

# OCD – its development and effect on Elbow Dysplasia

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## Definitions of terms:-

**Osteochondritis (OC)** arises from an error in conversion of cartilage to bone in the rapidly growing dog, resulting in the development of thickened and/or necrotic cartilage within joints and growth plates.

**Osteochondritis Dessicans (OCD)** is a progression of OC resulting in deep cracks and fissures within the cartilage, as well as joint swelling and lameness. OCD results from a combination of genetic, nutritional and rapid growth factors.

**Osteoarthritis (OA)** develops over time if there are remnants of cartilage flaps retained within joints and where incongruent joint surfaces have developed subsequent to the OCD lesions.

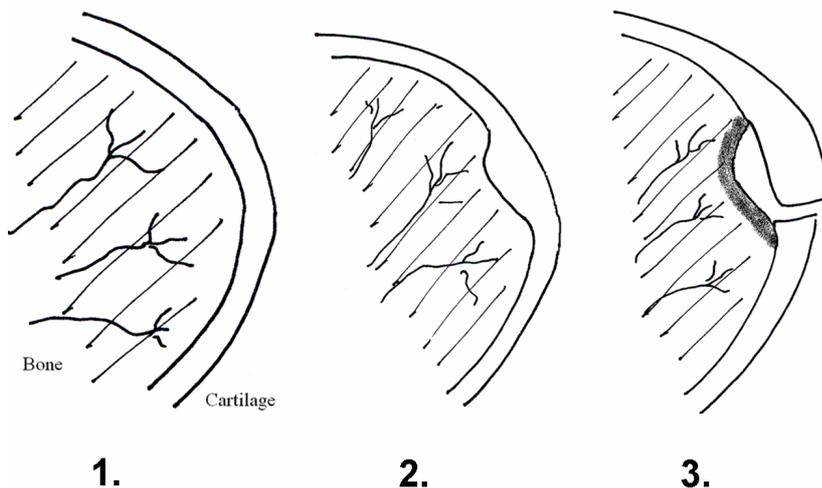
**Osteochondritis** is a developmental bone disease characterised by defective cartilage in various to multiple joints. The main feature of OC is **a failure of the cartilage to properly convert to bone** during the growth process. Due to rapid growth, the cartilage cells divide at a normal or increased rate, but the cells do not fully mature. This can result in thickening of the cartilage in various areas of the joint and a delay in bone formation.

As the cartilage continues to grow without being normally converted to bone, it loses part of its nutrition because of its increased thickness. Cartilage has no direct blood supply and relies on nutrition from the joint fluid and the underlying developing bone. The thicker cartilage results in loss of blood supply, in turn causing necrosis or death of the lower layer of cartilage.

Once this happens, the overlying cartilage separates from the underlying bone (which can also occur as a result of trauma) causing small cartilage “flaps” to lift. Such splits allow synovial (joint) fluid in direct contact with underlying bone, creating pain and an inflammatory response. This in turn causes the joint capsule to react and become inflamed as well, creating bursitis (joint swelling).

Cartilage flaps or loose sections that break away (often called “joint mice”) results in continuing pain and inflammation. Small loose fragments can attach to the joint capsule or migrate into a connecting tendon sheath connected to the joint capsule, causing chronic inflammation around the tendon sheath (most commonly seen in shoulder OCD).

Diagrams- Development of OCD



## Development of OCD

1. Normal Development of a joint surface

2. Failure of developing cartilage to convert to bone **results in a delay in bone formation** and a flattening effect and a thickening of the cartilage. Changes can be due to reduced blood supply, genetic and environmental factors.

3. Loss of nutrition to the lower sections of thickened cartilage results in necrosis and splits in the cartilage. Lifting of the cartilage exposes bone tissue to joint fluid, resulting in an inflammatory response and joint pain.

**One of the important features of OC is to remember that on X-ray, cartilage is radiolucent. Therefore, early changes to cartilage are not readily detectable until there is a delay in the bone formation underneath the actual cartilage defect, creating a “flattening affect” of the underlying developing bone.**

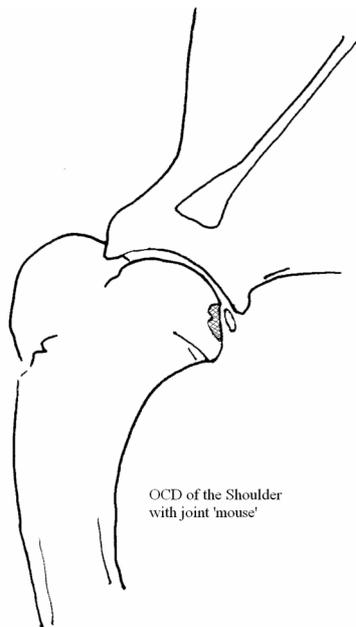
**Early lesions of OC can go three ways:**

1. Heals through rest and appropriate treatment such that there is a return to normal bone and cartilage development and maturation
2. Remains unchanged
3. Progress to OCD, the development of arthritis and of clinical lameness

**OCD** can occur in many different joints, most commonly in the elbows, followed by shoulders, and less commonly in the hocks. The affected joints can be single or bilateral. Different breeds have higher incidences or predilections for various joints to be affected.

**Osteoarthritis (OA)** or chronic joint disease develops further over time.

*Diagram - OCD of the Shoulder*



## **Joint Disease and Rapid Growth**

Many of the joint diseases discussed in this chapter occur in the younger dog and can arise as a consequence of rapid growth in an increasingly heavy breed of dog (over time).

Osteochondrosis and joint dysplasias have been studied in many species, in particular in pigs. In pigs, where the animals were selected for an increasingly heavy end weight and for rapidity of weight gain, the incidence of symmetrical lesions in joints and many growth plates. Experimentally in pigs, the incidence and severity of OCD was **directly related to rapid growth** ie. rate of weight gain. When the diet was restricted and they were grown at a slow growth rate, the incidence of OCD was dramatically reduced (almost to zero).

All dog studies in this area support the concept that high caloric intake rather, than the specific intake of protein, minerals or vitamins, influences the frequency and severity of osteochondrosis and HD. The causes of ED while not as thoroughly studied, show similarities and probably similar outcomes.

The common conclusion from studies on dogs is that excessive calcium, phosphorus and vitamin D along with a high energy diet and rapid weight gain causing rapid growth, are almost a sure fire recipe for pushing the parameters for normal structural growth and joint soundness well beyond their normal limits, resulting in joint disorders.

The higher incidence of osteochondrosis in males versus females is probably a direct reflection of this as males can be anywhere between 15-25% heavier than females at any one time (and certainly by full maturity), despite being born at a comparable weight.

Equally, this is not to say that genetics does not play a very important part in the body's structural soundness. Excessive rates of weight gain and thus rapid growth result in pushing the body's parameters beyond which they can cope, particularly if they were not the most structurally stable to start with. For example, an excessive rate of growth and weight will not create severe HD in itself but it can make an existing problem considerably worse.

### Rate of Weight Gain

*Rapid weight gain and rate of growth through excessive nutritional intake may cause a disparity of development of supporting tissues.*

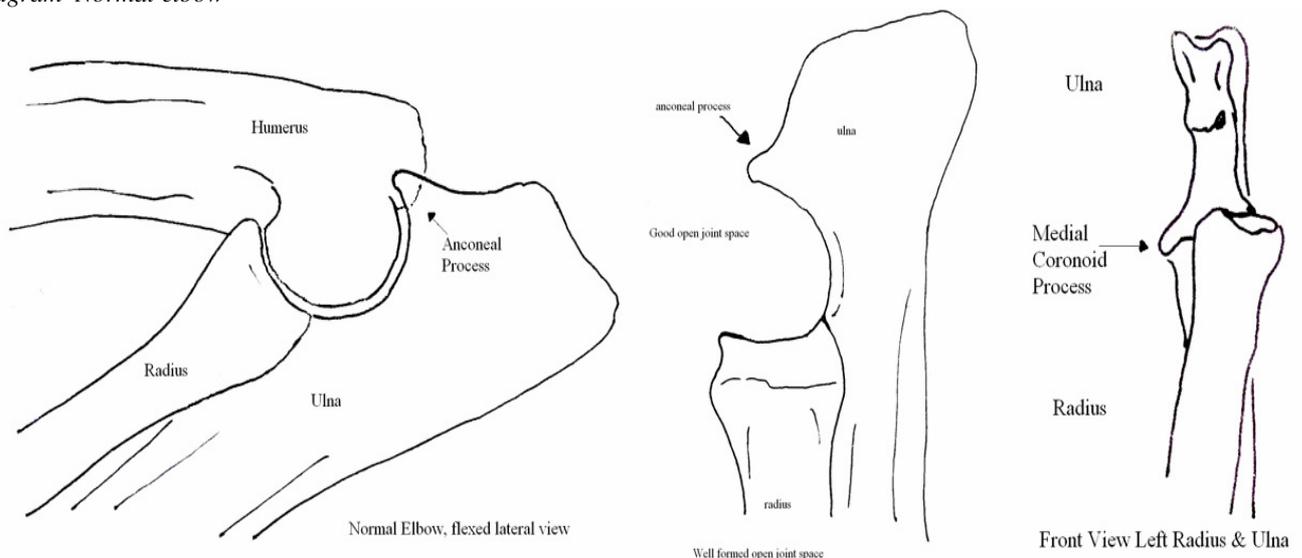
Factors affecting cartilage integrity (thickness and stability) and joint fluid composition, such as repeated trauma from excessive looseness of the joint, can increase joint fluid production, thickening of the joint capsule, resulting in both joint pain and reduction in joint stability. These factors contribute to the development of joint looseness and subsequent subluxation, resulting in early clinical signs and joint changes.

### Elbow Dysplasia (ED)

The elbow is quite a complicated joint, as there are 3 bones that must line up *precisely* in order to form a stable working joint.

*Any deviation from proper congruity between the various bones, either in length between the weight bearing surfaces of the radius and ulna, or a reduced ulna notch where the distal end of the humerus sits to form the top half of the joint, will result in arthritis.*

Diagram Normal elbow



- a) normal elbow, lateral view
- b) lateral view showing good open joint space
- c) Front view, left radius and ulna

ED results from an incongruity of the joint, mainly as a result of under development of the ulna trochlear notch (the inside surface of the joint at the top of the ulna). If the incongruity is minor, there may be minimal changes with no obvious clinical problems. The incongruity results in a step down effect from a poorly developed ulna notch in relation to the weight bearing surface of the top of the radius. If the incongruity is large, a combination of the following 3 major conditions may be seen – UAP, OCD, FCP.

Another form of elbow dysplasia can be seen from radial over growth (relative to the ulna) due to early closure of the distal growth plate of the ulna. Again this is where the cartilage core in this growth plate fails to get adequate nutrition causing a delay in growth and resulting in a shortened ulna and overgrowth of the radius. This causes incongruity in the elbow and in severe cases may result in bowing of the front legs, lateral luxation of the proximal end (near the elbow) of the radius. The resulting incongruity can lead to a combination of the above conditions. FCP (medial Fragmented Coronoid Process) Occasionally, after the anconeal process has united, there can be later development of joint incongruity. The micro-movement and stresses placed within the joint can result in a fragmented coronoid process, which nearly always affects the medial process.

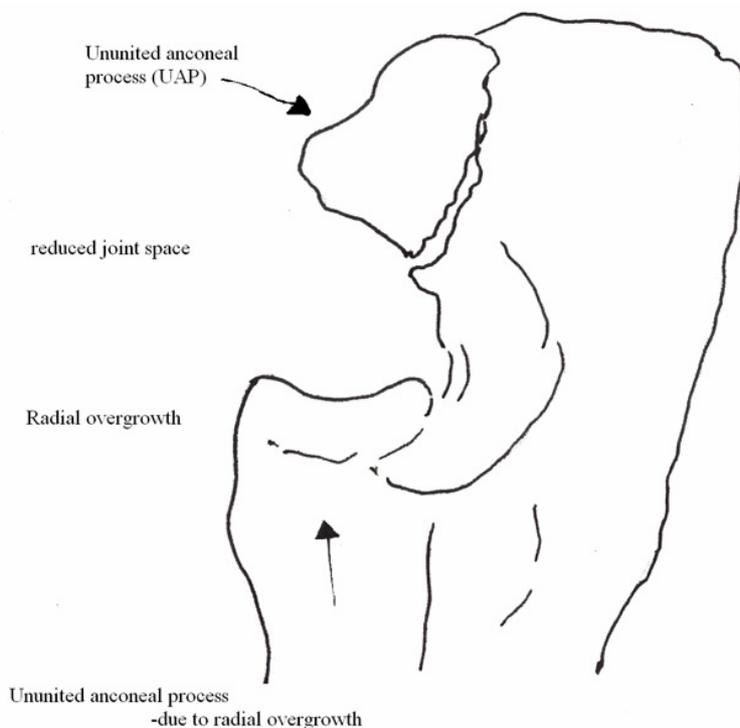
***There are 4 main subgroups of elbow dysplasia*** which may occur alone or in combination. Generally they are considered inherited in most breeds and the incidence may be adversely affected by incorrect diets in some breeds.

Excessive rate of weight gain can affect the incidence of many elbow conditions, often leading to joint incongruity (uneven rate of growth between the radius and ulna), and OCD, particularly in males.

### **i.UAP – Ununited anconeal process**

The anconeal process is a large triangular shaped piece of bone situated at the back of the elbow joint. This has a separate ossification centre in a handful of breeds mainly larger, heavier breeds notably the GSD, Labrador, Great Dane, Irish Wolfhound, Newfoundlands, Bloodhound, Basset and Dogge de Bordeaux. This process normally is fully ossified (sealed) by 16-18 weeks of age. This condition is considered inherited with a possible 3 different genes being involved.

*Diagram - UAP due to radial overgrowth*



The anconeal process forms the hinge at the back of the elbow. When the anconeal process has a separate ossification (growth area) in breeds such as in the GSD, this area becomes a target for poor conversion of cartilage to bone. The combination of the separate ossification centre and a too small circumference of the ulna trochlear notch (inside edge of the back of the joint) results in small movements along the cartilaginous bridge between the ossification centre of the anconeal process and the ulna. This results in failure of the anconeal process to unite. Chronic movement of the UAP results in ongoing development of joint arthritis and further wear and tear of the cartilage surfaces of the joint.

Closure of the ossification centre -The anconeal process in the GSD should be closed by 16-18 weeks of age, definitely by 20 weeks of age. If screening for UAP, X rays taken at 20 weeks should identify a problem if it is present.

**Symptoms** - Clinically signs are seen from as young as 5-6 months with intermittent lameness, which is exaggerated by exercise. Full flexion and extension of the elbow will elicit pain. There is often an accompanying lateral bursitis (fluid swelling).

**Diagnosis** - X rays of the elbow in the flexed lateral position will readily show whether the process has unified or not. Long standing cases often have bony arthritic changes as well.

**Treatment** (depending on other conditions/joint incongruities present) is either:

- a) removal of the process which, if done while young, usually has a reasonable prognosis with some arthritis later.
- b) fixation of the loose fragment by a lag screw will also give a reasonable prognosis in early cases.

As the anconeal process is not a weight bearing surface, early surgery gives better long term prospects than in the FCP cases (as loss of that process affects the weight bearing surfaces of the joint).

The majority of UAP cases in the GSD have good congruity of the head of the radius relative to the ulna. Where there is poor congruity, ie. the radius is too long for the ulna, an osteotomy (cutting) of the ulna to allow it to lengthen naturally is another option.

**Affected dogs should not be breed with.** UAP is generally considered to be inherited as a separate condition to OCD and FCP in the GSD and this may be true for other breeds as well.

## **ii. OCD – osteochondritis dessicans (see above as well)**

**OCD** occurs in many larger breeds and can affect almost any breed greater than 18-20kg at adulthood. There is a higher incidence in males versus females. This can affect many joints, the commonest being the elbow. Breeds that see with a reasonably high incidence of elbow OCD would include the following: Rottweiler (high incidence), GSD, Golden Retriever, St Bernard, Great Dane, Border Collie, Rhodesian Ridgeback and Labrador.

**Symptoms** – OCD is generally seen as a shifting lameness in the forequarter from around 5-8 months of age, some joint capsule swelling and usually a turning out of the front legs at stance as the inner edges of the elbow are most commonly affected. The cause of the problem is considered to be due to a faulty blood supply to the joint cartilage secondary to very rapid growth.

**Diagnosis** - On X ray the signs are often quite subtle in mild cases with minor “fluffiness” and/or flattening of the joint surfaces to the more distinct pot holes of larger lesions. It is generally diagnosed by X rays of a straight extended and slightly medially rotated view of the elbow.

**Treatment** - If the condition is mild, treatment with drugs such as Cartrophen\* which increases the blood supply to cartilage can be very effective along with complete rest, slowing down of the rate of weight gain and low doses of anti-inflammatories. Repair and recovery can take up to 6-8 weeks, depending on the severity of the condition. Very heavy puppies may have to be kept reasonably restricted until 9 months of age by which stage all rapid growth has slowed dramatically. Severe cases of OCD are often found in conjunction with FCP, and may require surgical intervention. Most cases >80% show good responses to medical, dietary and exercise management.

## **iii. FCP – fragmented coronoid process**

FCP of the ulna generally refers to the medial coronoid process, a process that stabilises the medial edge of the joint. Fragmentation of this process means that the inside edge of the elbow is not stable, hence the very typical lateral rotation of the leg away from the pain. It affects the same age group as above.

Diagram - FCP/OCD lesions – medial view, left elbow

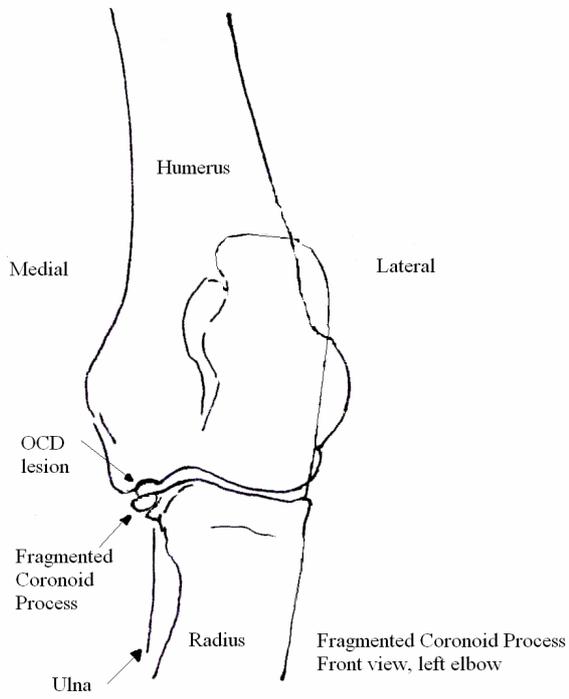
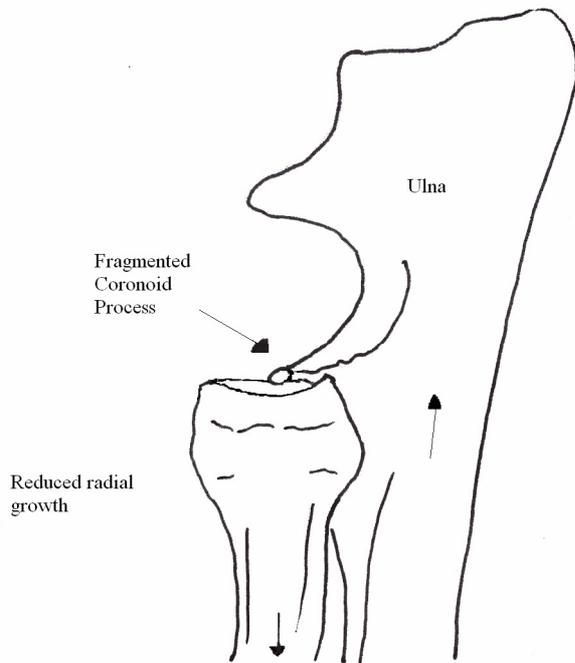


Diagram - FCP lesion - reduced growth of radius leading to FCP



**Diagnosis** - On X ray, with the elbow extended straight out and a second view with slight medial rotation the process can be visualised and assessed. The coronoid process can be seen as being separated from the main section of the ulna.

**Treatment** - If these are diagnosed early, surgical intervention can give reasonable results. In the older dog, where there are considerable secondary arthritic changes, medical management with tablets is probably preferred.

**Outcome** - Regardless of the treatment, the resulting joint incongruity (unevenness) will lead to osteoarthritis over time.

#### **iv. Joint Incongruity**

While most forms of elbow dysplasia can, by their development, result in joint incongruity, what we are looking at here is where there has been a possible early closure of a distal growth plate in the foreleg resulting in uneven growth of the radius (usually slightly shorter) in relation to the ulna. The resulting uneven ends of the bones within the joint can cause excessive wear on cartilages and in the worst cases, force the anconeal process distally (ie. create a UAP).

Other forms of elbow "dysplasia" exist which generally involve the lateral displacement of the top of the radius in relation to the ulna (giving a cabriole effect) but these are uncommon.

#### **General Treatment of Elbow Dysplasia**

As with any painful bone disorder, regardless of the age presented, common treatment is aimed at pain management, sensible diet and weight control and a restricted, suitable exercise regime. Where there are only minor changes in joint surfaces, medical management and conservative treatment with anti-inflammatory agents and rest is generally all that is needed.

Those animals with UAP require surgical intervention to minimise future arthritis. Younger dogs diagnosed with a FCP or loose cartilaginous flaps, should have these removed surgically in order to minimise future damage to the joint. Due to the incongruity of the joint, there will be ongoing arthritic changes. In the older dog with advanced arthritic changes, medical management with conservative exercise regimes is, generally, the preferred method of treatment.

#### **Diet and Elbow Dysplasia**

While diet may not of itself create elbow dysplasia, it can affect the severity of OCD seen. Rapid weight gain will push factors such as joint congruity, as well as affecting the blood supply to the cartilage within the joints. Reduce the energy levels in the diets, particularly for dogs that need to be confined, and ideally, change puppies onto an adult maintenance diet. Affected puppies should have their weight at or below the normal for their breed, sex and age.

#### **Rule outs (differential diagnoses)**

***Not all forelimb lameness is due to elbow dysplasia!*** Panosteitis and simple injuries should be checked for, particularly where there is a very sudden onset of lameness.

#### **Controlling the incidence of Elbow Dysplasia –**

The major task in reducing the severity and or incidence of any breed inherited disorder, is establishing the mode of inheritance. The inheritance of problems such as hip and elbow dysplasia where there are two or more genes affecting the inheritance pattern, one can only, at this stage use quantifying tests such as X rays and sires progeny results to assist in our control schemes. Some genes can behave incompletely, ie. blend effects with a normal gene and not be expressed, as the normal gene carries sufficient enzyme making ability to hide the effects of a defective gene. The animal will appear normal but is in fact a "carrier" of the abnormal gene.

*The more genes affecting a characteristic, the harder and slower it is to eradicate or affect the characteristic and the more environmental effects may come into play (weight, diet, rate of growth etc).* Where there are ways to **measure** the effect of the characteristic, then progress can be made in controlling the effect of the polygenes in the overall population eg. X raying of individuals and their progeny.

The schemes currently in use for control/reduction in severity of HD and ED aim to reduce the incidence and overall severity of these conditions across a breed a) as a whole and b) over time. Trying to shift the genetic

structure of polygenic conditions within a breed is a long term goal and cannot be pushed rapidly without severe consequences in other areas (eg. type, temperament etc).

. As this is a group of highly inherited conditions, fairly rapid improvements can be made over reasonably short periods of time. Breeding from severely affected dogs should be heavily discouraged. Generally dogs with a UAP, FCP and arthritis of greater than 4mm should not be used for breeding.

### **Grading of Elbow X rays**

When reading or scoring elbow X rays, the major measurement is done on the caudal (back) edge of the anconeal process. When this process is clean and shows no changes, then the elbow is graded normal. Grade 1 dogs are allowed up to just under 2mm of arthritic changes, Grade 2 dogs are allowed between 2mm to just under 4mm. UAP, 4mm of arthritic change and greater are automatic fails.

Occasionally a dog may have no changes on the anconeal process, but have minor changes on the ulna around the joint or the space between the top of the radius and ulna. While these dogs may receive a 0 from the anconeal process, they may then receive a Grade 1 due to minor arthritic changes elsewhere around the edges of the joint.

Breeding from dogs with significant arthritic changes (Grade 2) in the elbows should only be with care and *ideally always be to normal partners and preferably to those lines with low incidences of elbow problems*. Grade 1 animals can be bred to a wider range of animals, but, again, ideally to normal partners.

With the elbow results, as with the hips, the average score of the sire's progeny (where more than 20 progeny are scored) is of greater benefit in predicting the genetic worth of the dog than it is the score of the sire himself. To try and give an extra loading on results, we can loosely grade sires by the % of normal elbows being produced. Slight variations due to state averages (local population genetics) can effect these results.

Sires can further be loosely grouped on their progeny results and the % of normals. These sires can then be graded as to their effectiveness as sire for producing good elbows. I would suggest the following loose designation in assessing sires for the soundness of their elbow producing.

#### **Sires that produce greater than:-**

**>85% normal elbows should be considered excellent producers of good elbows**

**>70% normal elbows should be considered very good producers of elbows**

**>60% normal elbows should be considered good producers of elbows**

#### **Sires that produce less than :-**

**< 60 % normal elbows should be considered poor producers of elbows**

***The whole idea of these schemes is to give breeders information so as to give them knowledge prior to breeding an animal as to what one can afford to do.***

The conclusion one gets from these schemes, is that the more information one has for both of the parents and of their close relatives, particularly offspring of the sire, the better one can plan and get successful results across a litter. In breeds where such information is limited and/or sire statistics are not available, breeders have a much harder time selecting good sires and good breeding combinations.