Motor Control and Gait Characteristics in People with Type 1 and Type 2 Diabetes without Sensory Impairment in the Foot

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Abstract

Twenty-five control subjects, fifteen subjects with Type 2 and ten subjects with Type 1 diabetes were examined to compare motor control during walking in a linear path and during turns. To eliminate the contribution of sensory neuropathies to the feet and myopathies of the lower leg muscles, subjects were selected with no sensory impairment in their feet or muscle strength deficits. To measure deviations in gait and motor control errors, timing of gait was measured from foot sensors worn in the shoes, from accelerometers (2 axis) mounted on the head, shoulder, hips, knees and ankles, bilaterally and from the electromyogram of the major muscle groups in the legs. As a measure of peripheral reflex activity, the H reflex was also measured in the gastrocnemius muscle. The results showed that subjects with diabetes took more steps when walking in a linear path and during turns, significantly more time to walk a given distance than control subjects, and an increase in flexion/extension and lateral movement at the major joints in the body. Joint movement at the hip, knee, ankle and shoulders, showed a 50 to 100% increase during gait for subjects with diabetes compared to control subjects with significantly greater tremor occurring at the joints in the 8Hz and 16Hz bands indicating central motor control deficits in subjects with both Type 1 and Type 2 diabetes. H reflex was also about 50% less in subjects with diabetes compared to control subjects. These findings suggest that for individuals with Type 1 and Type 2 diabetes, impairments in both the timing and quality of gait are likely caused by dysfunction of central motor control found equally by both Types of diabetes.

Key words: accelerometry, diabetes, gait, neuropathy, polyneuropathy.

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Diabetes is a major health care problem in the United States affecting millions of Americans every year [43]. The prevalence of diabetes is approximately 6% of the population in the United States, while in some countries the prevalence is as high as 20% [1]. As many as 50% of people with diabetes develop significant peripheral neuropathies including both the somatic and autonomic nervous systems [10]. Such patients are 15 times more likely to report experiencing an injury during standing and walking and have an increased number of repetitive falls when compared to people without diabetes [10].

In individuals with sensory loss to the foot, gait abnormalities have been reported including improper pressure distribution on the foot and a longer stance phase and shorter steps than observed in people without diabetes [31, 32]. But these previous studies have only examined gait in patients with diabetes with impaired sensation in their feet [19, 21]. Significant sensory impairments in the feet are not seen until a number of years after the onset of diabetes [36]. Furthermore, gait is a complex process involving the visual, vestibular, sensory and motor control systems. Since vision [8] and the vestibular system [35] are also impaired in Type 1 and Type 2 diabetes, gait should therefore be impaired absent such sensory loss.

While research on gait and diabetes is scant, balance impairments in people with Type 1 and Type 2 diabetes due to loss of vestibular function have been described [15, 34, 35]. These balance impairments can precede sensory loss to the foot [9], showing a more central origin of the impairment of motor control. While it was once believed that only a small percentage of patients with diabetes display damage to the central and peripheral nervous systems, recent data shows that almost all patients with diabetes display early autonomic damage which was previously undetected [25, 26].

Therefore, since previous studies have been limited to patients with peripheral myopathies and neuropathies, a comprehensive study of gait in either patients with Type 1 or Type 2 diabetes has never been accomplished sepa-

rating motor control from sensory loss in the foot and peripheral myopathies. In the present investigation, we compared gait in control subjects to subjects with both Type 1 and Type 2 diabetes and examined timing during gait and movement at the knees hips, ankles, and shoulders assessed by accelerometers. Accelerometer data would provide a measure of adverse joint mobility during gait and motor control as might be associated with peripheral neuropathies or somatosensory neuropathies affecting the muscle spindle reflex loop. Data was analyzed for tremor as well as gross motor errors during gait. Muscle EMG and H reflex data was also collected to assess motor control. In patients with both Type 1 and Type 2 diabetes, H reflex has been shown to be depressed but testing usually is done in patients with sensory impairment [23, 30]. To isolate motor control from loss in feeling in the feet, subjects were selected with no sensory loss in the feet and no loss of muscle strength in the legs. Finally, no studies of gait have been accomplished on subjects with diabetes where subjects were making turns.

Falls usually do not occur during walking in a linear path but occur when varying the ground contact surfaces [12, 39]. Earlier studies on gait, suggest that analyzing gait during turns provides a much more sensitive analysis of the early onset of abnormalities [5, 6, 27]. This is probably due to the inability of the body to compensate for rapidly changing gravity vectors if any vestibular or sensory pathology is present [24]. Therefore in the present investigation, gait analysis was accomplished during walking in linear paths as well as during turns.

Subjects

Fifteen subjects between the ages of 40 and 70 who have been diagnosed with Type 2 diabetes for at least five years and 10 subjects with Type 1 diabetes were invited to participate in this study. Twenty-five subjects without a known history of diabetes between the ages of 40 and 59 years of age were controls. All subjects with diabetes were managed with medication. Both controls and diabetic subjects were free of any history of falling or gait abnormalities. Subjects were excluded if there was any clinical deficit in strength, range of motion, or sensation in the feet on testing. Sensory testing was accomplished by using Semmes Weinstein Monofilaments. Impairment was shown if a 10g filament could not be felt on any toes on either foot or on the heel or base of each of the five rays [3, 36]. All experimental procedures and methods were explained to each subject who then signed a statement of informed consent as approved by the Committee of Human Experimentation. The heights, ages and weights of the subjects are listed in table 1. Of the subjects with Type 2 diabetes, the length of time since diagnosis was 6.4 +/- 1.9 years. The average HbA1c of the groups with Type 1 and Type 2 diabetes was 7.8 +/- 1.2 and 7.4 +/- 1.4% respectively.

Table 1. Age, heights, and weights of subjects. Each point is shown +/- the SD.

	Mean		
	age(yrs)	height(cm)	Weight Kg)
Type 1 Diabetes	54.2 ± 6.9	173 ± 7	77.3 ± 6.7
Type 2 Diabetes	58.6 ± 7.9	179 ± 6	74.9 ± 7.3
Controls	57.3 ±11.2	175 ± 8	75.2 ± 5.8

Methods

Accelerometry

Two plane-accelerometers weighing 3g (Analog Devices Corp 202xb) were placed bilaterally on the shoulders (on the acromial end of the clavicle), hips (over the bony prominence of the greater trochanter), knees (just under the patella and on the head of the tibia), and ankles (on the lateral side and on the lateral malleolus), and one on the center of the forehead of each subject (figure 1). The accelerometers measured movement in the side-to-side (lateral) and forward back (flexion extension) direction in the range of 0-2 g (one g is the force of gravity at the earth surface). The output of the accelerometers was amplified with a gain of 2 with an

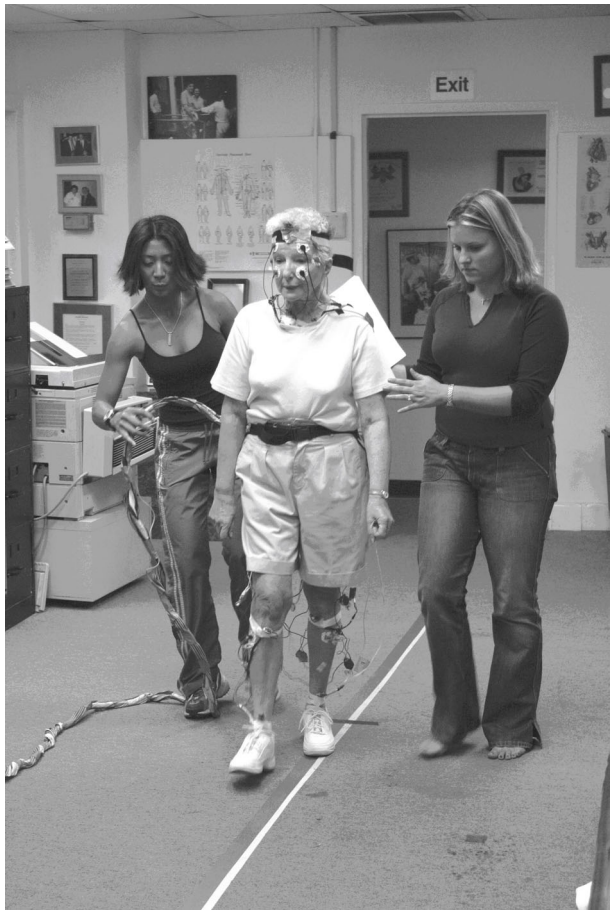


Figure 1. A typical subject.

amplifier whose frequency response was flat from DC to 1000 Hz. The amplified signal was digitized at 1024 samples per sec and stored in an IBM computer by a Biopac MP100 signal digitizer (Biopac Corp, Santa Barbara).

Electromyogram

EMG was recorded through 3 silver-chloride adhesive electrodes above the active muscles. Two of the electrodes were the active electrodes and were placed over the belly of the muscle and 2 cm distal. The final electrode was the guard and was placed laterally to the belly. Electrode output was amplified with a Biopac amplifier (Biopac Corp, Santa Barbara, California). EMG was amplified with a gain of 2000 and the frequency response was flat from 1 Hz to 500 Hz. The amplified signal was digitized at two thousand samples per second by a twelve bit A/D converter and stored for later analysis. For each muscle studied, the EMG was normalized by placing the body into a position to isolate muscle activity [16] and then performing three 3-second maximal efforts; 1 minute was allowed between the contractions. EMG during gait was then divided by the maximum EMG activity during the strength determination to normalize EMG as a percent of the maximum muscle activity.

The H-reflex

To quantify the H-reflex, electrical stimulation was applied through 2 electrodes above the motor nerve to the medial gastrocnemius muscle in the poplateal fosse, and one ground electrode was applied to the achilles tendon. An electrical stimulus (single impulse) was applied to elicit a muscle twitch. Stimulus pulse width was 200 microseconds. The amplitude of the stimulus was increased in 2v increments from 2v to 38v and the EMG above the muscle was recorded. The maximum amplitude of the H wave, M wave and the H wave latency were measured.

Before measurements, the leg was warmed in a 37 deg C water bath for 15 minutes to stabilize temperature to that of the core.

Measurement of timing during gait

Shoe insert transducers measured the timing during gait. Each load cell was composed of two thin layers of brass (0.4 mm) separated by a layer of conductive rubber (1.0 mm). The conductive rubber is sold by Zoplex Corporation (ZF60). The rubber varied in resistance from 200 meg ohms to 1 ohm when pressure was applied to the rubber. By sandwiching the rubber between two pieces of brass, pressure on the brass plates, caused a change in electrical resistance. [24] The sandwich was placed between 2 layers of masking tape. Six transducers were placed at various locations under the foot. The resistive output was converted to an electrical output via a Wheatstone bridge. The output was then recorded on a data recorder for later analysis.

Data analysis

Statistical analysis involved ANOVA and calculation of means, standard deviations, coefficients of variation and t-tests on Microsoft Excel. The level of significance was $P<0.05$. In addition to means and standard deviations of the individual groups of data, an additional data analysis was accomplished. During walking in a linear path, as described below under Results, data was digitized and analyzed over a 4 second period. During turns, data was analyzed over a 1 second period. For each second of data that was collected, the total number of data samples was 1,024 samples. As a means of analyzing the variation in acceleration (that is the joint movement) from each plane on each accelerometer, the RMS G force was calculated from all digitized data. This then provided the average acceleration over either the 4 second period or 1 second period associated with movement at that particular joint. However, the mean alone did not provide information as to the extent of the variation in acceleration during gait, only the average acceleration. Therefore, in addition to the mean, the standard deviation was calculated from the raw data. Because of variations in mean acceleration from subject to subject, the standard deviation is only meaningful when compared to the mean. Therefore, to avoid bias in the data, a final calculation, the coefficient of variation (standard deviation divided by the mean) was calculated. The average coefficient of variation for each accelerometer at each plane and for each subject could then be averaged together to finally determine the average coefficient of variation under each experimental condition and for each accelerometer.

Finally, a Fast Fourier transform was used to analyze the frequency components of the accelerometer data. The amplitude of the 8 and 16 hertz bands was used to analyze tremor by expressing the power in the 6-12 Hz bands as a percent of the total spectral power (8Hz band in analysis) and 12-20 Hz bands as the 16Hz band power.

Procedure

Subjects rested for 10 minutes for acclamation. During this period, demographic information was collected including, age, weight, height, and any falling history during the last 2 years. Subjects were excluded if they had 3 or more falls in the previous 2 years. Subjects were screened for bilateral lower extremity muscle strength deficits by manual muscle testing. Active range of motion was evaluated by using a Goniometer on the lower extremities and sensory evaluation was accomplished bilaterally on the legs and feet with nylon wires of various sizes. H reflex was measured on the medial gastrocnemius muscle. Nine accelerometers were placed on the lateral side of both ankles, knees, hips, and shoulders, on the lateral surface of the hips, and one accelerometer was placed on the anterior surface of the forehead. EMG electrodes were placed over the medial gas-

trocnemius, hamstring, quadriceps and tibialis anterior bilaterally. The subjects were asked to walk for 7 meters then make a pivot turn to the left and to the right side. This protocol was repeated with two different turn diameters, 0.33 and 0.66 meter in diameter to the left and right. Subjects were also asked to stop as quickly as possible on one run (without prior warning) when they saw a strobe flash. Reaction times were then recorded. During all these activities acceleration and timing during gait were recorded (Figure 1).

Results

The results of the experiments are shown in tables 2-7 and figures 2 through 8.

Timing during gait when walking in a linear path – Table 2 lists the results of the measurements of timing during gait in the 25 control and 25 subjects with diabetes. There was no significant difference in the data on timing and acceleration between subjects with Type 1 and Type 2 diabetes ($p>0.05$). As shown in table 2, the average ratio of the stance and swing times (stance-swing ratio) was significantly lower in the controls than in the subjects with either Type 1 ($p<0.05$) or Type 2 diabetes ($p<0.01$). The mean velocity of walking was also significantly greater in the controls subjects than the subjects with either Type 1 ($p<0.01$) or Type 2 ($p<0.01$) diabetes. The subjects with diabetes also took a greater number of steps to complete the seven meter walk. The average number of steps in the pooled subjects with diabetes was 6.1 +/- 1.2 to complete the seven meter walk, whereas in the control subjects, the average was only 4.61 +/- 1.1 steps to complete the seven meter track, this difference being significant ($P<0.05$). The reaction rates of the subjects with diabetes were also slower. As shown in table 2, the average "stop time" for the control subjects was just 2.1 seconds whereas the stop time for the subjects with diabetes was significantly ($P<0.01$) longer.

Timing data for gait during turns is shown in table 3. Subjects with Type 1 and Type 2 diabetes were not significantly different from each other so their data has been grouped together ($p>0.05$). Subjects with diabetes had much slower gait while turning than did the control subjects. Table 3 summarizes data for turns of 0.33 me-

Table 2. Descriptive gait data on gait in patients with Type 1 diabetes (Diab 1) and Type 2 diabetes (Diab 2) and control subjects (controls) all data is shown ± the S.D.

	velocity	Stance/Swing ratio	stop time	velocity	Stance/Swing ratio
Diab 2	0.76±0.22	1.63±0.62	4.3±0.72	0.42±0.13	1.85±0.39
Diab 1	0.71±0.19	1.67±0.51	4.1±0.9	0.38±0.11	1.71±0.31
Controls	1.18±0.37	1.43±0.39	2.1±0.6	0.81±0.21	1.51±0.43

Table 3. Turn data in diabetic and control groups. Mean data is shown for right pivot turns (RP), left pivot turns (LP) and turns of 0.33 and 0.66 meters diameter to the right and left.

	Diabetes			Controls	
Turn	Group Mean: # turn steps	Group Mean: Turn Time (sec)		Group Mean: # turn steps	Group Mean: Turn Time (sec)
RP	1.6±0.8	1.2±0.3	RP	1±0.2	1.1±0.2
0.33R	2.1±1.1	1.3±0.4	0.33R	1.2±0.2	1.3±0.3
0.66R	2.3±1.1	1.7±0.4	0.66R	1.7±0.4	1.6±0.6
LP	1.2±0.4	0.8±0.2	LP	0.9±0.4	0.9±0.2
0.33L	1.9±1.3	1.2±0.2	0.33L	1.3±0.3	1.2±0.4
0.66L	2.2±1.1	1.6±0.5	0.66L	1.5±0.4	1.2±0.3

Table 4. Mean group tremor data in subjects with diabetes and control subjects ± the S.D. at the 8Hz band.

Tremor 8 Hz	knee	ankle	hip	shoulders
Type 2	3.3±1.15%	3.1±0.94%	2.3±1.23%	1.2±0.28%
Type 1	3.1±1.1	3.2±1.3	2.3±0.8	1.3±0.34
controls	1.9±0.38%	2.2±0.41%	1.1±0.24%	0.9±0.21%

Table 5. EMG data walking in a linear path for the right and left tiabialus anterior (rtib, ltib) and right and left gastrocnemius muscles (lgast, rgast) in controls and patents with type 1 and type 2 diabetes.

	% rtib	%r gast	% ltib	% lgast
control	2.3±73	34.2±8.4	28.1±9.3	33.7±8.1
Type 1	63.7±17.2	57.4±11.5	51.4±14.7	53.9±13.4
Type 2	61.1±15.7	55.9±11.96	46.3±11.4	54.2±11.3

ter diameter 0.66 diameter turns pivot turns. Control subjects walked much quicker and used fewer steps during turns. Subjects with diabetes walked slower and with more steps than control subjects as shown in this table ($p<0.01$)

Accelerometry - Figure 2 shows typical accelerometer results. The top two traces in figure 2 show timing of gait. A positive excursion of the trace on the right leg, (most upper trace) shows toe contact whereas a negative deflection represents heel contact. The right leg is shown in the upper trace and the second trace below shows the left leg. Typical accelerometer data for the left (two middle traces) and right knee (two bottom traces) are also shown in this figure. The third trace

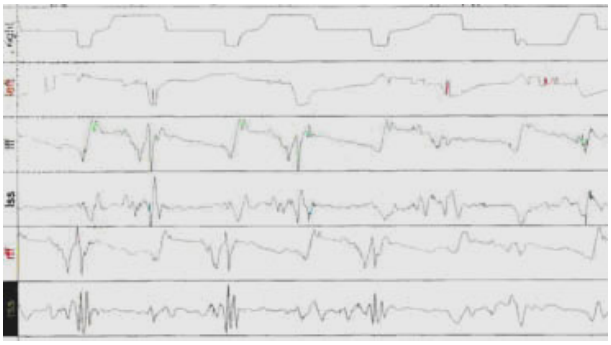


Figure 2. A typical tracing of toe and heel contact data (upper two traces) and accelerometer data (lower four traces) from the left knee (third and fourth trace from the top) and right knee (fifth and sixth trace from the top) for flexion extension (third and fifth traces) and lateral movement of the knee (fourth and sixth traces). In the top two traces toe contact is shown as an upper movement of the trace and heal contact is a downward movement of the trace.

Table 7. H reflex data on controls and subjects with type 1 and type 2 diabetes.

	H REFLEX DATA			
	H (MV)	M (MV)	Latency	H/M
Type 1	1.2	5.7	35.1	0.21
SD	0.1	1.1	16.2	0.07
Type 2	1.4	6.0	37.3	0.23
SD	0.3	1.3	14.2	0.08
Control	2.3	4.4	25.2	0.53
SD	0.3	1.1	7.9	0.14

from the top shows acceleration in the flexion extension direction for the left knee whereas lateral movement of the knee is shown in the fourth trace from the top. The means, standard deviations, and coefficients of variation of the accelerometers during walking in a linear path are shown in figures 3 and 4, and data during turns is shown in figures 5 and 6. For brevity, only data is shown for the knee and hip since data on other joints was similar. Further, data is shown for accelerometers on the outside leg of the turns in all cases.

Data in figures 3, 4, 5, and 6 had been grouped for subjects with diabetes since there was no statistical difference between the groups in any of the measurements made ($P>0.05$). As shown in figure 3 the greatest acceleration for subjects with diabetes at the hip while walking a linear path was 0.08g. In contrast, for control subjects, the g-loading was much less averaging 0.04g's; these differences were significant ($P < 0.05$). The standard deviations were also higher for subjects with diabetes averaging 0.16 whereas control subjects the standard deviation were 0.04. Thus the coefficient of variation was over twice as high for movement at the hip in subjects with diabetes when compared to control subjects. The same relationship was seen for lateral movement during walking in a linear path as shown in the bottom panel of figure 3. Lateral movement at the hip involved approximately half the g-loading as movement at the knee. For example, in the subjects with diabetes, the average lateral movement at the hip was 0.02g's, a figure approximately half that of flexion extension at the knee, these differences were significant ($P<0.01$). The same pattern was seen in figure 4 during walking in a linear path for flexion extension (Panel A) and lateral movement of the knee (Panel B). Here however g-loading was substantially higher. For example in the subjects

Table 6. Analysis of EMG data as the ratio of tibialus anterior and gastrocnemius (see text) during initial contact (IC) and toe off while walking in a linear path and during turns. Data is the mean +/- the SD.

STRAIGHT	IC	toe off	**TURN**	IC	toe off
control	8.1±1.3	7.5±2.1	Control	11.2±6.7	13.4±2.9
Type 1	34.8±9.7	36.9±11.2	Type 1	43.7±16.4	47.6±13.8
Type 2	37.3±14.2	34.8±16.7	Type 2	45.9±21.2	41.7±11.9

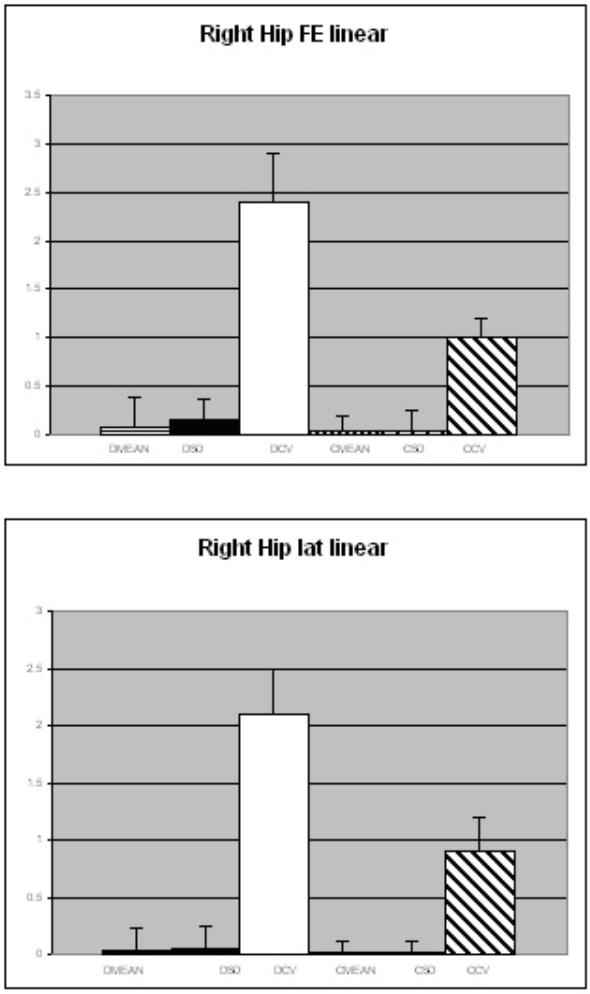


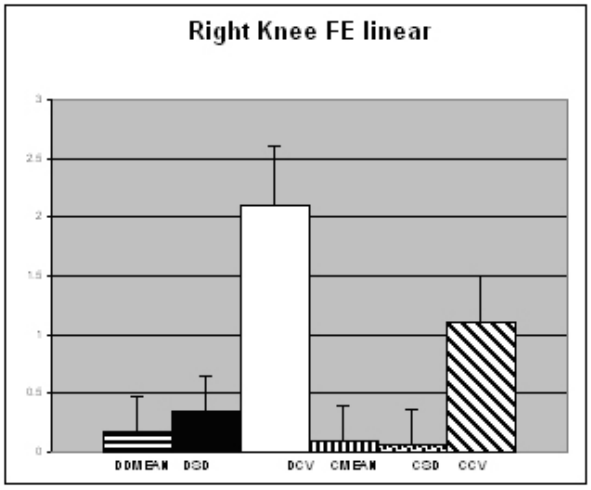
Figure 3. The mean g loading at the hip during flexion extension during walking in a linear path, the standard deviation of hip flexion extension and lateral movement and the coefficient of variation for subjects with diabetes (d) and control subjects (c) for flexion extension (panel A) and lateral movement (panel B).

with diabetes, the g-loading at the hip in the flexion extension direction was only 0.08g's whereas the g-loading at the knee in the flexion extension direction was 0.17g's; the difference was significant ($P < 0.01$). This approximate doubling in g-loading at the knee was seen both for subjects with diabetes and control subjects in the flexion extension direction, and in the lateral movement of the knee (Figure 4B). The coefficient of variation was still approximately double that in subjects with diabetes as was seen in control subjects. The same relationships were maintained in turns. For example, when looking at the hip in figure 5 flexion extension at the hip during turns of 0.66 meters diameter showed g-loading at the hip in the flexion extension direction of 0.06g's for subjects with diabetes whereas for control subjects the loading was approximately half at 0.03g's as shown in figure 5A. Lateral movement at the hip was less than flexion extension movement at the hip. The coefficient of variation was over double that of subjects with diabetes compared to control subjects. This is also true in figure 6A and B concerning flexion extension movement at the knee (Panel A), and lateral movement at the knee during turns of 0.66 meters diameter. The response at the other joints was similar to these joints. In all cases, the coefficient of variation and mean acceleration at joints in both the flexion extension and lateral direction was significantly greater in both groups with diabetes than was seen for control subjects ($P < 0.05$). Further, for all joints, there is no significant difference between the subjects with Type 1 and Type 2 diabetes ($P > 0.05$).

Tremor

As shown in figure 7, and 8, the accelerometer data was analyzed for frequency components. By using a fast fourier transform (FFT), a frequency component can be assessed from 1 hertz to 100 hertz. Typical power spectra are shown in subjects with diabetes (Figure 8) and in control subjects (Figure 7). As shown in table 4, in subjects with diabetes, there is more power at the 8 hertz band than for control subjects when analyzing adverse movement at the knee, ankle, hip, and shoulders respectively. For example, the power in the 8 hertz band was 3.3% of the total power in the EMG spectrum for subjects with Type 2 diabetes, 3.1% for the subjects with Type 1 diabetes, and 1.9% for the total band for control subjects. There was no statistical difference between the 8 hertz band power for the subjects with Type 1 and Type 2 diabetes for any joint ($p>0.05$). However, the power in either group or the combined group was statistically greater than that of control subjects ($P < 0.05$). There was no significant difference in tremor in the lateral direction and flexion extension data for each group as shown in table 4 ($p>0.05$). These end results were seen at the 16 hertz bandwidth. In all cases, muscle tremor at the 8 hertz and 16 hertz bands averaged almost doubles that of control subjects to subjects with Type 1 and Type 2 diabetes.

Muscle tremor in the 8 hertz bands was also nearly twice as high during turns. For example, at the knee, the tremor during walking on a linear course was 3.3% of the total power in the 8 hertz band for subjects with Type 2 diabetes. During turns, tremor increased to 4.3 ± 1.6 % of the total power. At the ankle, tremor for subjects with Type 2 diabetes increased from 3.1% as shown in table 4 to 4.4 ± 1.3% of the total power. The same increase was seen in control subjects. At the knee, tremor increased from 1.9 ± 0.38% of the total power of the spectrum to 2.2 ± 0.4%. At the ankle, in control subjects, power increased from 2.2 ± 0.4% to 2.5 ± 0.6%. At each of the joints, there was an increase in tremor associated with turns of either 0.33 or 0.66 meters diameter. However, the increase in tremor was significantly greater in subjects with diabetes than control subjects ($P < 0.05$). Further, for any one joint the increase



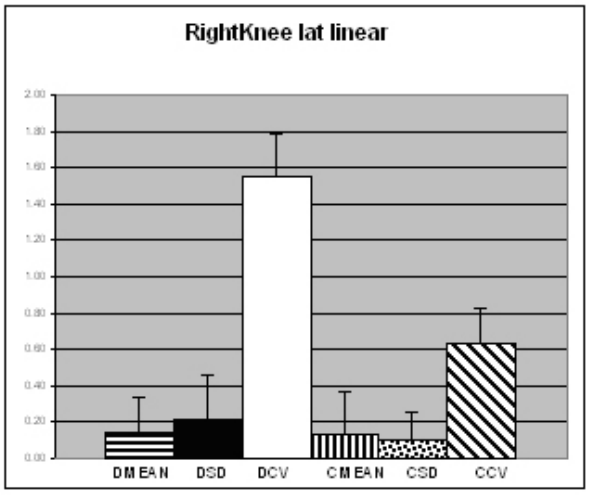
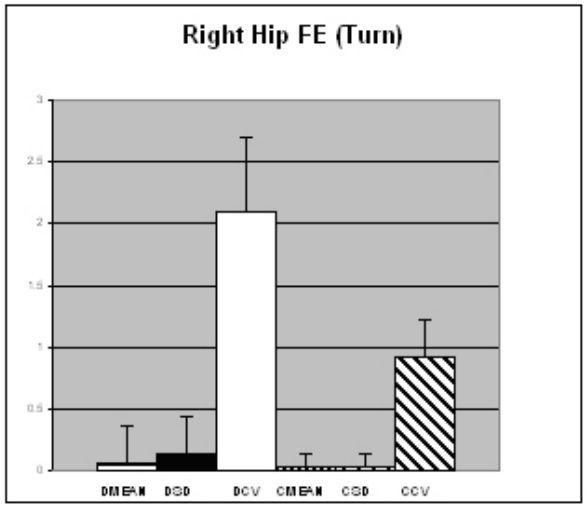


Figure 4. This figure illustrates the flexion extension and lateral movement of the knee during walking in a linear path. Illustrated here is the mean g loading at the knee, the standard deviations and coefficient of variation for control subjects (c) and subjects with diabetes (d). Data has been averaged for subjects with both Type 1 and Type 2 diabetes for flexion extension of the knee (panel a) and lateral movement of the knee (panel b).



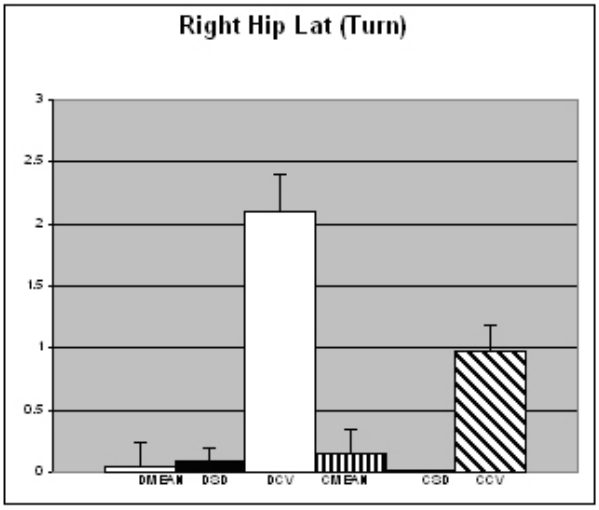
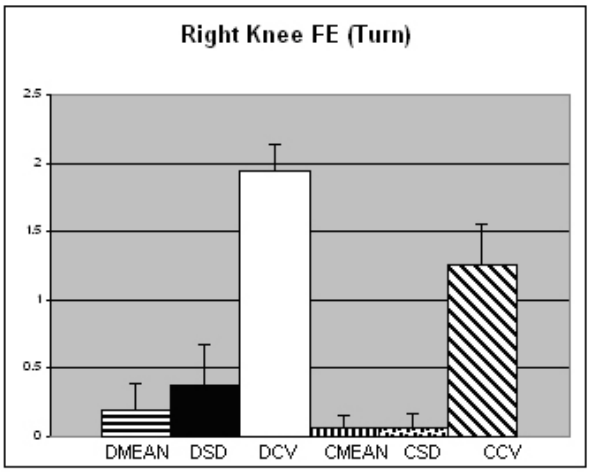


Figure 5. This figure illustrates the average flexion extension of the hip during turns of 0.33 meters diameter for subjects with diabetes (d) and control subjects (c) showing the mean data (mean), standard deviation (sd), and coefficient of variation (cv) for control subjects (c) and subjects with diabetes (d) concerning flexion extension of the hip (panel a) and lateral movement of the hip (panel b).

in tremor was greatest in the turn of 0.33 meters diameter compared to 0.66 diameter for the subjects with diabetes ($P < 0.01$) but there was no significant difference in the tremor between either diameter turn in control subjects ($P > 0.05$).

Analysis of EMG showed similar results. Table 5 states the average activity of the right and left tibialis anterior and right and left gastrocnemius muscle as a percent of total muscle activity during a maximal effort. When expressed as normalized muscle activity subjects with diabetes (both Type 1 and Type 2) had significantly greater muscle activity in all of the muscles examined during walking in a linear path as shown in this table. The same results were seen for the quadriceps and hamstring muscles. Muscle activity in the leg nearly tripled during gait in subjects with diabetes compared to control subjects; these differences were significant ($P < 0.01$). However the data for subjects with Type 2 and Type 1 diabetes was not different from each other ($P > 0.05$).

To understand the additional muscle activity further, during both linear walking and during turns, data was analyzed during only the phase of initial contact of the heel and during toe off during the gait cycle. During turns, data was averaged for both the 0.33 and 0.66-meter turn. As shown in table 6, a ratio was taken of gastrocnemius and tibialis anterior muscle activity. Dur-



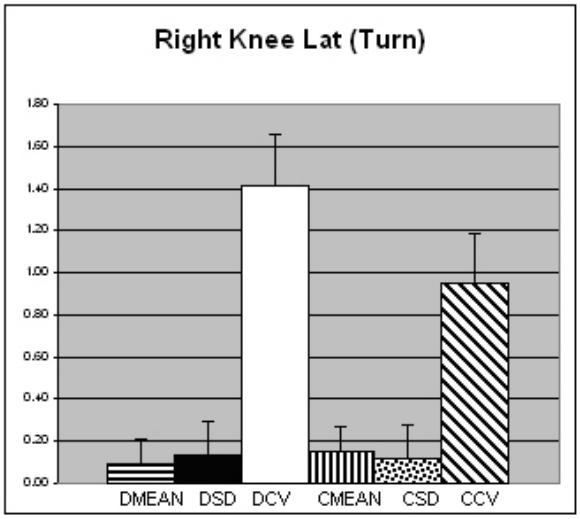


Figure 6. This figure illustrates the average flexion extension of the knee during turns of 0.33 meters diameter for subjects with diabetes (d) and control subjects (c) showing the mean data (mean), standard deviation (sd), and coefficient of variation (cv) for control subjects (c) and subjects with diabetes (d) concerning flexion extension of the knee (panel a) and lateral movement of the knee (panel b).

ing initial contact, tibialis anterior is active and the gastrocnemius should be quiescent. Therefore, the ratio of gastrocnemius to tibialis anterior activity should be low since the tibialis anterior is responsible for breaking the movement of the ankle on initial contact. However, during turns and during walking in a linear path, the ratio of gastrocnemius to tibialis anterior activity during initial contact (IC) was low for control subjects but was nearly 400% percent higher in subjects with Type 1 and Type 2 diabetes. For both walking in a linear path and turns, these differences were significant ($P < 0.01$). The same was found during toe off. During toe off, the prime mover is the gastrocnemius muscle and the tibialis anterior muscle is quiescent. While walking in a linear paths and during turns (table 6), muscle activity in the control

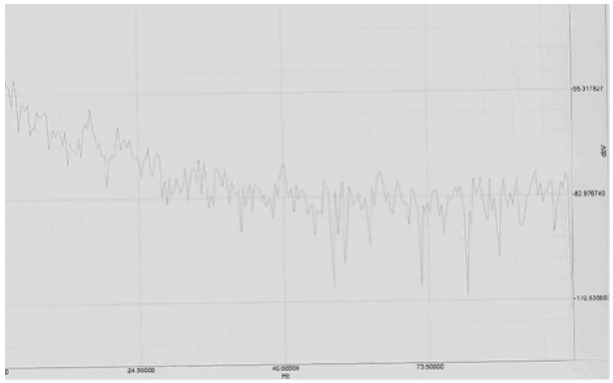


Figure 7. A spectral analysis (FFT) of the knee accelerometer data in a typical control subject in a frequency range of 0-100 Hertz.

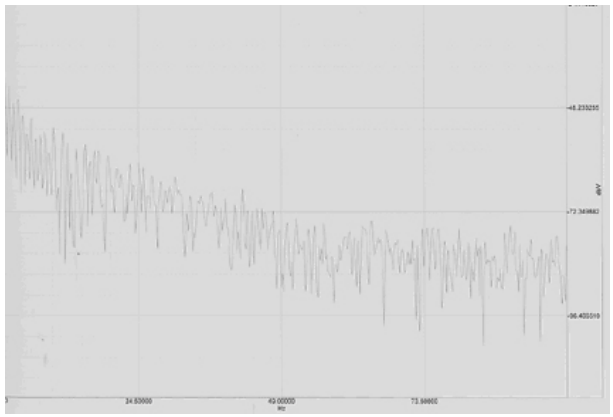


Figure 8. A spectral analysis (FFT) of the knee accelerometer data in a typical subject with diabetes in a frequency range of 0-100 Hertz.

subjects was 7.5 and 13.4% respectively. However, for subjects with Type 1 and Type 2 diabetes, the muscle activity ratio of the tibialis anterior to gastrocnemius was approximately four times higher. Thus, for walking in linear paths and turns subjects with diabetes displayed significantly more co-contraction of muscle in addition to the tremor noted above for accelerometer data.

Discussion

People with diabetes have been reported to have autonomic dysfunction [15, 25-27], visual loss [2] and loss of vestibular function that precedes, in many cases, significant sensory loss. [9] Visual and vestibular impairments are seen both in patients with Type 1 and Type2 diabetes [2, 13, 14, 20]. While the most common feature of diabetic gait is improper pressure distribution on the foot and a longer stance phase and shorter steps [21, 31, 32], the true effect of somatic and autonomic polyneuropathies on gait characteristics during gait in a linear direction and in turns had yet to be elucidated prior to the present investigation.

Some of the mechanism of the damage to the vestibular, somatic and autonomic nervous systems has been linked to damage to the microcirculation associated with

poor glycemic control in Type 1 and Type 2 subjects with diabetes. Precapillary damage occurs prior to any apparent damage to the somatic or autonomic nervous systems. This precapillary damage is due to dysfunction of endothelial cells, which impairs the normal nitric oxide pathways that cause vasodilatation. [37] This reduction in nerve blood flow, chronic inflammation, and glycolated end products has been linked to somatic and autonomic nervous system damage. [18, 33, 37, 41, 42] There is also some indication that there is a deprivation of nerve growth factors in people with Type 1 and Type 2 diabetes. [4] Whatever the mechanism, damage is widespread in the body in both Type 1 and Type 2 diabetes.

Gait is a complex process involving input from the eyes, the vestibular system, central data processing and the motor centers in the lower brain and spinal cord. It also relies on sensory input from muscle spindles and the bottom of the foot and viable skeletal muscle. Since all elements of the nervous system, vestibular and visual systems can be damaged in both Type 1 and Type 2 diabetes as sighted above, subjects in the present investigation were selected with no sensory impairment in the foot and no noticeable motor loss below the waist to isolate central neurological gait impairment. In this respect, then, subjects were chosen to eliminate myopothies and major peripheral sensory neuropathies to focus the present investigation on the effects of Type 1 and Type 2 diabetes to defects in central motor control and in the visual and vestibular systems on gait. There were several indicators of motor control deficits. First, during walking in a linear path and during turns, there was increased g loading in both the flexion extension and lateral directions of all joints examined. It is not surprising that the hip joint had a smaller percent increase in g loading in all subjects since the knee and the ankle are at the end of a long lever arm and any adverse movement at the hip will be reflected in greater movement at the knee and the ankle. However, at every joint that was examined for both walking in a linear path and turns, there was more movement as assessed by the average g loading and the coefficient of variation in g loading at each joint. This was made worse when subjects walked in the smallest turn (0.33 meters). While wider turns also showed enhanced g loading, the tighter turns showed a significant increase in g loading over walking in a linear path. When coupled with slower gait, it would appear that subjects with diabetes (both Type 1 and Type 2) seemed to display uncertainty during gait, even thought they had full sensation in their feet an no impairment in muscle strength in their legs.

The origin of this uncertainty seemed to be motor control itself induced by either vestibular, visual impairment, or by motor errors induced by spinal motor control centers or all three. The speed of gait, width of steps and coordination of agonist-antagonist activity should all be linked to central nervous control. Improper input from the vestibular or visual systems would cause the central nervous system to loose coordination. This uncertainty in placing the feet due to improper coordination would in turn cause gait to be slow with wider steps and co-contraction of muscles to stabilize the body to prevent falls. Data in the present investigation seems to support all of the above.

The vestibular system is impaired in both Type 1 and Type 2 diabetes [13]. The microcirculation to the vestibular system is high and probably damaged due to diabetes. As such, improper vestibular input with both Type 1 and Type 2 diabetes would probably lead to motor control errors. Further, slowing the nerve conduction velocity in the brain and spinal cord would also destroy coordination in gait in subjects with Type 1 and Type 2 diabetes [13, 22, 40]. Defects in the visual system and vestibular system have been linked to atrophied nerves and demylenation [7]. At a multi-organ level, visual-vestibular interaction in patients with both Type 1 and Type 2 diabetes has shown to be impaired [8]. It is not surprising then that if the visual and optical systems both show slowed conduction velocities and atrophy of neurons resulting in impairment and poor interactions between these two central brain centers, that gait would also be impaired absent losses in muscle strength or sensation. Perhaps the best indicator of loss of central motor impairment is in the 16 hertz band on analysis of accelerometer data. Here, the power in the 16 hertz band showed almost a 3 fold increase in activity during gait when walking a linear path and in turns in patients with diabetes compared to control subjects. The 16 hertz band is commonly associated with motor control errors generated in the brain [11]. In contrast, the 8 hertz band, which was also increased by diabetes, shows motor control errors in the spinal cords. The increase in error in both the 8 and 16 hertz band was similar which seemed to indicate a general reduction in motor control throughout the entire central nervous system.

In the peripheral nervous system, motor control is also impaired. Absent major sensory loss and even early in diabetes, motor and sensory nerve conduction velocity is impaired [40]. This impairment in some studies is associated with over a 50% reduction in nerve conduction velocity [28, 40]. The reduction in velocity is attributed to an increase in MAP kinase due to Polyol pathway flux in diabetes and not necessarily due to circulatory impairment which seems to alter only synaptic transmission [28]. Such a dramatic slowing would have the effect of slowing normal reflex control of movement. The best evidence for this peripheral component is the H reflex.

Here, and in other studies, there was an increase in H latency and magnitude [23, 40]. Such a slowing in reflex activity should cause errors during gait and cause the slowing and co-contraction of muscle seen here. While Nardone and Schieppati (2004) did not demonstrate an increase in side to side sway when standing

until medium and large fiber neuropathy was seen, gait, especially in turns, is for more challenging on the motor control system then standing and impairments should appear long before those seen during standing.

The result of poor motor control is that subjects used more agonist antagonist and co-contraction of muscles activity during gait in both Type 1 and Type 2 diabetes compared to control subjects. In the present investigation, analysis during initial contact and toe off during gait showed muscle activity to be at least five fold higher in antagonist muscle groups during each gait cycle. This would have the effect of increasing the energy during gait and slowing the speed of gait as was seen in the present investigation. In tight turns (0.033 meters diameter), the co-contraction of muscle was even worse, showing that subjects used co-contraction of muscle to stabilize the ankle and knee to prevent falls. It is not surprising, then, that gait is slower and, since agonist antagonist muscles are firing at the same time gait is much more unsteady in subjects with diabetes than control subjects.

Whatever the exact central mechanism, patients with diabetes should be more at risk for falls due to poor motor control than patients without diabetes. Curiously, the loss of motor control seems to affect gait in falls more than dealing with heavy equipment. For example, in a recent study, [17], Kennedy has shown that there is a increase risk of low impact falls in patients with both Type 1 and Type 2 diabetes. However, these same patients were at the same risk of motor vehicle accidents as control subjects that were aged matched. In the study, Kennedy and colleagues show the rate of accidents where insulin treated patients were estimated at 2.91 per hundred thousand populations per year compared to 148 for the control population making the relative risk 1.97 times as great for someone with Type 1 and Type 2 diabetes for low impact falls than control subjects. However, others have found the risk of falling even much higher. Some studies have noted that the increase risk of falls is ten to fifteen times higher in subjects with diabetes than control subjects. [10, 29] Patients in these studies had peripheral neuropathies impairing sensation in the feet. In the present investigation, although subjects were free of major peripheral sensory and motor neuropathies, subjects still had tremor, co-contraction of muscles, slow gait, and improper pressure distribution on the feet during gait. The fall risk in this population has been clearly established.

Obviously, the present investigation was limited to approximately 25 subjects with diabetes compared to control subjects. Questions remain as to quantifying the relationship between damage to various motor control systems and various portions of the central nervous system such as eyes, inner ear, and the motor control nuclei and the age and length of time since subjects had diabetes. Further, more detailed analysis of falls and the cause of the falls need to be correlated to get a better understanding of the increase risk associated with patients with Type 1 and Type 2 diabetes.

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