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Diabetic Driving Studies—Part 3: A Comparison of Mean Brake Response Time Between Neuropathic Diabetic Drivers With and Without Foot Pathology

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A R T I C L E I N F O

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A B S T R A C T

We have previously demonstrated an abnormally delayed mean brake response time and an increased frequency of abnormally delayed brake responses in a group of neuropathic diabetic drivers compared with a control group of drivers with neither diabetes nor lower extremity neuropathy. The objective of the present case-control study was to compare the mean brake response time between neuropathic diabetic drivers with and without specific diabetic foot pathology. The braking performances of the participants were evaluated using a computerized driving simulator with specific measurement of the mean brake response time and the frequency of abnormally delayed brake responses. We analyzed a control group of 20 active drivers with type 2 diabetes, lower extremity neuropathy, and no history of diabetic foot pathology and an experimental group of 20 active drivers with type 2 diabetes, lower extremity neuropathy, and a history of diabetic foot pathology (ulceration, amputation, and/or Charcot neuroarthropathy) from an urban U.S. podiatric medical clinic. Neuropathic diabetic drivers without a history of specific foot pathology demonstrated an 11.11% slower mean brake response time (0.790 ± 0.223 versus 0.711 ± 0.135 second; $p < .001$), with abnormally delayed reactions occurring at a similar frequency (58.13% versus 48.13%; $p = .0927$). Both groups demonstrated a mean brake response time slower than a suggested threshold of 0.70 second. The results of the present investigation provide evidence that diabetic patients across a spectrum of lower extremity sensorimotor neuropathy and foot pathology demonstrate abnormal automobile brake responses and might be at risk of impaired driving function.

The effect of lower extremity pathology and surgical intervention on automobile driving function has been a topic of contemporary interest in orthopedic studies. Several investigators have reported general guidelines and produced original data regarding the return to safe driving after lower extremity surgery (1–11). Others have specifically studied the effect of chronic musculoskeletal lower extremity pathology (12,13), the use of immobilization devices (14–17), the effect of major limb amputation (18–20), and the general effects of diabetes and hypoglycemia (21–24) on driving outcomes.

Our group has previously reported original data on the effect of diabetic sensorimotor neuropathy on driving function (25). We observed a statistically significant increase in the mean brake response time (0.757 versus 0.549 second) and an increased frequency of abnormally delayed braking reactions (57.5% versus 3.5%) in a group of neuropathic drivers with diabetes compared with a control group of drivers with neither diabetes nor lower extremity neuropathy. Even independent of a comparative statistical analysis, the observed mean brake response time in the experimental group was slower than a recommended safety threshold of 0.70 second, indicating that the combination of diabetes and lower extremity neuropathy might have a negative effect on driving performance.

Diabetic neuropathy presents as a symmetrical sensorimotor polyneuropathy preferentially affecting the distal lower extremities. The most apparent effects of this occur within the sensory system and contribute to the development of an insensate plantar foot, pedal ulcerations, soft tissue and bone infection, and limb amputations (26–29). However, involvement of the motor system can also lead to lower extremity weakness, skeletal muscle atrophy, slowing of movements, unstable gait, and an increased frequency of falls (30–38). Additionally, auditory and visual reaction times have been demonstrated to be impaired in the presence of diabetes (39–41).

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Given this, it is not difficult to envision how diabetic lower extremity neuropathy might affect automobile driving function.

Although providing original and unique data on potentially impaired diabetic driving function, an acknowledged limitation of our previous investigation was the heterogeneity within the experimental group. The experimental group consisted entirely of neuropathic diabetic drivers but ranged from those without any history of specific diabetic foot pathology to those with pedal ulcerations, partial foot amputations, and/or Charcot neuroarthropathy. The objective of the present case-control investigation was to assess the mean brake response times of neuropathic diabetic drivers with and without diabetic foot pathology. We specifically aimed to determine whether a group of neuropathic diabetic drivers with diabetic foot pathology had slower brake response times than a group of neuropathic diabetic drivers without a history of diabetic foot pathology.

Patients and Methods

After approval by our institutional review board (Temple University; protocol no. 22922), the braking performance of the subjects was evaluated using a computerized driving simulator (Stationary Simple Reaction Timer; Vericom Computers, Inc., Rogers, MN). This device has previously been used to evaluate the brake reaction times in the setting of lower extremity impairment and measures to a precision of 0.01 second (12,25). The simulator consists of a laptop computer, steering wheel, and accelerator and brake pedal system (Fig.). The participants were seated in a comfortable position with adjustment of the simulator construct as needed for individual preference. For the purposes of the present investigation, the participants depressed the accelerator pedal and maintained a constant speed with their right foot as a driving scene was displayed on the monitor. Next, at a random time within a 10-second window after initiation of a constant speed, red lights were activated on the monitor, which alerted the participants to depress the brake pedal as fast as they could with their right foot.

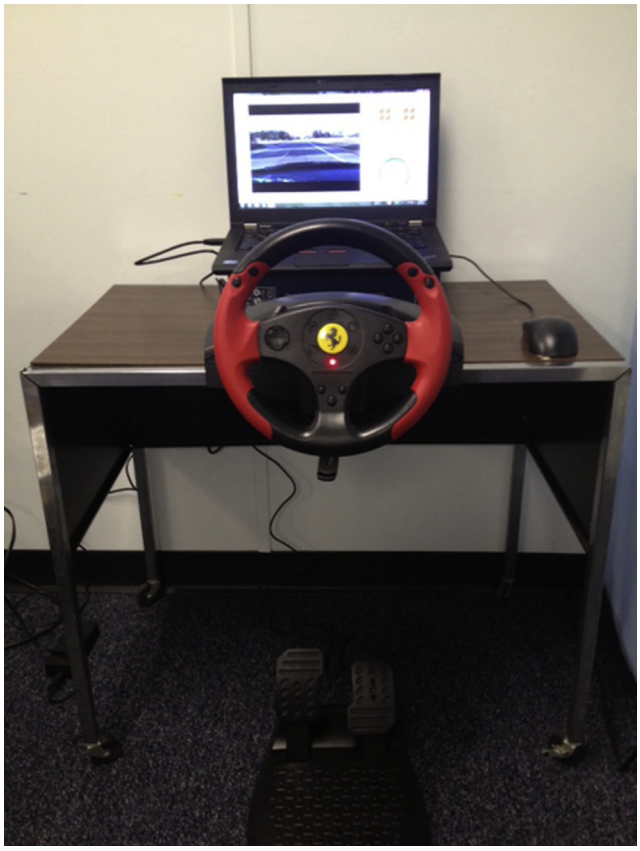

Fig. Driving simulator. The primary outcome measure of the present investigation was the mean brake response time using a computerized driving simulator. The brake response time was defined as the interval from red light activation on the computer monitor while maintaining a constant accelerator speed to initiation of the brake pedal.

The interval between the red light activation and initiation of the brake pedal was recorded as the brake response time.

Verbal instructions and a demonstration describing how to use the simulator were given, and the participants had the opportunity to undergo multiple practice trials before the actual brake response testing until they were comfortable with the equipment. Ten recorded trials were then performed for each participant, with elimination of the fastest and slowest trials from each set before data analysis. The primary outcome measure was the mean brake response time from the 8 recorded trials, and our sample size power estimate was based on this result. The frequency count of the abnormally delayed trials was the secondary outcome of interest.

Although a number of studies have reported on normal and abnormal brake response times (42,43), several investigators, government sources, and the driving simulator software we used have established a cutoff threshold for potentially unsafe brake response times at 0.70 second (8,12,43). In the present investigation, we considered brake reaction times $<$0.70 second as normal and those $\geq$0.70 second as abnormally delayed.

We had previously enrolled an experimental group of 25 active drivers with type 2 diabetes and lower extremity neuropathy from an urban U.S. podiatric medical clinic (Foot and Ankle Institute, Temple University School of Podiatric Medicine, Philadelphia, PA) (25). These participants were enrolled consecutively from eligible consenting patients and included those both with and without a history of specific diabetic foot pathology (a history of, or current, pedal ulceration, minor/major amputation, and/or Charcot neuroarthropathy). For the present investigation, we expanded the data collection of this cohort to consecutively enroll a control group of active drivers with a history of type 2 diabetes, lower extremity neuropathy, and no history of pedal ulceration, minor/major limb amputation, or Charcot neuroarthropathy and an experimental group of active drivers with type 2 diabetes, lower extremity neuropathy, and a history of, or current, foot ulceration, minor/major limb amputation, and/or Charcot neuroarthropathy. We considered active drivers to be those who identified themselves as current drivers, possessed an active driver's license, and who had driven at least twice in the previous month. The exclusion criteria were a history of any right-sided lower extremity surgery within 3 months, current requirement for any type of protective immobilization device on the right leg (i.e., surgical shoe, removable cast boot, short leg cast, Charcot restraint orthotic walker), and any current driving restrictions related to their care. All drivers were tested in their own footwear that they were wearing on that day. Additional information collected from the experimental group included age, gender, most recent hemoglobin A1c value ($<$6 months), any history or current evidence of lower extremity ulceration, any history of minor or major lower extremity amputation, any history of Charcot neuroarthropathy, and the side of any ulceration, amputation, or Charcot neuroarthropathy.

Neuropathy was defined using the Michigan Neuropathy Screening Instrument (MNSI), a validated measure of diabetic neuropathy encompassing sensory, motor, and autonomic components (44,45). The maximum score available is 5 points for each limb and 10 points for both limbs. Participants receive 1 point for each foot if any deformities are present (e.g., Charcot neuroarthropathy, hammertoes, previous amputation, plantar callus formation, fissuring, infection) and 0 points if no deformities are present. Participants receive 1 point for each foot if any ulceration is present and 0 points if no ulceration is present. Participants receive 1 point if the Achilles tendon reflex is absent, 0.5 point if the Achilles tendon reflex is present with reinforcement (clasping the hands and fingers together with the Jendrassik maneuver), and 0 points for an intact Achilles tendon reflex without reinforcement. Participants receive 1 point if vibratory sensation is absent as measured with a 128-Hz tuning fork at the dorsal hallux interphalangeal joint, 0.5 point if vibratory sensation is diminished (not able to sense after 5 seconds), and 0 points if vibratory sensation is intact (subject able to sense for $>$5 seconds). Participants receive 1 point if protective sensation is absent as measured with a 5.07-gauge Semmes-Weinstein monofilament (defined as the inability to sense 4 sites on the plantar foot [heel, first metatarsal head, fifth metatarsal head, and plantar hallux]), 0.5 point if 1 of the 4 sites is sensed, and 0 points if $\geq$2 sites are sensed. Vibratory and sensory testing was adjusted if any partial foot amputation was present. We considered participants who scored $\geq$2.5 of 10 as having neuropathy, and subjects who scored $<$2.5 as not having neuropathy (44,45).

An a priori power analysis based on a previous investigation using this simulator and participants with lower extremity pathology (12) was calculated, assuming a primary outcome measure standard deviation of 0.1 second and a detectable effect size of 0.2 second to ensure a power of 0.8 and an α of 0.05 with an independent Student's t test. We chose to collect data from 40 subjects (20 in each group). The data were stored in a password-protected personal computer for subsequent statistical analysis. All statistical analyses were performed using Statistical Analysis Systems software, version 9.2 (SAS Institute, Cary, NC). Descriptive statistics were calculated and consisted of the mean, standard deviation (SD), range, and frequency count. Comparative statistical analyses performed on the primary outcome measure (mean brake response time) used the independent Student t test. For the secondary outcome measure (frequency count of abnormally delayed reactions), we used Fisher's exact test of the null hypothesis.

Results

The control group consisted of 20 participants (15 males) with a mean $\pm$ SD age of 58.65 $\pm$ 11.41 (range 28 to 75) years and 160 brake

response trials. The mean ± SD MNSI score was 3.98 ± 1.27 (range 2.5 to 7.5). Hemoglobin A1c data were available from 18 of the 20 participants (90.0%; mean ± SD 7.7% ± 1.59%, range 5.5% to 10.3%). The mean ± SD brake response time was 0.790 ± 0.223 (range 0.46 to 1.68) second. An abnormally delayed brake response time was observed in 93 of these 160 trials (58.13%).

The experimental group included 20 participants (19 males [95.0%]), with a mean ± SD age of 55.35 ± 9.53 (range 28 to 69) years and 160 brake response trials. The mean ± SD MNSI score was 6.85 ± 1.70 (range 3.5 to 9.0). Hemoglobin A1c data were available from 18 of the 20 participants (90.0%; mean ± SD 7.97% ± 1.14%, range 6.4% to 10.1%). The mean ± SD brake response time was 0.711 ± 0.135 (range 0.50 to 1.30) seconds. An abnormally delayed brake response time was observed in 77 of these 160 trials (48.13%).

The results of a comparative statistical analysis between the control and experimental groups are listed in the Table. The control group demonstrated an 11.11% slower mean brake response time (0.790 versus 0.711second; $p < .001$), with abnormally delayed reactions occurring at a similar frequency (58.13% versus 48.13%; $p = .0927$) compared with the experimental group. The control and experimental groups both demonstrated a mean brake response time >0.70 second.

Discussion

The results of the present investigation provide evidence that drivers with diabetes mellitus, lower extremity neuropathy, and a history of specific diabetic foot pathology (ulceration, amputation, Charcot neuroarthropathy) do not demonstrate slower mean brake response times or more frequent abnormally delayed brake responses compared with a group of drivers with diabetes, lower extremity neuropathy, and no history of diabetic foot pathology. These findings provide evidence against our initial hypothesis that neuropathic diabetic drivers with a history of diabetic foot pathology would perform worse during brake response testing than neuropathic diabetic drivers without specific diabetic foot pathology. We had originally hypothesized that those diabetic drivers with specific foot pathology might represent a more advanced disease state and might perform worse with respect to brake response testing. The group of neuropathic diabetic drivers without foot pathology demonstrated statistically significant slower mean brake response times. We believe that although this finding demonstrated statistical significance, the clinical significance is limited because both groups demonstrated mean brake response times in excess of a recommended safety threshold of 0.70 second (8,12,43).

Our interpretation of this finding is that the onset of specific lower extremity pathology as a consequence of lower extremity sensorimotor neuropathy does not impart an additional effect on the brake response time compared with the neuropathy as an independent variable. We had previously observed a statistically significant slower mean brake response time and an increased frequency of abnormally delayed braking reactions in a group of neuropathic diabetic drivers compared with a control group of drivers with neither diabetes nor lower extremity neuropathy (25). We can now conclude that these findings appear to extend comparably across a spectrum of patients with diabetes and neuropathy, including those with and without lower extremity ulcerations, amputations and Charcot neuroarthropathy. Any diabetic driver with lower extremity sensorimotor neuropathy might be at risk of abnormal brake responses, regardless of the onset of any specific diabetic limb pathology.

Just as with any scientific investigation, critical readers are encouraged to review and assess the study design and specific results to reach their own independent conclusions. The preceding represents our conclusions based on the data. We also realize that all investigations have limitations, and the present study had several to consider. First, we believe it is important to appreciate that the brake response time represents only a single facet of total driving function. Also, although the brake response times are slower, this not mean that neuropathic diabetic drivers are necessarily at an increased risk of traffic accidents. Elderly drivers, for example, have been observed to compensate for the decreased reaction times associated with age by driving at slower speeds, driving during safe driving conditions, and by following vehicles at greater distances (46–51). Although it is possible that neuropathic diabetic drivers enact similar or different compensatory driving behaviors, this was not specifically studied within the investigational design.

Second, data were collected from a limited amount of subjects within an urban environment; therefore, these results might not be representative of a broader population sampling. Also, all participants were treated at a podiatric medical clinic; thus, at least some degree of selection bias was probable. We observed no difference between groups with respect to age ($p = .3272$), gender ($p = .1818$), or hemoglobin A1c ($p = .5673$), although the MNSI score was higher (6.85 versus 3.98; $p < .001$) in the experimental group with specific diabetic foot pathology. This should not be surprising, because variables such as ulceration, amputation, and Charcot neuroarthropathy are included in the calculation of this score.

Finally, our specific investigational method had inherent limitations. The participants were tested using a driving simulator consisting of stationary pedals and a laptop computer screen on a table,

Table
Primary and secondary outcome measure results

Variable	Drivers With Diabetes, Lower Extremity Neuropathy, and No History of Diabetic Foot Pathology (n = 160 Trials*)	Drivers With Diabetes, Lower Extremity Neuropathy, and History of, or Current, Diabetic Foot Pathology (n = 160 Trials*)	*p* Value
Age (yr)	58.65 ± 11.41	55.35 ± 9.53	.3272†
Male gender	15 (75.0)	19 (95.0)	.1818‡
HbA1c (%)	7.70± 1.59	7.97± 1.14	.5673†
MNSI score	3.98 ± 1.27	6.85 ± 1.70	<.001†,§
Brake response time (s)	0.790 ± 0.223	0.711 ± 0.135	<.001†,§
Frequency of abnormally delayed reactions (≥0.70 s)	93/160 (58.13)	77/160 (48.13)	.0927‡

Abbreviations: HbA1c, hemoglobin A1c; MNSI, Michigan Neuropathy Screening Instrument.
Data presented as mean ± standard deviation, %, or n/n (%).
* Data from 160 trials and 20 participants for all variables, except for HbA1c, for which data were available for 144 trials and 18 participants.
† Independent *t* test.
‡ Fisher's exact test.
§ Statistically significant at $p < .05$.

not a real-life driving situation. Our threshold for abnormally delayed brake response times could also be open to debate. We used a previously published study design and safety threshold that we have confidence in but that is possibly without universal acceptance within the automobile driving safety community.

In conclusion, the results of the present investigation provide evidence that all diabetic patients with lower extremity sensorimotor neuropathy, not just those with ulcerations, amputations, and/or Charcot neuroarthropathy, demonstrate abnormally delayed automobile brake responses and might be at risk of impaired driving function.

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