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# Altered accelerator pedal control in a driving simulator in people with diabetic peripheral neuropathy

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## What's new?

- Diabetes impacts driving safety through the influence of hypoglycaemia and diabetic retinopathy.
- Diabetic peripheral neuropathy (DPN) affects tactile sensation, proprioception and muscle strength primarily in the feet and the lower limbs.
- Drivers with DPN showed reduced ankle position sense, impaired muscle function and an altered use of the accelerator pedal.
- Despite driving more slowly, drivers with DPN experienced more loss-of-control events than other drivers, but demonstrated a residual ability to improve with practice.
- Control of the accelerator pedal and overall driving performance are affected by DPN, but this research opens up opportunities to devise technological solutions and training programmes to help people with DPN drive more safely.

## Abstract

**Aim** To investigate whether the sensory-motor impairment attributable to diabetic peripheral neuropathy would affect control of the accelerator pedal during a driving simulator task.

**Methods** A total of 32 active drivers, 11 with diabetic peripheral neuropathy (mean ± SD age 67±5.0 years), 10 with diabetes but no neuropathy (diabetes group; mean ± SD age 62±10 years), and 11 healthy individuals without diabetes (healthy group; mean ± SD age 60±11 years), undertook a test on a dynamometer to assess ankle plantar flexor muscle strength and ankle joint proprioception function of the right leg, in addition to a

driving simulator task. The following variables were measured: maximal ankle plantar flexor muscle strength; speed of strength generation (Nm/s); and ankle joint proprioception (ankle repositioning error, degrees). In the driving simulator task, driving speed (mph), accelerator pedal signal (degrees) and the duration of specific 'loss-of-control events' (s) were measured during two drives (Drive 1, Drive 2).

**Results** Participants with diabetic peripheral neuropathy had a lower speed of strength generation ($P$<0.001), lower maximal ankle plantar flexor muscle strength ($P$<0.001) and impaired ankle proprioception ($P$=0.034) compared to healthy participants. The diabetic peripheral neuropathy group drove more slowly compared with the healthy group (Drive 1 $P$=0.048; Drive 2 $P$=0.042) and showed marked differences in the use of the accelerator pedal compared to both the diabetes group ($P$=0.010) and the healthy group ($P$=0.002). Participants with diabetic peripheral neuropathy had the longest duration of loss-of-control events, but after one drive, this was greatly reduced ($P$=0.023).

**Conclusions** Muscle function, ankle proprioception and accelerator pedal control are all affected in people with diabetic peripheral neuropathy, adversely influencing driving performance, but potential for improvement with targeted practice remains possible.

## Introduction

Diabetic peripheral neuropathy (DPN) is one of the commonest complications of diabetes [1,2], with consequences for reduced or absent tactile sensation, vibration perception, proprioception, muscle strength and joint range of motion [2–7]. The cutaneous sensory loss, together with sensory-motor dysfunction, are most pronounced in the feet, and progress up to affect more proximal parts of the lower limbs, following a dying back pattern (distal–proximal direction) [8,9]. In people with DPN, muscle

strength of the ankle dorsal and plantar flexors is reduced by ~30% compared to people with diabetes but no neuropathy [10]. Nerve conduction velocities of peroneal and sural nerves significantly correlate with muscle response latencies, while a loss of movement perception, predominantly at the ankle joint, is attributable to altered function of muscle spindles [11–13]. The detrimental effects of diabetes and DPN in affecting neuromuscular and motor function with consequences for daily life activities has been reported in terms of marked unsteadiness, slower speed of strength generation and altered muscle activation timing while walking on level ground and on stairs [14,15].

In addition to locomotor tasks, driving is another common daily activity where the integration of motor and sensory function is important for successful performance. Previous studies investigating driving performance in diabetes have focused on the influence of hypoglycaemia (resulting in an impaired ability to drive), diabetic retinopathy (impairing the clear vision needed to operate a motor vehicle) and DPN, affecting the ability to feel foot pedals which could impact the ability to drive safely [16,17]. Concerning this last issue, a few recent studies have demonstrated that people with DPN had slower mean brake response times and have an increased frequency of abnormally delayed braking reactions compared with both people with diabetes but no DPN and healthy individuals, when driving on a simulator [18,19]. Possible reasons for this could be that sensory neuropathy impairs the ability to feel the pedals, or the ability to move the feet efficiently between the accelerator and brake pedals; thus, the impaired neuromuscular function of the plantar flexor muscles, together with the lack of adequate proprioceptive feedback, could influence driving performance [20].

Whilst previous studies of driving in diabetes have focused primarily on the influence of hypoglycaemia and diabetic retinopathy, no previous research has investigated the influence of DPN on sensory-motor function, its impact on the accelerator pedal control

during driving, and the overall effect these factors have on driving performance. We hypothesized that people with DPN would have impaired sensory-motor function around the ankle joint that would impact adequate control of the accelerator pedal during driving and, in turn, influence driving performance. The aim of the present study, therefore, was to investigate sensory-motor function in people with diabetes and DPN and driving performance using a driving simulator.

## Methods

### Participants

Thirty-two participants, all active drivers holding a UK driving licence and driving for not less than 30 min every day, were recruited into three groups: 11 participants with DPN (mean ± SD age 67 ± 5.0 years and BMI 32 ± 4.2 kg/m$^2$; nine men, two women), 10 participants with diabetes but no peripheral neuropathy (mean ± SD age 62±10 years and BMI 31±5.2 kg/m$^2$; nine men, one woman) and 11 healthy age-matched participants without diabetes (mean ± SD age 60 ± 11 years and BMI 27 ± 4.4 kg/m$^2$; nine men, two women). All participants gave their written informed consent to participate in the study, which received ethical approval from all relevant bodies. The principal inclusion criteria were: diagnosis of diabetes (diabetes group) or absence of diabetes in the healthy group (confirmed via random blood glucose test <7.8 mmol/l); holding a current full UK driving licence; driving a car at least once per week; and age >20 years (Table 1). The principal exclusion criteria were: active foot ulcers on either foot; lower limb amputation involving more than two toes on the right foot (foot applied to the accelerator pedal); dementia; visual acuity worse than 20/50; and proliferative diabetic retinopathy.

**Neuropathy assessment**

The presence and severity of DPN was measured using the modified Neuropathy Disability Score, a composite test of multiple sensory modalities, together with the detection of the vibration perception threshold using a neurothesiometer (Bailey Instruments Ltd, Manchester, UK) [1]. Based on these tests, participants with diabetes were divided into groups as follows: DPN group: Neuropathy Disability Score ≥6, vibration perception threshold ≥25 Volts; and diabetes group: Neuropathy Disability Score ≤3, vibration perception threshold ≤15 Volts. These tests were also performed in the healthy group to confirm the absence of peripheral neuropathy.

**Design**

During a 2.5-h experimental session, participants underwent a series of tests which were always presented in the same order. Firstly, information was taken about participants' medical history, and subsequently a maximum isometric contraction of the ankle plantar flexor muscles and an ankle proprioception test were performed on an isokinetic dynamometer (Cybex Norm, Rosemont, IL, USA). Visual acuity was assessed using a 'Snellen chart' and a random blood glucose test was undertaken to confirm the absence of diabetes in healthy individuals and to avoid any hypo- or hyperglycaemic influence during the subsequent driving task in those participants with diabetes. Participants with diabetes had blood glucose levels within the range 4.5–20 mmol/l (72–360 mg%) before the driving simulator task.

**Procedure**

*Motor function variables*

An isokinetic dynamometer (Cybex Norm) was used to assess the speed of strength generation and the maximal strength of the ankle plantar flexor muscles in addition to ankle proprioception function. The dynamometer was integrated with a data acquisition system Labchart 8® (AD Instruments, Sydney, NSW, Australia). Only the right foot and lower limb were selected for dynamometry testing because this is the side used to operate the accelerator pedal in all cars. The tests were performed according to standardized procedures. The ankle position selected to perform all motor function tests was 10° of plantar flexion, an angle corresponding to a mid-position of the accelerator pedal when driving [21]. Participants were positioned prone on the dynamometer couch and their right foot secured to the footplate of the dynamometer using non-elastic straps.

After several standardized submaximal contractions as a warm-up, participants completed a maximum effort isometric strength test of the plantar flexor muscles of the right leg at 10° plantar flexion. Participants were instructed to push against the dynamometer footplate as hard and as fast as possible, similar to an emergency brake when driving, and to maintain the contraction for ~3–5 s. This test included two trials, with a rest of 2–3 min in between each trial to avoid any fatigue effect.

With regard to proprioception function, the knowledge of the position (sense) and the ability to reproduce a specific joint position was objectively quantified recording the error value in degrees, the 'ankle repositioning error' [22]. We recorded the error value (i.e. the discrepancy between the target ankle joint position and the actual position participants selected in the trial). A higher error value indicates poorer proprioception function. The arm of the dynamometer moved passively to the target angle (10° plantar

flexion) and remained at this position for 5 s to demonstrate the target angle to the participant. Subsequently, the dynamometer foot plate was then moved into dorsiflexion and plantar flexion to disturb position sense. Participants were then asked to reproduce the same ankle joint position and maintain it for at least ~3 s while it was recorded. The proprioception test included some practice trials at different ankle joint angle positions to facilitate comprehension of the task, with a 1-min rest in between to avoid any learning effects. Participants were unable to see their feet during the experiment.

*Driving simulator task*

The driving simulator consisted of a 42-inch plasma screen, a steering wheel giving realistic feedback when turned, accelerator and brake pedals (automatic gear change), and an adjustable car seat. The setup was the same as that used in previous studies [23,24]. Participants were invited to find a comfortable driving position, with adjustment of the simulator construct as needed for individual preference. Specific instructions were given to participants to drive safely, as they would in a real car. Verbal instruction and a demonstration describing how to use the simulator were also given. The task consisted of a driving environment simplified by the absence of other vehicles and pedestrians, taken from the Colin McRae Rally 2 simulation (Codemaster, Leamington Spa, UK). The view was of the road ahead through the windscreen, with addition of the speedometer in the bottom right-hand corner. The route was a 3.1-mile winding country road, which included gentle and sharp bends with few straight sections. Each participant drove the same route twice.

**Data analysis**

*Motor function variables*

The speed of strength generation at the ankle was measured as the rate at which joint torque was developed, which reflects the speed of force being developed by the ankle plantar flexor muscles [15]. The speed of strength generation was assessed as the gradient of the torque–time (Δtorque/Δtime) curve over the first 150 ms after the onset of contraction. Onset of muscle contraction was defined as the time point at which the torque curve exceeded baseline torque by 5 Nm.

The maximal strength of the plantar flexor muscles was defined by the value of the 'peak torque' (Nm), i.e. the highest value exerted around the ankle joint, during a maximal isometric contraction of the plantar flexor muscles [25].

Proprioception function was assessed using the ankle repositioning error, which quantified the proprioception function using an error score obtained by the difference between the target position (10° plantar flexion) and the position reached by the participant. We considered the mean of three trials to be representative of the global proprioception function: higher error values correspond to lower proprioception function.

*Driving simulator task*

We analysed data from two repeats of the 3.10-mile driving course, quantifying driving speed (mph) and producing frequency distribution plots of the accelerator pedal position signal (degrees). This signal was recorded over a range from 0°, when no load was applied to the pedal, to a maximum of –20°, when the pedal was completely depressed to the floor. The pedal position plots represent the percentage of time that the

pedal was pushed down by different amounts. We considered each complete drive as a whole in order to observe the pattern of pedal usage and produced 'difference plots' by subtracting the frequency distribution of the healthy group or the diabetes group from the frequency distribution of the DPN group to identify and highlight any differences between the people with DPN and the other two groups. These difference plots, therefore, highlighted how differently drivers with DPN used the accelerator pedal when compared to drivers in the diabetes and healthy groups.

Lastly, the analysis of the steering wheel signal (degrees) for which we detected specific characteristics in terms of amplitude and repetitive frequency (Fig. 1) allowed us to estimate the total duration of any instance that we defined as a loss-of-control event. Loss-of-control events consisted of an extreme, unjustified and inappropriate use of the steering wheel, i.e. movements that reached the full range of motion of the steering wheel and/or exhibited a repetitive frequency, during which the driver continued to maintain this exaggerated motion. The total duration of these loss-of-control events for each group was then normalized to the number of participants in each group (seconds per person).

**Statistics**

The motor function variables (maximal ankle plantar flexor strength, speed of strength generation, and ankle proprioception), driving speed and loss-of-control events were analysed using one-way ANOVA, with Bonferroni *post hoc* tests to assess differences between groups (DPN, diabetes and healthy groups). We used a paired sample *t*-test to assess differences in the same group between drives (loss-of-control events, driving speed).

Differences between the accelerator pedal frequency distribution plots between groups (DPN vs diabetes group; or DPN vs healthy group) were assessed using an independent samples Student's *t*-test. Data were analysed using parametric statistics as the data were normally distributed. All statistical tests were performed using SPSS statistical package version 22 (IBM Corp., Chicago, IL, USA), with *P* values <0.05 taken to indicate statistical significance.

## Results

### Motor function variables

The mean ± SD speed of strength generation was significantly lower in the DPN group ($P<0.001$) and the diabetes group ($P=0.002$) when compared to healthy participants [DPN group: 80.30 ± 61.37 n/ms; diabetes group: 104.66 ± 43.36 n/ms; healthy group: 252.65 ± 28.37 n/ms (Fig. 2a)]. The maximal plantar flexor muscle strength values were significantly lower ($P<0.001$) only in the DPN group vs the healthy group [DPN group: 22±62; diabetes group: 39.98±14.33; healthy group: 54.44±20.31 (Fig. 2b)]. A lower ankle proprioception function, corresponding to a higher error value, was found in the DPN group ($P=0.034$) vs the healthy group [DPN group: 3.54±2.29; diabetes group: 3.30±1.63; healthy group: 1.45±1.34 (Fig. 2c)]. No significant differences were found between the diabetes and the healthy group for these two variables (maximal strength, $P=0.76$; proprioception, $P=0.81$).

### Driving simulator task

Analysis of the study drives showed a significantly slower mean ± SD driving speed in the DPN group compared to the healthy group in both the first ($P=0.048$; DPN group: 16.53 ± 5.39 mph; diabetes group: 20.41±4.68 mph; healthy group: 22.08±4.76 mph)

and the second drive ($P$=0.042; DPN group: 18.55 ± 4.15 mph; diabetes group: 21.56 ± 3.97 mph; healthy group: 24.06 ± 4.15 mph). Furthermore, only the healthy group had a significantly faster driving speed during the second drive compared to their first drive ($P$=0.013; Fig. 3a). The mean ± SD duration of loss-of-control events was significantly higher in the DPN group compared to both the diabetes ($P$=0.040) and healthy groups ($P$=0.012) during the first drive (DPN group: 59.07±64.71 s; diabetes group: 13.02±15.46 s; healthy group: 5.86±15.68 s). During the second drive, there was only a significant difference in the duration of loss-of-control events between the DPN and the healthy group ($P$=0.049; DPN group: 13.83 ± 20.23 s; diabetes group: 7.08 ± 8.11 s; healthy group: 0 ± 0 s) The DPN group was the only group that significantly reduced the duration of loss-of-control events during the second drive when compared to the first one ($P$=0.023; Fig. 3b).

The accelerator pedal 'difference plots' highlight significant differences in control of the accelerator pedal of the DPN group compared to both the diabetes (pedal position angle: 0–0.5°, $P$=0.010; 1–1.5°, $P$=0.039; 1.5–2°, $P$=0.044) and healthy groups [pedal position angle: 0–0.5°, $P$=0.002; 3–3.5°, $P$=0.041; 3.5–4°, $P$=0.036; 4–4.5°, $P$=0.046 (Fig. 4)]. Participants with DPN spent more of the time driving with the accelerator pedal closer to, or at the extremes of its range, that is, both barely depressed and at the other extreme, approaching full depression to the floor. They spent much less of the time with the pedal in its mid-range.

## Discussion

In the present study we show for the first time how sensory-motor deficits associated with DPN influence the use of the accelerator pedal during a driving simulator task. Despite driving more slowly, participants with DPN drove using the extremes of the

accelerator pedal range: either barely depressed at all, or near fully depressed to the floor. Furthermore, the DPN group experienced more loss of control (longer duration, in seconds) while driving compared to the participants with diabetes but without neuropathy and compared to the healthy participants. From a positive perspective, only the participants with DPN improved significantly from the first to the second drive in reducing the duration of loss-of-control events, highlighting that this might be modifiable by training.

In both drives, we found that the DPN group had a very different approach to the use of the accelerator pedal from that of both the diabetes and healthy groups. The DPN group, when driving, spent most of the time with the accelerator pedal depressed by ≤1°, i.e. much more time hardly pushing at all on it, while in the same drive, they also spent more time using the more extreme depressed position of the pedal right to the floor, tending to skip the middle range of pedal compression. The healthy participants and those with diabetes but no neuropathy, in contrast, used the extremes of pedal position less of the time. Rather, there was a gradual decrease in the time the pedal spent more and more pushed down, representing a more homogeneous, smoothly graded use of the middle range of the pedal.

Another interesting finding concerns driving speed; participants with DPN drove significantly more slowly than the healthy participants of the same age without diabetes, a mechanism already observed in older cohorts, attributed to a desire to compensate for their slower reaction times [26,27]. Interestingly, despite the fact that they were driving more slowly, the duration of loss-of-control events that occurred during the first drive was significantly higher in the DPN group compared to the healthy group. Although this observation indicates the potential of DPN to impair driving ability, analysis of the second drive led to a more positive finding. After one drive, the DPN group was able to

greatly reduce the loss-of-control events, indicating residual ability for improving driving performance, and the potential for practice or structured training to recover the lost ability in a functional way. Standard sensory-motor function tests, which all drivers undertook, confirmed the detrimental effects of the nerve damage attributable to diabetes in affecting plantar flexor muscle function and ankle proprioception, which may be the underlying mechanisms for the impaired control of the accelerator pedal. Participants with DPN had lower maximal ankle plantar flexor muscle strength values and impaired ankle proprioception function compared to the healthy group. As regards speed of strength generation, the participants with diabetes both with and without DPN developed less strength in the first 150 ms of a maximal isometric contraction of plantar flexor muscles compared to healthy participants. It has been demonstrated that the most disabling consequence of DPN is the continuous loss of motor axons, which, in combination with insufficient reinnervation, results in denervation of muscle fibres, responsible for the muscular atrophy and muscle weakness at the ankle [28]; thus, the formation of very large motor units leads to a progressive reduction in force steadiness and fine motor control in this population [6]. This might explain the observed loss of continuously graded control of the accelerator pedal when driving.

These observations support our initial hypothesis that the ability to know where the foot is in space (proprioception) and a reduced tactile sensation of the pedal against the foot, could be two major and important factors in achieving precise control and appropriate pressure on the accelerator pedal during driving.

We acknowledge that the present sample size may be considered relatively small, however, tests used to assess motor control were highly reproducible and there was adequate statistical power to detect significant changes in the main variables, as evidenced from our results. We acknowledge that there may be residual confounding

variables as a result of unknown differences between the groups examined that we might have been unable to control for. In this respect, a future longitudinal study may be useful in confirming the present findings.

Whilst people with DPN might represent an increased risk with regard to driving safely, our findings indicate that drivers with DPN potentially retain a residual ability to improve, but further research will determine whether this potential for improvement can be realized through a specific, standardized and systematic training programme. Through future research we intend to use the knowledge of the specific pattern of impairment to devise solutions that help drivers with DPN to drive more safely for longer, which would confer benefits with regard to quality of life and autonomy.

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**Competing interests**

None declared.

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FIGURE 1 Example from a single participant's steering wheel signal to illustrate how the 'loss-of-control events' (quantified in seconds) were identified from the steering wheel signal (degrees) during driving. Portions of the wheel signal (Start–End) that abruptly exceeded the range seen during normal driving in terms of amplitude and/or frequency were identified. These periods were then summed and quantified as the total duration (seconds) in one single drive, and then normalized for the number of participants in each group (to obtain seconds per person).

FIGURE 2 (a) Speed of strength generation (SSG). (b) Maximal strength values. (c) Ankle repositioning error (ARE). All motor function testing was performed on the right leg using a joint angle of 10° plantar flexion in the three groups: healthy participants, participants with diabetes but no neuropathy, and participants with diabetic peripheral neuropathy (DPN). Values are mean and SD, $n$=32. *Significantly different, $P$<0.05.

FIGURE 3 (a) Driving speed (mph) during the first drive (black bars) and second drive (white bars) for each group: healthy participants; participants with diabetes but no neuropathy; and participants with diabetic peripheral neuropathy (DPN). (b) Duration of the loss-of-control events (seconds per person) during the first (Drive 1) and second (Drive 2) drives. Values are mean and SD, $n$=32. *Significantly different, $P$<0.05.

FIGURE 4 Accelerator pedal position frequency distribution plots. Each bar represents the time (seconds) the accelerator pedal spent in a specific position, from 0 (no pressure on the pedal) to a maximum of –20° (maximal pressure applied on the pedal) during driving. The graphs on the left side represent the original frequency distribution plots of each group: Healthy individuals (Healthy), people with diabetes but no neuropathy (Diabetes), and people

with diabetic peripheral neuropathy (DPN); on the right side the 'difference plots' are obtained by subtracting one group plot from another. The change in colour indicates the significantly different DPN group's pedal use when compared to those of the Healthy and Diabetes groups ($P$<0.005). The upper panel represents the comparison between DPN and Healthy groups, while the lower panel represents the comparison between DPN and Diabetes groups.

Table 1 Characteristics of the healthy, diabetes but no neuropathy, and diabetic peripheral neuropathy groups included in the study

| Group | Participants, *n* | Age, years | VPT, Volts | Driving licence, years |
|---|---|---|---|---|
| Healthy | 11 | 60 ± 11 | 7 ± 3 | 40 ± 8 |
| Diabetes | 10 | 62 ± 10 | 11 ± 6 | 43 ± 9 |
| DPN | 11 | 67 ± 5 | 44 ± 10 | 45 ± 9 |

DPN, diabetic peripheral neuropathy; VPT, vibration perception threshold.

Values are mean and SD.