicondylar spur
- United medial epicondyle

Elbow dysplasia is a world-wide problem in intermediate and heavy set breeds. It is commonly seen in the rottweiler, Labrador and golden retriever, and German shepherd dog. Other breeds often affected are the Bernese mountain dog, Saint Bernard, Newfoundland, and bullmastiff . The condition is seen sporadically in many other breeds. Breeds may be predisposed to a particular form of elbow dysplasia, eg. German shepherd dog suffers more from UAP, the rottweiler rarely has OCD and the Labrador retriever is most likely to have combined OCD and FMCP. The condition is more likely to occur in males probably due to their faster rate of growth, subtle genetic influences or a sex linked factor. Elbow dysplasia is a polygenic and multi factorial condition, the incidence of which can be reduced by selective breeding. Affected dogs are more likely to have offspring with dysplastic elbows than normal dogs. The greater the degree of arthrosis in the parents, the greater percentage of puppies are likely to suffer from the condition.
Recognition of the condition by veterinarians and breeders and the institution of screening and breeding programmes are required to decrease the incidence of this often crippling disease. Elbow dysplasia is a progressive disease and owners must be made aware that medical or surgical treatment may result in improvement but not normality.

Diagnostic imaging techniques

Good quality well positioned radiographs remain the most cost-effective method of diagnosing elbow dysplasia.

Radiographs however do not show all abnormalities or often are only suggestive of an abnormality. This is often the case in the younger dog when arthrosis has not yet developed.. The contralateral elbow should always be imaged because of the high incidence of bilateral disease. If the origin of lameness is uncertain, radiographs of the rest of the limb should be made to rule out shoulder OCD, panosteitis and palmar metacarpal sesamoid pathology (mainly Rottweiler).

Pathophysiology

Osteochondrosis, the disturbed endochondral ossification of articular or physeal cartilage, is most likely the underlying cause for all the conditions making up elbow dysplasia. Underdevelopment of the trochlear notch with secondary incongruity is postulated by others to be the primary cause. In osteochondrosis, normal cartilaginous development and maturation fails in the hypertrophic zone resulting in thickened cartilage. In the elbow joint articular cartilage involvement results in OCD and non-articular cartilage alterations result in FMCP, UAP and elbow incongruity, probably as result of small growth abnormalities of the long bones making up the elbow joint. The cartilaginous growth disturbance is likely to have genetic and environmental, mainly traumatic and nutritional, causes. The most important nutritional factors are an excess supply of energy and relative over-nutrition with calcium. Trauma is usually minimal and associated with hyperactivity or excessive body weight. Severe trauma resulting in premature closure of a physis is a separate clinical entity causing severe growth disturbance of the affected long bone which may markedly influence joints adjacent to the traumatised physis, and is excluded from the elbow dysplasia syndrome.

Fragmented medial coronoid process

Fragmentation of the medial coronoid process is believed to be a manifestation of the osteochondrosis complex and is the most common clinical entity causing elbow arthrosis. The cartilaginous medial coronoid process ossifies from its base to the tip i.e. it has no separate ossification centre, and ossification is completed at 20-22 weeks. Osteochondrosis results in the deeper chondrocytes in the thick layer of cartilage being unable to survive and undergoing chondromalacia with eventual fissuring of the cartilage and subsequent fragmentation. The fragment often mineralises as a result of receiving blood supply through its fibrous connection with the annular ligament which inserts on the coronoid process. Mechanical stresses have been incriminated as a cause of FMCP. One theory postulates asynchronous growth of the radius and ulna with a relative overgrowth of the ulna in relation to the radius. This places an abnormal load on the medial humeral condyle and medial coronoid process and if these structures are weakened by delayed ossification, pathology occurs in both these structures. Fragmented medial coronoid process is thus commonly seen together with OCD or erosive lesions and rarely with UAP. The disparate growth may still be evident at the time of presentation or may have corrected itself.

Radiological changes

The medially located fragment should not be confused with the sesamoid in the origin of the supinator muscle on the lateral side or with osteophyte formation on the medial coronoid process. The fragment is rarely seen radiologically with fragment visibility reported as low as 9.8%. If visible, it may be seen as a single loose fragment or as several smaller fragments. Factors contributing to poor visibility include fragment location between the radial head and remaining intact coronoid process, minimal fragment displacement or that the fragment cleavage line is often oblique to the X-ray beam making it impossible to see. Additionally, the medial coronoid process may be just fissured and non-displaced, non mineralised or just abnormally shaped making radiographic evaluation impossible and requiring more sophisticated imaging techniques. An additional radiographic change which may be indicative of medial coronoid pathology is blunting of the medial coronoid process. Radiographic changes are often only seen from about 7 months and then only secondary arthritic changes may be all that are seen. The arthritic changes are similar to those caused by other conditions (see below). Osteophyte reactions tend to be less severe than with UAP and more severe than with OCD. Joint incongruity may also be evident with the lateral coronoid process displaced proximally to the radial head resulting in step formation. If more sophisticated imaging techniques are not available, the diagnosis can often only be confirmed by means of arthrotomy or arthroscopy. Dogs presented with clinical evidence of an elbow

problem at less than 7 months of age with normal radiographs should undergo arthroscopy or must return for follow-up radiographs 4-8 weeks later.

Osteochondritis dissecans of the medial humeral condyle
The pathophysiology is very similar to that described for FMCP. Osteochondritis dissecans without concomitant FMCP is very rare. The cartilage flap rarely mineralises and may separate and form a joint mouse.

Radiological changes
The defect is readily seen on the CrCd or pronated CrCd view as a radiolucent lesion or dished out defect on the medial humeral condyle and may be seen from 5-6 months of age and prior to secondary arthrosis. In more advanced cases the defect may be surrounded by a sclerotic rim. If OCD is the sole lesion, arthrosis is less marked than with FMCP but clinical signs may occur earlier and may be more disabling than with FMCP. Combined OCD and FMCP lesions result in the most severe arthrosis.

Erosive lesion/cartilage defect
An erosive lesion or cartilage defect, is seen most commonly on the medial humeral condyle and occasionally on the medial coronoid process and probably has the same aetiology as FMCP and OCD. It may not be possible macroscopically to determine if a medial humeral condyle lesion is primary OCD or a secondary erosive lesion. Erosive lesions commonly accompany FMCP. Erosive lesions are postulated to occur rather than FMCP or OCD when the ulnar growth disparity occurs at a slightly older age when the medial coronoid process and medial humeral condyle cartilage is almost mature. Alternative theories are that minor joint incongruity may result in the lesions or that they are earlier stages of FMCP and OCD. These lesions are also known as “kissing” lesions but Olsson differentiates this from an erosive lesion.

Radiological changes
Lesions are not seen on radiographs of affected joints but secondary changes may be seen.

Ununited anconeal process
The condition occurs mainly in the German shepherd dog. The separate ossification centre of the anconeal process, only present in larger breeds, appears at 11-14 weeks and the anconeal process is united with the olecranon at 20-22 weeks. The ununited anconeal process may be completely separated or joined to the ulna by fibrous or fibrocartilagenous tissue. Overgrowth of the radius with a relatively shorter ulna is believed to be the cause by Olssen. The radius forces the humeral trochlea in a proximal direction and the floor of the olecranon fossa exerts more pressure than normal on the anconeal process damaging the anconeal process ossification centre. If osteochondrosis is present, the whole structure is less resistant to trauma and a tear in the weakened cartilage prevents osseous bridging of the gap. Wind on the other hand proposes a theory of a primary incongruent joint with an abnormally developed slightly elliptical trochlear notch causing UAP (see elbow incongruity). Ununited anconeal process is rarely seen together with FMCP but if present, has important implications as the surgical approaches are different.

Radiological changes
The condition is readily seen on a flexed ML view as an irregular vertical radiolucent line through the caudal aspect of the anconeal process. Superimposition of the medial humeral epicondyle physis in views that are not fully flexed should not be confused with an UAP in dogs less than 8 months old. The fragment may separate completely from the olecranon. Arthritic changes become severe over time. It is essential to make multiple views to ensure no other causes of elbow dysplasia are present.

Elbow incongruity
On the semiflexed ML and CrCd view there should be small evenly spaced joints between the humeral trochlea and the ulnar trochlear notch and between the humeral condyle, radius and medial coronoid of the ulna. The joint space forms a continuous arc on the ML view. The lateral coronoid process should lie close to and run continuously with the adjacent radial head. Incongruity is characterised by a gap or step formation between

the lateral coronoid process and the adjacent proximal radius, a more proximally located medial coronoid process, humerus displaced cranially on the centre of the radial articulation, increased humeroulnar and humeroradial joint space and an indistinct outline of the trochlear notch. These findings suggest that the trochlear notch develops in a slightly elliptic shape with the articular curvature of the trochlear notch too small to fully encompass the humeral trochlea. If the incongruity occurs after 6 months it may be present on its own with only elbow arthrosis resulting. If present before 6 months, incongruity may also predispose the dog to FMCP, OCD and UAP due to increased mechanical forces on these structures and incongruity may thus be seen together with these conditions. Intermediate and heavy set breeds have been shown to have a relatively longer proximal ulna as compared to the adjacent radius in comparison to other breeds. Wind postulates that this reflects a need to accommodate a trochlear notch of sufficient size to encompass a heavier and larger humeral trochlea. Insufficient development of the trochlear notch with resultant incongruity is thus most likely in these larger dogs. Incongruity may not always be evident at the time of radiological examination due to compensatory adjustments during growth.

Radiological changes
Incongruity, as described above, is best seen on the normal ML and CrCd views

Arthrosis
Arthrosis is a chronic, degenerative, non-infectious joint disease involving joint cartilage, joint capsule and subchondral bone tissue and is usually the end result of the above conditions.

Radiological changes
Early changes are best seen on the ML views with osteophyte formation starting within the hollow of the anconeal process and lateral epicondylar ridge, followed by the margins of the cranioproximal radius and craniodistal humerus articular surfaces and sclerosis surrounding the trochlear notch. The latter tends to start distally and may be as result of osteophyte formation at the joint capsule insertion or subchondral sclerosis or both. The anconeal osteophytes correspond to the insertion of the olecranon ligament and joint capsule on the proximal non-articular surface of the anconeal process. On the CrCd views osteophytes are seen on the medial humeral epicondyle and on the medial aspect of the medial coronoid process. Severe arthrosis may develop over time and joint mice may be present cranial to the medial coronoid process or in the olecranon fossa. Arthrosis may be present without any radiographic evidence of a primary cause and could be due to erosive lesions, healed FMCP or minor incongruity.

Control programmes

Elbow screening programmes have been established to determine the degree of elbow involvement with a view to limiting breeding with severely affected dogs. The scoring system essentially only evaluates arthritic changes that occur secondary to the primary causes. It does not predict the type of lesion present for which multiple views may be required The grading also does not correlate with the degree of lameness that may be evident. To make a specific diagnosis multiple elbow views are usually required whereas for screening programmes most countries only require 1 or 2 views.
The International Elbow Working Group (IEWG) was established in 1989 to lower the incidence and promote a greater worldwide understanding of elbow dysplasia. The group consists of veterinarians, veterinary radiologists, geneticists and dog breeders and meets annually in different parts of the world to discuss current knowledge and promote elbow screening schemes. More information on the IEWG can be obtained from their web site at <http://www.vetmed.ucdavis.edu/iewg/iewg.html>.
The IEWG has established guidelines for elbow screening and these have been adopted by the Federation Internationale Cynologique and World Small Animal Veterinary Association as the official standard. Control programmes in different countries are encouraged to utilise the IEWG criteria when initiating screening programmes. The current IEWG elbow screening protocol includes submission of permanently identified good quality flexed ML radiographs of both elbows from 12 months onwards for osteophyte evaluation. The flexed ML view has been proven to be a sensitive predictor of elbow arthrosis resulting from elbow dysplasia even though

the inciting cause is not always evident. Grading is as follows:

Grade 1 (mild arthrosis) - osteophytes <2mm at one or more of the following sites :

a) within the hollow of the anconeal process
b) on the cranioproximal edge of the radius
c) on the proximal edge of the medial coronoid process
d) on the proximal edge of the lateral epicondylar ridge
e) sclerosis in the area caudal to the distal end of the ulnar trochlear notch and to the proximal radius

Grade 2 (moderate arthrosis) - osteophytes 2-5 mm high at one or more locations as described for grade 1.

Grade 3 (severe arthrosis) - osteophytes >5 mm high in one or more locations as described for grade 1.

Osteophytes may also be evaluated on a CrCd radiograph at the following locations:

f) distal medial aspect of humeral condyle
g) medial aspect of medial coronoid process

Additionally if the primary pathology is evident it should be stated. Screening programes should be performed at a standard and narrow age interval, eg. As close to 12 months as possible as increasing age has a significant influence of the prevalence and severity of elbow arthrosis.

Information gained from screening programmes should be available in an open registry system to researchers and responsible owners to improve the selection of breeding stock. It is ideal not to breed with any dog with arthrosis, but this is likely to result in breeder resistance and breeding programmes should thus be adapted to the incidence of elbow dysplasia in the breed to ensure breeder compliance.

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Nutritional influences on orthopedic conditions in the dog.

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Introduction

The growth process in dogs shows remarkable changes in a relatively short time compared to other animal species and man. Especially in the large and giant breeds the growth in length (long bone growth) is spectacular. At the age of 16 to 18 months these large dogs have reached their final body size (not weight). Growth in length in small breeds stops at around 6 months. During adulthood wear and tear stress will result in normal cartilage damage that can recover in the joints exposed. However over exposure and joints at risk will show increased arhrosis or osteo-arthritis (OA) formation. In the senior animal the normal process of cartilage degradation results in what could be called “physiological OA” in affected joint.

Does nutrition have an influence on the skeletal development and if so, what are the most important nutritional components involved and what are the effects? Based on series of research several factors have proven to be

important. These are the energy mineral and vitamin intake. The protein intake is often mentioned for its presumed role in inducing developmental skeletal disease, and has been the topic of research for that matter. In recent years attention has also been given to the role of nutraceuticals like glucosamin glycans and chondroitin sulphate in the protection and or degeneration of cartilage degeneration i.e., OA.

Bone and joint development in large and giant breed dogs.
During early rapid growth many things can go wrong, and some unfortunately do. It has been found that depending on the breed, several conditions related to the skeletal growth may occur. The diagnoses are made more often in some breeds than others because they have proven to be hereditary. These conditions include the well-known osteochondrosis (OCD), elbow dysplasia (ED) and hip dysplasia (HD). ED is the combined term that includes 1. Osteochondrosis (OC) of the elbow, 2. ununited anconeal process (UAP), 3. Fragmented medial coronoid process (FMCP) and elbow incongruency (INC). The hereditary basis for these diseases does not mean that they can be easily addressed by breeding measures only, as they have proven to be multifactorial. Breeding programs must be the basis of the program for eliminating these serious problems from the population.

It is important to realize that growth includes growth in body weight and growth in body length (height). These two are often confused, and in many cases where there is reference to rapid growth in dogs, this refers to bodyweight increase. Heavy dogs are not always tall dogs. Bassets, Bulldogs, dachshunds and beagles are examples of dog breeds with relatively short legs compared to their body weight.

The above mentioned skeletal problems OCD, ED and HD, that occur mainly in large breeds, can indeed be influenced by nutrition. Early research by Scandinavian workers was published before the Great Dane Cornell studies by Prof. A. Hedhammar in 1974. Most of the popular breeds are affected, for example Labrador & Golden Retrievers, Bernese Mountain Dogs, German Shepherd dogs, Mastiffs, Bordeaux Dogs, and St. Bernards. The racing type dogs are genetically not prone to the conditions and are typically not affected by these type problems. Not all problems occur to the same extent in the breeds mentioned above, which again is a signal for the hereditary nature of the conditions. Some breeds show more problems in the shoulder joint, others more in the elbow, while others typically are affected by shoulder or hip problems. Differences for sub populations of the same breed in different continents have been reported.
For the skeletal problems as we know them in small breeds, like the Legg Calve Perthes (hip) disease and medial patellar luxation, no influence by nutrition has been shown.

The growth in length is the result of growth in so called growth plates. The bones do not grow randomly, but only in these cartilage growth plates that are situated at the ends of the bones. Most bones have growth plates at both the top and the bottom ends. During the growth process cartilage cells at the top of each plate, split up and line up in columns. In the process, they shift to the bottom of each plate during their maturation. At the ‘mature side’ of the plate the cartilage cells die and are replaced by bone. This process goes on an on, until the bone has reached its final length.
It has been shown in University studies in Great Danes in Sweden (Hedhammer) and the Netherlands (Hazewinkel, Nap, Schoemakers, Tryfonidou) that nutrition can influence this growth process. The 2 key factors are 1. overnutrition (of a complete and balanced diet thereby supplying excess energy and excess minerals) and 2. excess calcium intake. Recent research revealed the importance of age of exposure to these factors (Schoenmakers 1998) and the influence of vitamin D intake (Tryfonidou 2002).

How does mineral intake affect bone development?
Excess calcium intake has been shown to be the key factor in OCD and ED. High calcium intake increases the risk, while lowering the intake decreases the risk. It has been shown also that the 1.1% calcium level which was generally advised for dog foods is relatively high for large and giant breeds, especially in the early growth phase (the first weeks, and months). When feeding 3 times normal levels of calcium, disturbances in the cartilage maturation in the growth plates was found, whereas lower calcium levels decreased the frequency and severity. Obviously, too low calcium levels coincide with the development of a weak skeleton that cannot

support the bodyweight and is subject to fractures. The risk for oversupply of calcium ("intoxication") is related to the fact that the intestine of immature dogs cannot 'refuse' calcium after ingestion. A minimum of about 40% is absorbed after intake, no matter the level of excess. This is true in both large and small breed puppies, but only results in clinical problems in the large breeds.

The disturbance of skeletal maturation results in disturbed endochondral ossification and in disturbances in bone remodeling. The ossification process is retarded and the hypertrophic zone gets wider in both the articular as well as in the growth plate cartilage. As a result the growth slows down and abnormalities may occur that are clinically relevant. This is the case when the wider growth plate results in a cartilage cone that slows down the growth (shorter bone) and especially when this occurs in the vulnerable distal V-shaped ulnar growth plate. The delicate balance between radial and ulnar growth gets disrupted and incongruenty may show up. A shorter ulna can result in radius curvus syndrome (short ulna, short antebrachium, incongruent elbow and carpal joint, and antecurvature of the radius). The elbow incongruenty can coincide with an ununited anconeal process (UAP), one of the conditions recognized as elbow dysplasia (ED). The other conditions under the ED umbrella are OCD, fragmented coronoid process the previously mentioned incongruenty.

Complete and balanced pet foods:
The challenge while manufacturing pet foods for growing large breed dogs is to find the optimal balance between a relatively low level of calcium to reduce the risk for osteochondrosis, while supplying enough calcium to build sufficient strength for bodyweight support. Studies (Lepine et al) have shown that 0.8% provides the optimal level to feed the large breeds (Great Danes) from weaning until maturity.
Although no research to date has shown an incremental risk with stepwise increases in calcium levels, the knowledge that 1. calcium is the risk factor in the development of OCD and related condition, 2. that at the level of 1.1% Ca more OCD type lesions were found than at 0.55% Ca and 3. that 0.8% calcium levels have been proven safe for raising large and giant breeds, makes any level higher than 1.4% - 1.5% questionable, and should be considered a potential risk in diets for growing dogs. Due to (hereditary) differences in "sensitivity" for OCD between breed exists, differences will occur in manifestation of the condition after similar exposure.

Providing optimal calcium levels in dog food is more complicated than providing a percentage of Ca as outlined above, because the amount of food is consumed on the basis of energy content (and requirement) and not on the basis of gross weight of the product. This implies that when feeding a complete and balanced diet, at the amount that the energy requirements are met, all other nutrient requirements (including calcium) should automatically be provided in the correct amounts by the diet at the same time (and should not exceed risk levels either). The consequence of this is that a 1.1% calcium level in an energy-dense and highly digestible diet results in a lower daily calcium intake compared to the same 1.1% level in a low-energy, poor quality product. Therefore the energy density has to be taken into account when judging the calcium level in the product.
For dog owners, the energy content of a product is not always available. It is often not written on the packaging (no guaranteed method for uniform declaration is available and therefore energy declarations are not allowed by law in most countries). The best one can do (apart from gathering information from the manufacturer or importer) is to look at the fat level. Generally speaking, high fat levels coincide with high energy levels. When comparing different products, the differences in ingredient digestibility have to be taken into account as well. Wet (canned) products should be compared to dry products on a dry matter basis, and high quality premium diets should be compared with low quality diets with care. High quality premium products might contain more than 25% greater energy than low quality products based on the same ingredient list and chemical analysis because the quality of the ingredients differs. Important is also the clinical experience with a given formulation. Large Breed growth product with a Ca level at 0.8% (17.3 MJ or 4138 kcal/kg has proven safe in far over 1 million dogs where the product has been used for optimally feeding pups from weaning till adulthood. Due to the high food intake, the calcium requirements are safely met, while reducing risks related to high Ca intake.

It has been shown that the influence of phosphorus levels are secondary in importance to the calcium levels. The phosphorus intake will result in skeletal problems only in case of major imbalance. This may occur when feeding a meat-only diet to a growing dog. This results in an extremely low calcium intake (0.03% calcium) combined with an inbalanced calcium : phosphorus ratio. The result is a weak skeleton and high risk for pathological fractures.

Can we influence hip dysplasia by nutrition?
The development of hip dysplasia (HD) is not related to the longitudinal growth but is nevertheless influenced by nutrition to a high extent. As mentioned, high food intake during growth results in higher risk for HD to develop. The mechanism for this is related to the rapid increase in bodyweight and the relatively immature skeleton to support this bodyweight. At birth the skeleton is largely cartilaginous and is gradually transformed into bone. Cartilage, as compared to bone, is flexible and it can and will change shape under the influence of loading forces. When the immature skeleton, and thus the immature hip joint (dorsal acetabular rim) is 'overloaded' by a dog's excessive bodyweight (compared to its age) it is at risk to adapt the shape of its hip joints and become dysplasic. HD is however, a hereditary disease and when the genes are not in the breed, it is believed that excess body weight for a given age will not result in increased HD severity. On the other hand, when the breed is genetically at risk for HD, it has been shown that the frequency and severity can be dramatically increased by overfeeding the puppy. Reality today is that many breeds (and breed standards) expect a large body weight at an earlier age, thus expecting maximal body (weight) development rather than optimal development. The "orthopedic" optimal development of bodyweight is often not in accordance with breeders and judges desire where a larger volume / size (teddy bear) at an earlier age is perceived to be better. A relatively lean bodyweight at the age of one year for breeds that are at risk for HD, does not coincide with optimal chances for winning shows and prizes. However, it might be better for the long-term well-being of the individual animal. Dog breeders associations, kennelclubs, individual breeders and dog owners have to consider these research findings and balance them against the short term early age show results. The final body size is not reduced by a less steep bodyweight growth curve.

Can we influence OCD and ED by nutrition?
The risk factor for OCD and ED are dietary Ca intake and total food intake. The Ca intake relative to the energy intake in large and giant breed dogs has to be relatively low compared to adult dogs or dogs of small and medium breed, that are not at risk for OCD and ED. A complete and balanced diet with Ca at 0.8% and 4138 Kcal or 17.3 MJ/kg has been shown to be the optimal balance between concerns for over and under supply of the mineral and can be fed from 6 weeks until 18 months of age. Too rapid growth in bodyweight increases the risk for these conditions. A balanced growth with a moderate bodyweight growth velocity is optimal for the end result; that being an optimally healthy dog. The additional benefit for the owner / breeder is that feeding restricted amounts (and not maximal amounts) reduces the daily feeding costs. Limiting the food intake to 75 or 80% of the maximum intake (ad libitum feeding) should not be confused with anything like starvation. It will in most cases result in feeding levels that are in line with 'normal' guidelines. It is important to understand that this will not impact the final adult size (shoulder height / growth in length) of the dog.

Does high protein increase the risk for OCD?
Ingredient levels in a complete and balanced diet are balanced to the energy density of the food. In case of energy dense diets, such as performance or growth products, the relatively high energy primarily the result of high fat levels, must be balanced with relatively high protein levels. In high fat diets protein percentages can be up to 32%, without delivering high protein intake per day. Protein intake as well as other diet components is in grams, and not in percentages. Performance (30% protein and 20% fat) and growth diets (small and medium breed diets: fat / protein at 32 / 21 and 29 / 18 respectively), are no high protein diets, but they are high energy diets with balanced protein levels to assure sufficient protein on a daily basis. For Large Breed puppy products are formulated with lower protein levels (26%) since the fat level is lower (14%).

In addition, university studies in Great Danes (Nap et al 1993) have shown that a true high dietary protein level

(as compared to an isocaloric normal protein diet) had no negative influence on the skeletal development. In other words, high protein intake does not result in increased risk for OCD and / or ED. In these studies the test diet contained 32% protein compared to the normal of 21%. No differences were seen in skeletal development or OCD during the critical first 6 months of life. Also the longitudinal growth was not affected. As a conclusion: high protein intake does not create a risk factor for OCD or ED. It has to be kept in mind, that when aminoacid (AA) requirements are met, there is no need for additional protein. Excess protein will be used as an energy source by the body. Since the amino acid profile of animal protein best reflects the dogs' requirements for essential AA, high quality, nutrition that provides animal-derived proteins will optimally support growth and development.

What if my dog gets old?

Abnormalities in joint development, whether they are related to OCD, ED or HD or any other disabling joint condition, will result in so-called secondary degenerative joint disease, or osteo-arthritis (DJD and OA respectively). The results of the "wear and tear" process will be a painful condition of the joint that is increasingly disabling. First of all, it has to be stressed that the best one can do is to prevent the development of DJD or OA by creating the optimal environment for the joint development early on in the dog's life. This includes managing bodyweight and nutritional composition as previously discussed. Management of clinical arthritis is typically related to balancing exercise and bodyweight management via physical activity and nutrition protocols.

Animals suffering from OA should be kept lean and exercised regularly. Several short walks a day, avoid excess loading of the joints and are better than long, weekend walks where peak joint loading occurs. The level of exercise can eventually be relatively high, when the build-up has been done gradually. Animals with OA have been reported to enjoy jogging with the owner. However, when the training schedule is interrupted (the owner falling ill or travelling for example, or in case of any other reason why the exercise regime changes abruptly) the animal will suffer the consequences when the training is picked up again after the break, and will not be able to perform anywhere close to the level before the break. Balancing the fatty essential omega 6 and 3 fatty (n6 and n3) acid levels in the dog's diet might also add to the management of the inflammation process that occurs in the affected joints. The optimal balance between n6 and n3 fatty acids has been proven to be between 5 and 10 n6 to 1 n3. These levels of 5 to 10 times more n6 than n3 ratio, indicate a relatively high n3 level compared to conventional diets). This has been proven to provide the optimal ratio between the inflammatory and less inflammatory mediators involved in the OA processes.

For the effects of neutraceuticals on cartilage we quote the summary of one of the few world expert research groups of the world (Academic Hospital University Liege, Belgium; Henrotin et al 2001): "Several entities have been carefully investigated for the symptomatic and structural management of osteoarthritis. The most compelling evidence of a potential for inhibiting the structural progression of osteoarthritis has been obtained with glucosamine sulfate while some preliminary results also suggest that chondroitin sulfate could be used in the same indication. At any rate, these two compounds have clearly demonstrated a symptomatic action, mainly in osteoarthritis of the lower limbs. Symptomatic effect, on pain relief and improvement of functional disability, was also reported with the use of avocado/soybean extracts. This article summarizes the evidence indicating that some nutraceutical compounds can effectively interfere with the structural progression of OA". It was concluded that "Several compounds classified as nutraceuticals have shown symptomatic or structural efficacy in OA. So far, the most compelling evidence of a potential for inhibiting the progression of OA is obtained with glucosamine sulfate while some hints also suggest that chondroitine sulfate could be used in the same indication. These two compounds, however, have clearly demonstrated a symptomatic action, mainly in OA of the lower limbs. Symptomatic effect, on pain relief and improvement of functional disability was also reported with the use of avocado/soybean extracts. The beneficial effects obtained with nutraceuticals on cartilage degradation justify their use in the animal nutrition. Nevertheless, an important issue is that all the conclusive studies reported herein resulted from the use of prescription medicines, and not over-the-counter pills or food supplements. Therefore, the extrapolation of these findings to food supplements must be done with caution"

These citations are in agreement with reports in the veterinary literature and with clinical reports from veterinarians around the world and conclusions in review articles (Anderson 1999) on the subject. Levels and duration needed for optimal efficacy and preventive and therapeutic protocols have to be further studied both in research settings as well as in field studies. There is no longer doubt about the beneficial effects of so-called chondro-protective neutraceuticals. We are now entering the period of fine-tuning dosages and indications. The availability of animals for these studies, the open environment of client owned dogs and the long term commitment and compliance of owners participating in these studies are a serious complicating factor for execution of quality double blind studies.

Table 1: does nutrition affect skeletal integrity in the dog ?

	growth	adult & senior
fats		
energy source	yes / overweight	yes / overweight / arthrosis
fatty acid (FA) source	yes / n3:n6 and essential FA joint inflammations	yes / n3:n6 and essential FA joint inflammations
vitamin carrier	yes / vit ADEK	yes / vit ADEK
protein	no for OCD, ED & HD	no
amino acids	* essential Az's yes !	* essential Az's yes !
carbohydrates (CH)	no	no
(non) fermentable fibres	* CH fermentability is important for GI health	* CH fermentability is important for GI health
vitamins		
vitamin D	yes / high levels & Rickets	yes
minerals		
calcium	yes / high Ca: OCD, ED, low Ca: hyper PTH	no high Ca risk yes low Ca: hyper PTH / over long period
phosphorus	yes / OCD, ED / P is important at young age and at high levels / second to Ca	no
neutraceuticals		
glucosamin glycans	yes / cartilage, arthrosis	yes / cartilage, arthrosis
chondroitin sulphate	yes / cartilage, arthrosis * in young animal at risk for cartilage deformation ** subject to further studies	yes / cartilage, arthrosis * early stages of OA ** subject to further studies

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Large Breed Nutrition on the World Wide Web:

The reader is also invited to visit the website > eukanuba-scienceonline.com < for more information and further reading on the topics related to feeding large breed dogs especially during the important period of rapid growth. Several proceedings and the audio and video recording of the Iams Clinical Nutrition Symposium Venice 2001 are directly available at your convenience via your home computer system.

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

Forthcoming IEWG meetings

Rhodos [October 2004]

IEWG 2003

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website: **www.vetmed.ucdavis.edu/iewg/iewg.html**