

## Comparison of cycling kinetics during recumbent bicycling in subjects with and without diabetes

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**Abstract**—We compared recumbent bicycle kinetics in diabetic peripheral neuropathy and nondiabetic men (nine per group). 3D kinematic and force pedal data in a linked-segment model were used. The generalized muscle moment (GMM) patterns were similar between the two groups except for (1) decreased maximum knee flexor moment, (2) increased minimum knee flexor GMM, and (3) maximum hip extensor GMM by the diabetic subjects. Similar to the walking support moment, a summation moment immutable pattern was observed, although the groups accomplished it differently. The diabetic group utilized the hip during the power phase and the knee during the recovery phase. The nondiabetic group utilized both joints together during both phases. Differences in ankle GMM were not observed, suggesting further research using the recumbent bicycle as an exercise modality for diabetic peripheral neuropathy patients to enhance ankle range of motion and strength, commonly observed walking deficits.

**Key words:** *bicycling, biomechanics, diabetes, peripheral neuropathy.*

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### INTRODUCTION

Type 2 diabetes is the most common and the most rapidly increasing type of diabetes (1). There are 798,000 new cases of diabetes diagnosed every year, and approximately six percent of the entire U.S. population has diabetes (2). The prevalence of type 2 diabetes increases with age (3,4). Poor glycemic control has been identified as the factor most responsible for increasing the risk for amputation (5–7). Specifically, complications of type 2 diabetes are related to poor glycemic control and involve microvascular and neuropathic consequences (8). There is a strong causal relationship between poor glycemic control, poor microcirculation, and peripheral neuropathy (9,10), leading to insensate feet, poor skin circulation, high plantar pressures, foot ulceration (11,12), and eventually, amputation (6,13). The disease sequellae for diabetic peripheral neuropathy are angiopathy, neuropathy, ulceration, infection, and amputation (14).

Fifty percent of diabetic patients present some degree of peripheral sensory neuropathy (9). Additionally, the percent of patients suffering from nerve damage increases with increased duration of disease (11). The most common type of peripheral neuropathy affecting diabetic patients is symmetric polyneuropathy, involving distal sensory and motor fibers (10).

This causes sensory loss and motor abnormalities in the distal parts of the limbs. The presence of clinical neuropathy is correlated with increased age, longer duration

of diabetes, and male gender (9). Diagnosis of peripheral sensory neuropathy is based mainly on clinical examination. Most commonly, peripheral sensory neuropathy is defined as insensitivity to the 5.07 monofilament on the foot (9). Subjects unable to correctly identify all six sites tested on the foot with the 5.07 monofilament are diagnosed with loss of protective sensation or peripheral sensory neuropathy (15). Numerous articles suggest that peripheral neuropathy is the most important pathologic precursor for the development of foot ulcers in diabetic patients (11,12,16) due to its role in muscle atrophy, foot deformities, abnormal plantar pressure distribution (16), and gait deficits (17).

Although exercise cannot reverse severity of peripheral neuropathy or the associated symptoms, exercise can prevent the loss of physical fitness associated with disuse syndromes (18,19). Reports in the diabetic care literature suggest that non-weight-bearing activities, such as bicycling, may be beneficial, since repetitive weight-bearing activities, such as treadmill walking on insensate feet, may ultimately lead to ulceration and fracture (19,20). There is debate as to the effectiveness of ankle muscle strengthening exercises for subjects with severe peripheral neuropathy. Mueller (17) argued that it is doubtful that increasing ankle muscle strength is possible or desirable, since increased plantar flexor strength may lead to a more vigorous pushoff and more stress on the forefoot. Cavanagh (17), in an invited commentary to the Mueller article, however, suggested that strength training may have a meaningful effect on the existing muscle fibers to maximize the output of remaining muscle fibers. Additionally, he proposed that interventions, which potentially increase range of motion, may also be beneficial (17).

The consequences of peripheral neuropathy (e.g., limited ankle mobility and strength) contribute greatly to the walking deficits of diabetic peripheral neuropathy patients. Specifically, decreased ankle range of motion is associated with higher plantar pressures during walking (21–23).

Additionally, muscle atrophy of the peroneal muscles and subsequent decreased muscle strength, caused by peripheral neuropathy result in a supinatory force and moment, creating increased plantar pressure under the fourth and fifth metatarsal heads (24). Walking deficits, coupled with balance problems caused by decreased sensation and proprioception, increase the falls risk in this population (25,26).

Bicycling offers many advantages as an exercise modality as well as a tool to evaluate movement patterns. It is similar to gait because it requires reciprocal use of the lower limbs, fosters symmetry of movement, has a rate similar to walking (revolutions per minute vs. step rate), is rich in proprioceptive and timing cues, provides alternating muscle activation of antagonists (27,28) and is non-weight-bearing. Bicycling is a unique tool to study the motor control characteristics in the diabetic population. It is a task which has well-defined and well-controlled mechanical constraints (29) without concern for balance and gait problems or assistive devices utilized.

Additionally, comparable cycling velocities can be used for both diabetic and nondiabetic subjects. While much work has been reported on the kinetics of upright cycling in elite and recreational riders (30), little has been done with the recumbent bicycle or with older populations. The recumbent bicycle with a large bucket seat, which is low to the ground, has advantages over the upright bicycle in older and diabetic populations in terms of comfort and safety. The purpose of this study was to compare recumbent bicycle kinetics in subjects with diabetes and neuropathic complaints and age-matched nondiabetic control subjects.

## METHODS

### Subjects

Eighteen men voluntarily agreed to participate and signed informed consent forms approved by the Veterans Affairs Greater Los Angeles Healthcare System Institution Review Board. Nine of these subjects with a history (greater than 5 years) of Type 2 diabetes and neuropathic complaints of numbness and/or tingling in their feet were unable to correctly identify at least one site (of six sites tested) on the plantar surface of the foot in one or both feet with the 5.07 monofilament (15,21,31,32). None of these subjects had a history of plantar ulceration. Nine subjects with no history of diabetes or neuropathic complaints served as nondiabetic control subjects.

Average demographic information for each group is presented in **Table 1**. Exclusion criteria for both groups included (1) inability to stand unaided; (2) inability to walk >50 feet without an assistive device; (3) inability to understand verbal instructions; (4) severe cardiac problems which limited physical activity; (5) neurologic

disease (e.g., multiple sclerosis, CVA, Parkinson's disease); (6) pain or trauma of the lower limb, which limited range of motion or physical activity (e.g., amputation, rheumatoid arthritis, symptomatic arthritis); and (7) history of foot ulceration.

**Table 1.**

Demographic Information. Average and standard deviation (ranges) are presented for each group.

CHARACTERISTICS	DIABETIC GROUP	NONDIABETIC GROUP
Age	70 ± 8 (58 – 79)	65 ± 7 (58 – 79)
Weight (kg)*	89.2 ± 14.6 (73.4 – 116.6)	75.3 ± 14.1 (57.6 – 99.0)
Height (m)	1.77 ± 0.10 (1.63 – 1.94)	1.67 ± 0.11 (1.52 – 1.91)
BMI (kg/m <sup>2</sup> )	28.4 ± 4.0 (25.6 – 38.1)	26.0 ± 3.2 (19.3 – 29.8)

\* $p < 0.05$

## Instrumentation

**Bicycle:** The bicycle apparatus was a recumbent bicycle (Cateye Ergociser EC-3600) with cadence, pulse, and work rate digital readouts. The bike seat was adjustable for each subject's lower-limb length, allowing for the knee to be within 20° of flexion, the ankle to be in a neutral position, and the trunk to rest against the back rest of the bicycle seat when the crank was at 180° (farthest away from the subject) (33). This position allowed for the most comfortable distance between the seat and the pedals and prevented stretching of the lower-limb musculature during the end of the power phase when the crank was farthest away from the subject. Attached to the crank were custom-built pedals, capable of measuring normal and tangential components of the applied load (Konigsberg Inc., Pasadena, CA). Each pedal included a double cantilever design instrumented with standard foil strain gauges (350 Ω) with signals conditioned by a fully active Wheatstone bridge amplifier. The pedals used a potentiometer to monitor pedal and crank angle. The pedals were calibrated at 6-month intervals. Voltage outputs were calibrated against known loads with forces measured in Newtons. Previous calibrations have shown the pedals to be linear in both the normal and tangential directions ( $r^2 = 0.99$ ) through the expected range of applied loads (33).

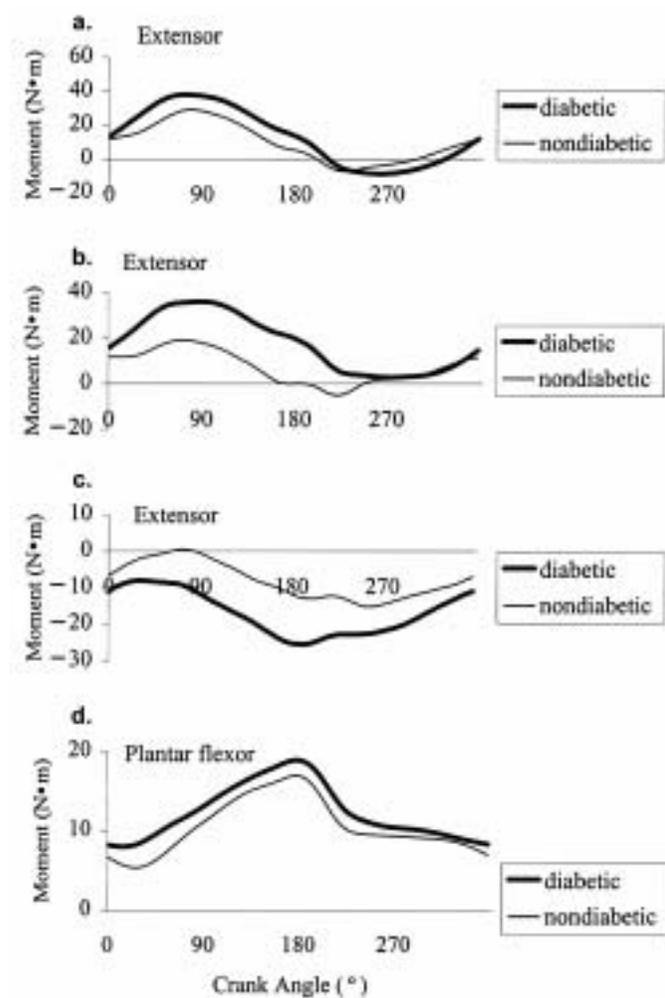
**Kinematic Analysis:** A six-camera high-resolution video-based motion analysis system (Motion Analysis Corp., Santa Rosa, CA) was used to collect kinematic data. The 64-channel 12-bit resolution analog data acqui-

sition system for use with the SUN workstation included software to time-synchronize the force pedal (240 Hz) and kinematic (120 Hz) data. Subjects had retroreflective hypoallergenic markers with adhesive backing placed on the hip (approximating the superior border of the greater trochanter), knee (lateral femoral epicondyle), ankle (inferior tip of the lateral malleolus), and fifth metatarsal-phalangeal (MP) joints (head of the fifth metatarsal) bilaterally.

**Data Collection Protocol:** Cadence was determined by relating a comfortable self-selected walking speed to rotations per minute. The time of a 20-foot walk was converted to revolutions per minute by division of the circumference created by the path of the pedals during a complete pedal revolution by the calculated time required to walk a distance equal to the circumference at the self-selected walking speed. All subjects walked at a speed between 3.6 and 3.9 seconds over a distance of 20 feet. Consequently, the comfortable pedal speed was between 60 to 65 rpm. All subjects pedaled within this range at 1.0 kg\*m workload (60–65 W). A 2-minute warm-up period allowed time for subjects to develop a consistent pedaling speed and pattern. Data were collected during a 15-second period following the 2-minute warm-up. Fifteen seconds of data allowed for approximately 9 to 15 revolutions to be studied. Subjects were tested with their own comfortable shoes.

**Analysis:** Three-dimensional coordinates of the segment endpoint markers were obtained with the use of frame-by-frame 3D trajectory tracking system software (EVA HiRes, Motion Analysis Corporation, Santa Rosa, CA). Interpolation algorithms for resolving gaps in the trajectories because of marker obstruction were included in the software. KinTrak software (Motion Analysis Corporation, Santa Rosa, CA) was used to calculate intersegmental and right horizontal joint angles. Custom Lab View software was used to calculate the generalized muscle moment (GMM) for right lower-limb joints with the use of force pedal and kinematic data as inputs (33). All revolutions were normalized to 360 points and averaged together to form an averaged GMM pattern for the right limb for each subject. Peak generalized muscle moments at the ankle, knee, and hip, as well as the summation of the moments analogous to the support moment during walking (support moment = knee GMM + ankle GMM + hip GMM when all positive

moments are extensor) (34), were obtained from the averaged GMM patterns of the right limb of each subject and averaged together for subjects in each group. Subjects within each group were additionally grouped into LOW and HIGH weight groups defined by above and below the median weight (74.25 kg) for all subjects. Comparisons of peak GMM parameters across group and weight were made with 2 factor ANOVA (StatView Ver. 5.0, SAS Institute, Cary, NC). The level of significance used in this study was  $p \leq 0.05$ .



**Figure 1.**

Average GMM profile for (a) summation moment (knee GMM—ankle GMM—hip GMM) for diabetic and nondiabetic groups, (b) hip, (c) knee, and (d) ankle. A positive moment represents an extensor (ankle plantar flexor) moment, while a negative moment represents a flexor (ankle dorsiflexor) moment.

## RESULTS

**Figure 1** represents the averaged GMM pattern for each group throughout the pedaling revolution from top dead center ( $0^\circ$ ) to top dead center ( $360^\circ$ ) for each joint and the summation moment. The power phase was defined as the phase from top dead center ( $0^\circ$ ) to full extension ( $180^\circ$ ) as the lower limb extended and the crank rotated away from the subject. The recovery phase was defined as the phase from full extension ( $180^\circ$ ) back to top dead center ( $360^\circ$ ) as the lower limb flexed and the crank rotated toward the subject. Extensor moments were considered positive for all joints for purposes of presentation in **Figure 1** and **Table 2**.

Overall patterns at each joint are similar across subjects and groups (**Figure 1**). The ankle GMM remained plantar flexor throughout the pedaling cycle, with peak plantar flexor GMM occurring at approximately bottom dead center ( $180^\circ$ ). With the exception of a small extensor moment occurring prior to  $90^\circ$  in some subjects and on average in the nondiabetic subjects, the knee GMM was flexor throughout the pedaling cycle with peak flexor moment occurring after peak plantar flexor GMM during the recovery phase (approximately  $230^\circ$ , although quite variable ( $196^\circ$ – $274^\circ$ )). The hip GMM was extensor during the power phase (peak occurring before  $90^\circ$ ) and slightly flexor during the recovery phase (peak occurring approximately  $250^\circ$ ). The summation of the three moments, analogous to the support moment in walking (34), was extensor during the power phase and flexor during the recovery phase, although the transition point was quite variable (range:  $140^\circ$  –  $250^\circ$ ). One subject in the diabetic group and two subjects in the nondiabetic group maintained extensor summation moments throughout the pedaling cycle.

While patterns were quite similar between the two groups, magnitudes, particularly at the knee and hip, were significantly different (**Table 2**). The diabetic group had a greater plantar flexor ankle GMM (minimum ankle GMM) during the first  $30^\circ$  of the pedaling cycle than the nondiabetic group, but this was not significant when taking weight into account. Minimum ankle GMM, however, occurred earlier in the pedal cycle in the diabetic group ( $13^\circ$  vs  $26^\circ$ ). At the knee, only one subject in the diabetic group was able to produce an extensor GMM, while three subjects in the nondiabetic group produced an extensor GMM. Consequently, the average maximum knee GMM was negative for the diabetic group and

**Table 1.**  
Average and standard deviations for peak GMM parameters within each group.

		DIABETIC	NONDIABETIC
ANKLE	Maximum (N•m) <sup>1</sup>	18.52 ± 3.58	17.54 ± 1.97
	Timing <sup>(0)</sup>	176 ± 7	181 ± 6
	Minimum (N•m) <sup>2</sup>	7.71 ± 2.35	5.73 ± 1.63
	Timing <sup>(0)</sup>	13 ± 13	26 ± 6*
KNEE	Maximum (N•m) <sup>3</sup>	-5.50 ± 6.13	1.82 ± 2.74*
	Timing <sup>(0)</sup>	46 ± 25	68 ± 25*
	Minimum (N•m) <sup>4</sup>	-27.24 ± 4.00	-17.81 ± 6.03*
	Timing <sup>(0)</sup>	233 ± 53	224 ± 33
HIP	Maximum (N•m) <sup>5</sup>	35.35 ± 11.32	26.04 ± 8.89*
	Timing <sup>(0)</sup>	77 ± 38	67 ± 29
	Minimum (N•m) <sup>6</sup>	-7.10 ± 19.00	-4.77 ± 17.68
	Timing <sup>(0)</sup>	249 ± 57	248 ± 64
SUMMATION	Maximum (N•m) <sup>7</sup>	39.29 ± 11.33	35.31 ± 10.03
	Timing <sup>(0)</sup>	103 ± 60	84 ± 23
	Minimum (N•m) <sup>8</sup>	-11.38 ± 9.19	-7.21 ± 12.27
	Timing <sup>(0)</sup>	236 ± 52	244 ± 24

\* $p < 0.05$

1. Maximum ankle GMM represents maximum plantar flexor moment (maximum positive value).
2. Minimum ankle GMM represents minimum plantar flexor moment (least positive value) or maximum dorsiflexor moment (maximum negative value).
3. Maximum knee GMM represents minimum flexor moment (least negative value) or maximum extensor moment (maximum positive value).
4. Minimum knee GMM represents maximum extensor moment (maximum positive value).
5. Maximum hip GMM represents maximum flexor moment (maximum positive value).
6. Minimum hip GMM represents maximum flexor moment (maximum negative value).
7. Maximum summation moment represents maximum extensor moment (maximum positive value).
8. Minimum summation moment represents the maximum flexor moment (maximum negative value or minimum extensor moment (minimum positive value)).

slightly positive for the nondiabetic group ( $p < 0.05$ ). Timing of this maximum knee GMM occurred significantly earlier in the pedaling cycle for the diabetic group than in the nondiabetic group. While unable to produce comparable knee extensor GMMs, the diabetic group produced greater knee flexor GMM ( $p < 0.05$ ) than the nondiabetic group. Timing was quite variable within both groups. At the hip, the diabetic group created greater hip extensor GMM than the nondiabetic group ( $p < 0.05$ ). The maximum and minimum summation moments were similar in magnitude and timing between the two groups.

## DISCUSSION

The description of lower limb kinetics presented in this paper revealed that the hip extensor GMM was

higher and knee extensor GMM was lower while subjects rode a recumbent bicycle, the description of lower-limb kinetics in diabetic subjects with neuropathic complaints and in age-matched nondiabetic control subjects revealed that the hip extensor GMM was higher and knee extensor GMM was lower in the diabetic group, suggesting differing strategies were used to accomplish the cycling task between the two groups.

While the summation moments were not statistically different between the two groups, the strategies to accomplish the cycling task varied between the groups. Stein et al. (35) proposed that movements to accomplish a specific task can involve a combination of strategies. The nondiabetic group utilized both the hip and knee GMM to accomplish the cycling task throughout both power and recovery phases. Kinematically, the hip and knee move in concert with each other throughout both the

power and recovery phases (36). Kinetically, less knee flexor GMM appears during the power phase and less reliance on the hip extensor GMM as the sole contributor to the extensor thrust. This resulted in a reduced hip extensor GMM observed in the nondiabetic group during the power phase. Additionally, during recovery, the hip generated a small flexor GMM in the nondiabetic group along with the knee flexor GMM. Thus, the two joints worked in combination to produce the overall summation moment throughout both phases of the cycle.

In the diabetic group, the greatest contributor to the magnitude and pattern of the extensor summation moment during the power phase was the hip, because the knee never produced an extensor GMM and was increasingly flexor.

During the recovery phase, however, the knee seemed to be primarily responsible for pulling the limb closer to the body. This was achieved in the diabetic group by producing greater knee flexor GMM but producing no hip flexor GMM during the recovery phase. The preferential use of the hip extensor GMM by the diabetic subjects may be a compensation for limited ankle range of motion (37) during the first 30° of the pedaling cycle when the ankle was dorsiflexing and the limb was in the most flexed position. Possible muscle atrophy of the peroneal muscles caused by peripheral neuropathy (24) may also limit the dorsiflexion produced during this pedaling phase. Although electromyographic data have not been reported for the peroneal muscles during cycling, tibialis anterior on/off patterns were consistent with activity during the phase of the pedaling cycle (90° of the pedaling cycle approaching top dead center) on an upright bicycle (30,38,39). This time frame for the upright bicycle is analogous to the first 90° of the pedaling cycle on a recumbent bicycle when top dead center was defined as the position when the limb was most flexed. Unpublished data on recumbent bicycling also showed electromyographic activity of the tibialis anterior during the first 90° of the pedaling cycle (40) when top dead center was defined as the position when the limb was most flexed. Further studies are warranted to evaluate the electromyographic activity of the peroneal muscles during recumbent cycling and the potential training effects with the use of biofeedback regarding ankle range of motion or peroneal electromyographic activity given the ankle deficits which have been demonstrated in the diabetic population during walking (31).

Mueller et al. (17) observed that the greatest deficits between diabetic peripheral neuropathy subjects and nondiabetic control subjects occurred at the ankle during walking. They demonstrated decreased plantar flexor strength, decreased ankle mobility, and diminished plantar flexor GMM and power, although subjects with diabetic peripheral neuropathy were walking slower than the nondiabetic control subjects. Consequently, Mueller et al. (32) proposed that the use of a hip strategy (pulling the limb forward using hip flexor muscles) rather than an ankle strategy (pushing the limb forward using plantar flexor muscles) reduced peak plantar pressures. Additionally, they demonstrated a relationship between dorsiflexion range of motion and ankle plantar flexor power during walking (31). They suggested that limited ankle plantar flexor power resulted in increased reliance on a hip strategy during walking (17), which may explain the reliance on the hip GMM in the summation moment during cycling.

Similar to walking (34), however, the summation moment was immutable, regardless of group. Winter suggested that in walking, there was a flexible trade-off between the hip and the knee creating an immutable support moment (41). In cycling, this trade-off appears to also be apparent. While the nondiabetic group utilized both joints throughout both the power and recovery phases, the diabetic group selectively utilized the hip during the power phase and the knee during the recovery phase to a greater degree than the nondiabetic control group.

## CONCLUSIONS

Unlike observations during walking, differences in ankle GMM were not observed during recumbent bicycling, suggesting further research using the recumbent bicycle as an exercise modality for the diabetic peripheral neuropathy patients to enhance ankle range of motion and strength, commonly observed walking deficits. It may be used as a modality to continue to develop ankle strength and mobility without placing the patient at risk for high plantar pressures or falls, since recumbent bicycling is a non-weight-bearing task. Additionally, differences in kinematic and kinetic parameters can be documented at similar workloads and velocities to indicate true changes in motor control parameters between groups. It is well established that joint kinematics and

kinetics are related to walking speed (42) and reductions in joint kinematics and kinetics during walking in diabetic subjects may have been a function of reduced walking speeds. In cycling, pedaling speed was easily controlled, yet significant differences in motor control parameters existed between the diabetic and nondiabetic groups.

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