Canine stifle joint biomechanics associated with tibial plateau leveling osteotomy predicted by use of a computer model

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Objective—To evaluate effects of tibial plateau leveling osteotomy (TPLO) on canine stifle joint biomechanics in a cranial cruciate ligament (CrCL)-deficient stifle joint by use of a 3-D computer model simulating the stance phase of gait and to compare biomechanics in TPLO-managed, CrCL-intact, and CrCL-deficient stifle joints.

Sample—Computer simulations of the pelvic limb of a Golden Retriever.

Procedures—A previously developed computer model of the canine pelvic limb was used to simulate TPLO stabilization to achieve a tibial plateau angle (TPA) of 5° (baseline value) in a CrCL-deficient stifle joint. Sensitivity analysis was conducted for tibial fragment rotation of 13° to –3°. Ligament loads, relative tibial translation, and relative tibial rotation were determined and compared with values for CrCL-intact and CrCL-deficient stifle joints.

Results—TPLO with a 5° TPA converted cranial tibial translation to caudal tibial translation and increased loads placed on the remaining stifle joint ligaments, compared with results for a CrCL-intact stifle joint. Lateral collateral ligament load was similar, medial collateral ligament load increased, and caudal cruciate ligament load decreased after TPLO, compared with loads for a CrCL-deficient stifle joint. Relative tibial rotation after TPLO was similar to that of a CrCL-deficient stifle joint. Stifle joint biomechanics were affected by TPLO fragment rotation.

Conclusions and Clinical Relevance—In the model, stifle joint biomechanics were partially improved after TPLO, compared with CrCL-deficient stifle joint biomechanics, but TPLO did not fully restore CrCL-intact stifle joint biomechanics. Overrotation of the tibial fragment negatively influenced stifle joint biomechanics by increasing caudal tibial translation. (Am J Vet Res 2014;75:626–632)

Degeneration and rupture of the CrCL lead to instability of the stifle joint in dogs.1–3 The CrCL limits hyperextension of the stifle joint as well as internal rotation and cranial displacement of the tibia relative to the femur.2 Rupture of the CrCL, which most commonly results from degeneration of the CrCL over time,4 leads to inflammation, pain, and osteoarthritis.1,2,5,6 It is a leading cause of stifle joint lameness and is one of the most commonly diagnosed orthopedic conditions.7,8 Prevalence of CrCL deficiency was reported as 2.55% across all breeds,9 and management of CrCL-deficient dogs in the United States in 2003 was estimated to cost > $1 billion.10 Large dogs are at greater risk for CrCL damage and typically have less successful outcomes after CrCL rupture without surgical intervention.11 Several corrective stabilization techniques have been proposed to enhance stifle joint stability following CrCL deficiency; these techniques include intracapsular and extracapsular stabilization and osteotomy reconstruction of the proximal aspect of the tibia such as TPLO.4,11 However, because of the lack of comparative long-term data, no surgical stabilization technique prevents osteoarthritis progression or can be considered superior to other stabilization techniques,1 and complication rates during TPLO range from 7% to 31%.12,13 The lack of
reaction forces and moments.\textsuperscript{22} Pelvic limb muscle force analysis was conducted to determine pelvic limb joint reaction forces during the stance phase of gait.\textsuperscript{22–26} These models have been validated in the context of intact and CrCL-deficient stifle joints and have distinct biomechanics of intact and CrCL-deficient stifle joints.\textsuperscript{1,3,11,17–20} Furthermore, TPLO may shift the vertical force vector cranially and thereby influence the extent of alteration of the tibial plateau slope, patient weight, and location of the center of the TPLO radial cut.\textsuperscript{3,15,16} However, TPLO can convert cranial tibial translation to caudal tibial translation, which possibly predisposes the CaCL to increased strain and injury.\textsuperscript{1,3,16} The impact of TPLO on loads of other stifle joint ligaments during the stance phase of a walking gait is unknown.\textsuperscript{1,3,11,17–20} Furthermore, TPLO may shift femorotibial contact pressure caudally and thereby impose a detrimental effect on the menisci, which may lead to medial meniscus damage and osteoarthritis.\textsuperscript{21,27}

Computer simulation models of the canine pelvic limb have been used to parametrically investigate biomechanics of intact and CrCL-deficient stifle joints during the stance phase of gait.\textsuperscript{22–26} These models have predicted an increase in CaCL loads in CrCL-deficient stifle joints. Furthermore, computer simulation models of the canine pelvic limb have been used to predict sensitivity of stifle joint biomechanics to ligament pre-strain, TPA, and femoral condyle diameter.\textsuperscript{22,23,26}

The purposes of the present study were to implement TPLO stabilization into a previously developed quasistatic 3-D computer simulation model of a canine pelvic limb; evaluate loading patterns of stifle joint ligaments, RTT, and RTR; and investigate the influence of tibial fragment rotation angle in the sagittal plane on stifle joint biomechanics. We hypothesized that TPLO would reduce cranial tibial translation and internal tibial rotation, compared with that for a CrCL-deficient stifle joint, and result in ligament loads with values similar to those for a CrCL-intact stifle joint. Additionally, we hypothesized that rotation angle of the tibial fragment in the sagittal plane would affect stifle joint biomechanics.

Materials and Methods

Computer model of a canine pelvic limb—A 3-D computer model of a canine pelvic limb was previously developed by use of computer-aided design and modeling software.\textsuperscript{22,24} The model was based on the left pelvic limb of a 5-year-old 33-kg neutered male Golden Retriever that had no known orthopedic or neurologic disorders. To develop the model, the dog was walked on a leash in a straight line; motion-capture kinematics and force platform kinetics were collected during the stance phase of a walking gait.\textsuperscript{22} Inverse-dynamics analysis was conducted to determine pelvic limb joint reaction forces and moments.\textsuperscript{25} Pelvic limb muscle forces that crossed the stifle joint were represented as linear force vectors following each respective muscle path (n = 22 muscles), and each muscle force was calculated throughout the stance phase by means of the minimization of maximal muscle stress strategy\textsuperscript{22} and the 3-D constraint equation as follows:

\[
M = \sum_{j=1}^{n} F_i \times r_i
\]

where \(M\) is the stifle reaction moment determined via inverse-dynamics analysis, \(F_i\) is the muscle force, and \(r_i\) is the moment arm of muscle \(i\). Muscle forces were constrained so that they did not exceed 60% of maximal contraction force.\textsuperscript{22,27} Ligaments were represented as tension-only elements that carried a load when stretched beyond their neutral length, as determined by use of the following equations\textsuperscript{28}:

\[
F = \begin{cases} 
0; & \epsilon \leq 0 \\
0.25k (\epsilon^3 - \epsilon); & 0 < \epsilon \leq 2\epsilon^\text{m} \\
k (\epsilon^\text{m} - \epsilon); & 2\epsilon^\text{m} < \epsilon 
\end{cases}
\]

where \(F\) is the ligament load, \(\epsilon\) is the strain in ligament \(j\), \(k\) is the stiffness value for ligament \(j\), and \(\epsilon^\text{m}\) is the parameter that defines the ligament response transition from the toe region to the linear region. Ligament stiffness values were approximated as the product of each ligament cross-sectional area and the mean CrCL longitudinal tendon modulus.\textsuperscript{29}

Femoromeniscal contact elements were included to prevent penetration between the femur and menisci. Femoromeniscal forces were determined by use of the following equation:

\[
F_r = (k \times g^\prime) + (dg/dt)f(g, c\text{max}, d\text{max})
\]

where \(F_r\) is the contact force, \(k\) is the stiffness, \(g\) is the contact penetration during simulation, \(\epsilon\) is the elastic component exponent, \(dg/dt\) is the instantaneous rate of change of \(g\) with respect to time, and \(f(g, c\text{max}, d\text{max})\) is a contact penetration step function whereby \(c\text{max}\) is the maximum damping and \(d\text{max}\) is the penetration at which \(c\text{max}\) occurs.\textsuperscript{22} Femoromeniscal contact equation constants used in the present study were as follows: \(k = 1,500\ N/mm, \epsilon = 1.1, c\text{max} = 0.5\ N/s/mm,\) and \(d\text{max} = 0.8\ mm\). Penetration between the tibia and femur and between the patella and femur were prevented, with contact represented by use of this equation. Femorotibial and femoropatellar contact equation constants used in the present study were as follows: \(k = 30,000\ N/mm, \epsilon = 1.5, c\text{max} = 28\ N\times s/mm,\) and \(d\text{max} = 0.1\ mm\).

Canine pelvic limb model with implemented TPLO—Tibial plateau leveling osteotomy, which reduces the TPA through rotation of the fragment of the proximal aspect of the tibia,\textsuperscript{14} was implemented in the computer simulation model of the canine pelvic limb. Motion-capture kinematics, ground reaction forces, and optimized muscle forces used in the previously developed model\textsuperscript{22} were applied to the canine pelvic limb model incorporating the TPLO. A 30-mm radial osteotomy was centered on the midpoint between the intercondylar tubercles of the tibia\textsuperscript{25} in the sagittal plane (Figure 1). The tibial fragment was concentrically rotated 17° caudally in the sagittal plane to reduce the TPA from 22° to 5° relative to the perpendicular of the tibia long axis (5° was defined as the baseline TPA).\textsuperscript{15} However, rotation of the fragment in the sagittal plane introduced slight medial femoromeniscal interference.
Model simulation—The canine pelvic limb model was developed by use of rigid body motion software and was used to simulate the stance phase of gait. The CaCL, MCL, and LCL loads; tibial translation; and tibial rotation at discrete 10% intervals were determined. The RTT between the CrCL-intact and CrCL-deficient stifle joints was defined by use of the following equation:

$$\text{RTT} = \text{FT}_{\text{deficient}} - \text{FT}_{\text{intact}}$$

where $\text{FT}_{\text{deficient}}$ represents fixed point tibial translation (ie, the distance between the tibial tuberosity position relative to a fixed point on the femur in the craniocaudal direction) for the CrCL-deficient stifle joint and $\text{FT}_{\text{intact}}$ is fixed point tibial translation for the CrCL-intact stifle joint. The RTT between the CrCL-intact and TPLO-managed stifle joints, $\text{RTT}_{\text{TPLO}}$, was defined by use of the following equation:

$$\text{RTT}_{\text{TPLO}} = \text{FT}_{\text{TPLO}} - \text{FT}_{\text{intact}}$$

where $\text{FT}_{\text{TPLO}}$ represents fixed point tibial translation for the TPLO-managed stifle joint.

The RTR between the CrCL-intact and CrCL-deficient stifle joints was defined by use of the following equation:

$$\text{RTR} = \text{R}_{\text{deficient}} - \text{R}_{\text{intact}}$$

where $\text{R}_{\text{deficient}}$ is the internal-external rotation (as defined in another study) for the CrCL-deficient stifle joint and $\text{R}_{\text{intact}}$ is the internal-external rotation for the CrCL-intact stifle joint. The RTR between CrCL-intact and TPLO-managed stifle joints, $\text{RTR}_{\text{TPLO}}$, was defined by use of the following equation:

$$\text{RTR}_{\text{TPLO}} = \text{R}_{\text{TPLO}} - \text{R}_{\text{intact}}$$

where $\text{R}_{\text{TPLO}}$ is the internal-external rotation for the TPLO-managed stifle joint. A positive RTR value corresponds to internal rotation, and a negative RTR value corresponds to external rotation.

Data analysis—Outcome measures for a TPLO-managed stifle joint with baseline TPA were compared with outcome measures for both CrCL-intact and CrCL-deficient stifle joints by use of the peak value test. An ANOVA or similar statistical tests were not performed because model-predicted outcomes were single values without variability. Peak outcome measures for the various models were considered significantly different when the absolute difference was $> 20\%$ or differences between stance-phase intervals were $> 10\%$. Model sensitivity to rotation angle of the tibial fragment in the sagittal plane was assessed by use of a sensitivity index (defined as the percentage change in outcome measure divided by the percentage change in input parameter [ie, a 5% increase in outcome measure attributable to a 10% increase in input parameter represented a sensitivity index of 0.5]).

Higher absolute values for sensitivity indices indicated that outcomes had greater sensitivity to that parameter.

Results

Ligament loads for a CrCL-deficient stifle joint managed with TPLO were determined throughout the stance phase (Figure 2). Similarly, $\text{RTT}_{\text{TPLO}}$ and $\text{RTR}_{\text{TPLO}}$ were calculated.
RTT<sub>TPLO</sub> for a TPLO-managed CrCL-deficient stifle joint were determined throughout the stance phase (Figure 3). Peak CaCL, LCL, and MCL loads in the TPLO-managed stifle joint were 54% of body weight (at 30% stance phase), 108% of body weight (at 20% stance phase), and 58% of body weight (at 50% stance phase), respectively. Also, the model predicted that peak LCL and MCL loads increased 640% and 520% from the CrCL-intact stifle joint to the TPLO-managed stifle joint, respectively. Following TPLO, peak LCL load occurred at 20% stance phase, with a simultaneous increase in external rotation from 0.2° to 2.5° and peak vertical ground reaction force. The model predicted that TPLO reduced peak CaCL load by 70%, compared with the peak CaCL load for the CrCL-deficient stifle joint, whereas peak LCL and MCL loads increased 4% and 56%, respectively. Ligament loads for the TPLO-managed stifle joint exceeded the peak value test criteria when compared with ligament loads for CrCL-intact and CrCL-deficient stifle joints. Thus, TPLO-managed stifle joint biomechanics differed from CrCL-intact and CrCL-deficient stifle joint biomechanics.

Caudal RTT<sub>TPLO</sub> was present throughout the stance phase and peaked at −4.8 mm (at 30% stance phase). The RTT was 20.2 mm at 50% stance phase, whereas RTT<sub>TPLO</sub> was −2.9 mm at the same point of the stance phase, which represented a reduction of 114%. The RTR<sub>TPLO</sub> peaked at an internal rotation of 5.2° (at 50% stance phase) and ranged between −2.9° and 1.9° for other stance phases. Comparison of RTT to RTT<sub>TPLO</sub> indicated that the peak value test criteria were exceeded. The RTR and RTR<sub>TPLO</sub> did not differ because their difference was within the peak value test criteria.
Ligament loads throughout the stance phase were compared for each rotation angle of the tibial fragment in the sagittal plane (Figure 4). Similarly, RTT\textsubscript{TPLO} and RTR\textsubscript{TPLO} were determined for each rotation angle of the tibial fragment in the sagittal plane (Figure 5). Each outcome measure was sensitive to the rotation angle of the tibial fragment in the sagittal plane as indicated by sensitivity indices (Table 1).

**Discussion**

Little is known regarding in vivo CaCL, LCL, and MCL loading following TPLO. Findings from the computer model simulations in the present study predicted differences in peak CaCL, LCL, and MCL loads between TPLO-managed and CrCL-intact stifle joints, with increases in all ligament loads after TPLO. Therefore, TPLO did not return biomechanics to those of a CrCL-intact stifle joint. Instead, peak CaCL loads increased by 88% in a TPLO-managed stifle joint, compared with those for a CrCL-intact stifle joint. Similarly, results of an in vitro study indicated that CaCL strain increased following TPLO, compared with CaCL strain for an intact stifle joint at midstance. In another study, a mathematical model indicated an increase in CaCL loads following tibial plateau rotation that corresponded to TPLO. In the present study, the likelihood of ligament injury or degradation attributable to increased loading was higher in all ligaments following TPLO, compared with the likelihood for the CrCL-intact stifle joint. However, the likelihood of CaCL injury or degradation may decrease following TPLO, compared with the likelihood for a CrCL-deficient stifle joint.

The RTT\textsubscript{TPLO} decreased, compared with RTT. Tibial translation after TPLO was converted to the caudal direction, which is consistent with findings of other studies. The model in the present study predicted that RTT\textsubscript{TPLO} was reduced by 114% from RTT at 50% stance phase. In comparison, in vitro studies have indicated a reduction in RTT\textsubscript{TPLO} at midstance ranging from 90% to 133%. At midstance, RTR\textsubscript{TPLO} increased by 14% (from 4.6° to 5.2°), compared with RTR. Although RTR\textsubscript{TPLO} was predicted as being greater than RTR at 50% stance phase in the present study, this magnitude was lower than RTR\textsubscript{TPLO} measured in vitro (10°) in another study. In the present study, model results suggested that TPLO precedes in reducing tibial translation and CaCL loading in a CrCL-deficient stifle joint, but ligament loads appeared to exceed those of their natural state in a TPLO-managed stifle joint.

Outcome measures predicted by the computer model in the present study were sensitive to rotation angle of the tibial fragment in the sagittal plane of a TPLO-managed stifle joint. Incrementally rotating the tibial fragment to achieve a 13° to –3° TPA generally increased CaCL loads, which is consistent with in vitro CaCL strain data. In that study, investigators found that CaCL strain increased from the minimum rotation angle of the tibial fragment necessary to prevent cranial tibial translation (TPA = 6.5°) through a level tibial plateau (TPA = 0°) to the maximum rotation angle evaluated (TPA = –13.5°). Similarly, the computer model in the present study predicted that LCL loads generally increased with rotation of the tibial fragment from a TPA of 13° to –3°. However, MCL loads decreased from a TPA of 13° to 9° and then increased from a TPA of 3° to –3°, with a local minimal region of stability between 3° and 9°. The authors are unaware of any in vitro studies that have been conducted to evaluate the effect of tibial fragment rotation on LCL or MCL loads.

In the present study, the computer model predicted that cranial tibial translation during the stance phase was eliminated and converted to caudal tibial translation for all TPAs evaluated (13° to –3°). These findings are in agreement with in vitro findings that indicated cranial tibial translation was eliminated at midstance for a TPA of 5°, 6°, and 6.5° ± 2.6° (mean ± SD). Although in vitro studies have indicated that a TPA ranging from 5° to 6.5° after TPLO will eliminate cranial tibial translation and restore stifle joint stability, the model in the present study predicted that cranial...
tibial translation was converted to caudal tibial translation for TPAs of 5° and 7° at midstance. This difference could have been attributable to the inclusion of active semimembranosus and semitendinosus muscles in the model that were not represented in in vitro studies. Inclusion of these additional muscles in the model applied caudally directed forces that may have led to caudal tibial translation.

Peak RTR ranged from 4.9° to 7.0° of internal rotation for all TPAs evaluated, except for 13° whereby RTR was −2.3° (which represented external rotation). This local minimum in RTR may indicate a region of stability in a TPLO-managed stifle joint. The preoperative CrCL-deficient stifle joint is unstable at midstance in vivo and in vitro, but after the tibial fragment is rotated for TPLO, stifle joint stability improves.3,15,32 However, there are differences in the recommended extent of tibial fragment rotation necessary for joint stability.1,3,12,15,21 Additionally, the integrity of the medial meniscus can influence stability in TPLO-managed stifle joints.32 Findings from the present model suggested that a TPLO-managed stifle joint was stable for a TPA < 13°, and sensitivity analysis indicated trade-offs among the outcome measures for the range of TPAs evaluated. Although in vitro1,15 and in vivo2 studies have indicated that cranial tibial translation is eliminated for TPAs of 5° to 6.5°, normal ground reaction forces in TPLO-managed hind limbs have been reported in dogs with postoperative TPAs as high as 14°.31 These findings suggest that the TPA necessary for stifle joint stability after TPLO may differ among dogs on the basis of subject-specific anatomic and morphological characteristics. Morphological parametric variation can affect stifle joint biomechanics in intact and CrCL-deficient stifle joints.31

Tibial plateau leveling osteotomy was implemented in the canine pelvic limb computer model. However, predicted findings from the computer model should be interpreted with consideration for the following limitations. Clinically, TPLO is implemented by use of TPA measurement and identification of bony landmarks for preoperative planning, intraoperative execution, and postoperative assessment. Therefore, identification of TPA and bony landmarks in the model may differ across individual patients, and TPA measurements can differ on the basis of surgeon experience and measurement technique.3 The sensitivity analysis evaluating rotation angle of the tibial fragment in the sagittal plane was conducted to assess its influence on stifle joint biomechanics in TPLO. We found that the rotation angle of the tibial fragment influenced stifle joint biomechanics for the range of angles evaluated. The influence of additional TPLO implementation parameters on stifle joint biomechanics should also be investigated.

Meniscal elements in the computer model were intended to represent healthy intact menisci. The integrity of the meniscus was maintained and did not reflect degradation over time that could result from CrCL deficiency or TPLO. Neither meniscal release nor meniscectomy was represented in the model. These procedures result in detachment, transection, or removal of portions of the medial meniscus15 and are performed by some surgeons during TPLO to prevent future meniscal damage. Moreover, no surgical complications were represented in simulations, and the postoperative stifle joint was represented as fully healed with no associated lameness or functional defects. Additionally, the present computer model represented a single point in time and did not account for longitudinal in vivo changes in conditions. For example, osteoarthritis can progress in CrCL-deficient stifle joints after TPLO,36 but the effects of osteoarthritis on stifle joint biomechanics were not investigated in the present study. Finally, the computer model represented an approximation of a complex biomechanical system. Muscle forces were estimated with an optimization algorithm, paths of muscle forces were described as linear vectors, each ligament was represented as a single element rather than multiple ligament bundles, and the menisci were modeled as rigid bodies that may not have represented the inherent flexibility of menisci. However, despite these limitations, CrCL-intact stifle joint kinematics measured in vivo22 are similar to stifle joint kinematics predicted by use of the present computer model.

Stifle joint biomechanics were generally improved after TPLO, compared with CrCL-deficient stifle joint biomechanics. However, TPLO did not fully restore CrCL-intact stifle joint biomechanics. Rotation angle of the tibial fragment in the sagittal plane used for TPLO influenced stifle joint biomechanics.

References