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GENETICS OF CANINE ELBOW DYSPLASIA

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INTRODUCTION

Elbow dysplasia (ED) is one of the most common hereditable diseases in canines, it is well known by both veterinary surgeons and breeders of our time. It not only affects the gait and locomotion of dogs that develop it but also affects their welfare and interferes with the use of working and sport dogs. However is it one condition or several? The condition was first studied in the 1970's (K Make, et al., 2000) and the term ED now refers to several abnormal developments within the elbow joint. An elbow is considered to be positive for ED when one or more of the following primary lesions are present: Osteochondritis dissecans (OCD), ununited anconeal process (UAP), Elbow incongruity and fragmented coronoid process (FCP) (Jedee Temwichitr, et al., 2010). Despite being a common disease it has not yet been widely documented. ED has been overshadowed by similar diseases such as hip dysplasia (HD) due to the connection and similarity between the two diseases. ED generally first presents itself in dogs between the ages of 4 to 12 months old. Often the primary symptoms shown are pain and lameness in their forelimb, however this is not exclusively the course of the disease as it can also be subclinical or chronic; occurring in later life up to and later than 6 years of age. Once the disease is present and is allowed to progress it can lead to the development of secondary lesions to which there is no surgical treatment as yet therefore will affect the animal for the rest of its life (Matthew Pead and Sue Guthrie, 2013).

As ED is seen throughout an extensive range of breeds, I have chosen to base my studies on a select list of highly documented breeds which have particular affinity for ED and it is due to this that they are considered predisposed to developing ED. These breeds are: the Bernese Mountain Dog, Labrador Retrievers, Golden Retrievers and German Shepherd breeds. As this disease has been documented in such a vast number of breeds, noted in over one hundred different breeds (The orthopaedic Foundation for Animals) it has been concluded that it commonly affects medium to large, fast growing breeds.

ED is believed to be a hereditary disease influenced by multiple genes alongside environmental factors (I.C.M. Lavrijsen, et al., 2012). Currently ED is diagnosed by a screening process implemented by the British Veterinary Association (BVA) the

Kennel Club and International Elbow Working Group (IEWG) where dogs of a certain age are radiographed under general anaesthetic. The radiographs are then graded by two members of a certified panel, grades range from zero to three depending on condition of the joint or the severity of the disorder (T.W. Lewis, et al., 2011). Work is currently underway to use genetic analysis and selection in breeding programs as a method to reduce the occurrence of ED (J.A. Woolliams, 2011). This will be elaborated on later in this paper.

The goal of this paper is to review several research papers, journals and books based on my topic with an objective to obtain information to support the belief that ED is in fact a genetic disease and to outline measures which can be implemented to reduce the prevalence of the disease. I wish to elaborate on the history and current status of this condition in a hope to provide clear chronological data from which research can progress.

METHODS AND MATERIALS
(REVIEW OF LITERATURE)

In this thesis I have chosen to base my knowledge on several journals which I have collected and are listed in my references. I utilised many journal databases for my search; such as 'pubmed', 'Science Direct' and 'CAB abstracts' to obtain several up to date publications related to my topic. I then analysed and investigated the result which I have referenced when relevant throughout my work. I have also used several reliable webpages including that of the British Veterinary Association along with webpages belonging to both the Irish and United Kingdom kennel club and the International Elbow Working Group's official website.

Furthermore I referenced several books "Veterinary Genetics and Animal Breeding" written by Dr. Zöldág, László, "The Merck Veterinary Manual" edited by Cynthia M. Kahn and Scott Line, "The genetics of the dog" written by A. Ruvinsky & J. Sampson, 'Small Animal Orthopaedics' written by Zoltán Diószegi and 'Guide to small animal clinics' written by Chris Pasquini & Susan Pasquini.

I have used all this information available to me in order to illustrate the disease of elbow dysplasia to the best of my ability.

GENERAL

Elbow dysplasia (ED) is the generic term given to the generalised incongruence of the elbow joint. ED is comprised of several malformations which have been studied since the 1950's and ED itself was first described by Olsson in 1974 (K Mäki, et al., 2000). It is considered a hereditary developmental disorder due to the characteristic inherited incongruencies within the joint and is one of the most common heritable diseases of canines. There are three common malformations that comprise ED which affect many areas of the complex hinge joint. These malformations are referred to as primary lesions. The three primary lesions in question are; Fragmented Coronoid Process (FCP), Ununited Anconeal Process (UAP) and Osteochondritis Dissecans (OCD) of the medial humeral condyle. Often elbow incongruities are also treated as a primary

lesion also. One or more of these primary lesions may be present in any one case of ED. These affect the semilunar notch causing the articulation with the humeral trochlea to be altered; this can lead to abnormal wear of the cartilage and bones found within the joint causing irreversible secondary lesions for example Degenerative Joint Disease (DJD) and osteoarthritis (OA) (Zoltán Diószegi, 2006).

Fragmented Coronoid Process

Fragmented Coronoid Process is a condition of the medial elbow, in which the coronoid process fails to unite either completely or partially with the ulnar diaphysis (figure 1.). This prevents it from becoming part of the articular surface of the trochlear notch causing joint instability and irritation which can result in osteoarthritis. Most often FCP presents in puppies from the age of 4 to 6 months due to their rapid growth period during this stage of life (Aldo Vezzoni, 2006). FCP can manifest due to ulnar overgrowth causing overloading on the medial coronoid process as it lies higher than the radius compared to that in a healthy elbow joint where the radius normally bears the majority of the weight (Cynthia M. Kahn and Scott Line, 2010). FCP is diagnosed using both radiographic means using craniocaudal; craniocaudal oblique and medial views and by the use of Computed Tomography (CT) scans. The early detection of FCP is disputed as being beneficial, as the initial joint incongruity may improve spontaneously as the dog continues to grow and develop (Aldo Vezzoni, 2006). The first clinical signs of ED are often caused by FCP presenting as lameness, exorotation, crepitation during movement and a reduced range of motion of the affected legs. This condition can be unilateral or bilateral (A. Ruvinsky and J. Sampson, 2001). If the condition is bilateral it can hinder the detection on both symptomatic and radiologic means as lameness becomes more difficult to diagnose. The radiographs of each elbow are considered and compared however if the condition is bilateral a third radiograph from another dog of the same age and breed with a healthy elbow joint is necessary for comparison.

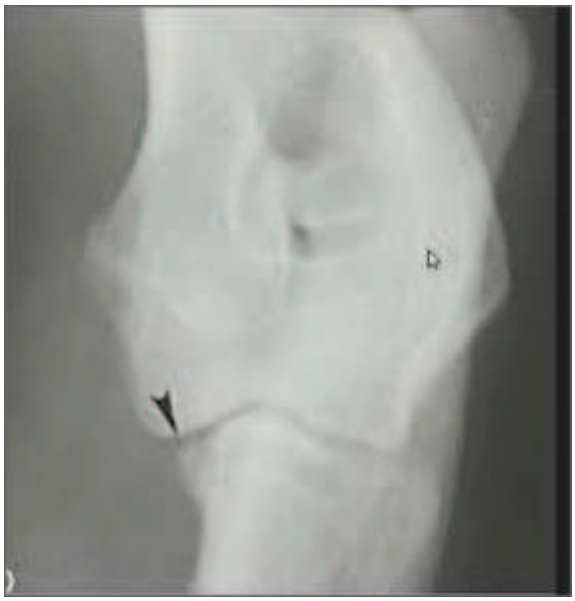


Figure 1. Fragmentation of the coronoid process can occasionally be seen on a craniolateral-caudomedial view indicated by the black arrow (www.studydriod.com)

Ununited Anconeal Process

Ununited Anconeal Process (UAP) occurs when there is a separation of the anconeal process from the proximal ulnar metaphysis. Typically the fusion of the anconeal process and the ulnar metaphysis is completed at 5 to 6 months of age. UAP occurs due to an imbalance in the joint as the anconeal process is initially held in place by bridging fibrous tissue; the imbalance can cause the fibrous tissue to fracture leading to joint instability. In advanced cases osteoarthritis and joint effusion can occur (Cynthia M. Kahn and Scott Line, 2010). This condition regularly manifests at the ages of 5 to 8 months (figure 2.) and presents with swollen joints, painful movement, and lameness along with a restricted range of motion (A. Ruvinsky & J. Sampson, 2001). UAP is diagnosed by comparing radiographs of the affected limb and the opposite limb, however similar to FCP this condition can too be bilateral therefore comparing radiographs of a healthy animal of the same age and breed may be necessary. Performing ulnar osteotomy can be an early treatment for UAP as this releases the pressure on the anconeal process. In more severe cases a screw fixation may be necessary (Aldo Vezzoni, 2006).

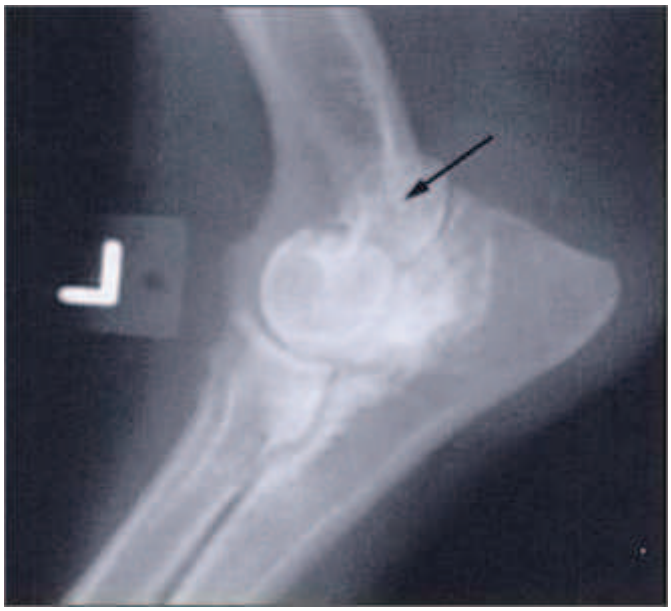
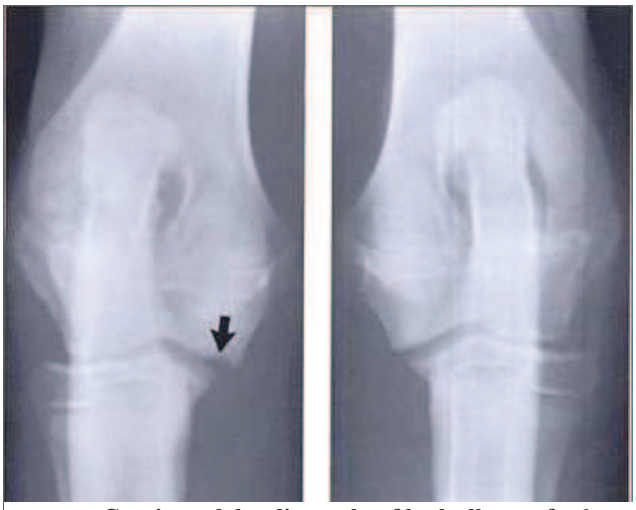


Figure 2. Lateral radiograph of an 8 year old male German Shepherd dog with a malformed UAP indicated by the arrows (Karen M. Tobais, et al., 2012).

Osteochondritis Dissecans

Osteochondritis dissecans (OCD) is a defect in the joint cartilage formation and maintenance of said cartilage, in which a segment of the cartilage can begin to raise from the bone or even break off into the joint space which is known as a "mouse" (figure 3.). This ailment is usually reported from the ages of four to ten months and results from a disturbance of the endochondral fusion of the epiphysis of the medial epicondyle at the distal point of the humours either side of the trochlear ridges. OCD can exhibit swollen joints, painful movement, lameness and reluctance to move, these are similar to the symptoms of the previous lesions (A. Ruvinsky and J. Sampson, 2001; Cynthia M. Kahn and Scott Line, 2010). The diagnosis is based on radiographic signs and CT scans. Surgical removal of the cartilage flap, mouse or joint fragment and debridement on the underlying bone to promote fibrous scarring are necessary treatments for OCD. If left untreated secondary lesion of DJD may develop (Aldo Vezzoni, 2006).



Craniocaudal radiographs of both elbows of a 6-d dog showing signs of OCD lesion in (A) seen on al aspect of the humeral condyle, (B) shows the ontours (Karen M. Tobais, et al., 2012).

Genetics of an animal will predispose them to developing these above malformations which comprise ED. However it can be exacerbated by other factors such as growth rate, abnormal cartilage and bone development, diet, level of exercise and joint stress. It is most prevalent in young rapidly developing large breed canines such as German Shephards, Bernese Mountain Dogs, Rottweilesr and Golden Retrievers. However if these factors were eliminated it would not prevent the condition from developing nor reduce the potential to pass the disorder to their offspring but may delay the development. Symptoms can be present from as young as four months of age (I.C.M. Lavrijsen, 2012) up to one year old and can even present in later life after six years of age (Jacob Michelsen, 2013). The canines that develop this disorder frequently display forelimb lameness, swollen joint and exhibit pain in one or both forelimbs with reluctance to move, these symptoms are regarded as the first signs of ED.

DIAGNOSIS OF ELBOW DYSPLASIA

Diagnosis of ED is not an easy task as we are not just concerned with one disease affecting a certain area within the elbow joint but several different malformations that affect the joint at different stages of development as well as different areas of the joint itself. Diagnosis is also hindered by the fact that ED can be clinical, subclinical or chronic presenting with mild lameness. As ED is composed of a number of primary lesions the clinical signs for each malformation can lead to various symptoms which

are not exclusive to one lesion alone making diagnosis on symptomatic basis alone very difficult. OCD can present with lameness, joint distension and pain on manipulation, UAP is often associated with crepitation and pain whereas FCP presents with lameness, abnormal gait and pain (Chris Pasquini and Susan Pasquini, 2011). These malformations may be bilateral which further hinders the diagnosis making detection of clinical symptoms more difficult. Early diagnosis is much sought after to prevent further deterioration of the elbow joint; however there are pros and cons associated with this also. If it is recognised at an early stage treatment can be performed in order to rectify the malformation and to prevent the development of secondary lesions which are irreversible and would have a lifelong effect on the animal in question. Alternatively if the condition is diagnosed and treated at an early stage, the treatment might be an unnecessary stressor and expense as the malformations that occur at a young age during the rapid growth period may stabilize as the animal reaches full skeletal maturity. Early diagnosis can be performed as young as the fifteenth or twentieth week of life, however if no signs of elbow dysplasia are discovered at this point further evaluations should be performed at the sixth or seventh month of age as ED can develop later on (Aldo Vezzoni, 2006). The ossification of the anconeal process is complete on average at four and a half months of age. However it is advised to complete an orthopaedic evaluation after one year has been reached according to the IEWG as this is when skeletal maturity is expected therefore the mean screening age is twelve months or older.

One of the most beneficial and economical methods of diagnosis is by radiographic means. After a great deal of discussion by screen authorities, breeders and veterinarians; taking into consideration radiation exposure, length of anaesthesia, cost and accuracy of diagnosis it was decided that a minimum of two radiographic views are necessary according to the IEWG's protocol which requires one medio-lateral flexed projection and one craniocaudal view for evaluation (figure 4.). However according to the BVA three radiographs of each elbow are necessary to diagnose. The views always include a Medio-lateral view of the elbow in either 15^{o} or 90^{o} flexion to visualise the outline of the anconeal process, this is where the first osteophyte formation is commonly seen and it is also used to assess osteosclerosis of the elbow. The second radiographic view necessary is a medio-lateral view with the elbow in an extended position to inspect the coronoid process and elbow incongruity. The third

being a craniocaudal and/or craniolateral-caudomedial view which are used to diagnose the presence or absence of OCD, osteophytes and OCD-like lesions (kissing lesions).

Animals can only be screened once in their lifetime and are graded according to the IEWG's grading scheme outlined by the same protocol on a zero to three graded scale, which is discussed later in this paper. However despite multiple views of radiographs along with the expertise and experience of the scrutineers, lesions can still be missed. Therefore diagnosis with computed tomography (CT), magnetic resonance imaging (MRI), scintigraphy and arthroscopy are considered to be more reliable (I.C.M. Lavrijsen, et al., 2012) although they are more costly.

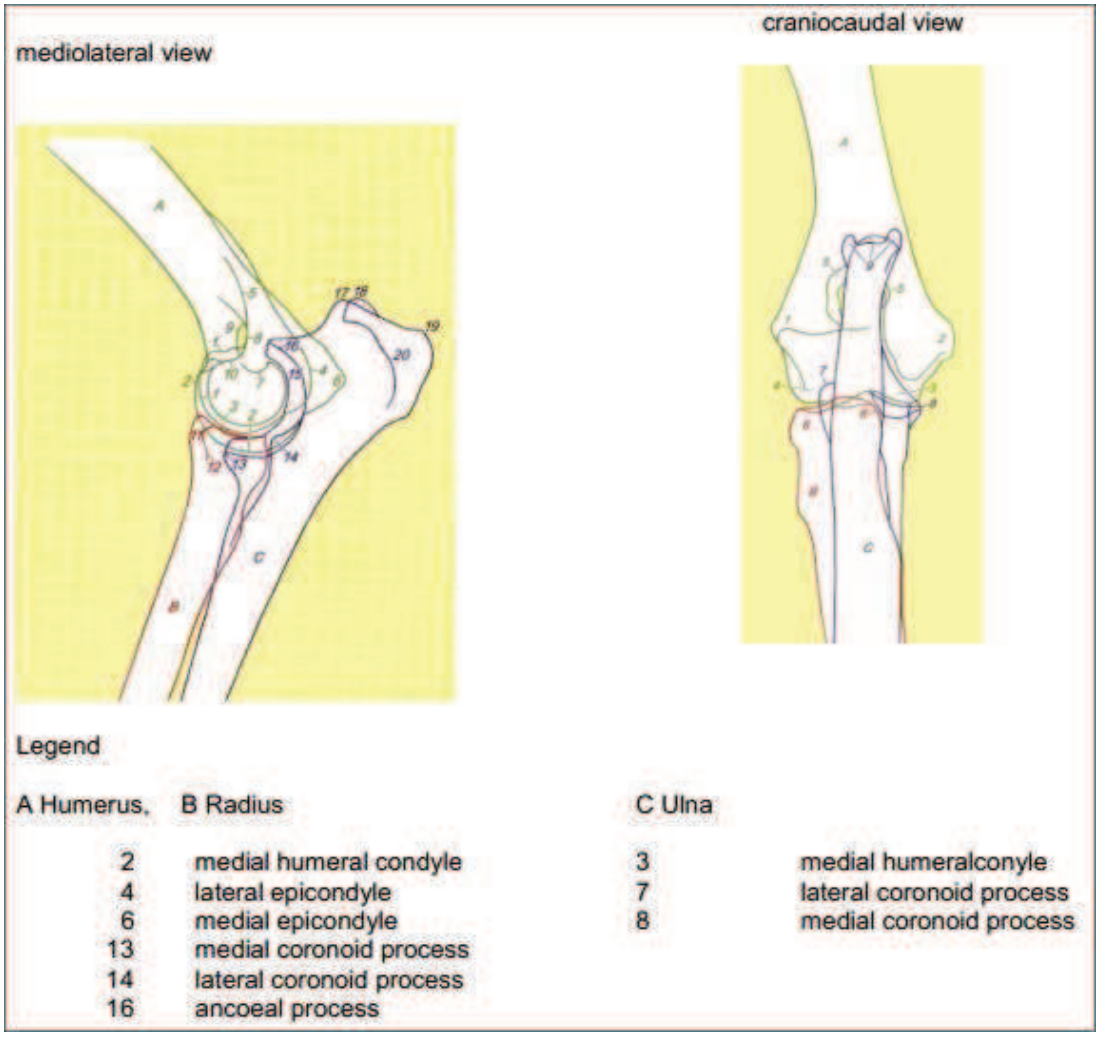


Figure 4. Normal elbow joint (Dr. H.A.W. Hazewinkel, 2012).

Computed Tomography

CT scanning can help establish a firm diagnosis when radiographs are obscure. In the area of the medial coronoid process fragmentation, fissure, abnormal shape, sclerosis and osteophytes can be detected. As for the medial humeral condyle sclerosis and flattening can be identified. Ununited anconeal process with or without incongruity can be identified along with incongruities of the humeroradial, humeriulnar and/or radioulnar joints and the shape of the trochlear notch can also be examined using CT scanning. However the positioning is very important and an experienced scrutineer is necessary.

Magnetic Resonance Imaging

MRI evaluation of the elbow joints are limited by the size of the joint containing a number of complex articulations within, these limitations vary depending on the strength of the MR device and skill of the examiner. However MRI can be useful in identifying variation in the soft tissue surrounding the joint and the subchondral bone pathology which could be beneficial in the diagnosis of ED.

Ultrasonography

Ultrasound (US) can also be a valuable imaging technique for diagnosis. An experienced sonographer using linear transducers emitting frequencies higher than 7.5 MHz are necessary (Gielen Ingrid, et al., 2012).

All these methods of examinations could improve the diagnosis of ED but are still subjective similar to the current screening methods and are costly, therefore are not part of the routine screening schemes.

TREATMENT

Following diagnosis, treatment methods vary depending on the primary lesion present and the age of the animal in question as shown in the algorithms below (figure 5 &6.). Often ED is treated using a conservative method involving weight loss to reduce the stress on the joint, exercise restrictions and medication which can be used to provide pain relief and act as an anti-inflammatory. This method of treatment is popular due to the expense of the corrective surgery therefore a conservative method often proves more economic for pet owners. In some cases surgery is necessary to remove fragments of the cartilage and bone from the joint, however if secondary lesions like joint degeneration has already begun this may lead to further problems having a lifelong effect on the animal.

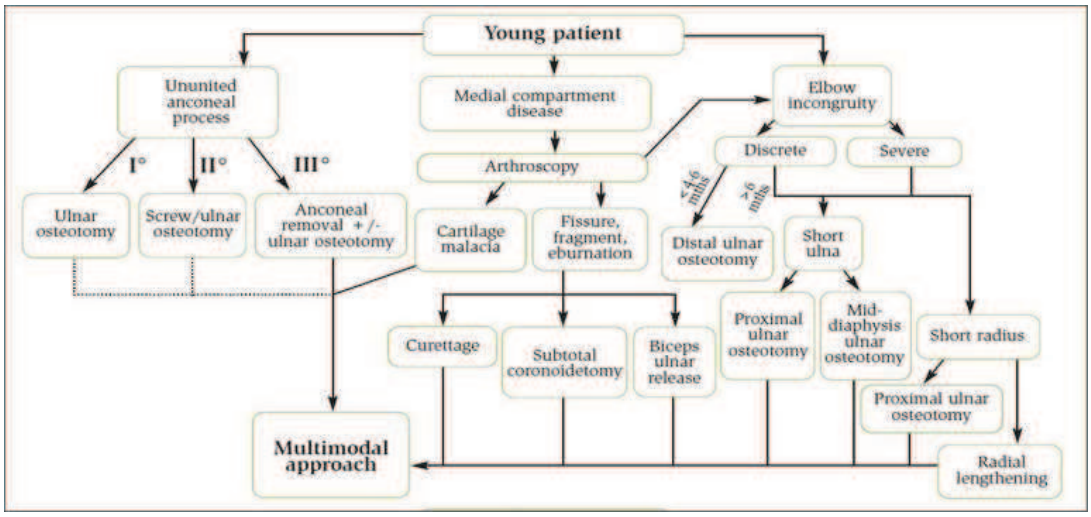


Figure 5. Algorithm for the treatment of ED in young patients (Vet grad website).

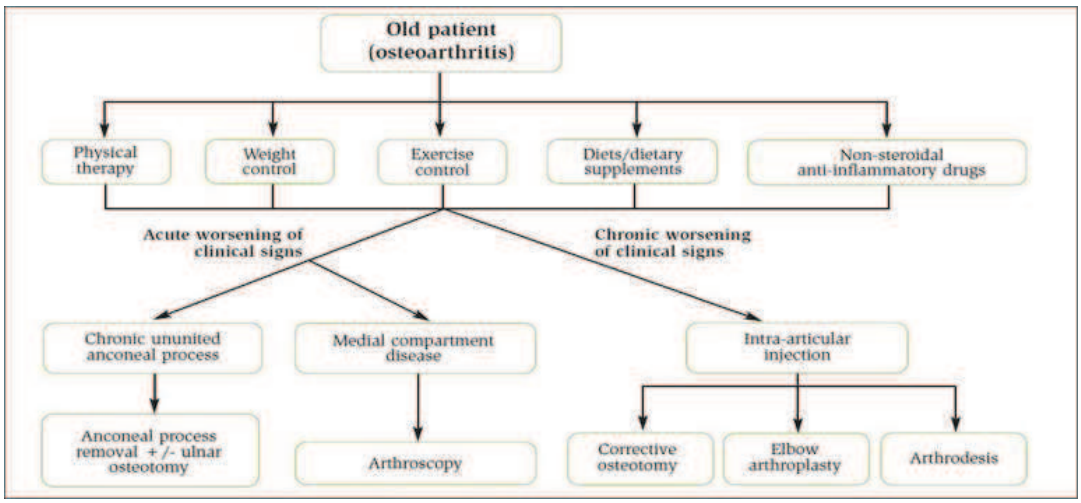


Figure 6. Algorithm for the treatment of ED in older patients (Vet grad website).

The early treatment of UAP involves dynamic ulnar osteotomy to release the pressure on the anconeal process; screw fixation in lag fashion can be performed when chances of bony fusion are high for example when the procedure is performed when the animal is approximately four to six month old (figure 7.). At this stage fusion of the anconeal process and the restoration of the joint congruity is very likely. If this procedure is carried out in older animals however where joint degeneration is already present, the increased motility of the anconeal process may impede its healing.

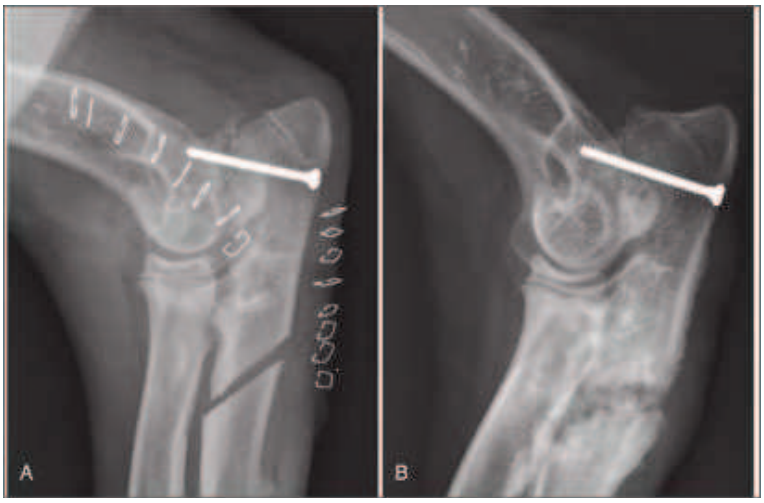


Figure 7.Proximal ulnar osteotomy and screw fixation for the treatment of UAP, A) postoperative medio-lateral radiograph of a 4 month old puppy with UAP, B) Medio-lateral radiograph of the same puppy 6 weeks later (Karen M. Tobais, et al., 2012).

In cases caused by FCP in canines aged between four and six months suffering from incongruity, subchondral bone sclerosis of the subtrochlear notch with no osteophytes can be treated by performing a distal dynamic ulnar osteotomy (DUO). A DUO involves removing four to five millimetres of the ulna approximately two to four centimetres proximal to the distal ulnar physis. Doing this will release the pressure on the medial and lateral coronoid processes. This is then monitored using postoperative radiographs three to four weeks later; if clinical signs persist or worsen, further treatment may be necessary. In more severe cases treatment with arthroscopy or mini-arthrotomy in conjunction with DUO is performed in canines less than eight months of age. After eight months of age the interosseous ligament between the radius and ulna thickens and hardens which renders this procedure ineffective.

Cases with radiographic signs of OCD are treated by arthroscopy or mini-arthrotomy means. This can also be treated alongside the distal dynamic ulnar osteotomy. Because of the wide exposure of the subchondral bone and activation of the inflammatory mediators, low-dose of corticosteroids and an NSAID are given (prednisolone 0.2 mg/kg, PO, SID, for one to two weeks, meloxicam 0.075 mg/kg, PO, SID, for three to four weeks). This study found that using low-doses of both these drugs aided the therapeutic effect and presented with little or no side-effects (Aldo Vezzoni, 2006). Treatment however does not prevent the condition being passed to potential offspring.

SCREENING AND GRADING ON ELBOW DYSPLASIA

ED is considered a complex disease caused by a combination of genetic and environmental factors. Pedigree information and population-wide data on disease has been/is being collected by associations including the BVA and the IEWG in an effort to control the prevalence of ED occurring. These associations have collected their data through health screening schemes. The information collected is then analysed by statistical programs to calculate the extent to which the disease is genetically based. This information is used to determine the estimated breeding values (EBVs) of an individual dog. By using the EBVs breeders can decipher and select sires and dams with the best genotype, therefore those who have a high genetic risk of passing the disease to their offspring are excluded from the mating program.

As there is not yet a blood or soft tissue test available to detect the animal with the best genotype for mating, screening is carried out by radiographic means. The IEWG and the BVA have issued guidelines to be followed in order to comply with the screening needs. Minimum age of the animal submitted for screening must be at least twelve months of age to ensure the animal has reached skeletal maturity (Sofia Malm, et al. 2007). Three views are required of each elbow to ensure the best analysis of the joint condition, a medio-lateral view in both flexed and extended positions along with a crainocaudal (or caudocranial) view is required. It is preferred that the dogs are manually restrained, however anaesthesia may be used in order to perform flawless radiographs for grading (I.C.M. Lavrijsen, et al., 2012).

The grading scheme of the radiographs is according to the protocol of the IEWG, two scrutineers must agree on the grade of each radiograph basing their opinions on the IEWGs grading criteria as outlined in table below (table 1.) (Matthew Pead and Sue Guthrie, 2013). The films are evaluated in a two stage process, first the degree of secondary joint disease e.g. arthrosis is assessed. Secondly the radiographs are checked for sign of a primary lesions and any other abnormalities are also noted. The joints are scored on a grade system from zero to three; zero is considered a healthy joint with no signs of disease, one is a mild case of disease with osteophytes of less than 2 mm found anywhere in the joint, grade two is a moderate case where osteophytes of 2 -5 mm are found and grade three is a severe case of joint disease where osteophytes of greater than 5 mm are seen.

Table 1: scoring canine ED according to IEWG scheme.

Amended new elbow dysplasia grading based on degree of arthrosis and/or presence of primary lesion. Scoring mode effective immediately.		
Elbow Dysplasia Grading		Radiographic findings
0	Normal elbow joint	Normal elbow joint, No evidence of incongruencies, sclerosis or arthrosis
1	Mild arthrosis	Presence of osteophytes < 2 mm high Suspect sclerosis of the base of the coronoid processes Step of up to 2 mm between radius and ulna
2	Moderate arthrosis or suspect primary lesion	Presence of osteophytes of 2 – 5 mm high Obvious sclerosis of the base of the coronoid processes Step of >2-5 mm between radius and ulna (obvious INC) Suspect presence of a primary lesion (UAP, FCP, OCD)
3	Severe arthrosis or evident primary lesion	Presence of osteophytes of > 5 mm high Step of >5 mm between radius and ulna (obvious INC) Obvious presence of primary lesion (UAP, FCP, OCD)
A borderline (BL) score between ED 0 and ED 1 is allotted in some countries to dogs with minimal anconeal process modelling of undetermined aetiology.		

GENETICS OF ELBOW DYSPLASIA

One of the earliest studies carried out on the topic of ED was in 2000 a study on the German Rottweiler population since screening for ED had started in 1997. A total of 2114 dogs were tested 974 were male and 1140 were female. They followed the guidelines of the IEWG and graded them accordingly they used the REML algorithm taking into account variances in month of birth, gender and year of examination. The result of this study revaeled that ED is more prevalent in male dogs than female dogs. Only 39.1% of male dogs examined were free from ED versus 51.5% of female dogs. This study suggests that this is due to the higher body weight of male dogs. Using the REML algorithm the estimated genetic variance component was calculated to be 28% which indicated that the genetic background was strong enough to select by simple mass selection despite not having information of relatives (R.Beuing, et al., 2000).

One year later in 2001 a publication stated that ED has a polygenic mode of inheritance. OCD was caused by the failure of endochondral ossification of the epicondylar articular-epiphyseal complex which may be due to the ulnar over growth and elbow incongruity. This causes an abnormal load on the medial humeral trochlear ridge preventing the successful ossification from occuring. The same study also stated that factors including nutritional imbalance, rapid growth and genetics affect the development of ED. This publication confirmed that all breeds that were predisposed to OCD (Labrador Retrievers, German Shepherds and Rottweilers) of the medial humeral trochlear ridge also appear to be predisposed to FCP, however the same could not be said for the other way round as several breeds were predisposed to FCP only this supported the concept that FCP and OCD are inherited independently and therefore ED has a polygenic mode of inheritance (A. Ruvinsky, et al., 2001).

Three years on and a study carried out on four dog populations (German Shepherd, Golden Retriever, Labrador Retriever and Rottweiler) based on polygenic models that included both fixed and random environmental effect and additive genetic effects was performed. In applying the mixed inheritance models the effects of a major gene was added to the polygenic models. The major gene was considered to be an autosomal bi-allelic locus with Mendelian transmission probabilities. Using both Gibbs sampling and a Monte Carlo Markov Chain algorithm a polygenic model was determined for each breed. The polygenic models revealed that the distribution of all grades were not

normal within the breeds therefore seven possible values for grades of ED resulted. The distributions were skewed for ED in all breeds except Rottweilers which displayed bimodal distribution. This supports the existence of a major gene in Rottweilers. However the results for the other breeds involved showed very large estimates of major gene variance and allele frequencies for ED, especially in Labradors Retrievers. This did not indicate evidence supporting the existence of a major gene in these breeds. Major genes are recessive therefore it can be concluded that advances in genetic progress would occur if selections were made against a major gene. However data of this study would be more accurate if the most severe clinical cases were also included, these cases are usually operated on at a younger age before skeletal maturity and therefore screening occurs (K Mäki, et al., 2004).

A study carried out on the golden retriever population for FCP discovered that FCP frequently occurred among Labradors who shared common ancestors. 1194 dogs were studied from the Netherlands a mix of 570 males and 585 females discovered that males were 1.6 times more likely to be affected than females. This study also performed radiographic investigation of complete litters of Labradors which ruled out FCP being caused by an autosomal dominant trait (J. Temwichitr, et al., 2010). As Jedee Temwichitr et al. (2010) discovered Labradors have an 18% genetic risk of suffering from FCP this percentage risk could even increase up to 50% within certain families. This further supports the theory of ED being a genetically inherited disease and with such high prevalence seen in certain families selection against this could prove very effective.

A study carried out by T.W. Lewis et al in 2011, studied the EBVs of untransformed elbow scores from 1997 to 2007 and discovered that total grades decreased on average by 0.075 grades and a decrease of EBVs by 0.0031 ± 0.0014 grades per annum based on that year of birth as shown in the chart below (figure 8.). These figures are collected from selections made based on the phenotype alone, despite this still progress is being made.

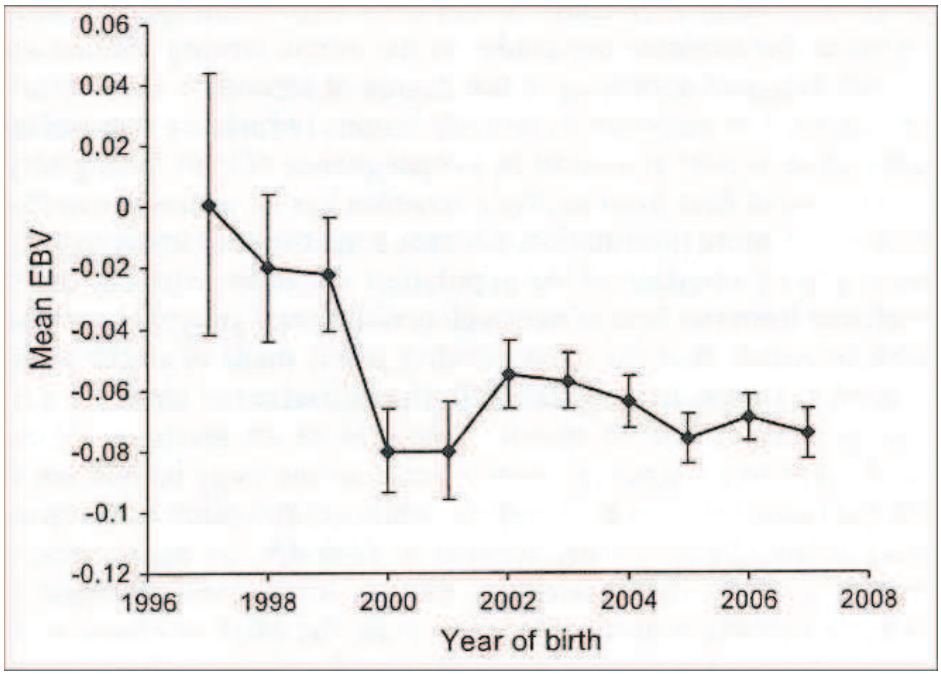


Figure 8. Mean EBVs for untransformed elbow scores according to year of birth for all records of dogs scored at >1 and <3 years old from the years 2000 to 2008 (error bars +/- 1 standard error). (T.W. Lewis, et al., 2011).

This study also states that the inheritance pattern of ED is still not certain, there has been some evidence to support a major gene effect with varying disposition of primary lesions across a range of pedigree breeds. This suggests that each primary lesion is a different genetic syndrome. T.W. Lewis et al. also noted that the genetic correlation between HD and ED is weak; therefore selection against one disease will elicit little response on the other disease (T.W. Lewis, et al., 2011).

In 2012 a group of researchers analysed the screening results from 2693 Labrador retrievers (LR), 1213 Golden Retrievers (GR) and 974 Bernese Mountain Dogs (BMD) from the Netherlands. They had an average screening age of one and a half years to two years old and were screened between the years 2002 until 2009. These screening results were based on radiographic images which have been assessed by three to five board certified examiners following guidelines set out by the IEWG and grading the ED on the zero to three scale. They discovered that the incidence of OA was most frequent in BMD than that of the other two breeds (LR and GR) examined, FCP was the most common primary lesion across three breeds, INC was very

common in BMDs at 50% where as it was rarely seen in Retrievers in only 6% of all cases. OCD was most common in GR at 25% and LR at 13% although it was rarely seen in BMDs at 3%, UAP was absent from this study (I.C.M. Lavrijsen, et al., 2012). These results once again support that hypothesis that all primary lesions are different genetic disorders and can be inherited independently. From this study it was concluded that the heritability of FCP was 0.17 in LR, 0.24 in GRs and 0.03 in BMDs, OCD had a heritability of 0.07 in GRs and INC had a heritability of 0.10 in BMDs. The FCP heritability in Retrievers is high enough (0.24) to predict that removing the dogs with FCP from breeding programs would in fact lead to genetic improvement (I.C.M. Lavrijsen, et al., 2012).

A paper in 2013 noted that many studies favoured the use of EBVs as a more accurate estimation of genetic liabilities for a specific trait compared to that of the individual phenotype. This paper states that improvements in the rate of genetic progress could be made through the use of EBVs, which can act as a more accurate predictor of genetic risk and by increasing the intensity making more EBVs available for every dog pedigree could bring about a huge decline in the incidence of ED in canines. Currently breeding selections are still being made on the bases of ancestral phenotypes and two of the dogs' own phenotype and although this has made slight improvements in the genetic progress against ED, the use of EBV would be more accurate. Currently only four to eight percent or less of the higher risk animals are being excluded from breeding programs; yet despite the lower levels of restrictions they still yielded a positive result as against ED. Estimates made on the heritability of the untransformed average elbow score ranged from 0.14 (ROTT) to 0.30 (GR). Although regression of the EBVs has been made to date with a decline in genetic susceptibility for ED of between -0.18% per year (LR) to -0.72% per year (BMD), all breeds included in the study showed an improving genetic trend with respect to elbow score, as illustrated in the table below(table 2.). A drawback has been reported in the use of EBVs for selection as it can promote greater rates of inbreeding in the course of generating more progress therefore awareness to inbreeding should also be considered during selection for breeding (Lewis, et al., 2013).

Table 2 Parameter estimates of elbow score (Lewis, et al., 2013).

	σ_P^2	σ_A^2	h^2	s.e.	r_A	s.e.	r_E	s.e.
BMD	0.760	0.201	0.26	0.054	0.005	0.134	0.122	0.051
GR	0.278	0.084	0.30	0.054	0.137	0.098	0.095	0.050
GSD	0.265	0.048	0.18	0.062	0.203	0.140	−0.054	0.055
LAB	0.196	0.037	0.19	0.028	0.344	0.064	−0.003	0.024
ROTT	0.533	0.073	0.14	0.106	0.550	0.299	−0.091	0.091

Estimates of phenotypic and genetic variance (σ^2_P and σ^2_A respectively) and heritability (h^2) of elbow score and genetic and residual correlations (r_A and r_E respectively, with standard errors) with hip score for 5 breeds. Breed abbreviations: Bernese Mountain Dog [BMD], Golden Retriever [GR], German Shepherd Dog [GSD], Labrador Retriever [LAB], Rottweiler [ROTT].

CONCLUSION

For many decades now ED has been posing a problem for canines across a wide range of pedigree breeds due to our lack of knowledge and assertiveness in controlled breeding measures. History of ED dates back as far as the 1950's and the syndrome that we now know today as elbow dysplasia was first described in the 1970's and yet despite decades of existence exact inheritance is unclear. It has been concluded that ED is not a single compound disorder but a multifactorial disorder comprising of a variety of primary lesions with the majority of the research focused on FCP, UAP and OCD as the main issues involved. Since the 1950's much progress has been achieved thanks to associations such as the IEWG and the BVA who have brought ED to the forefront of the canine medical field encouraging recent research methods against the disease. These associations have implemented health screening protocols across all breeds to which are predisposed to ED, these protocols yielded grading of elbow joint based on the severity of each aspect of ED resulting in valuable information for breeders and researchers alike. Several research papers have produced results confirming ED to be a genetically linked disorder however the exact causative gene still evades our understanding. With results from I.C.M. Lavrijsen et al. discovered heritability rates were high enough to warrant successful results would be obtained with selection against the occurrence of ED. IT has been proven for many years to be a genetic disease however in 2001 this was elaborated on by A. Ruvinsky et al. to be a polygenic syndrome and it is due to this fact that the exact cause still eludes us. Although research to obtain a causative major gene is nearing a breakthrough as evidence supporting their existence was concluded by T.W. Lewis et al. with some breeds proving to be more advanced for the presence of a major gene than others due to bimodal distribution of ED scores, this has not yet been confirmed.

There has been a huge increase in the awareness of breeders and the general public to breeding programs which have been proven to decrease the incidence of ED. This awareness has also lead to an increase in voluntary examinations of breeding animals, which in return widens the database on ED making research more accurate. Studies completed on the selection methods against the disease based on phenotypes have thus far proved beneficial causing a decrease in prevalence over the years. Results from these studies have acted as a platform for further advances and it is now believed

that EBVs would be a more accurate depiction of an animal's genetic effects therefore a more defined base for selections across predisposed breeds. Furthermore it would increase the momentum of progress started by phenotype selection alone. One flaw that was noted with the proposed use of EBVs for selection was the increased incidence of inbreeding however with close attention and deliberate selection opposing inbreeding this should not be a concern (Lewis, et al., 2013).

Future propositions

I believe that there are many steps which could be easily implemented to improve the genetics against ED for the future. First off I feel a wider more elaborate screening method could be implemented to ensure a more accurate base for selection, as the selection has a direct link to genetic progress in breeding for health (Lewis, et al., 2013). As phenotype selection has been proven to have a beneficial effect on the genetic progress across all breeds and that EBVs have been heavily researched and are believed to be more accurate than solely phenotype selection; providing an index for ED from which breeding animals can be selected on their high or low genetic merits against ED based on their EBVs could accelerate improvements. These improvements to reduce the prevalence could also be increased if the percentage of animals that are high risk for the disease was increased to restrict a wider range from the breeding programs. As I feel restricting less than ten percent of the high risk breeders is a passive approach and a more aggressive approach would yield a larger impact on the results thus far. Although research on DNA scanning and genome mapping is inconclusive as of yet, I feel that this may be the future of screening against ED as it would provide a non-subjective, definitive report on the breeding animal's genetics for development and heritability potential.

The IEWG hold annual meetings outlining new developments made on the disease and with their hard work along with the co-operation of breeders I feel ED is soon going to become a condition of the past.

ABBREVIATIONS AND ACRONYMS

BVA – British veterinary association

BL – Borderline

BMD – Bernese Mountain Dog

CT – computer tomography

DJD – Degenerative joint disease

DNA - Deoxyribonucleic acid

DUO – Dynamic ulnar osteotomy

EBV – Estimated breeding value

ED – Elbow dysplasia

FCP – fragmented coronoid process

GR – Golden Retriever

HD – Hip dysplasia

IEWG – International Elbow working Group

INC – Incongruity

KC – Kennel club

LR – Labrador Retriever

MRI – Magnetic resonance imaging

ROTT – Rottweiler

OA – Osteoarthritis

OCD – Osteochondritis dissecans

UAP – Ununited anconeal process

US – Ultrasounds

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