

Effects of tibial plateau angle and spacer thickness applied during in vitro canine total knee replacement on three-dimensional kinematics and collateral ligament strain

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Objective—To quantify the 3-D kinematics and collateral ligament strain of stifle joints in cadaveric canine limbs before and after cranial cruciate ligament transection followed by total knee replacement (TKR) involving various tibial plateau angles and spacer thicknesses.

Sample—6 hemi-pelvises collected from clinically normal nonchondrodystrophic dogs (weight range, 25 to 35 kg).

Procedures—Hemi-pelvises were mounted on a modified Oxford knee rig that allowed 6 degrees of freedom of the stifle joint but prevented mechanical movement of the hip and tarsal joints. Kinematics and collateral ligament strain were measured continuously while stifle joints were flexed. Data were again collected after cranial cruciate ligament transection and TKR with combinations of 3 plateau angles (0°, 4°, and 8°) and spacer thicknesses (5, 7, and 9 mm).

Results—Presurgical (ie, normal) stifle joint rotations were comparable to those previously documented for live dogs. After TKR, kinematics recorded for the 8°, 5-mm implant most closely resembled those of unaltered stifle joints. Decreasing the plateau angle and increasing spacer thickness altered stifle joint adduction, internal rotation, and medial translation. Medial collateral ligament strain was minimal in unaltered stifle joints and was unaffected by TKR. Lateral collateral ligament strain decreased with steeper plateau angles but returned to a presurgical level at the flattest plateau angle.

Conclusions and Clinical Relevance—Among the constructs tested, greatest normalization of canine stifle joint kinematics in vitro was achieved with the steepest plateau angle paired with the thinnest spacer. Furthermore, results indicated that strain to the collateral ligaments was not negatively affected by TKR. (*Am J Vet Res* 2014;75:792–799)

Cranial cruciate ligament deficiency is the leading cause of stifle joint osteoarthritis in dogs.^{1–3} In 2003, the annual economic impact related to this condition was estimated to be \$1.32 billion in the United States alone.⁴ The reported prevalence of CrCL deficiency in canids has more than doubled over the last 30 years because of longer life expectancies and improved diagnosis of this joint condition.⁵ Rupture of the CrCL causes stifle joint instability and abnormal joint motions that can damage surrounding cartilage and tissues. In a study⁶ of cadaveric canine pelvic limbs, both cranial tibial thrust and internal tibial rotation increased immediately after transection of the

ABBREVIATIONS

CrCL	Cranial cruciate ligament
LCL	Lateral collateral ligament
MCL	Medial collateral ligament
OKR	Oxford knee rig
TKR	Total knee replacement
TPA	Tibial plateau angle

CrCL and failed to improve over time. For dogs with prior surgical procedures that failed or severe stifle joint osteoarthritis, TKR (a more invasive surgical procedure) may be required to restore the function of the joint.⁷ To date, TKR has been shown to improve postoperative joint function in canids, but full function of the stifle joint after TKR has not been achieved.⁷

The TPA is a measure of the angle between the articulating surface of the tibia and the femur. It is typically measured on radiographic views as the angle between the long axis of the femur (extending from the midpoint of the intercondylar eminences to the center of the tarsal joint) and the medial tibial plateau (extending from the most cranial to caudal radiographic margins of the proximal portion of the tibia).⁸ Although the normal canine TPA is approximately 24°, implants are currently being inserted at 6° in an effort to mini-

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minimize excess cranial tibial thrust that develops as a result of the loss of the CrCL.⁷ For dogs, a TPA of 6° has been promoted as optimal for tibial plateau leveling procedures to prevent development of cranial tibial thrust, but the clinical importance of this angle has been questioned.^{7,9,10} Thus, the assumption that a TPA of 6° is the optimal for TKR in dogs may not be correct because the caudal cruciate ligament remains intact during a tibial plateau leveling osteotomy but is removed during TKR. Decreasing the TPA to such a small angle may induce excess caudal tibial thrust and affect joint kinematics. To simulate the natural kinematic motions of the stifle joint in a cadaveric pelvic limb, the joint must travel through all 6 degrees of freedom clinically described as 3 rotations (flexion and extension, adduction and abduction, and internal-external rotation) and 3 translations (cranial-caudal, medial-lateral, and proximal-distal translations). The OKR was first designed for the simulation of knee kinematics in cadaveric human specimens and has been shown to allow all 6 degrees of freedom of that joint.¹¹

To date, limited kinematic data obtained by means of an OKR for canine stifle joints are available from previous *in vitro* studies^{6,9,12,13} that mainly focused on cranial-caudal translation and axial rotation during a static pose at the midpoint of the stance phase. Results obtained from cadaveric pelvic limbs have varied greatly and are difficult to compare with *in vivo* data. It has been suggested that techniques applied to cadaveric pelvic limbs may cause distortion of the stifle joint's natural motions because of the loss of muscle tensions and constraints placed across the joint.^a By retaining both the soft tissue around the joint and the adjacent hip and tarsal joints and by continuously collecting data during a full motion cycle, *in vitro* findings may be more comparable to *in vivo* data.¹¹

The primary purpose of the study reported here was to quantify the 3-D kinematics and collateral ligament strain of cadaveric canine stifle joints before and after CrCL transection and subsequently after TKR involving various TPAs and spacer thicknesses. It was expected that different TPAs and different-sized tibial spacers applied during TKR would affect the *in vitro* stifle joint kinematics and ligament strain.

Materials and Methods

Sample—Six hemi-pelvises were collected from clinically normal nonchondrodystrophic dogs (weight range, 25 to 35 kg) euthanized for reasons unrelated to the study or orthopedic disease. Each hemi-pelvis was radiographed (Figure 1), and TKR templates were superimposed on the radiographic views to determine implant size. Hemi-pelvises were stored at -40°C and thawed at room temperature (approx 22°C) for 24 hours before testing.



Figure 1—Lateral radiographic views of 1 of 6 cadaveric canine hemi-pelvises (from stifle joint to tarsus) used to quantify the *in vitro* 3-D kinematics and collateral ligament strain of stifle joints before and after CrCL transection followed by TKR involving various TPAs and spacer thicknesses. A—Preoperative view to illustrate a TPA of 25°. B—View obtained after CrCL and TKR to illustrate a TPA of 10°. Implant components (white component at the distal end of the femur), screws, and Kirschner wire pins (white thick lines) are visible. Imaging software was used to measure the TPA angle (shown as a white thin line nearest the joint on the radiographic view) and the estimated length of the tibia (shown as a white thin line along the bone on the radiographic view).

Modified OKR—To mount each specimen to a modified OKR, a custom-made bar of angle iron was bolted through the ischial tuberosity and sacroiliac joint of the ilium and attached to the crosshead of a materials-testing machine^b (Figure 2). The testing rig traveled at a rate of 50 cm/min to provide controlled vertical motion that simulated stifle joint flexion and extension. The paw was attached to a platform with a dog boot that tightened below the tarsus without visibly impeding range of motion of the tarsal joint. Sliders were adjusted to align the tibial crest directly over the



Figure 2—Photograph of a hemipelvis collected from a clinically normal nonchondrodystrophic dog and mounted on a modified OKR for use in a study to quantify the 3-D kinematics and collateral ligament strain of stifle joints in canine cadavers before and after CrCL transection followed by TKR involving various TPAs and spacer thicknesses. The paw is attached rigidly to a platform, and an angle iron is bolted through the ischial tuberosity and sacroiliac joint of the ilium.

paw at full extension of the stifle joint. The angle iron was free to rotate in the craniocaudal plane, simulating angular spinal movement. The specimen was also free to translate in the mediolateral and craniocaudal planes through the use of sliders, allowing the full 6 degrees of freedom of the stifle joint. Each specimen was cycled from full extension to full flexion of the stifle joint and returned to full extension for 5 cycles while data were collected. Because the specimen was in a static position when cycling commenced, the first cycle was not used in data analysis.

Kinematic data collection—After each specimen was mounted to the modified OKR, a Kirschner wire (diameter, 1.6 mm) was drilled into the bone at 6 anatomic locations (the greater trochanter, lateral epicondyle, medial epicondyle, tibial crest, lateral malleolus, and medial malleolus), and reflective markers were attached to the end of each wire. Prior to testing, the stifle and tarsal joints underwent full range of motion to en-

sure no impingement from the wires. During the motion cycle, 3-D marker positions were recorded with a set of 5 infrared cameras^{c,d} at 200 Hz and data collection software.^e From the raw marker location data, the kinematics of the stifle joint was calculated with a custom program^f and the joint coordinate system technique described by Fu et al.¹⁴ All kinematic parameters are reported in terms of motion of the tibia relative to the femur. Translations were calculated from the tibial crest to the midpoint of the femoral epicondyles along the long femoral axis. Except for flexion-extension data, kinematics was calculated in reference to the initial position at full extension with an angle of 0° or a translation of 0 mm (indicating the joint was in the same orientation as the initial frame).

Kinematic parameters—The kinematic rotation of the stifle joint was described by the adduction-abduction, internal-external rotation, and flexion-extension data. For purposes of this study, stifle joint translation was measured as the change in physical distance between the tibial crest and the midpoint of the epicondyles; data collected were cranial-caudal, medial-lateral, and proximal-distal translations.

Collateral ligament strain measurement—Displacement sensors^g are comprised of a main body and freely moving core and detect core position by measuring the coil's differential reluctance by use of a sine wave excitation and synchronous demodulator.¹⁵⁻¹⁷ Sensors were connected directly to a signal conditioner.^c Both the sensors and signal conditioner were calibrated by the manufacturer as a single unit. The displacement sensors were attached to the collateral ligaments by means of barbs on the sensors; the barb locations were marked with a fine-tip marker pen to allow reattachment of the barbs if removed. The long axis of each sensor was aligned with the long axis of the ligament with the entire pelvic limb at full extension. The sensor wire was loosely sutured to the overlying tissue to prevent sensor displacement as a result of wire movement. For strain calculations, the original distance between the sensor barbs with the entire pelvic limb at full extension was measured with calipers (accuracy, ± 0.01 mm). The sensors remained on the ligaments for the duration of the experiment. Displacement data were collected at 200 Hz. Displacement for each sensor was calculated from the output voltages with the factory calibration polynomial fit equations. Strain on each ligament was calculated as the ratio of sensor displacement to original distance between the barbs. Strain data were coordinated with the kinematic data by means of a custom program^h that determined the length of a motion cycle from the flexion-extension kinematic data and the point at which displacement values deviated from the baseline data (ie, data obtained before CrCL transection).

TKR components—Tibial baseplates^h simulating 3 TPAs that accepted a snap fit of the stock trial tibial spacers were custom manufactured (Figure 3). Combinations of 3 TPAs (8°, 4°, or 0°) and 3 tibial spacer thicknesses (5, 7, or 9 mm) were used to create 6 tibial plateau constructs for evaluation; these construct combinations were 0° and 5 mm, 4° and 5 mm, 4° and 7 mm,

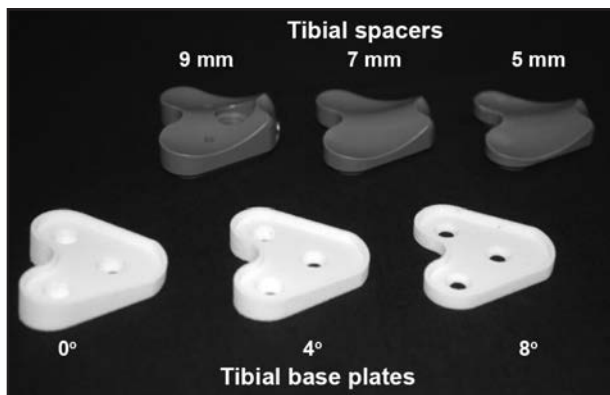


Figure 3—Photograph of commercially available stock tibial spacers (5 to 9 mm in thickness [top]) and custom-made tibial baseplates that simulate 8°, 4°, and 0° tibial osteotomies (bottom).

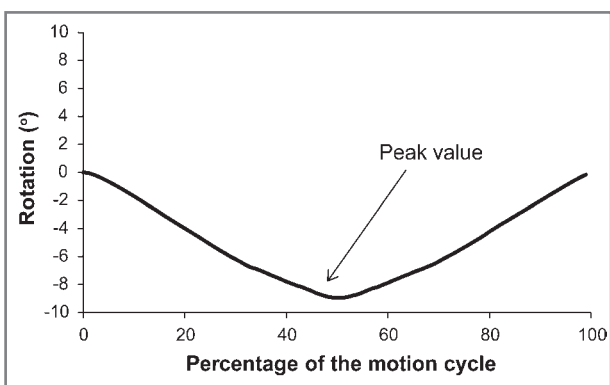


Figure 4—Representative plot of internal-external rotation of the tibia relative to the femur in a cadaveric canine hemi-pelvis mounted in a modified OKR during cycling of the stifle joint from full extension to full flexion and return to full extension (designated as 0% through 100% of the motion cycle). Positive rotation values represent external rotation; negative rotation values represent internal rotation. Five motion cycles were performed and data were collected; because the specimen was in a static position when cycling commenced, the first cycle was not used in data analysis. Peak values were defined as the largest absolute value on the plot; for each specimen, a mean peak value from the 4 motion cycles was calculated and used in statistical analyses of differences among treatments.

8° and 5 mm, 8° and 7 mm, and 8° and 9 mm. Tibial baseplates were designed to be implanted on a 10° tibial cut that was generated by means of an extramedullary tibial alignment guide. The custom tibial baseplates were manufactured with screw holes that allowed them to be screwed into the tibial osteotomy without altering the position of the baseplates between components. Femoral implant components and tibial spacers used during the procedure were stock components.^h

Experimental procedure—Baseline data for each stifle joint were obtained prior to CrCL transection. Each hemi-pelvis was manipulated so that the unaltered stifle joint moved through full extension to full flexion and was returned to full extension for 5 cycles while baseline kinematic and strain data were collected. With the specimen on the modified OKR, the surgeon (SCB) opened the joint capsule and completely severed the CrCL. The hemi-pelvis was again manipulated through 5 motion cycles while stifle joint data were collected; the reflective markers were then removed, but

the displacement sensors and Kirschner wires were left in place.

The same surgeon performed the TKR procedure for all specimens as previously described.⁷ For the first TKR experiment, the 8° tibial baseplate was screwed into place and the 5-mm custom tibial spacer was snap fit onto the baseplate. With the stifle joint reduced, the patellar tendon was replaced in its natural position, and the joint capsule and overlying tissue were closed with sutures.

The specimen was remounted on the rig with the stifle joint at full extension and was run through 5 motion cycles for data collection. The specimen remained on the rig with markers and sensors in place while the custom tibial spacer implants were sequentially exchanged to create tibial plateau constructs of increasing thickness. To change the spacer, the sutures were removed, the joint was flexed, and the tibia was cranially luxated for access to the implant. With the joint still in flexion, the spacer present was snapped out and replaced with the next largest spacer. The pelvic limb was reduced with pressure applied on the spacer to move the femoral component into the spacer tracks. The joint was sutured closed after each implant exchange. With the 7-mm spacer and then the 9-mm spacer inserted, the specimen was run through 5 motion cycles for data collection.

Once all 3 spacer thicknesses (5, 7, and 9 mm) were tested at 8°, the tibial baseplate was removed, and the 4° baseplate was implanted into the same holes previously drilled into the tibial osteotomy to ensure identical alignment among baseplates. With the 4° baseplate, the 5- and 7-mm spacers were inserted into the stifle joint and tested in order of increasing thickness. The 4° baseplate was then replaced with the 0° baseplate, and the stifle joint was tested with only the 5-mm spacer. Baseplates with decreasing angles were tested with only the thinner spacers to avoid overexpansion of the joint and damage to the surrounding ligaments during testing. Each specimen was run through 5 motion cycles with each set of implants. A final radiographic view was obtained to determine any differences between target and actual TPA (Figure 1).

Data collection and analysis—A complete motion cycle started with the specimen in a position at which the stifle joint was in maximum extension, then moved to a position of maximum flexion, and then moved back to its bent starting position. That is, 0% of a motion cycle was when the pelvic limb was stretched to the greatest normal extension allowed by the stifle joint, 50% of a motion cycle was when the pelvic limb was bent to its lowest position allowed by the stifle joint (ie, full flexion), and 100% of a motion cycle was when the pelvic limb was returned to the position that coincided with 0% of the motion cycle (ie, return to full extension). Curves of the percentage of the motion cycle versus external-internal rotation (Figure 4) were used to determine the peak rotation that occurred during testing. Analysis of the adduction-abduction rotation indicated that this peak value occurred at the same location of the motion cycle. The peak values were also at the same location of the motion cycle for the external-internal rotation, cranial-caudal translation, lateral-medial translation, and proximal-distal rotation.

Table 1—Mean ± SD peak kinematics and collateral ligament strain for the stifle joints of 6 canine cadaver hemi-pelvises before (unaltered) and after CrCL transection and after subsequent TKR with various TPAs and spacer thicknesses.

Stifle joint status or TKR construct applied	Rotation (°)		Translation (mm)			Strain (%)	
	Adduction (+) and abduction (–)	External (+) and internal (–)	Cranial (+) and caudal (–)	Lateral (+) and medial (–)	Proximal (+) and distal (–)	MCL	LCL
Unaltered	4.3 ± 2.8 ^a	–8.9 ± 5.5 ^a	–20.0 ± 5.5 ^a	6.2 ± 5.9 ^a	–14.0 ± 4.3 ^a	0.11 ± 0.03 ^a	5.72 ± 1.08 ^a
CrCL transected	1.2 ± 5.2	–7.8 ± 4.5	–20.4 ± 5.5	4.9 ± 4.3 [*]	–15.5 ± 4.0 ^a	0.25 ± 0.23	7.02 ± 4.90
8°, 5 mm	4.3 ± 2.5 ^b	–3.9 ± 4.3 ^b	–22.0 ± 5.3 ^{a,b}	2.1 ± 7.3 ^{a,b}	–8.4 ± 3.6 ^b	0.02 ± 0.03 ^a	0.49 ± 0.53 ^b
8°, 7 mm	1.6 ± 5.8 ^{b,c}	1.9 ± 1.0 ^c	–24.4 ± 4.4 ^b	–4.7 ± 4.9 ^{c,d}	–7.5 ± 5.3 ^b	0.07 ± 0.09 ^a	0.09 ± 0.12 ^b
8°, 9 mm	1.6 ± 1.6 ^b	0.5 ± 4.4 ^c	–24.9 ± 5.5 ^b	1.1 ± 3.1 ^{b,d}	–8.9 ± 6.8 ^{a,b}	0.62 ± 0.88 ^a	1.25 ± 1.23 ^b
4°, 5 mm	–2.8 ± 4.6 ^d	5.1 ± 6.9 ^c	–24.4 ± 3.6 ^b	–7.3 ± 3.1 ^c	–8.0 ± 4.3 ^b	0.67 ± 0.99 ^a	0.21 ± 0.09 ^b
4°, 7 mm	–0.6 ± 2.3 ^{b,d}	2.7 ± 4.4 ^c	–24.4 ± 5.0 ^b	–4.9 ± 3.2 ^{c,d}	–7.0 ± 5.3 ^b	NR	NR
0°, 5 mm	–4.0 ± 3.7 ^{c,d}	6.5 ± 8.0 ^c	–23.3 ± 4.0 ^{a,b}	–8.7 ± 4.7 ^c	–7.9 ± 3.9 ^b	0.50 ± 0.40 ^a	7.63 ± 1.65 ^a

Each specimen was mounted on a modified OKR; the paw was attached rigidly to a platform, and an angle iron was bolted through the ischial tuberosity and sacroiliac joint of the ilium. Before and after CrCL transection and after subsequent TKR, data were collected during cycling of each stifle joint from full extension to full flexion and return to full extension. Five motion cycles were performed; because the specimen was in a static position when cycling commenced, the first cycle was not used in data analysis. Kinematics is reported as motion of the tibia relative to the femur. Peak values were defined as the largest absolute value; a mean peak value from the 4 motion cycles for each specimen was calculated. Data obtained after CrCL were not compared statistically with data obtained after subsequent TKR.

*For this variable, mean peak values for stifle joints before and after CrCL transection differed significantly ($P < 0.05$).

NR = Data were not reported owing to malfunction of instruments used during testing.

^{a-d}For a given variable, mean peak values with different superscript letters differed significantly ($P < 0.05$).

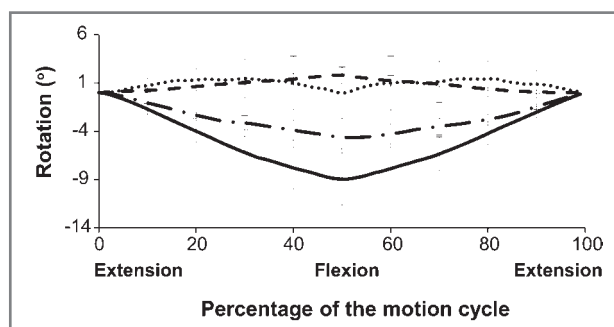


Figure 5—Plot of internal-external rotation (mean ± SEM) of the tibia relative to the femur in 6 cadaveric canine hemi-pelvises before (solid line) and after CrCL transection followed by TKR involving an 8° baseplate and a 5-mm spacer (dotted and dashed line), 7-mm spacer (dashed line), or 9-mm spacer (dotted line). Specimens were mounted in a modified OKR, and data were collected during cycling of the stifle joint from full extension to full flexion and return to full extension (designated as 0% through 100% of the motion cycle). See Figure 4 for remainder of key.

In a similar manner, peak strains for the MCL and LCL were determined.

Mean peak values were calculated for each trial with each specimen and used in paired *t* tests of data within specimen among treatments. The questions of interest included whether there was significant intraspecimen variability in kinematics or strain among TKR trials with differing TPAs and with differing tibial spacer thicknesses. Comparison with a paired *t* test was performed to determine whether there were significant differences in kinematic and strain data attributable to TPA or spacer thickness. Statistical analyses were performed by use of software.¹ A value of $P < 0.05$ was considered significant for all analyses.

Results

Unaltered versus CrCL-deficient stifle joints—

Among the 6 stifle joints, CrCL transection caused a significant decrease in lateral translation (Table 1). The CrCL transection did not significantly alter any other kinematics or strain to the MCL or LCL.

TPA—After the TKR was implanted, the mean TPA among the 6 specimens was $10.2 \pm 0.8^\circ$. All tibial plateau constructs (ie, baseplate plus spacer) caused a decrease in peak distal translation (determined on the basis of movement of the tibia relative to the femur), compared with findings for the unaltered stifle joints; the decrease was significant for all but the 8°, 9-mm construct (Table 1). The change in peak distal translation did not vary significantly among constructs. Compared with findings for the unaltered stifle joints, the 8°, 7-mm; 8°, 9-mm; 4°, 5-mm; and 4°, 7-mm tibial plateau constructs caused an increase in peak caudal translation, whereas the effect of the 8°, 5-mm or 0°, 5-mm construct was not significant.

Compared with findings for the unaltered stifle joints, TKR with an 8° baseplate resulted in overall decreases ($P < 0.05$) in both internal rotation (Figure 5) and lateral translation during the motion cycle. Post-TKR peak internal rotation was decreased significantly from preoperative values for each 8° construct. Peak lateral translation was decreased from preoperative values for each 8° construct, but this change was not significant for the 8°, 5-mm construct. Both the 4° and 0° implants caused a significant ($P < 0.05$) reversal of peak values from internal to external rotation, from adduction to abduction, and from lateral to medial translation, compared with findings for the unaltered stifle joints (Table 1); the changes from internal to external rotation were also evident from the motion cycle curves (Figure 6). Compared with findings for the unaltered stifle joints, the 8° and 4° baseplates significantly decreased peak LCL strain, whereas the 0°, 5-mm construct resulted in restoration of LCL strain to normal. There were no significant differences in peak MCL strain between joints before or after treatment.

Tibial spacer thickness—The 8°, 5-mm spacer tibial plateau construct caused significant decreases in peak internal rotation and proximal translations, compared with findings for the unaltered stifle joints (Table 1). The 8°, 7-mm and 8°, 9-mm spacer tibial plateau

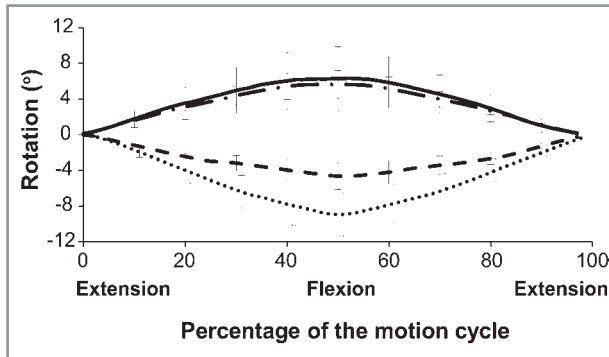


Figure 6—Plot of internal-external rotation (mean \pm SEM) of the tibia relative to the femur in the 6 cadaveric canine hemipelvises in Figure 4 before (solid line) and after CrCL transection followed by TKR involving a 5-mm spacer and a 0° baseplate (dotted and dashed line), 4° baseplate (dotted line), or 8° baseplate (dashed line). See Figure 4 for remainder of key.

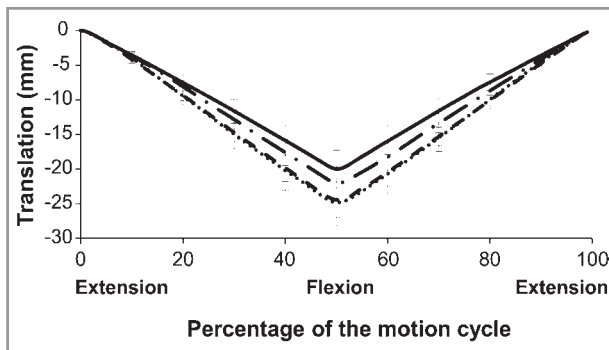


Figure 7—Plot of cranial-caudal translation (mean \pm SEM) of the tibia relative to the femur in the 6 cadaveric canine hemipelvises in Figure 4 before (solid line) and after CrCL transection followed by TKR involving an 8° baseplate and a 5-mm spacer (dotted and dashed line), 7-mm spacer (dashed line), or 9-mm spacer (dotted line). Positive translation values represent movement in the cranial direction; negative translation values represent movement in the caudal direction. See Figure 4 for remainder of key.

constructs caused significant changes in adduction, internal rotation (Figure 5), and caudal translation (Figure 7). Total knee replacement with each of the 3 tibial plateau constructs with the 8° baseplate resulted in a decrease in proximal translation; this decrease did not vary with spacer used. Compared with findings for the unaltered stifle joints, each of the tibial plateau constructs with the 8° baseplate significantly decreased LCL strain; this decrease did not vary with the spacer used. There was no significant difference in MCL strain after application of the 8° baseplate with the 5-, 7-, or 9-mm spacer, compared with findings for the unaltered stifle joints.

Discussion

Joint rotations of the unaltered stifle joints in the cadaveric canine hemipelvises used in the present study were comparable to in vivo data published by Fu et al¹⁴; for 6 dogs during a walking gait in that study,¹⁴ mean stifle joint extension was approximately 140° and flexion was 115°. For those stifle joints in vivo, internal rotation of the tibia relative to the femur during flexion was approximately 10°, a finding similar to the internal rotation of 8.9° determined in the present study. The

stifle joints in vivo also had adduction of 10°, compared with mean adduction of 4.3° in the present study.

To our knowledge, there are no previous studies that have quantitatively investigated the effects of TKR on canine stifle joint kinematics either in vitro or in vivo; however, some studies^{18,19} have evaluated the effects of CrCL transection and other reconstruction techniques in canine stifle joints. In vitro studies^{20,21} of the knee in humans in which the pull of the gastrocnemius and quadriceps femoris muscles was simulated revealed significant increases in cranial tibial translation and internal rotation after transection of the anterior cruciate ligament (corresponding to the CrCL in dogs), which were not identified in the cadaveric canine stifle joints of the present study. However, in an in vivo study²² of 5 dogs, it was determined that excess cranial tibial thrust in CrCL-deficient stifle joints occurs only during the stance phase, with the joint returning to normal craniocaudal alignment during the non-weight-bearing swing phase. It has also been suggested that the quadriceps femoris muscle pull generates a force in the CrCL at certain flexion angles, indicating that the quadriceps femoris muscle is a contributor to cranial tibial thrust.^{20,23,24} One limitation of the present study was its use of passive flexion and extension of the joint without simulating weight bearing or active quadriceps femoris muscle forces. Therefore, it was not surprising that cranial tibial thrust was not observed after CrCL transection.

Kinematics after CrCL transection in the present study was similar to results of the in vitro study by Chailleux et al,¹³ in which the quadriceps femoris tendon was pulled but weight bearing was not simulated. In that study,¹³ no significant changes were seen in cranial tibial thrust, adduction, or internal rotation after CrCL transection in canine stifle joints. These results were attributed to the fact that only the non-weight-bearing phase of gait was simulated. It was concluded that quadriceps femoris muscle force is not the main proponent of cranial tibial translation and that a weight-bearing simulation coupled with quadriceps femoris muscle pull would have likely caused greater cranial tibial thrust. This conclusion is further supported by results of another study¹² in which an axial load was applied to cadaveric canine stifle joints without simulating muscle forces. Significant cranial tibial translation was observed after CrCL transection, indicating that weight bearing without simulation of quadriceps femoris muscle forces places a major load on the CrCL. Although limited by the lack of dynamic stabilization provided by live muscles, results from these previous in vitro studies^{12,13} support the results from the present study, in which CrCL transection did not result in significant changes in cranial translation when treated canine stifle joints were flexed passively.

With the exception of the 8°, 9-mm construct, all evaluated tibial plateau constructs (implant combinations) caused a significant decrease in distal tibial translation, compared with findings in the unaltered joints; the decreases detected did not differ among constructs. These data indicated that the TKR components did not completely replace the tissues that had been removed during the surgical procedure and that, during the ex-

periment, the joint space was not overexpanded (overstuffed). Conversely, one may argue that the decrease in distal tibial translation indicated an inappropriate sizing of the implants, thereby creating inappropriate or inaccurate joint dynamics. In the present study, sizing was performed to specifications currently recommended for the clinical TKR procedure, and this may reveal a flaw in the current recommendations. However, during the study, we did attempt to place the larger spacers in with the shallower baseplates (eg, 0° baseplate and 7- or 9-mm spacers) but could not reduce the stifle joint into normal articulation. Thus, overexpansion may be limited by the inability to reduce the joint.

Compared with the unaltered joints evaluated in the present study, there was an increase in caudal tibial translation (ie, caudal movement of the tibial tuberosity and plateau relative to the femoral condyle while the joint was cycled from extension to flexion) in joints after TKR with each implant (although the difference was not always significant). This may be attributed to the mean TPA of $10.2 \pm 0.8^\circ$ after the TKR was implanted, compared with the typical TPA of 24° in normal canine stifle joints. When neither cruciate ligament is intact, the shallower slope may prevent cranial tibial thrust but cause caudal tibial thrust, even when the stifle joint is flexed passively. Tibial plateau angles steeper than 8° should be investigated further to determine whether use of such angles restores stifle joint kinematics more closely to normal.

In the present study, use of the 8° , 5-mm implant did not alter adduction, compared with findings in unaltered stifle joints. Although this implant also significantly decreased internal rotation by 5° , it was the only implant that maintained internal rotation of the stifle joint rather than creating external rotation.

Medial collateral ligament strain was minimal in the unaltered stifle joints and was not significantly different after TKR. The use of the 8° , 5-mm and 4° , 5-mm constructs significantly decreased LCL strain from 5.72% in the unaltered joints to 0.49% and 0.21%, respectively. The 0° , 5-mm implant returned LCL strain to 7.63%, which was not significantly different from that in unaltered joints. These results indicated that LCL strain is minimal with steeper TPAs and that the LCL may benefit from steeper tibial osteotomies. With the 8° baseplate, all 3 spacer thicknesses resulted in a significant decrease in LCL strain (mean strain for the three 8° constructs, 0.61%); LCL strain did not differ significantly with increasing spacer thickness. This finding was expected because the ligament would be stretched as a thicker spacer is placed into the stifle joint prior to flexing the joint. An *in vitro* investigation²⁵ of TKR in human joints revealed similar relationships between the LCL strain and the gap in the knee joints due to the implant spacers and between the MCL strain and this joint gap. The variability of the ligament strain data obtained in the present study and in the human study²⁵ was similar. The decreased strain on the collateral ligaments may be attributed to decreases in distal tibial translations after TKR because the joint is more compact, causing less strain on the ligaments. Results of the present study indicated that strain to the collateral ligaments in cadaveric canine stifle joints is not affected by TKR.

Findings of the present study suggested that both TPA and spacer thickness affect the kinematics of the canine stifle joint. In general, both decreasing the TPA and increasing the tibial spacer thickness caused joint kinematics to deviate from those of the normal canine stifle joint. Abnormal knee kinematics in humans causes a change in the contact mechanics of an implant, leading to premature implant loosening and wear as well as excess strain on the surrounding ligaments.^{22,26,27} Abnormal kinematics attributable to these tibial implant characteristics may be a contributor to the implant complications and failures that have been observed clinically in the dog. Surgeons should take care not to cut the tibial plateau too shallow during surgery or overstuff the joint with large tibial spacers, because postoperative stifle joint kinematics and success rates may be negatively affected.

The study of this report had several limitations, largely owing to the use of cadaveric preparations that did not include the dynamic joint stabilization provided by muscles. The study findings should be further investigated with methods that include a weight-bearing force and better simulate *in vivo* conditions that affect strain to the collateral ligament and joint kinematics. Further studies should investigate TPAs $> 8^\circ$ to determine whether there is an angle at which joint kinematics is more closely restored to normal. The findings of the present study may provide insight into the design specifications needed for TPAs in dogs undergoing TKR.

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- b. Instron Testing Machine, Model 4201, Norwood, Mass.
- c. T-series Cameras, Vicon Motion Systems Inc, Lake Forest, Calif.
- d. Motus Software, Vicon Motion Systems Inc, Lake Forest, Calif.
- e. Matlab R2008a, MathWorks Inc, Natick, Mass.
- f. 3-mm differential variable reluctance transducer, MicroStrain, Burlington, Vt.
- g. DEMOD-DVTR Signal Conditioner, MicroStrain, Burlington, Vt.
- h. BioMedtrix, Boonton, NJ.
- i. Minitab, release 13 for Windows, Minitab Inc, State College, Pa.

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