Morphometric characteristics of the pelvic limbs of Labrador Retrievers with and without cranial cruciate ligament deficiency

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Objective—To evaluate skeletal characteristics of pelvic limbs with and without cranial cruciate ligament (CCL) deficiency in Labrador Retrievers.

Animals—30 adult purebred Labrador Retrievers.

Procedures—Pelvic limbs (n = 28) of 14 dogs without CCL deficiency were classified as control limbs, whereas the limbs of 16 dogs with CCL deficiency were considered affected by (18 limbs) or predisposed to (10 contralateral limbs of dogs with 1 affected limb) CCL deficiency. Skeletal characteristics were evaluated via physical examination, radiography, and computed tomography. Radiographic and computed tomographic variables were compared among limb groups by use of a mixed-model ANOVA.

Results—The tibial plateau slope was steeper in CCL-deficient limbs but not in predisposed limbs, compared with the slope in control limbs. The angle between diaphyseal and proximal tibial axes was increased in both CCL-deficient and predisposed limbs. The relative width of the proximal portion of the tibia and the inclination of the patellar ligament did not differ among limb groups. The overall and distal femoral anteversion angles were greater in CCL-deficient and predisposed limbs, whereas the femoral condyle trochanteric angle was decreased in those limb groups, compared with findings in control limbs.

Conclusions and Clinical Relevance—Cranial angulation of the proximal portion of the tibia, excessive steepness of the tibial plateau, and distal femoral torsion appeared more likely to be associated with CCL deficiency than femoral angulation, tibial torsion, intercondylar notch stenosis, and increased inclination of the patellar ligament. (Am J Vet Res 2009;70:498–507)

Cranial cruciate ligament deficiency is the leading cause of osteoarthritis in the stifle joint of dogs.1–5 The annual economic impact of medical and surgical management of CCL insufficiency of dogs in the United States has been estimated at $1.3 billion.6 Most dogs with CCL deficiency develop this condition during the repetitive performance of normal daily activities, have a typical history of chronic progressive lameness, and have pathologic changes consistent with degenerative joint disease.7,8

In a recent study,9 27% of the phenotypic expression of CCL disease was attributable to genetics, whereas 73% of the phenotypic expression was linked to environmental factors. Most CCL instabilities that are diagnosed in dogs are believed to be caused by repetitive microtrauma that secondarily weakens the CCL, especially in large-breed dogs such as Labrador Retrievers.10–12 The cranial tibial thrust, a biomechanical force generated by contraction of the gastrocnemius muscle, is a proposed underlying cause for the repetitive microtrauma to the CCL.13 The magnitude of the cranial tibial thrust depends on the amplitude of the compressive force (70% of body weight at trot) and the slope of the tibial plateau with respect to the axis that joins the centers of motion of the stifle and tarsal joints.

The steepness of the tibial plateau has an apparent role in the pathophysiology of CCL deficiency. However, steepness of the tibial plateau as the sole dependent variable cannot predict the risk of CCL deficiency development in dogs, especially in Labrador Retrievers.14,15 Instead, stress injuries are believed to result from a combination of conformation characteristics.
within the entire pelvic limb, which results in a biomechanical imbalance among the factors that influence the cranial tibial thrust. The exact role of each of these factors and their relative importance in CCL deficiency have not been defined. This gap in knowledge prevents the identification of appropriate preventive measures against CCL deficiency in dogs.

The objective of the study reported here was to identify the morphometric skeletal characteristics associated with CCL deficiency in Labrador Retrievers. The working hypothesis was that the skeletal conformation in Labrador Retrievers with nontraumatic CCL deficiency is different, compared with the skeletal conformation of unaffected Labrador Retrievers.

Materials and Methods

Dogs—The Institutional Animal Care and Use Committee of the University of Illinois approved all study procedures prior to enrollment of dogs. Informed consent was obtained from the owners of 30 adult purebred Labrador Retrievers that were examined at the University of Illinois veterinary medical teaching hospital from June 1, 2005, through May 30, 2006. The control group included dogs that were ≥ 6 years old, that had no history or clinical signs of stifle joint disease, and for which orthopedic, radiographic, and CT examinations of the stifle joints revealed no abnormalities.15 Dogs with unilateral or bilateral CCL disease were included in the study if they had no history of trauma and the diagnosis was confirmed during surgery. There was no age criterion for dogs with CCL disease. The sound contralateral limbs of CCL-deficient dogs were considered as predisposed to CCL deficiency on the basis of the high incidence of bilateral CCL disease in dogs.11,16,17 Contralateral limbs that had undergone previous surgery to treat CCL disease were excluded from the study. Each limb included in the study was analyzed separately and classified as unaffected (control limbs), CCL deficient (deficient limbs), or predisposed to CCL deficiency (predisposed limbs).

Physical examination—The degree of lameness was scored as 0 (no clinical lameness), 1 (mild lameness that was evident only after strenuous exercise), 2 (moderate lameness that was evident at all gaits during normal activity or exercise; considerable weight bearing on affected limb with each step), 3 (severe lameness that was evident at all gaits with characteristic toe-touching lameness), and 4 (no weight bearing on the affected limb).18,19 All dogs underwent a complete physical examination, including orthopedic and neurologic examinations, while awake. The general conformation of the pelvic limbs and the standing angles of the tarsal, stifles, and hip joints were also evaluated while the dogs were awake. Assessment of the range of motion of the tarsal, stifles, and hip joints; cranial drawer sign test; and tibial compression test were performed on each pelvic limb in sedated dogs. Sedation was induced with morphine (0.5 mg/kg, IM), atropine sulfate (0.04 mg/kg, IM), and medetomidine hydrochloride (8 µg/kg, IM).

Radiographic evaluation—Radiography of the pelvic limbs was performed for all dogs. Mediolateral and caudocranial radiographic views of each tibia, which were awake. Assessment of the range of motion of the tarsal, stifles, and hip joints; cranial drawer sign test; and tibial compression test were performed on each pelvic limb in sedated dogs. Sedation was induced with morphine (0.5 mg/kg, IM), atropine sulfate (0.04 mg/kg, IM), and medetomidine hydrochloride (8 µg/kg, IM).
The tibial plateau angle,\textsuperscript{14,19,26,27} patellar ligament angle,\textsuperscript{28,29} and DTA-PTA\textsuperscript{30} were assessed on the mediolateral radiographic view of each tibia by use of previously reported techniques. The length of each femur was defined as the distance between the greater trochanter and the extensor fossa of the long digital extensor muscle on a mediolateral radiographic view of the femur.

The caudocranial view of the tibia was used to evaluate joint alignment and tibial rotation. Tibial rotation was measured as the distance between the medial edge of the calcaneus and the center of the talus.\textsuperscript{31} A lateral displacement of the calcaneus was described as internal tibial rotation and assigned positive values, whereas medial displacement of the calcaneus was described as external tibial rotation and assigned negative values.\textsuperscript{31} An extended ventrodorsal radiographic view of the pelvis was used to assess the conformation of the hip joint (angle of inclination\textsuperscript{32–34} and Norberg angle\textsuperscript{34,35}), the alignment of the patella (quadriceps angle),\textsuperscript{34,36} and the angulation of the femur (femoral varus angle)\textsuperscript{37} by use of previously established techniques.

CT evaluation—Examination of dogs with CCL-deficient stifle joints via CT was performed during the anesthetic episode required for surgical management of the disease, whereas CT examination of control dogs was performed during the anesthetic episode required for a dental hygiene procedure. Dogs were positioned in dorsal recumbency with the pelvic limbs extended and...
parallel to each other. Images were acquired on a helical scanner; mean slice thickness was 4 mm, and there was 50% overlap between slices. The data were reconstructed to form a 3-dimensional image from the hip joint distally to the metatarsal bones. The 3-dimensional images were used to obtain measurements via both black-and-white and transparent bone protocols.

Tibial and femoral lengths, the angle of inclination, and Norberg angle of each hip as well as the quadriceps angle were measured via 3-dimensional CT by use of the same landmarks as those used for radiography. Tibial torsion was assessed in a transverse plane by measuring the angle between the transcondylar axis of the proximal portion of the tibia and the cranial tibial axis of the distal portion of the tibia (Figure 3). The alignment of the tibial crest was assessed in a transverse plane as the angle between a line drawn perpendicular to the midpoint of the caudal condylar axis between the 2 proximal tibial condyles and a line between the same midpoint and the center of the cranial proximal tibial crest (Figure 4).

The conformation of the intercondylar notch relative to the femoral condyles was assessed, as previously described. The widths and heights of the femoral condyles and intercondylar notch were measured and used to calculate the intercondylar notch width and height indices. These indices were calculated by dividing the width (or height) of the central intercondylar notch by the width (or height) of the femoral condyles.

Femoral torsion was evaluated on the basis of the anteversion angle, and the proximodistal location of this torsion was further defined via determination of the proximal and distal anteversion angles, femoral head trochanteric angle, and femoral condyle trochanteric angle (Figure 5). The overall anteversion angle has traditionally been defined as the angle between the axis of the femoral head and neck and the transcondylar axis of the distal femur in a transverse plane. A line was then drawn from the intersection of the axis of the femoral head and neck and the transcondylar axis of the distal portion of the femur to the lesser trochanter, which divides the overall anteversion angle into proximal and distal anteversion angles. The intertrochanteric axis was defined as the line connecting the greater and lesser trochanters at their highest points. The angle between this intertrochanteric axis and the axis of the femoral head and neck was defined as the femoral head trochanteric angle, whereas the femoral condyle trochanteric angle was defined as the angle between the intertrochanteric axis and the transcondylar axis of the distal femur.

**Statistical analysis**—Data are reported as mean ± SD, and a value of $P < 0.05$ was considered significant. A 95% confidence interval was calculated for selected measurements from control dogs to provide breed-specific standards. For the variables of interest, deficient and predisposed limb group means were each compared with the control limb group mean by use of a mixed-
model ANOVA. With limb nested within a dog because the analysis was based on 1 or 2 limbs for each dog. Variables that were not normally distributed were logarithmically transformed before the ANOVA was performed. A Spearman rank correlation coefficient was calculated to evaluate the linear relationship between 2 variables. The effect of stifle joint angle on patellar ligament angle was examined by use of multivariable regression to perform regression of the patellar ligament angle versus the stifle joint angle. The square of the patellar ligament angle was also regressed against the stifle joint angle to evaluate a potential curvilinear relationship.

Results

No significant difference was detected between the mean ± SD weight of dogs without (36.9 ± 8.9 kg) or with (37.5 ± 7.4 kg) CCL disease. The sex distribution did not differ between the 2 groups of dogs; among the control dogs, there were 8 females and 6 males, and among the dogs with CCL disease, there were 10 females and 6 males. Dogs in the control group were older (mean age, 8.30 ± 1.94 years) than dogs with CCL disease (mean age, 4.4 ± 1.8 years). The control group was composed of 14 Labrador Retrievers that met the criteria for inclusion; data from 28 clinically unaffected stifle joints were used in the analyses. These dogs were admitted for routine dental hygiene procedures and had no evidence of concurrent orthopedic problems or gait abnormalities.

Among the 16 Labrador Retrievers included in the CCL-deficient group, 10 had unilateral CCL disease. No abnormalities in the contralateral limb were detected during the orthopedic examination, and these 10 limbs were designated as predisposed limbs for the purposes of the study. Four dogs with CCL disease in 1 limb had undergone surgeries (2 tibial plateau leveling osteotomies and 2 lateral tibial suture repairs) for treatment of CCL disease in the contralateral limb 7 months to 2 years before recruitment for the study of this report. Two dogs had bilateral CCL disease at the time of initial evaluation; thus, 18 limbs in 16 dogs were classified as deficient. Ten of these 18 stifle joints had a partial tear of the CCL, whereas 8 stifle joints had a complete CCL tear. Clinical lameness was scored as 1 (mild) in 3 limbs, 2 (moderate) in 7 limbs, 3 (severe) in 7 limbs, and 4 (non-weight bearing) in 1 limb. The duration of lameness before the initial examination varied (range, 3 days to 11 months), but 13 dogs were lame for 1 to 3 months. Joint effusion and laxity (indicated by positive results of the cranial drawer sign and tibial compression tests) were detected during orthopedic examination of all affected stifle joints.

Radiographic measurements—Radiographic evidence of osteoarthritis was identified in all deficient stifle joints and 4 of 10 predisposed stifle joints. Patellar enthesophytes, periaricular osteophytes in the proximal portion of the tibia, sesamoid osteophytes, and condylar periarticular osteophytes (lateral, medial, or both) were the most common determinants of osteoarthritis in affected stifle joints (Table 1).

Compared with the control limbs, the deficient or predisposed limbs did not differ with regard to femoral length, tibial length, femoral angulation, angle of inclination, or quadriceps and Norberg angles. However, the DTA–PTA was greater in both deficient (P = 0.008) and contralateral stifle joints that were considered predisposed to CCL disease (10) in Labrador Retrievers. The radiographic findings are listed in descending order of frequency of detection.

Table 1—Results of radiographic evaluation of osteoarthritis in stifle joints with CCL deficiency (n = 10) and contralateral stifle joints that were considered predisposed to CCL disease (10) in Labrador Retrievers. The radiographic findings are listed in descending order of frequency of detection.

<table>
<thead>
<tr>
<th>Radiographic determinant of osteoarthritis</th>
<th>No. of deficient stifle joints</th>
<th>No. of predisposed stifle joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patellar enthesophytes</td>
<td>17</td>
<td>3</td>
</tr>
<tr>
<td>Proximal tibial periaricular osteophytes</td>
<td>17</td>
<td>3</td>
</tr>
<tr>
<td>Sesamoid bone periaricular osteophytes</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>Condylar periarticular osteophytes</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td>Femoral trochlear groove periaricular osteophytes</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>Femoral intercondylar notch sclerosis*</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Capsular thickening or buttressing</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Subchondral cystic lucency</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Proximal tibial subchondral sclerosis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Proximal tibial subchondral cystic lesions</td>
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<td>0</td>
</tr>
<tr>
<td>Central tibial plateau osteophytes</td>
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<td>0</td>
</tr>
<tr>
<td>Intra-articular mineralized osseous fragments and intercondylar avulsion fracture fragments</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Meniscal mineralization</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Femoral subchondral sclerosis</td>
<td>0</td>
<td>0</td>
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</tbody>
</table>

*Congenital subchondral bone cyst (4 mm in diameter) was seen in the femoral intercondylar notch in 1 predisposed stifle joint. TCould not be differentiated.
in control limbs; there was no difference in tibial plateau angles between the predisposed and control limbs ($P = 0.062$). The 95% confidence intervals for the tibial plateau angle were 20° to 30° for control limbs, 25° to 38° for deficient limbs, and 21° to 31° for predisposed limbs.

The amount of internal tibial rotation in deficient limbs was greater ($P = 0.023$) than that in control limbs; there was no difference ($P = 0.222$) in tibial rotation between the predisposed and control limbs (Table 2). The 95% confidence intervals for tibial rotation were −5.5 to 3.9 mm for control limbs and −3.3 to 6.5 mm for deficient limbs. The relative width of the tibia, the patellar ligament angle, and the degree of flexion of the stifle joint did not differ between the deficient or predisposed limbs and the control limbs. There was no significant correlation ($r = 0.20; P = 0.132$) between the patellar ligament angle and the angle of the stifle joint.

**CT measurements**—The overall and distal anteversion angles were greater in both deficient ($P = 0.014$ and $P = 0.007$, respectively) and predisposed ($P = 0.010$ and $P = 0.008$, respectively) limbs, compared with these
angles in control limbs (Table 3). The 95% confidence intervals for the overall anteverision angle were 19° to 34° for control limbs, 23° to 47° for deficient limbs, and 24° to 43° for predisposed limbs; the 95% confidence intervals for the distal anteverision angle were 13° to 23° for control limbs, 18° to 37° for deficient limbs, and 18° to 34° for predisposed limbs. The angle between the transcondylyar and the intertrochanteric axes (femoral condyle trochanteric angle) was decreased in both groups (P = 0.003 and P = 0.036 in deficient and predisposed limbs, respectively), compared with the same angles in control limbs. The 95% confidence intervals for the femoral condyle trochanteric angle were 20° to 37° for control limbs, 19° to 30° for deficient limbs, and 18° to 32° for predisposed limbs. All other CT measurements for the deficient or predisposed limbs did not differ from measurements in the control limbs.

**Discussion**

The main findings of the study reported here were that the pelvic limbs of Labrador Retrievers with, or predisposed to, CCL disease had greater angulation of the proximal portion of the tibia (increased DTA–PTA in both deficient and predisposed limbs and increased tibial plateau angle in deficient limbs) and internal torsion of the distal portion of the femur (increased overall anteverision and distal anteverision angles and decreased femoral condyle trochanteric angle), compared with findings in unaffected pelvic limbs of other Labrador Retrievers. Additionally, the conformations of the patellar mechanism and intercondylar notch in deficient and predisposed limbs appeared to be similar to those of apparently normal limbs. Labrador Retrievers were selected for investigation because they are commonly affected with CCL disease, are frequently admitted to our clinic, and are more uniform in their conformation than are chondrodystrophic dog breeds.\(^\text{14,15}\) The mean age of the dogs with CCL disease in the present study was consistent with the mean age of large-breed dogs with this condition in a previous study.\(^\text{14}\) The difference in the mean age between Labrador Retrievers with and without CCL disease in our study was expected because the minimum age requirement for inclusion in the control group was based on the decreased risk for CCL disease in older Labrador Retrievers.\(^\text{14}\) Although this selection did not guarantee that each dog included in the control group was not affected by CCL disease, the characteristics of this condition support the inclusion of sound, older Labrador Retrievers as a low-risk population. Development of CCL disease in the contralateral limb in 42 of 114 (37%) dogs with CCL disease in 1 limb has been reported;\(^\text{15}\) therefore, the contralateral limbs in the dogs with 1 CCL-deficient pelvic limb were considered predisposed to CCL disease for the purposes of the present study.

The angulation of the proximal portion of the tibia in relation to its distal axis (ie, DTA–PTA) in CCL-deficient limbs in the present study confirmed the previously reported\(^\text{14,15}\) association between this tibial characteristic and CCL disease. Osmond et al\(^\text{16}\) described a strong correlation between an angulation of the proximal portion of the tibia and a steep tibial plateau in dogs with CCL disease. The fact that DTA–PTA in predisposed limbs was greater than that in control limbs in the dogs in the present study is a reflection of limb symmetry within each dog and provides some evidence to support the role of this deformity in the pathogenesis of CCL disease. The proposed mechanism for this contribution relies on a premature closure of the caudal portion of the tibial growth plate with a subsequent increase in the steepness of the tibial plateau. The resulting increase in cranial tibial thrust would eventually lead to fatigue failure of its passive restraint, the CCL. On the basis of the 95% confidence intervals calculated for control dogs in the study of this report, DTA–PTA > 9° appear to be consistent with a caudal angulation of the proximal portion of the tibial shaft in Labrador Retrievers. This reference limit is less than that previously suggested (11.2°),\(^\text{11}\) most likely because a single breed of dog was used in our study, which minimizes morphologic variations among the evaluated tibia.

The contribution of the tibial plateau angle to the pathogenesis of CCL disease remains controversial. The findings of the present study are in agreement with results of 2 studies;\(^\text{21,22}\) in those investigations, the tibial plateau angle in stifle joints with CCL disease was greater than the tibial plateau angle in stifle joints without CCL disease. However, the findings of the present study differ from data obtained in 2 other studies \(^\text{14,15}\) of Labrador Retrievers with and without CCL disease. In our study, the mean tibial plateau angle was 28.1 ± 3.4° in CCL-deficient stifle joints, compared with values of 25.6 ± 0.6°\(^\text{14}\) and 23.5 ± 3.1°\(^\text{15}\) determined in CCL-deficient stifle joints in the previous studies. This discrepancy among study findings may be related to the large proximal angulation of the tibia in 6 of the 18 affected stifle joints in the present study. Although this deformity was not evaluated in the other 2 studies\(^\text{14,15}\) involving Labrador Retrievers, there is a correlation between the proximal angulation of the tibia and the presence of a steep tibial plateau in dogs with CCL disease.\(^\text{30}\) The lack of significant difference between the tibial plateau angle in the control and predisposed limbs in the dogs of the present study most likely reflects the fact that the predisposed limbs will not all be affected by CCL disease. Alternatively, a type II error may be attributed to the smaller number of the predisposed limbs (n = 10) evaluated, compared with the number of deficient limbs (18).

An abnormal relationship between the tibial plateau and the patellar ligament has been proposed as contributing to the pathogenesis of CCL disease.\(^\text{20,28}\) In that biomechanical model, the joint force generated during weight bearing is distributed along the patellar ligament. Cranial tibial thrust is increased if this ligament is angled more than 90° in relation to the tibial plateau. A tibial tuberosity–advancement procedure has consequently been developed to realign the patellar ligament and neutralize shear forces at a standing angle.\(^\text{29}\) This proposed pathogenic model is supported by the increase in patellar ligament angle detected in 3 dogs with CCL disease, compared with the patellar ligament angle in 9 dogs without CCL disease.\(^\text{29}\) We were not able to confirm these findings in our study. The conformation of structures that could affect the inclination of the patellar ligament (eg, the femoral condyles

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and proximal portion of the tibia) was evaluated by use of measurements of femoral condyle length, femoral condyle length-to-femoral width ratio, proximal tibial width, and proximal tibial width-to-distal tibial width ratio; the findings for the deficient or predisposed limbs were similar to those for the control limbs. We were also unable to detect a correlation between the patellar ligament angle and the degree of flexion of the stifle joint, as previously reported.27,28 This discrepancy may be attributed to differences in methods used in those studies27,28 and in the study reported here; in the present study, positioning of the limbs was standardized, which limited the range of stifle joint flexion (68° to 105°), compared with the ranges (44° to 132°) in previous reports.39,31

In the study of this report, the femoral angulation (in varus) of the control dogs (7° ± 4°) was consistent with the measurements obtained from radiographic views (9 ± 2°) and bone specimens (7° ± 4°) in a previous study.37 Although the alignment of the stifle joint in relation to the axis of the pelvic limb was not specifically evaluated in our study, the lack of differences in the femoral angulation between the deficient or predisposed limbs and control limbs indicated that varus deformation of the femur is unlikely to contribute to the development of CCL disease in Labrador Retrievers.

The alignment of the extensor mechanism also appeared similar between the deficient or predisposed limbs and control limbs, based on the quadriceps angle and tibial crest alignment measured in all limbs. Although limbs affected with CCL disease appeared to have a greater amount of internal tibial rotation than control limbs radiographically, this result was not confirmed with the tibial torsion measurements obtained via CT. This discrepancy confirms the previously reported31 limitations of traditional radiography and the superiority of CT for evaluation of overall tibial alignment. Indeed, the linear displacement of the medial border of the calcaneus in caudocranial radiographic views does not distinguish between an anatomic malformation or malpositioning as causes of internal tibial rotation.31 Although the patella and fabellae appeared correctly positioned in all caudocranial radiographic views in the present study, rotational malpositioning of the tibia may have occurred without concurrent rotation of the femur because CCL disease is associated with an increased range of motion in internal stifle joint rotation.31

Although internal torsion of the tibia did not appear to contribute to CCL disease in the affected dogs in the present study, the degree of internal femoral torsion (as measured via CT) in deficient limbs, as well as in predisposed limbs, was greater than that in control limbs. The anteversion angle has traditionally been used to evaluate the relative orientation of the femoral neck in relation to the femoral condyles.34,30,31 To our knowledge, this is the first study to evaluate the anteversion angle in dogs with and without CCL disease via CT. Measurements that relied on the lesser and greater trochanters (distal anteversion, femoral condyle trochanteric, and femoral head trochanteric angles) were also included in the present study to differentiate version of the femoral neck from torsion located distal to the lesser trochanter of the femur. The increase in overall and distal anteversion angles and decrease of the femoral condyle trochanteric angle in both deficient and predisposed limbs, compared with these angles in control limbs, were consistent with an internal torsion of the femur distal to the lesser trochanter. The femoral head trochanteric angle and proximal anteversion angle did not differ between the deficient or predisposed limbs and the control limbs, thereby confirming that version of the femoral neck did not appear to contribute to CCL disease.

A previous study39 revealed no differences in the distal portion of the femur between dogs with and without CCL disease. This finding was based on measurements performed on mediolateral radiographic views of the stifle joint in which femoral torsion could not be evaluated. In the present study, CT measurements provided evidence that femoral torsion might have a role in the pathogenesis of CCL disease in Labrador Retrievers. Although the mechanism of action between this deformation and the pathogenesis of CCL disease warrants further investigation, internal rotation of the femoral condyles could affect the relationship between the proximal aspect of the CCL and the intercondylar notch.

On the basis of reports39,40,41,32 of data obtained from Labrador and Golden Retrievers, impingement on a narrow intercondylar notch has been suggested to contribute to CCL disease development in dogs. However, controversy remains as to whether intercondylar notch stenosis is a primary cause of CCL disease or a secondary change attributable to chronic CCL insufficiency and degenerative joint disease.30,40,32 The findings of the present study support a secondary change because dimensions of the intercondylar notch did not differ between the deficient or predisposed limbs and control limbs. Although affected stifle joints had radiographic evidence of degenerative disease, these changes consisted predominantly of patellar enthesophytes and may not have been severe enough to significantly decrease the dimensions of the intercondylar notch.

Cranial angulation of the proximal portion of the tibia, excessive steepness of the tibial plateau, and distal femoral torsion appear more likely to contribute to the pathogenesis of CCL disease than femoral angulation, tibial torsion, intercondylar notch stenosis, and increased inclination of the patellar ligament. Labrador Retrievers with cranial angulation of the proximal portion of the tibia (ie, DTA–PTA) > 9°, a tibial plateau angle > 30°, or an anteversion angle > 34° with a femoral condyle trochanteric angle < 20° may be predisposed to CCL disease. A prospective study involving young Labrador Retrievers appears warranted to investigate the relationship between these findings and the development of CCL disease. Prevention of this disease in dogs via modification of skeletal growth and deformation by use of surgical alteration of the growth plates requires further investigations.

a. General Electric high speed F/X scanner, General Electric Medical Systems, Milwaukee, Wis.
b. General Electric Advantax Workstation, General Electric Medical Systems, Milwaukee, Wis.
c. PROC MIXED, SAS, version 9.1, SAS Institute Inc, Cary, NC.

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d. PROC CORR, SAS, version 9.1, SAS Institute Inc, Cary, NC.

e. PROC REG, SAS, version 9.1, SAS Institute Inc, Cary, NC.

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