GASTROCNEMIUS DOMINANCE AS A CAUSE OF CRANIAL CRUCIATE LIGAMENT DISEASE (CCLD)

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Although CCL disease is the leading cause of lameness associated with the knee in large dogs, its pathogenesis remains unknown. The dynamic imbalance in dogs with CCL deficiency supports the concept of a relative dominance of factors contributing to the cranial tibial thrust over active restraints. Among these, the role of the gastrocnemius muscle, which is loaded during the stance phase, has not been well explored. The goals of this presentation are to present the evidence obtained in large dogs regarding this issue and discuss its clinical relevance as it relates to the pathogenesis of CCLD.

We enrolled thirty mature pure-bred Labrador Retrievers with (n=16) and without (n=14) CCLD in a cross sectional study. Pelvic limbs of dogs without CCLD were classified as normal (n=28 limbs), whereas those with CCLD were considered as diseased (n=18 limbs) or sound contralateral to CCLD (n=10 limbs). Muscle conformation of pelvic limbs was evaluated via physical examination, radiography (widths of quadriceps, hamstring and gastrocnemius were expressed relative to tibial length and to each other), and dual-energy X-ray absorptiometry (DEXA, lean contents of quadriceps, hamstring and gastrocnemius were expressed relative to tibial length and to each other). Computed tomographic (CT) images of the hindlimbs were analyzed with a solid modeling software to determine the body segment parameters (BSP, center of mass, mass, mass moment of inertia) of each segment of the limb (thigh, crus and foot). These BSP were integrated with kinetic and kinematic data obtained on the same dogs during overground trotting to perform an inverse dynamics analysis of the gait (joint reaction forces, power and moment around the hip, stifle and hock). At the end of this study, we identified the combination of factors that best discriminated contralateral limbs (predisposed to CCLD) from limbs at low risk to CCLD. A predictive equation was consequently developed, combining tibial plateau angle and femoral angle of anteversion.

To eliminate the effects of compensation in contralateral limbs of dogs with CCLD, we evaluated the gait mechanics of 20 sound Labrador Retrievers, categorized as low versus high risk to CCLD based on the predictive equation identified in our initial study.

We first found evidence of a morphometric imbalance between the gastrocnemius and the hamstring muscles:

- The ratio of the lean content of gastrocnemius to hamstring muscles was greater in diseased (P = 0.007) and sound contralateral (P = 0.013) limbs, than in normal limbs.
- The center of mass of the crus was located more proximally (at a distance from the stifle equal to 28% of the tibial length) in CCL deficient and contralateral limbs compared to normal limbs (31% of the tibial length). Based on the distribution of the gastrocnemius muscle in relation to the tibia, these results would be explained by a greater development of this muscle in limbs affected or predisposed to CCLD.

We found gait alterations in limbs affected or predisposed to CCLD that are consistent with an increased function of the gastrocnemius:

- The standing position of the hock was increased in CCLD and contralateral limbs
- In sound Labradors, the energy generated around the hock and stifle joints were increased in limbs predisposed to CCLD compared to limbs at low risk.
• In sound Labradors, the net extensor moment of the hock and the net flexor moment of the stifle were both increased in limbs scored as predisposed to CCLD compared to limbs scored as low risk to the disease

Clinical relevance:
A relative predominance of the gastrocnemius over the hamstring muscle group in a semi-flexed stifle would increase the caudodistal traction of the distal femur over the slope of the tibial plateau. This femoro-tibial compression would amplify the cranial tibial thrust normally counteracted by the CCL. This chronic strain placed on the CCL each time the limb is loaded could eventually lead to fatigue failure of the ligament in dogs. The origin of this dominance of the gastrocnemius and the contribution of the tibial plateau angle to this dynamic imbalance remain unclear.

Selected references: