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Review

Canine elbow dysplasia: Aetiopathogenesis and current treatment recommendations

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ABSTRACT

Elbow dysplasia is a common debilitating condition of large and giant breed dogs. Environmental factors and a complex genetic heritability play a role in predisposing dogs to elbow dysplasia with two aetiopathogeneses suggested for the development of the disease. Osteochondrosis was initially thought to cause elbow dysplasia, but more recent evidence has strongly supported various forms of joint incongruity as the most likely cause in most cases. Radioulnar length discrepancies and humeroulnar curvature mismatch have been implicated as the cause of medial coronoid disease and ununited anconeal process, but radial incisure incongruity and biceps/brachialis muscle forces could possibly play a role in some dogs.

Treatment of elbow dysplasia should address articular pathology, such as fragmented coronoid process, osteochondrosis, cartilage damage and ununited anconeal process as well as any identified underlying causes. Finally, several palliative procedures have been developed to address more advanced elbow disease and might offer improved outcomes compared to conventional medical management.

Introduction

Heritable canine elbow dysplasia was defined by the International Elbow Working Group (IEWG)[1] in 1993 to include fragmented medial coronoid process, osteochondrosis of the humerus, ununited anconeal process, articular cartilage injury and incongruity of the elbow joint. Elbow dysplasia is common and has a reported prevalence of 17% in UK Labradors (Morgan et al., 1999, 2000) and 70% in Bernese mountain dogs in The Netherlands (Hazewinkel et al., 1995). It is typically seen in young large and giant breed dogs, but is reported in smaller chondrodystrophic breeds like the Dachshund and French bulldog (Narojek et al., 2008; Sjostrom, 1998). Males are affected at about twice the rate of females (Meyer-Lindberg et al., 2006).

Most cases first present at 6–12 months of age because of persistent forelimb lameness, but some dogs present later in life (>6 years old), with clinical manifestations of medial coronoid disease and little or no prior history of lameness. A further group presents with lameness due to continuing or progressing joint pathology (Fitzpatrick et al., 2009a; Vermote et al., 2010).

Aetiopathogenesis

Genetics

Several large epidemiological studies have examined the genetic basis of elbow dysplasia, which appears to be inherited differently in different breeds. To complicate matters further, there is evidence that the different manifestations of elbow dysplasia could be inherited independently (Clements, 2006; Grandalen and Lingaas, 1991; Hazewinkel, 2006; Lewis et al., 2011; Ma et al., 2004). The differences in inheritance suggest that the syndrome that is currently designated as elbow dysplasia is a common end point for a variety of genetic disorders which disturb elbow development through various mechanisms. Because of the complexity of inheritance and the effects of environmental variables in disease expression, it is unlikely that genetic testing for elbow dysplasia will be possible in the foreseeable future.

Three mechanisms have been suggested for the development of elbow dysplasia including osteochondrosis (OC) (Nap, 1995; Olsson, 1983), various joint incongruities (Gemmill et al., 2005; Kramer et al., 2006), and a biomechanical force mismatch across the elbow joint (Hulse, 2008). All are hypothesised to occur as a result of a genetic predisposition with secondary environmental influencing factors, such as high energy diets, leading to rapid growth rates or excessive exercise (Nap, 1995). Recent evidence

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[1] See: http://www.vet-iewg.org (Accessed 1 April 2012).

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has supported various forms of joint incongruity as the most likely mechanism, but OC still appears to play a role in some dogs. The biomechanical force mismatch hypothesis is under development but the evidence to support it is currently weak.

Joint incongruity

There are three joints in the elbow, the humeroradial, humeroulnar and radioulnar (Fig. 1). Three types of joint incongruity have been proposed or demonstrated, namely, (1) radioulnar length mismatch; (2) humeroulnar incongruity, and (3) radioulnar incisure incongruity (Burton et al., 2008). The diagnosis of elbow incongruity by conventional radiology is relatively imprecise, with one small prospective cadaveric study demonstrating that a step of 1.5–4 mm is required to achieve an 86% specificity and 90% sensitivity for detection (Mason et al., 2002). The insensitivity of radiographic diagnosis, coupled with differences between study populations and the lack of standardised imaging protocols, has led to reported incidences of joint incongruity from 14% to 100% in dogs with fragmented coronoid process (Meyer-Lindberg et al., 2006; House et al., 2009; Moores et al., 2008; Samoy et al., 2006). The increasing use of arthroscopy and computerised tomography (CT) imaging over the last decade has refined the understanding of joint incongruity.

Radioulnar length mismatch

Radioulnar length mismatch, with the radius terminating either proximal to or distal to the level of the coronoid processes of the ulna, has been reported in numerous studies (Böttcher, 2011b; Gemmill et al., 2005; Morgan et al., 2000). It has been hypothesised that a short radius transfers weight in excess of normal physiological loads to the medial coronoid process of the ulna (Preston et al., 2000). Excessive cyclic loading leads to fatigue microdamage of the subchondral bone (Olsson, 1993; Wind, 1986a). A level 2 evidence based medicine (EBM) study (Table 2) examined the excised medial coronoid processes of 38 dogs with fragmented medial coronoid process (FCP) and described histological findings of fatigue microdamage and osteocyte loss consistent with this hypothesis (Danielson et al., 2006).

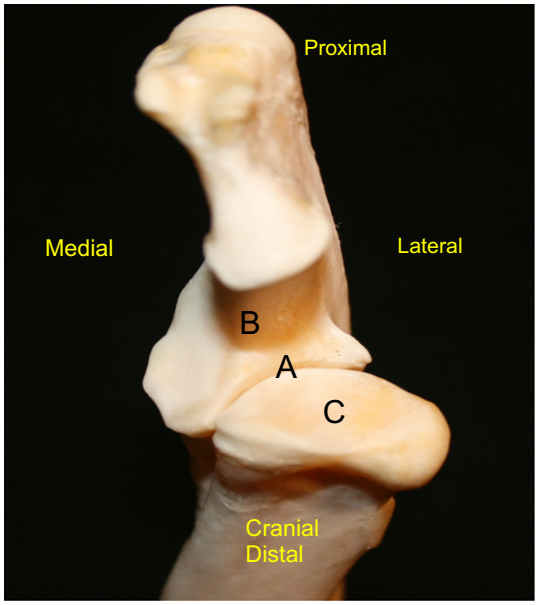

Fig. 1. Craniodistal view of the ulna and humeral head. A, radioulnar joint; B, humeroulnar joint; C, radiohumeral joint.

Numerous studies have also identified an association between FCP and a short radius (Böttcher, 2011b; Kramer et al., 2006; Meyer-Lindberg et al., 2006). The presence of FCP without significant incongruity has been explained by the observation that incongruity is not static, but will increase and decrease at different stages of skeletal growth, often leading to a congruent joint at maturity (Böttcher, 2011b; Trostel et al., 2003). However, the evidence for this hypothesis is anecdotal and studies describing the sequential development of the elbow joint in a number of dogs are necessary for confirmation.

In contrast to a short radius, a short ulna displaces the humeral head proximally relative to the ulna and places excessive loads on the anconeal process. This interferes with bony union of the anconeal process by 20–22 weeks of age in large dogs where there is a separate ossification centre, leading to ununited anconeal process (UAP; Sjöström et al., 1995; Van Sickle, 1966). Once again, this hypothesis is poorly supported by evidence and requires confirmation.

Humeroulnar incongruity

Humeroulnar incongruity is proposed to occur either when the radius of curvature of the ulna notch is less than the curvature of the humeral trochlea, or when a relatively long radius displaces the humeral head cranially from the ulna notch, causing subluxation of the joint (Morgan et al., 2000; Proks et al., 2011). The most frequent cause of humeroulnar incongruity is radial displacement of the humerus, as several studies have found a relationship between joint subluxation and clinical elbow dysplasia, but no relationship to the shape of the ulna notch has been reported, despite breed differences (Collins et al., 2001; Kirberger and Fourie, 1998; Proks et al., 2011). Humeroulnar incongruity is most commonly defined as a form of radioulnar incongruity, where the radius displaces the humerus from the ulnar notch, but further research is required to definitively describe the cause of humeroulnar incongruence, as the current evidence is primarily level 3b to 5.

Radioulnar incisure incongruity and biceps/brachialis mismatch

Radioulnar incisure incongruity and biceps/brachialis muscle mismatch have been proposed to account for the presence of clinical elbow disease limited to the incisure alone. Two potential mechanisms are hypothesised, namely (1) a radioulnar conflict at the incisure leading to crushing of the coronoid process against the radius, and/or (2) compression of the medial coronoid process against the radius by the eccentric pull of the biceps/brachialis muscle group, particularly in flexion (Fitzpatrick, 2006, 2009; Fitzpatrick and Yeadon, 2009; Hulse, 2008; Palmer, 2011; Fig. 2).

Incisure incongruity was suggested to occur through one of several ill-defined mechanisms, such as ligament laxity, a poor fit between the radius and ulna leading to localised force concentration, or compression of the lateral aspect of the medial coronoid process during weight bearing (Fitzpatrick and Yeadon, 2009; Fig. 3). The evidence for these incongruities is limited to three reports, describing clinical disease consistent with the theory and to a biomechanical analysis of the muscle forces of the racing Greyhound (Fitzpatrick et al., 2009a; Samoy et al., 2012; Van Ryssen and Van Bree, 1998). The proposed mechanisms are speculative, which from an EBM perspective is considered level 5. Significant biomechanical testing will be required before the theory can be confirmed or clinically applied.

Osteochondrosis

Osteochondrosis results from a failure of endochondral ossification (Bennett et al., 1981) and as it is a developmental pathology