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Preactivation of the quadriceps muscle could limit cranial tibial translation in a cranial cruciate ligament deficient canine stifle



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ABSTRACT

Cranial cruciate ligament (CrCL) deficiency is the leading cause of lameness of the canine stifle. Application of tension in the quadriceps muscle could trigger cranial tibial translation in case of CrCL rupture. We replaced the quadriceps muscle and the gastrocnemius muscle by load cells and turn-buckles. First, eight canine limbs were placed in a servo-hydraulic testing machine, which applied 50% of body weight (BW). In a second phase, the CrCL was transected, and the limbs were tested in a similar manner.

In a third phase, a quadriceps pretension of 15% BW was applied and limbs were again tested in a similar manner. Cranial tibial translation was significantly decreased in CrCL deficient stifles ($p < 0.05$) when quadriceps pretension was applied.

These findings indicate that quadriceps pretension could play a role in the stability of a CrCL deficient stifle and should then be considered in rehabilitation programs and conservative treatment of CrCL rupture in dogs.

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1. Introduction

As a dog walks, forces are applied on the tibia by muscles, body weight and ground. Depending on geometrical parameters such as stifle flexion angle and tibial plateau angle (TPA), the resulting force has a component that tends to load the stifle joint in the cranio-caudal direction, giving rise, in certain conditions, to a cranial tibial thrust. Cranial tibial thrust is balanced by active components (the pull of the stifle flexor muscles of the thigh) and passive components (the cranial cruciate ligament (CrCL), the caudal horn of the medial meniscus, and the capsular and collateral ligaments of the stifle) (Slocum and Slocum, 1993).

The role of the quadriceps and hamstring muscles in the human knee joint has been extensively studied (Alentorn-Geli et al., 2009; Berchuck et al., 1990; Beynon and Fleming, 1998; Bodor, 2001; Caltafamo et al., 2010; Draganich and Vahey, 1990; Elias et al., 2003; Fleming et al., 2001; Hashemi et al., 2010; Liu and Maitland, 2000; MacWilliams et al., 1999; Myer et al., 2009; O'Connor, 1993; Renström et al., 1986; Shelburne et al., 2005; Torry et al., 2004;

Williams et al., 2005). In particular, the hamstring muscles have been shown to act as an anterior cruciate ligament (ACL) agonist, decreasing strain on the ACL in in-vitro studies (O'Connor, 1993). The role of the quadriceps muscle however is less clear; both ACL agonist (Bodor, 2001; Hashemi et al., 2010) and antagonist (Alentorn-Geli et al., 2009; Beynon and Fleming, 1998; Draganich and Vahey, 1990; Myer et al., 2009; Renström et al., 1986; Shelburne et al., 2005; Williams et al., 2005) actions have been proposed.

In the dog, a recent study suggested that the semitendinosus (one of the hamstring muscles) acts as a CrCL agonist, preventing cranial tibial translation in a CrCL deficient stifle (Kanno et al., 2012). The quadriceps muscle has previously been considered by some authors as a CrCL antagonist (Kanno et al., 2012; Mostafa et al., 2010). Conversely, other authors have suggested that the patellar tendon itself, the distal extension of the quadriceps, could counteract cranial tibial thrust (Gibbons et al., 2006; Griffon, 2010; Moore and Read, 1996).

On the basis of previous ex-vivo experiments carried out in our laboratory we have suggested that quadriceps preactivation could contribute to the stabilization of a CrCL deficient stifle (Ramirez et al., 2011).

The objective of this study was to evaluate the effect of quadriceps preactivation on the stabilization of the CrCL deficient stifle joint in dogs. We hypothesized that quadriceps preactivation would oppose cranial tibial thrust resulting in decreased cranial tibial translation, and that this effect would be more pronounced in limbs with low TPA.

A part of this study has been accepted for oral presentation as an Abstract in the 22nd ECVS Annual Scientific Meeting, Rome, Italy, July 2013.

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2. Materials and methods

2.1. Sample

Fourteen hind limbs were harvested from 7 dogs weighing 21–37 kg by disarticulation of the coxofemoral joint. All dogs were euthanized for reasons unrelated to this study and had no radiographic evidence of orthopedic diseases in the hip, stifle, or tarsal joints.

2.2. Specimen preparation

Soft tissues, except for the stifle and hock joint capsule and collateral ligaments, were carefully removed from the collected hind limbs. Soft tissues distal to the tarso-metatarsal joint were left intact.

Two 2 mm Kirschner wires were inserted into the bone, in a direction perpendicular to the cortex surface at the origin and insertion of the medial collateral ligament to serve as radiological markers for evaluation of the cranial tibial translation.

2.3. Origin and termination of quadriceps muscle and gastrocnemius muscle

The quadriceps and gastrocnemius muscle were both replaced by structures composed of a stainless steel wire or cable, a turnbuckle and a load cell (Sensy, Model 5930 150 DN¹).

2.3.1. Quadriceps

Using a 1.5 mm drill bit a hole was drilled in the patella at its widest point in a lateromedial direction, perpendicular to the patellar tendon. A 1 mm stainless steel wire loop was passed through the hole and connected to a load cell, prolonged proximally by a custom made turnbuckle which was attached to the cranioproximal aspect of a custom designed jig holding the proximal femur, at the base of the greater trochanter.

2.3.2. Gastrocnemius

Using a 2.5 mm drill bit, two holes were drilled in the medial and lateral fabellar facets. A short 3.5 dynamic compression plate was placed over two washers and secured by two 3.5 monocortical screws.

Using a 2.0 mm drill bit a hole was drilled into the tuber calcanei, in a lateromedial direction, perpendicular to the common calcaneal tendon.

A custom made hook was used to connect the center of the dynamic compression plate to a load cell prolonged by a turnbuckle, assembled to a triangular shaped 1.8 mm Kirschner wire passed through the hole in the calcaneal tuber.

2.4. Specimen montage in the sevohydraulic press

The proximal femur was securely fixed via three pairs of screws and a 2.5 mm transfixing Kirschner pin to a custom-made aluminum jig connected to the horizontal bar of the press. The limbs were positioned in a neutral position (between breaking and propelling phase) in a sevohydraulic press (Instron 3366, 10 KN²), the foot being maintained in a custom-made shoe that prevented cranial or lateral displacement (Fig. 1).

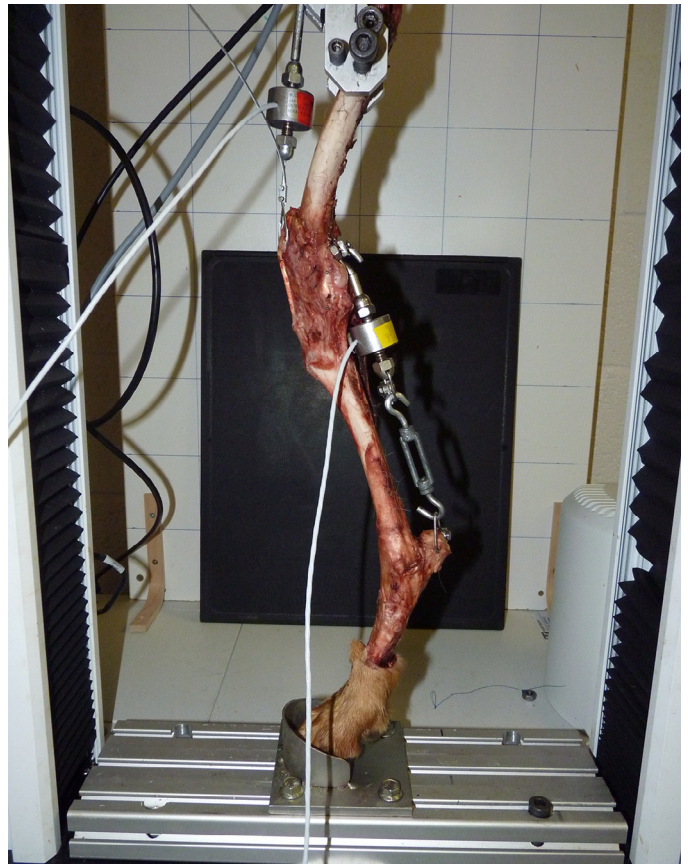


Fig. 1. Photograph of the sevohydraulic press illustrating the limb in a neutral position. A custom designed shoe prevented cranial and lateral displacement of the paw.

2.5. Joint angles and placement of the specimens

Joint angles were measured manually using a goniometer after positioning the limbs. The hip joint angle was defined as the angle between the line connecting the greater trochanter to the fibula head and a horizontal line, parallel to the horizontal bar of the sevohydraulic press. The stifle joint angle was defined as the angle between a line connecting the center of the greater trochanter to the center of the fibula head and a line connecting the fibula head to the center of the tarsal joint. The tarsal joint angle was defined as the angle between the line connecting the fibular head to the center of the tarsal joint and the line connecting the center of the tarsal joint to the center of the distal epiphysis of the third metatarsal bone.

The hip joint angle was set at 70° to the horizontal. The stifle and tarsal joint angles were set at 137 ± 5° and 143 ± 5° respectively before loading the limbs.

2.6. TPA

Prior to biomechanical testing, standard true lateral radiographs of the limbs were taken. True lateral radiographic views of the tibia were defined by superimposition of the femoral and tibial condyles, as defined elsewhere (Reif et al., 2004). The functional axis of the tibia was represented by a line drawn from the midpoint between the two apices of the tibial intercondylar eminence to the center of the talus. The medial tibial plateau was represented by a line drawn from its most cranial to its most caudal margin. TPA was measured as the angle between the medial tibial plateau and a line

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² Instron 3366: Instron, Boechout, Belgium.

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