Elbow dysplasia in the dog: pathophysiology, diagnosis and control

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ABSTRACT

Elbow dysplasia is a non-specific term denoting abnormal development of the elbow. Elbow dysplasia encompasses the clinical and radiographic manifestation of ununited anconeal process, fragmented medial coronoid process, osteochondritis dissecans, erosive cartilage lesions and elbow incongruity. The net result is elbow arthrosis, which may be clinically inapparent or result in marked lameness. These conditions may be diagnosed by means of routine or special radiographic views and other imaging modalities, or the precise cause of the arthrosis or lameness may remain undetermined. Breeds most commonly affected are the rottweiler, Bernese mountain dog, Labrador and golden retriever and the German shepherd dog. Certain breeds are more susceptible to a particular form of elbow dysplasia and more than 1 component may occur simultaneously. The various conditions are thought to result from osteochondrosis of the articular or physeal cartilage that results in disparate growth of the radius and ulna. Heritability has been proven for this polygenic condition and screening programmes to select suitable breeding stock have been initiated in several countries and have decreased the incidence of elbow dysplasia.

Key words: elbow dysplasia, elbow incongruity, fragmented medial coronoid process, osteochondritis dissecans, ununited anconeal process, screening programmes.

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INTRODUCTION

Elbow dysplasia is the abnormal development of the elbow joint. The term dysplasia is derived from the Greek words *dys* meaning abnormal and *plasia* meaning development. Elbow dysplasia is an all-encompassing term comprehensible to the dog-breeding fraternity and has gained popular recognition, particularly in communicating scientific information to the public[2,10,40]. In scientific publications, however, specific aetiologies should be referred to where possible[10] 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[40]. 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 to undetected elbow dysplasia may only be diagnosed later in life. In particular, lameness in both elbows may result in a peculiar gait that the owner may not have realised was abnormal. Often minor elbow dysplasia resulting in mild arthrosis may be present with no clinical evidence and breeders are unaware of the problem in their breeding stock[36]. In a study of rottweilers, 57 % of dogs had radiographic signs of elbow dysplasia but only 15 % were lame[36].

Developmental elbow abnormalities included in the term elbow dysplasia are fragmented medial coronoid process (FMCP), osteochondrosis and osteochondritis dissecans (OCD) of the medial humeral condyle, ununited anconeal process (UAP), humero-ulnar, humeroradial and radio-ulnar incongruity and articular cartilage erosion. These conditions, singly or in combination, result in irreversible elbow arthrosis with resultant pain and lameness. Osteochondrosis, the disturbed endochondral ossification of articular or physeal cartilage, is most likely the underlying cause for all the conditions included in elbow dysplasia[31-33]. Underdevelopment of the trochlear notch with secondary incongruity is postulated by others to be the primary cause[45,47,50]. In osteochondrosis, normal cartilaginous development and maturation fails in the hypertrophic zone, resulting in thickened cartilage[31]. In the elbow joint, articular cartilage involvement results in OCD while non-articular cartilage alterations are assumed to result in FMCP, UAP and elbow incongruity, probably as result of small growth abnormalities of the long bones making up the elbow joint[32]. The cartilaginous growth disturbance is likely to have genetic and environmental, mainly traumatic, and nutritional causes[31]. The most important nutritional factors are an excess supply of energy and relative over-nutrition with calcium[31]. Trauma is usually minimal and associated with hyperactivity or excessive body weight[31]. Figure 1 denotes the relationship of factors involved in elbow dysplasia and the development of arthrosis. Severe trauma resulting in premature closure of a physis is a separate clinical entity causing severe growth disturbance of the affected long bone that may markedly influence joints adjacent to the traumatised physis, and is excluded from the elbow dysplasia syndrome.

Elbow dysplasia is a worldwide problem in intermediate and heavy-set breeds and is commonly seen in South Africa in the rottweiler, Labrador and golden retriever, and German shepherd dog. Other breeds commonly affected worldwide are the Bernese mountain dog, Saint Bernard, Newfoundland, and bull mastiff. The condition is seen sporadically in many other breeds. Breeds may be predisposed to a particular form of elbow dysplasia, *e.g.* the German shepherd dog suffers more from UAP[16], the rottweiler rarely has OCD[11,17] and the Labrador retriever is most likely to have combined OCD and FMCP[17]. The condition is more likely to occur in males, probably owing to their faster rate of growth or a sex-linked factor[5,11,17,20]. Elbow dysplasia is a polygenic and multifactorial condition, the incidence of which can be reduced by selective breeding[15,20,40]. 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

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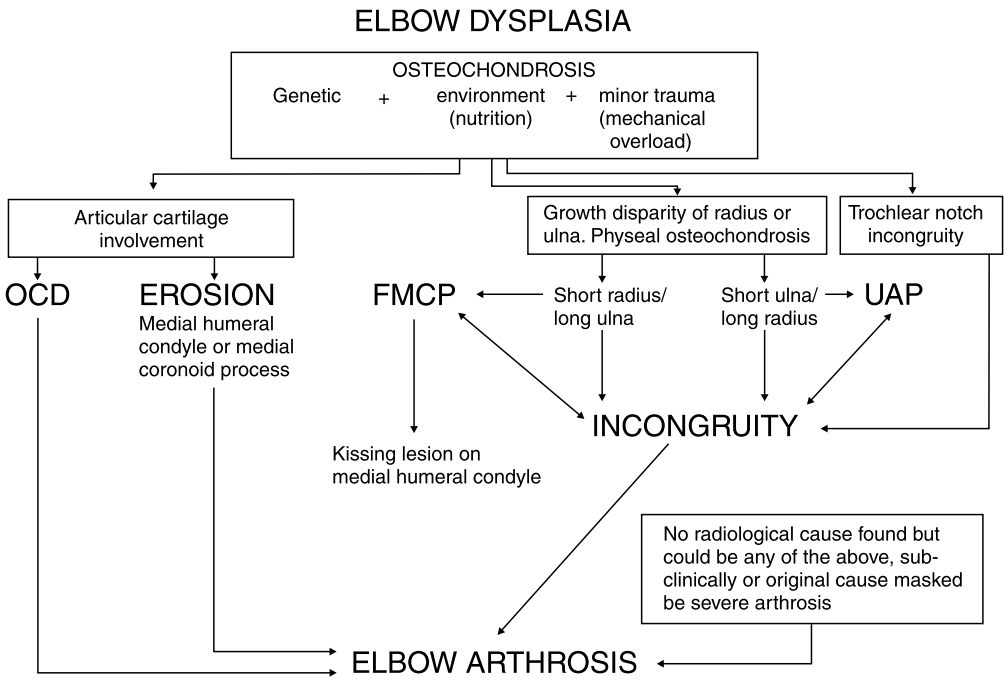


Fig. 1: **Flow diagram of interrelationship between elbow dysplasia and elbow arthrosis**.

owners must be made aware that medical or surgical treatment may result in improvement but not normality[48]. The objective of this article is to review the pathophysiology and diagnosis of the various conditions involved in elbow dysplasia, briefly consider therapy and discuss measures to prevent and control the condition.

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 are often only suggestive of an abnormality. This is often the case in the growing dog when arthrosis has not yet developed. Three-dimensional imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) are the most reliable methods, as they allow slices of the affected joint to be evaluated. Linear tomography has also proven to be of benefit[9,44]. Positive contrast arthrography, although technically fairly easy to perform, does not contribute to evaluating FMCP but could assist in the diagnosis of OCD[27]. The contralateral elbow should always be imaged because of the high incidence of bilateral disease or as a control[11,26,32,48]. If the origin of a forelimb lameness is uncertain, shoulder radiographs should also be made to exclude OCD of this region. A small number of dogs may have lesions in both the shoulder and elbow[32]. In the rottweiler, the palmar metacarpal region should be evaluated to exclude palmar metacarpal sesamoid pathology[36]. Radiographs of the rest of the limb may be required to exclude other conditions such as panosteitis.

Radiography

Optimal radiographic detail is essential to accurately evaluate elbow pathology. This is obtained by using table-top techniques, non-grid exposures, collimating to the elbow joint, centring the primary beam on the medial epicondyle of the humerus, using detail-intensifying screens and short-scale contrast (low kVp and high mAs exposure technique). Sedation or anaesthesia may be indicated for accurate positioning. Standard craniocaudal and mediolateral views have been supplemented by numerous additional views in an attempt to highlight certain anatomical locations or pathological conditions. Minor lesions such as erosion of the medial humeral condyle cartilage will not be visible in any of these views. Microfocal radiography of the elbow has been described and yielded more information than standard radiography but some lesions were still not detectable and this is not a readily available technique[19].

Lateral recumbency – routine mediolateral view of the joint (semi-flexed ML) (Fig. 2a)

The angle between the humerus and radius and ulna is about 110°. This view allows for the most accurate evaluation of joint incongruity but true lateral positioning is vital. Humeral condyles can be evaluated for OCD but minor changes may not be detected. The medial coronoid process is seen superimposed on the proximal radius with its cranial tip at the level of the radial physis. Osteophyte reactions on the cranial aspect of the joint are seen more readily than in the flexed ML view. Early osteophyte formation dorsally on the anconeal process may be obscured by the medial humeral condyle, particularly on suboptimal radiographs.

Lateral recumbency – mediolateral view with the joint maximally flexed (flexed ML) (Fig. 2b)

The angle between the humerus and radius and ulna is about 45°. This view maximally exposes the anconeal process and thus optimises visibility of UAP and early osteophyte formation within the dorsal hollow of the anconeal process. This view does not contribute much to the demonstration of OCD and FMCP compared to the other views[44].

Lateral recumbency – mediolateral view with the joint maximally extended and the limb supinated approximately 15° (extended supinated ML = Cd75°MCrLO)[30,44] (Fig. 2c)

This projection results in a true lateral view of the medial coronoid process. The radiograph allows the cranial border of the medial coronoid process to be seen in 94 % of cases as compared to 50 % and

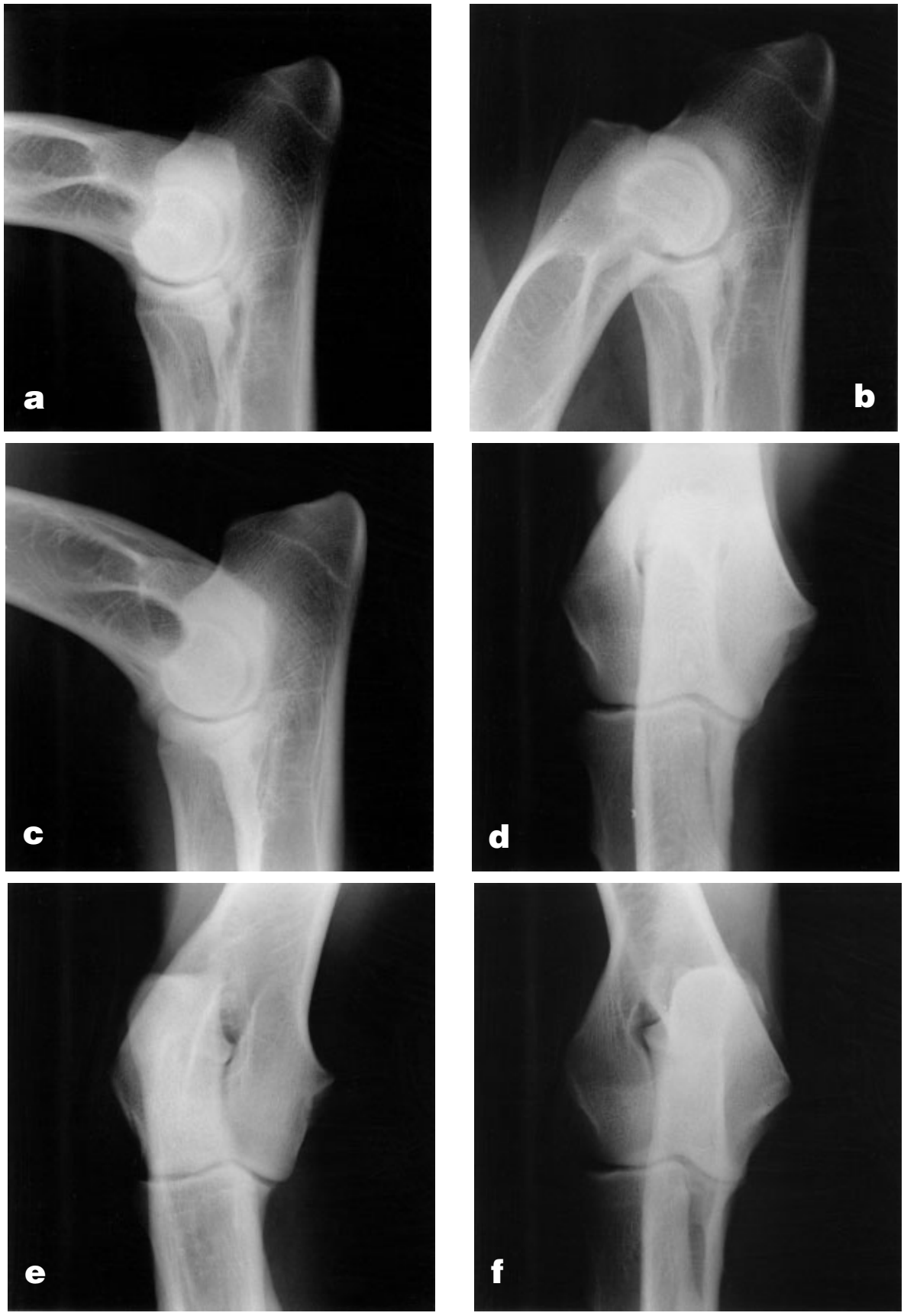


Fig. 2a: **Lateral recumbency – mediolateral view with the joint semi-flexed** (ML).
Fig. 2b: **Lateral recumbency – mediolateral view with the joint maximally flexed** (flexed ML).
Fig. 2c: **Lateral recumbency – mediolateral view with the joint extended and the limb supinated approximately 15°** (extended supinated ML=Cd75°MCrLO).
Fig. 2d: **Sternal recumbency – true craniocaudal view** (CrCd).
Fig. 2e: **Sternal recumbency – craniolateral to caudomedial oblique view** (Cr30°LCdMO).
Fig. 2f: **Sternal recumbency – craniomedial to caudolateral oblique view** (CrMCdLO).

56 % respectively in the semi-flexed ML and flexed ML views[30]. One study proved this view to be more reliable in detecting FMCP than the ML view[31].

Sternal recumbency – true craniocaudal view (CrCd) (Fig. 2d)

Useful to detect osteochondral defects of the medial humeral condyle but some lesions may only be seen in the extended supinated ML view, or if they are located more cranially, only in the ML view[44]. Osteophyte reactions on the medial humeral epicondyle and medial coronoid process are readily seen.

Sternal recumbency – craniolateral to caudomedial oblique views (Cr30°LCdMO)[30,44] (Fig. 2e)

This view is also known as the pronated CrCd view and is used to skyline the medial coronoid process and medial humeral condyle. The cranial aspect of the medial coronoid process is seen *en face* in this view making it difficult to see cleavage lines[30]. Osteophyte reactions on the medial humeral epicondyle and medial coronoid process and osteochondral defects of the medial humeral condyle are readily seen.

Sternal recumbency – craniomedial to caudolateral oblique view (CrMCdLO)[44] (Fig. 2f)

Also known as the supinated CrCd view and highlights the lateral humeral condyle and as such does not contribute to the evaluation of conditions involved in elbow dysplasia.

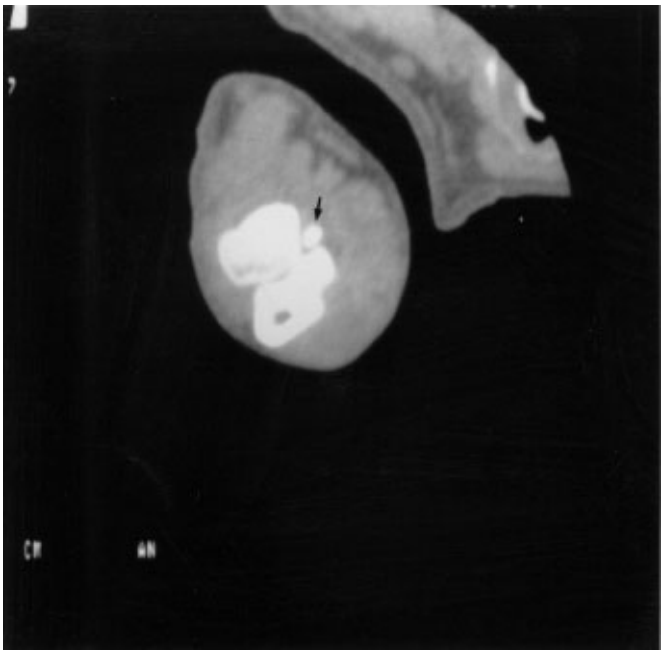


Fig. 3: **Computed tomography transverse cross-section of the radius and ulna, illustrating a fragmented medial coronoid process** (arrow) (courtesy of P Steyn, Colorado State University).

Computed tomography (Fig. 3)

Computed tomography is significantly more accurate than radiography, linear tomography and xeroradiography for the diagnosis of FMCP owing to its exceptional contrast resolution and ability to image the medial coronoid process in a 3rd plane[6]. Non-mineralised cartilage fragments occur more commonly than mineralised cartilage fragments and are much more likely to be seen with CT than with radiographs[6].

Magnetic resonance imaging

Owing to cost considerations, MRI is rarely used for elbow evaluations but is more accurate and sensitive than radiographs for the detection of FMCP and OCD. Additionally it allows non-displaced, non-mineralised fragments to be seen[39].

Linear tomography (Fig. 4)

Linear tomography is a useful technique to demonstrate potential disease areas that are obscured by super-

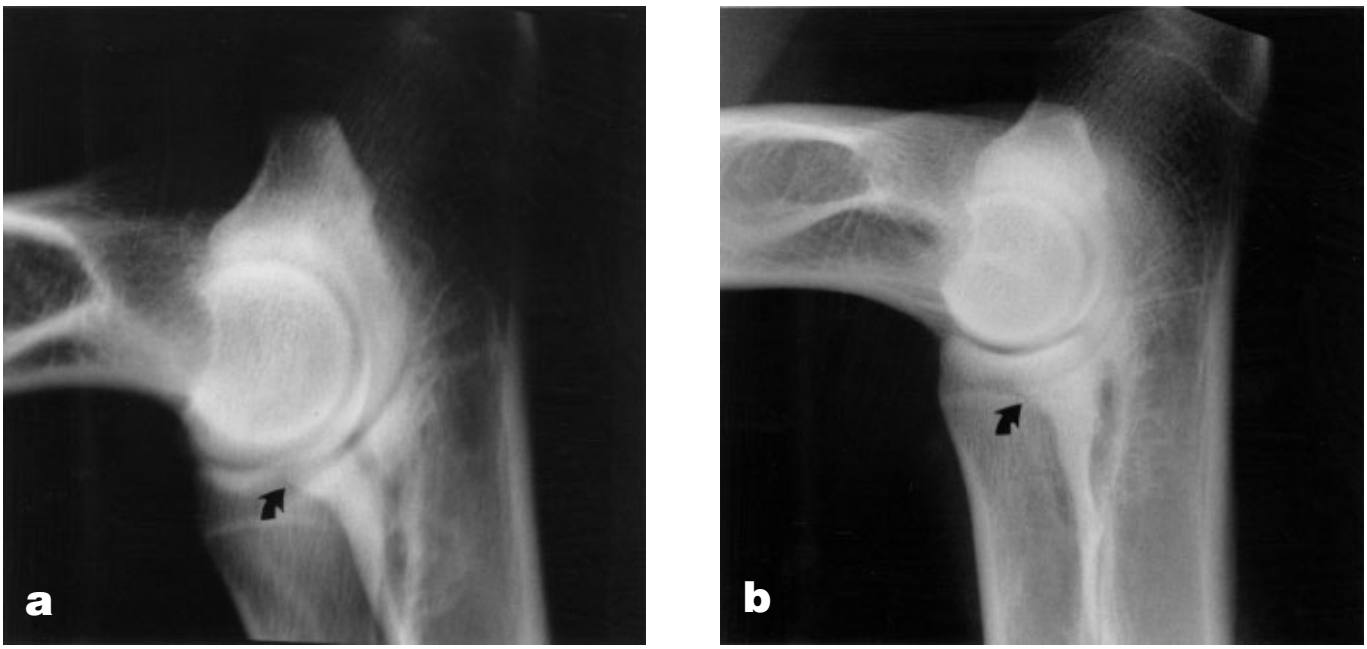


Fig. 4a: **ML linear tomography image of a normal medial coronoid process** (arrow).
Fig. 4b: **ML radiograph of same dog. Note improved visibility of medial coronoid process in Fig. 4a**.

imposed structures. Linear tomography is performed in the extended supinated ML view at the level of the medial coronoid process with the tube movement perpendicular to the axis of the leg and a swing angle of 40° [44]. The technique is used to see FMCP not visible in other views[9,44] and may detect up to 30 % more FMCP cases[44]. Another study showed no significant difference between linear tomography and radiographs but the combination of the 2 imaging modalities approached an accuracy similar to CT for detecting FMCP[6].

CLINICAL CONDITIONS

Clinical signs are usually seen from 5–6 months of age. A stiff, slightly stilted forelimb gait after rest progresses to lameness after exercise and pain on flexion and extension of the elbow joint[33]. The lower limb and elbow are slightly abducted and supinated. As clinical signs are mild and insidious, the patient is often only presented at about 6–9 months and after having been lame for 3–4 weeks[11,33]. Occasionally dogs are only presented in later life due to bilateral arthrosis[33]. The inciting condition results in an inflammatory reaction within the joint and arthrosis[33] and the primary cause is often only established after arthrotomy[17] or arthroscopy[1].

Fragmented medial coronoid process

Pathophysiology

Fragmentation of the medial coronoid process is believed to be a manifestation of the osteochondrosis complex[26,33] and is the most common clinical entity causing elbow arthrosis[14]. 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[33]. Osteochondrosis results in the inability of the deeper chondrocytes in the thick layer of cartilage to survive, and they undergo chondromalacia with eventual fissuring of the cartilage and subsequent fragmentation[26]. 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[33]. Mechanical stresses have also been incriminated as a cause of FMCP[33]. A recent popular hypothesis postulates asynchronous growth of the radius and ulna[32]. Overgrowth of the ulna relative to the radius appears to be the main cause of FMCP[32]. It 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[32]. Fragmented medial coronoid process is thus commonly seen together with OCD[6] or erosive lesions and rarely with UAP. The disparate growth may still be evident at the time of presentation or may have corrected itself.

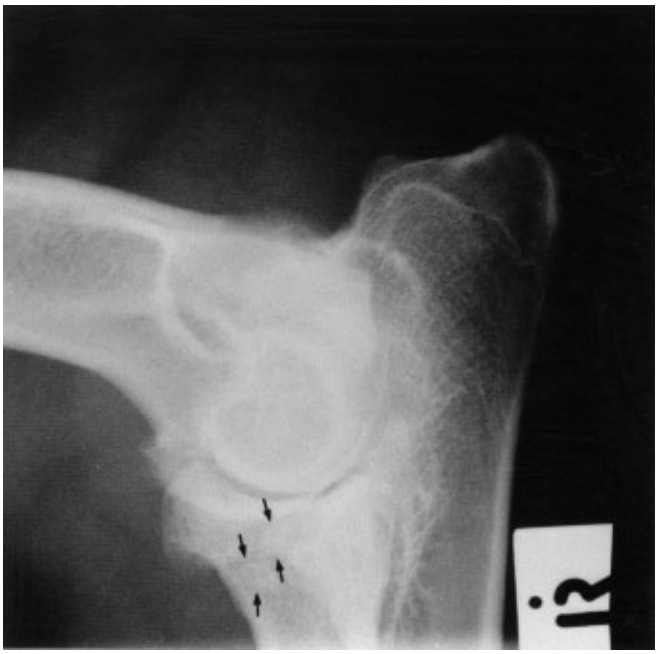


Fig. 5: **ML view with 2 separate medial coronoid fragments** (arrows) **superimposed on the proximal radius, and severe arthrosis**.

Radiographic changes

The medially located fragment should not be confused with the sesamoid bone 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 on radiographs[24,26,32], with fragment visibility reported as low as 9.8 %[13]. If visible, it may be seen as a single loose fragment or as several smaller fragments (Fig. 5). Factors contributing to poor or non-visualisation of the fragment include partial fragmentation, minimal fragment displacement[30,] small fragment, fragment location between the radial head and remaining intact coronoid process[31], or that the fragment cleavage line is often oblique to the X-ray beam, making it impossible to see[30,48]. Additionally, the medial coronoid process may be only fissured[1,14,24,31] and non-displaced, non-mineralised[39] or abnormally shaped[24], making radiographic evaluation impossible and requiring more sophisticated imaging techniques. Additional radiographic changes that may be indicative of medial coronoid pathology include loss of sharp delineation of the cranial edge of the medial coronoid (Fig. 6a) or blunting of the coronoid process[3]. Medial coronoid changes are most likely to be seen in the extended supinated ML and pronated CrCd (Fig. 6b) views. Radiographic changes are often seen only from about 7 months and then secondary arthritic changes may be all that are seen[11,32,33]. 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[26]. Joint incongruity may also be evident, with the lateral coronoid process displaced proximally to the radial head, resulting in step formation (Fig. 7). If more sophisticated imaging techniques are not available, the diagnosis can often only be confirmed by means of arthrotomy or arthroscopy[1,43]. Arthroscopy allows excellent visualisation of joint incongruity, free fragments, joint mice, abrasions, fissures and osteophyte formation of the medial coronoid process[1]. Dogs presented with clinical evidence of an elbow problem at less than 7 months of age with normal radiographs should undergo arthroscopy[1] or must return for follow-up radiographs 4–8 weeks later[32].

Treatment

Surgical exploration by means of arthrotomy, or preferably arthroscopy, with the removal of fragments is recommended in dogs less than 1 year old before the development of severe arthrosis[1,12,26,28,48]. Older dogs with extensive arthrosis may improve clinically after surgery[26]. Weight control, exercise restriction and analgesic therapy are all

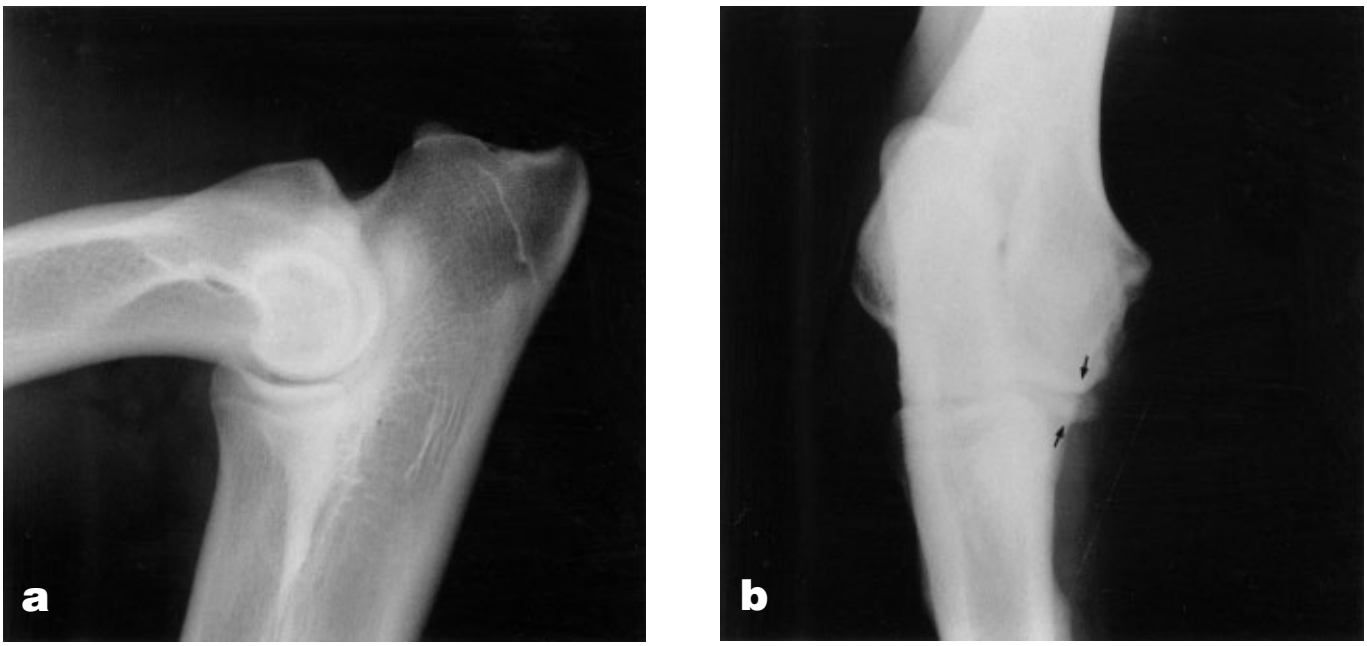


Fig. 6a: **ML view of 8-month-old German shepherd dog with poorly-visible medial coronoid process, degenerative changes and mild joint incongruity.**
Fig. 6b: **Slightly pronated CrCd view of same dog with separate medial coronoid fragment** (arrows).

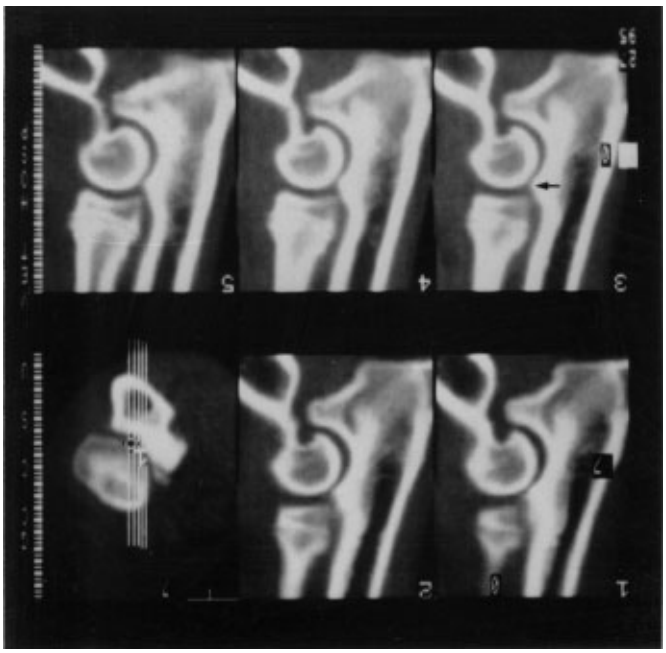


Fig. 7: **Sagittal computed tomography images to illustrate proximal displacement of lateral coronoid process** (arrow) **in relation to caudal radial head and incongruent humero-ulnar joint space** (courtesy of P Steyn, Colorado State University).

important additional therapeutic aids[26,48]. Owners should be made aware of the progressive nature of the condition. Several surgical approaches are possible but muscle separation between the pronator teres and flexor carpi radialis currently appears to be the method of choice[28]. Other studies cast doubt on the benefits of surgery. A study comparing surgical to medical treatment with pentosan polysulphate showed no difference in clinical status after 9 months, but a more rapid recovery in the medically treated cases[4]. Radiographic and range of motion scores, however, still showed progression of arthrosis. A study comparing surgery to treatment with rest and aspirin had similar results[23]. In early cases with a step between the radial head and the coronoid process, a proximal ulna sliding osteotomy will reduce the incongruency[1]. Treating the incongruency is probably more beneficial than removing the fragment.

Osteochondritis dissecans of the medial humeral condyle

Pathophysiology

The pathophysiology is very similar to that described for FMCP. Osteochondritis dissecans without concomitant FMCP is very rare[6]. The cartilage flap rarely mineralises[33]. The cartilage flap may separate and form a joint mouse.

Radiographic changes

The defect is readily seen in the CrCd or pronated CrCd views as a radiolucent lesion (Fig. 8a) or dished-out defect on the medial humeral condyle and may be seen from 5–6 months of age and prior to secondary arthrosis[5,11,33]. In more advanced cases the defect may be surrounded by a sclerotic rim[32]. In ML views it may be seen as a flattening of the caudodistal edge of the medial humeral trochlea[47] (Fig. 8b). If OCD is the sole lesion, arthrosis is less marked than with FMCP alone[11,21] but clinical signs may occur earlier and may be more disabling than FMCP on its own[21]. Combined OCD and FMCP lesions result in the most severe arthrosis[17,21].

Treatment

Surgical exploration and removal of fragments followed by curettage is performed as described for FMCP. The medial coronoid process should be evaluated at the same time.

Medical treatment is the same as for FMCP. Surgical results may be better than

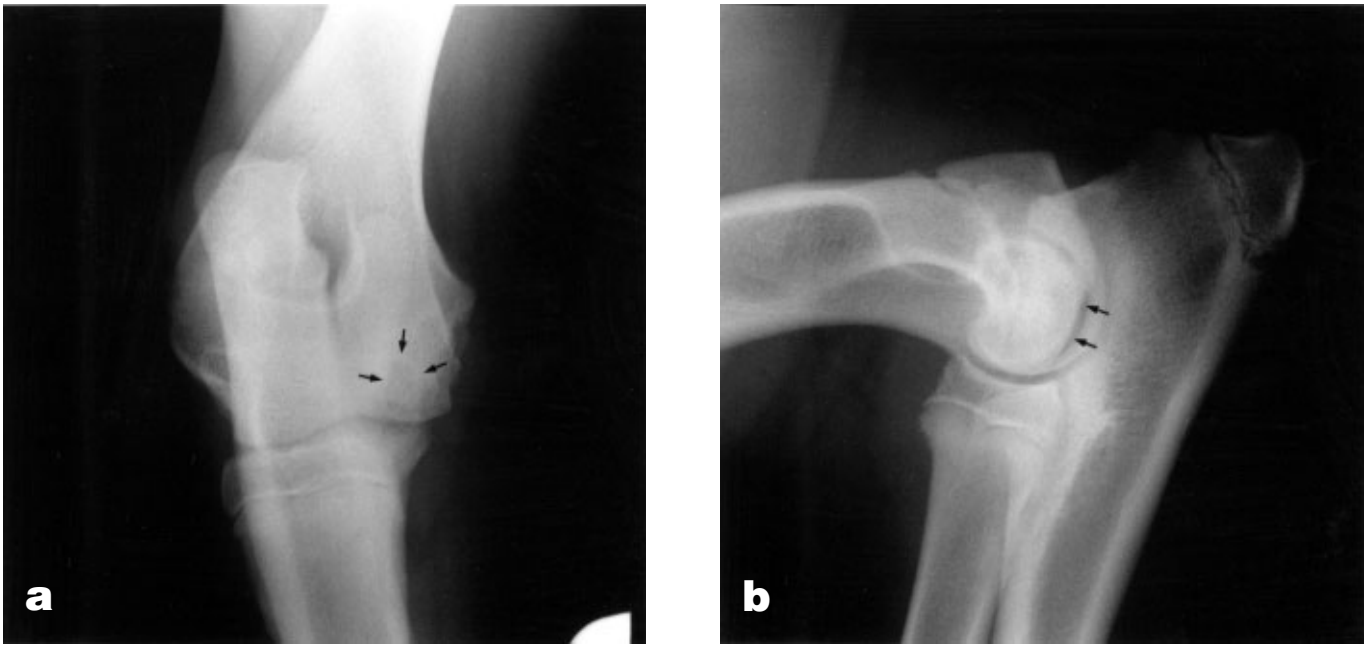


Fig. 8a: **CrCd view with a triangular radiolucent defect in the medial humeral condyle** (arrows) **in a 4-month-old boerboel with bilateral elbow OCD**.
Fig. 8b: **ML view of same dog with flattening of the medial condyle** (arrows).

for FMCP, particularly if surgery is performed at a young age[12].

Erosive lesion/cartilage defect

Pathophysiology

An erosive lesion or cartilage defect is seen most commonly on the medial humeral condyle and occasionally on the medial coronoid process[1,14,32] and probably has the same aetiology as FMCP and OCD and is due to ulnar overgrowth[32]. It may not be possible macroscopically to determine if a medial humeral condyle lesion is primary osteochondrosis or a secondary erosive lesion and histopathology may be required[32]. Erosive lesions commonly accompany FMCP[17]. Erosive lesions are postulated to occur rather than FMCP or OCD if the ulnar growth disparity occurs at a slightly older age when the medial coronoid process and medial humeral condyle cartilage are almost mature[32]. Alternative hypotheses are that minor joint incongruity may result in the lesions[48] or that they are earlier stages of FMCP and OCD[14]. These lesions are also known as 'kissing' lesions but Olsson differentiates this from an erosive lesion[32].

Radiographic changes

Lesions are not seen on radiographs of affected joints[13] but secondary arthrotic changes may be seen.

Treatment

Surgical removal of affected cartilage is recommended[32].

Ununited anconeal process

Pathophysiology

The condition occurs mainly in the German shepherd dog[16,18,38]. 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 (greyhound 14–15 weeks, German shepherd dog 16–20 weeks)[33,41]. The ununited anconeal process may be completely separated or joined to the ulna by fibrous or fibrocartilagenous tissue[38,41]. Overgrowth of the radius with a relatively shorter ulna is postulated by Olsson[22] to be the cause. 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 entire structure is less resistant to trauma and a tear in the weakened cartilage prevents osseous bridging of the gap[32]. In 2 separate studies, measurements made of the olecranon in German shepherd dogs with UAP showed a significantly shorter olecranon or proximally displaced radius in affected limbs[18,38]. Weis[45] and Wind[47], on the other hand, propose a hypothesis 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[6,24,50].

Radiographic changes

The condition is readily seen in flexed ML view as an irregular vertical radiolucent line through the caudal aspect of the anconeal process (Fig. 9a). Superimposition of the medial humeral epicondyle physis in views that are not fully flexed should not be confused with a UAP in dogs less than 8 months old[7]. The fragment may separate completely from the olecranon (Fig. 9b). Arthritic changes become severe over time. It is essential to make multiple views to ensure no other causes of elbow arthrosis are present[7].

Treatment

Conservative therapy is unrewarding and results in persistent lameness and progressive arthrosis[18,37]. Surgical therapy is the treatment of choice but at present no technique can ensure a functionally normal joint[7]. Surgical excision *via* a lateral arthrotomy has been the traditional treatment. This, however, results in instability with progressive arthrosis, but if performed early, results in acceptable longterm function[7,16,37]. Surgical re-attachment by means of a lag screw aims to prevent arthrosis by providing stability[7]. This technique may, however, aggravate arthritic changes if incongruity caused by asynchronous growth is present. Proximal ulnar osteotomy is a recently-proposed technique that aims to redress joint incongruity caused by

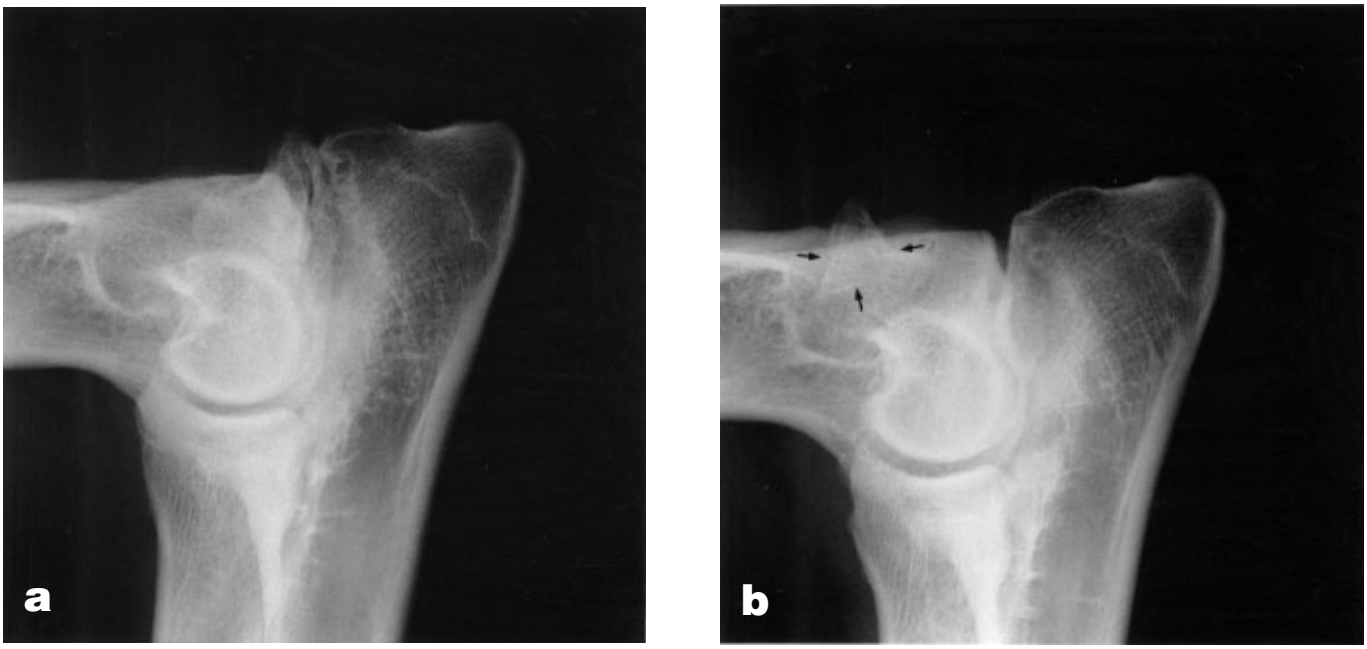


Fig. 9a: **Flexed right elbow ML view of a 2-year-old German shepherd with bilateral ununited anconeal process. The dog had a history of being mildly lame in the left front limb. Note vertical radiolucent line through anconeal process and secondary osteophyte formation. Visibility of pathology can be enhanced by making a fully flexed ML view.**
Fig. 9b: **Flexed left elbow ML view of same dog. Note cranially displaced united anconeal process** (arrows).

asynchronous radius or ulnar growth[7,32]. Results appear good, with olecranon fusion taking place in up to 95 % of cases[38], although in 27 % of these a narrow radiolucent line remained. The clinical response appears superior to that achieved by the other 2 techniques[7,38]. Olsson's short olecranon/overgrown radius hypothesis is supported by the ulnar ostectomy treatment, which relieves the pressure on the anconeal process, resulting in fusion of the ununited anconeal process with the ulna[32,38]. This treatment is unlikely to have a similar effect in relieving olecranon pressure if the UAP was due to trochlear notch with a smaller radius of curvature, as proposed by Weis[45] and Wind[47].

Elbow incongruity

Pathophysiology

According to Weis[45] and Wind[47], incongruity (described under radiographic changes) is due to the trochlear notch developing 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[47]. If present before 6 months, incongruity may also predispose the dog to FMCP, OCD and UAP owing to increased mechanical forces on these structures and incongruity may thus be seen together with these conditions[47]. Intermediate and heavy-set breeds have a longer proximal ulna relative to the adjacent radius than in other breeds[50]. Wind[50] postulated 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[50]. Incongruity may not always be evident at the time of radiographic examination, due to compensatory adjustments during growth[48]. According to Olsson[32], the incongruity is caused by asynchronous growth as discussed under FMCP.

Radiographic changes

In the semi-flexed ML and CrCd view of a normal elbow joint there should be small, even joint spaces between the humeral trochlea and the ulnar trochlear notch and between the humeral condyle, radius and medial coronoid process of the ulna[47]. The joint space forms a continuous arc in the ML view[47]. The lateral coronoid process should lie close to and run continuously with the adjacent proximal radius[47]. 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 humero-ulnar and humero-radial joint space and an indistinct outline of the trochlear notch[46,47] (Fig. 10). These changes are well illustrated in a radiographic guide of the elbow joint[49].

Treatment

Treatment is determined by concurrent FMCP, OCD and UAP, while the trochlear notch incongruity is treated conservatively[28].

Arthrosis

Arthrosis is a chronic, degenerative, non-infectious joint disease involving joint cartilage, joint capsule and subchondral bone tissue[10].

Radiographic changes

Early changes are best seen in the ML views with osteophyte formation commencing on the dorsal anconeal process and lateral epicondylar ridge, followed by the margins of the craniproximal radius and craniodistal humerus articular surfaces and sclerosis surrounding the trochlear notch[24,48] (Figs 5, 6, 9, 11). The latter tends to start distally and may be the result of osteophyte formation at the joint capsule insertion or subchondral sclerosis or both[47]. The anconeal osteophytes correspond to the insertion of the olecranon ligament and joint capsule on the proximal non-articular surface of the anconeal process[24]. In CrCd view, 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[14]. Arthrosis may be present without any radiographic evidence of a primary cause and could be due to erosive lesions, healed FMCP or minor incongruity.

HERITABILITY

Elbow dysplasia is inherited as multifactorial polygenic traits[15,20,34,40], *i.e.* requires more than 1 gene to cause the phenotype to be expressed in an individual[34]. A simple autosomal recessive mode of inheritance has been ruled out for the inheritance of OCD and FMCP and they appear to be inherited independently from each other as polygenic traits[34].

The frequency of elbow arthrosis caused by elbow dysplasia varies from 30–50 %, with males generally more frequently and severely affected[15,21]. However, in one study in Bernese mountain dogs, the female was shown to be more susceptible[40]. Radiographic studies are usually performed at 12 months. Severely affected cases diagnosed at a younger age and possibly destroyed are therefore excluded and consequently the incidence of elbow dysplasia may be underestimated[15]. The rottweiler is the breed with the highest incidence but the degree of arthrosis tends to be more severe in the Bernese mountain dog[15,22]. The risk of developing elbow dysplasia is higher in dogs with affected parents compared to dogs with unaffected parents[15,40]. Heritability varies from 0.10–0.48[15], 0.28–0.40[40], 0.45 (females)[20], and 0.77 (males)[20]. In rottweilers and Bernese mountain dogs, breeding parents that are both normal resulted in a 31 % incidence of elbow dysplasia in the offspring, breeding 1 normal with 1 arthritic parent resulted in 44–48 % of offspring affected and breeding affected parents with each other resulted in 59–62 % of offspring being affected[22]. In Sweden, concerted efforts to decrease the prevalence of elbow arthrosis have reduced the incidence from 60 to 38 % in the Bernese mountain dog and from 60 to 45 % in the rottweiler over a period of 5 years[40]. During the same period the incidence of moderate to severe arthrosis decreased from 42 to 14 % in the Bernese mountain dog and from 29 to 14 % in the rottweiler[40].

Heritability is a definite factor in the development of elbow arthrosis and elbow joints should be radiographed in susceptible breeds at the same time as hip dysplasia evaluations are performed[15]. Breeding animals, particularly in susceptible breeds, should be selected by their phenotypic status and by information on the elbow status of parents, grandparents, litter mates and offspring already born[15,34].

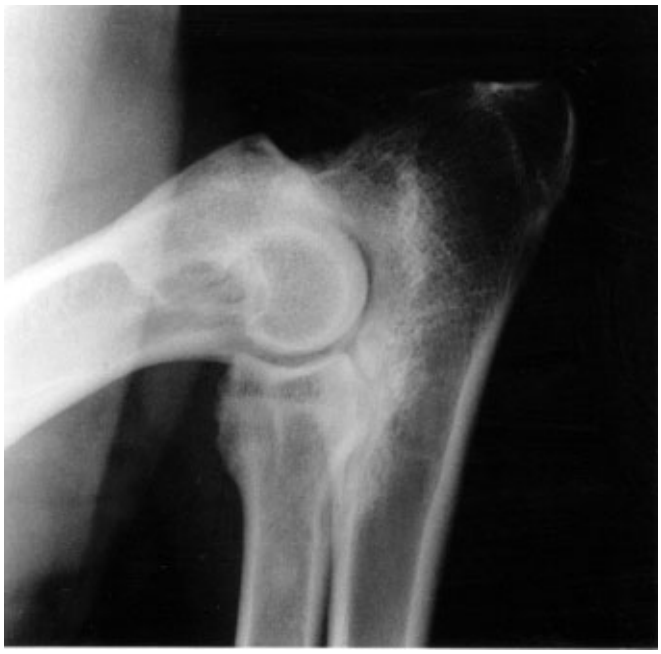


Fig. 10: **ML view of 8-month-old German shepherd dog with incongruent elbow joint. Compare to Fig. 2a. Note slightly proximally displaced lateral coronoid process and widened humero-ulnar and humero-radial joint space. Osteophyte reactions present and medial coronoid process poorly seen. Three medial coronoid fragments found at surgery**.

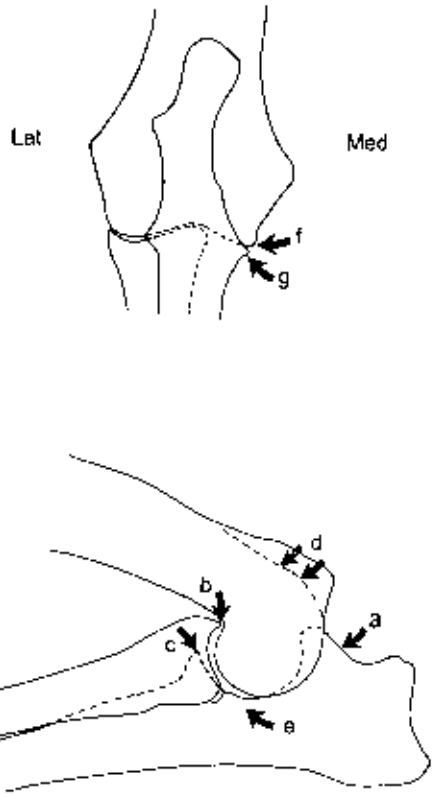
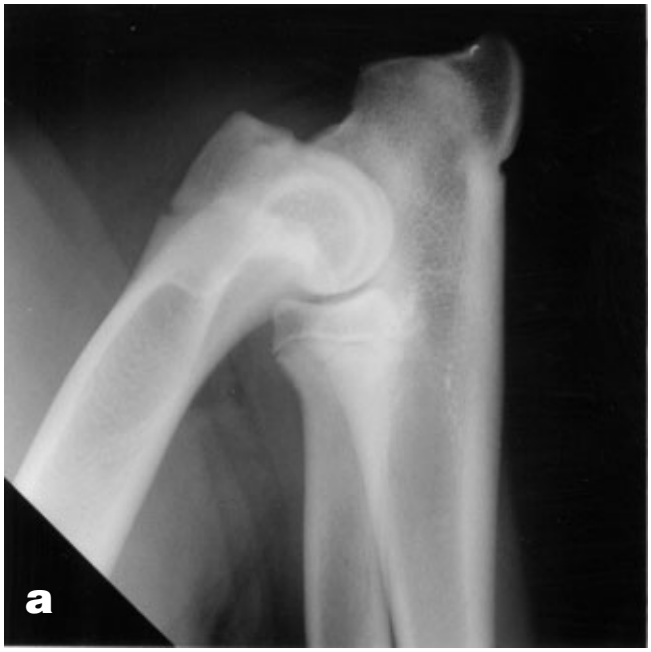
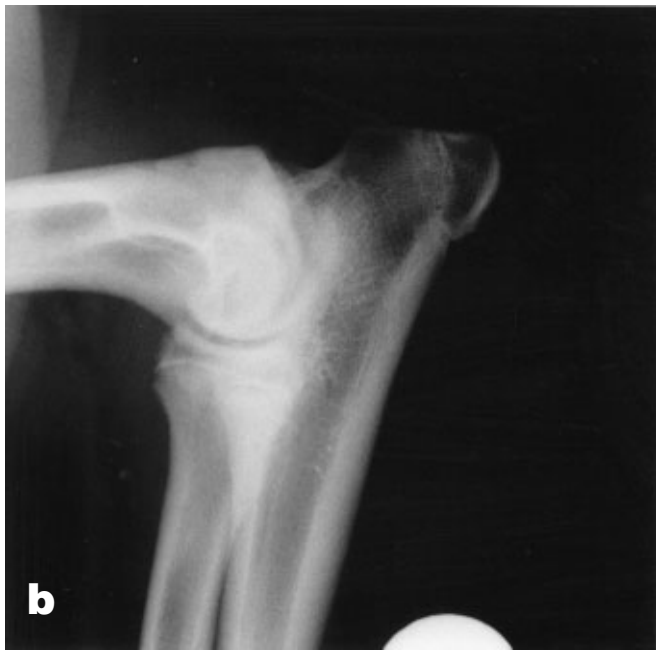


Fig. 11: **Schematic illustration of International Elbow Working Group elbow arthrosis grading scheme illustrating osteophyte location in flexed ML and CrCd views**.

CONTROL PROGRAMMES

Screening programmes to grade elbow arthrosis have been established to determine the degree of elbow involvement, with the intent to limit breeding with severely affected dogs. Elbow grading for degree of arthrosis has been performed by numerous investigators who often devised their own grading





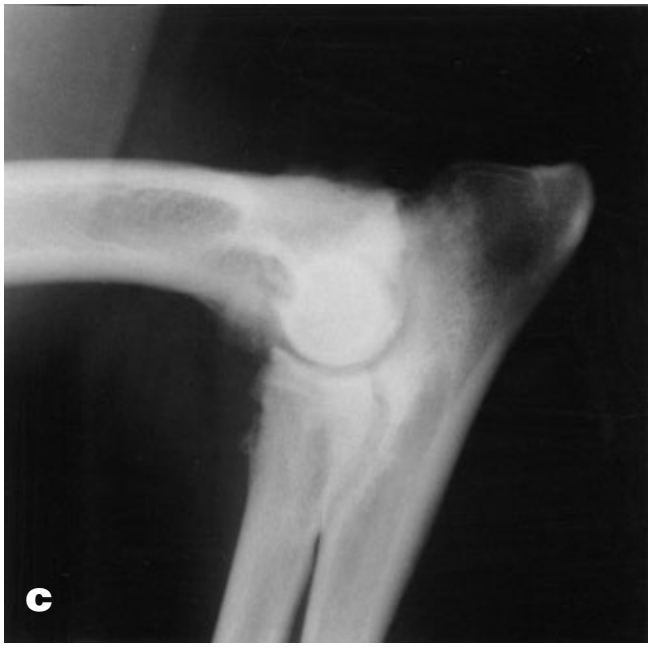


Fig. 12a: **Grade 1 elbow arthrosis**.
Fig. 12b: **Grade 2 elbow arthrosis**.
Fig. 12c: **Grade 3 elbow arthrosis**.

systems[15,17,21,25]. Many of these programmes involved certain breeds or organisations *e.g.* Bernese mountain dogs in Switzerland[25], Bernese mountain dogs and rottweilers in Scandinavia[15,22,40], rottweilers in Australia[36] and Dutch seeing-eye dogs[42]. The grading system does not, however, predict the type of lesion present and thus does not correlate with the degree of lameness that may be evident[17].

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 Fédération Internationale Cynologique and World Small Animal Veterinary Association as the official standard[2,29]. 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 (Fig. 11). 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[24]. Grading is as follows (Figs 11, 12):

Grade 1 (mild arthrosis) – osteophytes <2 mm anywhere in the elbow but

particularly at 1 or more of the following sites :

a) on the dorsal border 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) and/or sclerosis in the area caudal to the distal end of the ulnar trochlear notch and the proximal radius.

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

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

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

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

In addition, if the primary pathology is evident it should be noted. Screening programmes should be performed at a standard and narrow age interval, *i.e.* as close to 12 months as possible, as increasing age has a significant influence of the prevalence and severity of elbow arthrosis[40]. Dogs with clinical signs of elbow pathology or offspring bred with a high risk of developing elbow arthrosis are radiographed at an earlier age so that affected dogs may be submitted for surgery before arthrotic changes develop.

The benefit *versus* cost ratio of an elbow arthrosis screening programme has been extensively studied in Sweden[40]. The cost of screening and registration of dogs for elbow dysplasia was found to be less than the value of dogs estimated to have been saved from moderate to severe 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 a dog with any grade of 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. In Australia, rottweilers have a high incidence of elbow arthrosis and, in accordance with current IEWG recommendations, it was suggested that dogs with evidence of grade 2 and 3 elbow arthrosis should not be used for breeding[36].

DISCUSSION

Evidence appears to favour osteochondrosis in combination with asynchronous growth of the radius and ulna to be a major contributing factor causing elbow arthrosis. This is corroborated by the small number of cases that have simultaneous FMCP and UAP that would, according to this hypothesis, tend to be mutually exclusive. However, asynchronous radius and ulnar growth could possibly occur alternatively with first the ulna and then the radius, or *vice versa,* growing past each other. This hypothesis requires further investigation. The hypothesis of an abnormally developed slightly elliptical trochlear notch proposed by Weis and Wind seems unlikely to be a major contributing factor, as combined UAP and FMCP should then occur in much larger numbers of dogs and ulnar ostectomy would have no effect in cases with UAP[7,38]. It is, however, also possible that the time at which incongruency develops may affect the outcome. Trochlear notch malformation under 20 weeks of age may result in UAP, while such malformation after the anconeal process has fused to the ulna could result in FMCP.

Open registries are essential to combat elbow dysplasia. Evaluating the progeny, particularly of sires, will also make a marked difference as it has been shown that there is a lower incidence of elbow arthrosis in the progeny of unaffected sires[40]. In Sweden, an open registry is sponsored by the Swedish kennel club and has resulted in a marked decrease in the incidence of elbow arthrosis[48]. In the United States an open registry system for elbow dysplasia is controlled by the Institute for Genetic Disease Control (GDC) which also maintains open genetic registries for a multitude of other inherited diseases. The GDC is cooperating with Canada, Australia, Japan and has been asked to assist in establishing an open registry in Europe as 'shared knowledge is the greatest tool in the world for understanding'[35].

In South Africa, screening programmes should be introduced in predisposed breeds such as the rottweiler, Labrador retriever, golden retriever and German shepherd dog. The Kennel Union of South Africa, affected-breed clubs, veterinary clinicians, radiologists and surgeons should combine their resources and expertise to initiate such programmes. Cooperation with the GDC can be considered. As conditions involved in elbow dysplasia are being recognised earlier and more frequently and some progress is being made in their treatment, screening programmes will benefit those dogs that have elbow dysplasia as well as reduce the incidence of this often crippling disease.

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