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Diabetic Driving Studies—Part 2: A Comparison of Brake Response Time Between Drivers With Diabetes With and Without Lower Extremity Sensorimotor Neuropathy

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ABSTRACT

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 drivers with diabetes 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 2 groups of drivers with diabetes with and without lower extremity sensorimotor neuropathy. 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 the abnormally delayed brake responses. We compared a control group of 25 active drivers with type 2 diabetes without lower extremity neuropathy and an experimental group of 25 active drivers with type 2 diabetes and lower extremity neuropathy from an urban U.S. podiatric medical clinic. The experimental group demonstrated an 11.49% slower mean brake response time (0.757 ± 0.180 versus 0.679 ± 0.120 second; $p < .001$), with abnormally delayed reactions occurring at a greater frequency (57.5% versus 35.0%; $p < .001$). Independent of a comparative statistical analysis, diabetic drivers with neuropathy demonstrated a mean brake response time slower than a suggested safety threshold of 0.70 second, and diabetic drivers without neuropathy demonstrated a mean brake response time faster than this threshold. The results of the present investigation provide evidence that the specific onset of lower extremity sensorimotor neuropathy associated with diabetes appears to impart a negative effect on automobile brake responses.

The effect of lower extremity pathologic entities 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.

The diabetic neuropathy presents as a symmetrical sensorimotor polyneuropathy preferentially affecting the distal lower extremities. The most apparent effects 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 movement, 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). Given this, it is not

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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 initial investigation was the demographic differences between the control and experimental groups. The control group was younger and had no history of diabetes, and the experimental group was older, predominantly male, and entirely composed of patients with neuropathic diabetes. The objective of the present case-control investigation was to assess the effect of lower extremity neuropathy on the mean brake response time of drivers with diabetes. We specifically aimed to determine whether a group of drivers with diabetes with lower extremity neuropathy had slower brake response times compared with a group of drivers with diabetes without neuropathy.

Patients and Methods

After approval by our institutional review board (Temple University; protocol no. 22922), the braking performance of the subjects was evaluated with a computerized driving simulator (Stationary Simple Reaction Timer; Vericom Computers, Inc., Rogers, MN). This device has previously been used to evaluate 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, 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. 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 considered the mean brake response time from the 8 recorded trials, and our sample size power estimate was determined from this result. The frequency count of abnormally delayed trials was considered the secondary outcome of interest.

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 time from red light activation on the computer monitor while maintaining a constant accelerator speed to initiation of the brake pedal.

Although a number of studies have been reported with respect to 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 0.700 second (8,12,43). In the present investigation, we considered brake reaction times <0.70 second as normal and those ≥0.70 second as abnormally delayed.

We had previously enrolled and collected data from a group of 25 active drivers with a history of 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). For the present investigation, we subsequently enrolled a second group of active drivers with a history of type 2 diabetes without lower extremity neuropathy. This was considered the control group for the present investigation, and the diabetic drivers with neuropathy were considered the experimental group. Eligible consenting participants were consecutively enrolled without preselection. We considered active drivers as 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 consisted of a history of any right-sided lower extremity surgery within 3 months, the requirement for any type of protective immobilization device on the right leg (i.e., surgical shoe, removable cast boot, short leg cast), 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 participants included age, gender, most recent hemoglobin A1c value (<6 months), and a history of any specific diabetic foot pathology (history or current evidence of lower extremity ulceration, any history of minor or major lower extremity amputation, and any history of Charcot neuroarthropathy).

Neuropathy was defined using the Michigan Neuropathy Screening Instrument, 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. The participants receive 1 point on 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. The participants receive 1 point on 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. The 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 with the examiner). The participants receive 1 point if protective sensation is absent when measured with a 5.07-gauge Semmes-Weinstein monofilament (defined as an 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 ≥2 sites are sensed. The vibratory and sensory testing was adjusted if any partial foot amputation was present. We considered participants who scored ≥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 a total of 50 subjects (25 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 25 subjects (14 males [56.0%]) with a mean ± SD age of 58.16 ± 12.62 (range 32 to 75) years and 200 brake response trials. The mean ± SD Michigan Neuropathy Screening Instrument score was 1.06 ± 0.917 (range 0 to 2). A hemoglobin A1c value was available for 22 of the 25 subjects (88.0%; mean ± SD 7.32% ± 1.44%; range 5.6% to 11.5%). No participants in the control group had a history of specific diabetic foot pathology. The mean ± SD brake response time was 0.679 ± 0.120 (range 0.45 to 1.30) seconds. An