**IN-VIVO KINEMATICS OF THE ELBOW**

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**Introduction**

Elbow dysplasia (ED) is a common condition mainly in young dogs, being bilateral in up to 35% of dogs. Repetitive mechanical overload of the medial compartment is thought to induce fatigue micro damage of the subchondral bone and subsequent overt fragmentation of subchondral bone and destruction of overlying articular cartilage. Radio-ulnar incongruence (RUI), mainly in the form of positive radio-ulnar incongruence with the radius being shorter relative to the ulna, has been stressed as one potential causative factor of mechanical overload within the medial compartment. In 2011 Guillou et al. reported proximo-distal movement of the radius and ulna relative to each other in sound dogs at the walk and the trot. Pathological dynamic change of the radio-ulnar joint cup under load might be the reason why elbow joints without detectable RUI shows comparable pathologies at the medial coronoid process than incongruent joints. Biplanar fluoroscopic kinematography allows for highly precise movement analysis, superior to computer assisted gait analysis using skin mounted marker. Using this technique we investigate the three-dimensional (3D) in vivo kinematics comparing sound and dysplastic canine elbow joints, expecting dynamic RUI in joint with medial coronoid disease.

**Material and Methods**

The 3D kinematic pattern of six sound (five dogs) and seven dysplastic elbow joints (six dogs) was acquired. Dysplastic dogs had evidence of medial coronoid disease based on clinical, radiological as well as computed tomographic (CT) examination. The control dogs were orthopedically sound, based on history, orthopaedic examination and orthogonal radiographs of the elbow joints. After implantation of at least three 0.8mm Tantulum markers into the proximal radius, proximal ulna and distal humerus, synchronised biplanar fluoroscopic image sequences (500 fps, shutter 0.5 ms) of the elbow joint were acquired while the dogs were walking on a treadmill. A third synchronised high-speed video camera was used to define stance phase. Using XROMM software, marker 3D trajectories starting 30 frames before ground contact and ending 120 frames afterwards were collected and transferred to 3D-CT bone models of the individual elbow joint. Based on the so gained 3D animations of the elbow joints, relative axial (proximo-distal) movement of radius and ulna, as well as axial (internal-external) rotation between humerus and ulna were calculated for the 120 frames of stance phase. Axial motion and rotation were expressed as maximal amplitude. Furthermore the humero-ulnar joint contact pattern was calculated based on the animated bone models and visually compared between groups. Due to the small sample size median and interquartile range was used to summarize continuous data. Inter-group comparison was performed using Mann-Whitney test. Alpha was set to 5%.

**Results**

Breeds in the control groups were one Australian Shepard dog and Labrador Retriever each and three mix-breed dogs. Two dogs were male and three female. Breeds represented in the dysplastic group were one Bernese mountain dog and Golden Retriever each, two Labrador Retrievers and two mix-breed dogs. Two dogs were male and four female. With a median age of 1.4 years (1.2 to 2.8) for the control dogs and 1.5 years (1.3 to 1.8) for the dysplastic dogs, age did not differ significantly between groups (P = 0.7922). Same was true for body weight (control 26.0 kg [25.0 to 32.0] vs. dysplastic 26.0 kg [24.6 to 31.0]; P = 0.6623). No difference could be found for axial movement of the radius and the ulna relative to each other between groups (maximal amplitude control 0.06 mm [0.02 to 0.1] vs. dysplastic 0.03 mm [0.02 to 0.07]; P = 0.2334). Relative humero-ulnar rotation was significantly higher in dysplastic elbows than in controls (maximal amplitude control 2.9 degree [1.8 to 4.9] vs. dysplastic 5.2 degree [3.1 to 6.2]; P = 0.0016). Humero-ulnar contact at the medial coronoid process was evenly distributed over the medial coronoid in control elbows (see fig. 1-top row), while contact area in dysplastic elbows was reduced and shifted to the lateral aspect of the medial coronoid process (see fig. 1-bottom row).

**Discussion**

The hypothesis that relative radio-ulnar motion in terms of dynamic RUI would be increased in dysplastic elbows compared to unaffected dogs can be rejected. Therefore, the assumption that dynamic RUI under in vivo loading might be responsible for medial coronoid disease in dysplastic dogs doesn’t appear to be valid. However, our observation that the in vivo humero-ulnar contact pattern differs between sound and dysplastic dogs underlines the basic principle of joint incongruence in elbow dysplasia. Similar to hip dysplasia in which soft tissue laxity results in joint instability and subsequent degenerative joint disease, the documented increase in humero-ulnar rotation could be interpreted as rotational joint instability, too. Visually it appears that the
increased humero-ulnar rotation in dysplastic elbows results in a relative cranio-lateral shift of the humeral trochlea, smashing the trochlea against the lateral aspect of the medial coronoid process (see fig. 2). This visual impression is supported by the alteration in contact pattern with reduction in contact area and shift towards lateral, observed in every dysplastic elbow joint. Further studies are warrant to quantify the reduction in contact area at the medial coronoid process as well as the observed shift.

Having investigated axial radio-ulnar motion as well as humero-radial rotation only, other kinematic alterations should not be neglected as potential sources of mechanical disturbance within the dysplastic elbow joint. However, the clinical observation that the pathologies almost exclusively occurs within the humero-ulnar compartment points to some sort of humero-ulnar conflict. Valgus deformity either static or dynamic is probably the most significant differential to rotatory joint instability at this moment and will have to be focus of future studies.

Fig. 1: Typical joint contact pattern (every 10th frame starting with ground contact) in a sound (upper row) and dysplastic elbow joint (bottom row). Notice the reduction of contact area and its shift towards the lateral border of the medial coronoid process in the dysplastic joint. This could explain why fragmentation of the medial coronoid process commonly occurs at the lateral border of the medial coronoid process.

Figure 2: Image sequence of a dysplastic elbow (right side, medial view) while walking on a treadmill (Images 1-6). At the beginning of stance phase (image 2) relative humero-ulnar rotation shifts the humeral trochlea cranio-laterally. Image A and B illustrate the concentration of humero-ulnar contact area at the lateral border of the medial coronoid process under load in another dysplastic elbow (left side, frontal view. Image A - before stance phase, Image B - during stance phase)

References


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