Nerve Conduction Velocities Of Toll Collectors Who Are Exposed To Exhaust Particles In Highways

A Ozturk, B Tokmak, A Tokmak

Citation

A Ozturk, B Tokmak, A Tokmak. *Nerve Conduction Velocities Of Toll Collectors Who Are Exposed To Exhaust Particles In Highways*. The Internet Journal of Neurology. 2006 Volume 8 Number 2.

Abstract

Background: The nervous system is the most vulnerable system for lead poisoning. In literature mostly occupational lead intoxication has been investigated in workers who are exposed to exhaust gas.

Methods: 33 highway toll collectors (HTCs) between 25-60 years old were participated in this study. All 33 participants underwent a clinical neurological assessment and laboratory tests including electroneurography.

Results: There was no significant correlation between the exposure duration and the neurophysiological measurements.

Conclusion: The association between symptoms and exposure to chemicals is still controversial. We strongly recommend highway patrol workers to have neurological examinations at least once a year and electrophysiological tests more frequently.

INTRODUCTION

Traffic exhausts contains a complex mixture of byproducts including hundreds of pollutants in gaseous and particulate phases. Most of the particulate phase constitutes fine-mode particulate matter, which is derived from (a) combustion processes that has volatilized and then condensed to form primary particles or from (b) precursor gases reacting in the atmosphere to form secondary particles. Pollution is becoming a significant public health problem as well as an important political issue, due to the rapid growth in world population ([1]). Epidemiological studies have suggested that exposure to atmospheric particulate matter is associated with an increased risk of morbidity and mortality, but the mechanism remains unclear ([1, 2]).

Concerns raised in international context regarding the adverse health effects of air pollution. Of the motor vehicle generated air pollutants, exhaust particles (EPs) account for a highly significant percentage of the particles emitted in many towns and cities ([2]). Complete combustion of EPs produce water and the formation of various gases, liquids, and solid particles. EPs contain a complex mixture of gases such as carbon monoxide, nitric oxides, sulphur dioxide, hydrocarbons, formaldehyde, transition metals and carbon particles ([3]). These gases may cause harmful effects like stroke, cancer, autoimmune disorders, coagulation defects, and cardiovascular diseases ([2, 4, 5]).

The best known heavy metals in exhaust particles are Cu, Pb, Cd, Fe, and Zn. In literature, mostly occupational lead intoxication has been investigated in workers who are exposed to exhaust gas ([6,7,8,9,10]).

In this study, our aim is to visualize the effects of exhaust particles on peripheral nervous system in highway toll collectors by electroneurography. The overall objective of the present study was to study the relation between exposure to high levels of traffic exhausts and the differences of nerve conduction velocities.

MATERIALS AND METHODS

33 highway toll collectors (HTC) between 25-60 years old (mean 38.1 ± 7.7 years), were participated in this study. Clinical exclusion criteria were hypertension, diabetes mellitus, chronic illnesses which can cause peripheral neuropathy such as chronic renal failure, cerebrovascular and peripheral vascular diseases, drug use for systemic diseases, and consumption of narcotics and/or excessive alcohol. Participants's characteristics are shown in Table 1.

Figure 1

Table 1: Participants's characteristics (n = 33)

AGES (y)	
Mean (SD)	38.1 (7.7)
Range	25-60
20-39	8 (%24)
30-39	10 (%30)
40-49	14 (%43)
50-60	1 (%3)
DURATION OF OCCUPATIONAL EXPOSURE (y)	
Mean (SD)	14.2 (6.6)
0-10	11 (%33)
11-20	19 (%58)
21-30	2 (%6)
31-40	1 (%3)

All 33 participants underwent a clinical neurological assessment as outlined below :

CLINICAL ASSESSMENT

* Systemic questionnaire contains mood and sleep disturbances, fatigue, colicky abdominal pain, and constipation.
* Physical examination contains; coldness, skin color, marmorisation, and blood pressure in lying and standing positions.
* Neurological examination contains;
** Symptoms of paraesthesiae and pain distally in the limbs: 0 = absent, 1 = mild and intermittent, 2 = continuous without interference with movement or sleep.
** Limb power: 0 = normal, 1 = minor symptoms or signs, 2 = incapable of work or independent walking.
** Tendon reflexes: 0 = normal, 1 = diminished ankle jerk compared with other tendon reflexes, 2 = absent ankle jerk.
** Pin prick sensation: 0 = normal, 1 = blunted distally, 2 = lost distal pain sensation.
** Vibration sense (metatarsophalangeal joints): > 10 seconds = normal.

Fourteen subjects did not have paresthetic complaints and neuropathic symptoms or signs suggesting polineuropathy. Of the remaining 19 subjects, all had paresthetic complaints and features of polyneuropathy. The systolic and diastolic blood pressures and heart rates of all cases were in normal limits. All 33 subjects were investigated including full blood count, urinary analysis, liver enzymes, ECG and electroneurography. 19 subjects were investigated for other causes of neuropathy by detailed history, physical and neurological examination and blood chemistry. The examinations and the laboratory findings were in normal ranges. Clinical features are presented in Table 2. Electrophysiological measurements were studied in upper extremities bilaterally and in right lower extremity by using EBNeuro. Motor conduction velocity (MCV), distal motor latencies (DMLs) and amplitudes (CMAPs) were measured in median, ulnar, peroneal and tibial nerves. Sensory nerve action potential latencies (DSLs), sensory conduction velocity (SCV) and amplitudes (SNAPs) were measured in the median, ulnar and sural nerves with an average of 10 stimulus responses.

Figure 2

Table 2: Participants's clinical features (n = 33)

SYMPTOMS		
Paraesthesiae	Absent	19
	Mild	14
Pain	Absent	19
	Mild	14
SIGNS		
Pin prick	Normal	22
	Reduced	11
Vibration sensation	Normal	33
	Reduced	0
Achilles tendon reflexes	Normal	23
	Diminished	9
	Absent	1
Postural hypotension	Present	10
	Absent	23

RESULTS

All subjects in this study were male. A typical HTC works 8 hours a day. The mean year of HTCs lifetime work period is 14,2 ± 6,6. All electroneurographic parameters, including motor nerve conduction velocity (MCV), distal latency (DML), and compound muscle action potential (CMAP) of median, ulnar, tibial, and peroneal nerves as well as sensory nerve conduction velocity (SCV), sensory nerve action potential (SNAP) and distal latency of median, ulnar and sural nerves substained normal ranges are shown in Table 3. There was no significant correlation between the exposure duration and the neurophysiological measurements.

Figure 3

Table 3: Nerve conduction studies (mean (SD))

	Median		Ulnar		Tibial		Peroneal		Sural	
Parameter		Normal		Normal		Normal		Normal		Normal
DML®	3,4 (0,9)	<4,4	2,6 (0,5)	<3,3	4,6 (1,2)	<5,8	4,1 (0,9)	<6,5		
DML(L)	3,4 (0,7)	<4,4	2,6 (0,5)	<3,3						
MCV®	58 (8)	>49	57,2 (9,5)	>49	48,5 (8,4)	>41	46,4 (6)	>44		
MCV(L)	56,1 (6,6)	>49	57,9 (7,8)	>49						
CMAP®	6,4 (2,6)	>4	6,1 (1,8)	>6	9,1 (4,9)	>4	5,3 (3,3)	>2		
CMAP(L)	6,1 (3)	>4	6,2 (2)	>6						
SNAP®	52 (22,1)	>19	47,5 (31,4)	>17					12,3 (10,7)	>6
SNAP(L)	58,7 (21)	>19	44,2 (22,1)	>17						
DSL®	2,3 (0,4)	<3,4	2,1 (0,9)	<3,1					2,5 (1,1)	<4,4
DSL(L)	2,2 (0,3)	<3,4	2 (0,3)	<3,1						
SCV®	59 (7,3)	>50	54,4 (7,3)	>50					43,3 (19,1)	>40
SCV(L)	59,5 (6,6)	>50	55,8 (5,8)	>50						

DML: Distal motor latency (ms), MCV: Motor conduction velocity (m/s), CMAP: Compound muscle action potential (mV), SNAP: Sensory nerve action potential (μV), DSL: Distal sensory latency (ms), SCV: Sensory conduction velocity, ®: Right, L: Left

DISCUSSION

The nervous system is the most vulnerable system for lead poisoning. Automobile emissions are the most responsible source for lead exposure, especially for urban residents. As electrophysiologically, both sensory and motor peripheral nerve involvement is seen in adults with chronic lead intoxication. Sensory complaints include paresthesias and spontaneous pain ([11]). Motor signs include local weakness, atrophy, and fasciculations ([12]).

The clinical picture of lead neuropathy is probably dependent on the duration and level of exposure. The classic form, which is unique among toxic neuropathies, is seen following subacute exposure to high environmental concentrations of lead. This disorder is purely motor in its clinical manifestations and the distribution of the weakness is extremely unusual, with early and severe involvement of wrist and finger extensors, usually before any other muscle involvement. Most toxic neuropathies are predominantly sensory and the sensory loss is in a length-dependent pattern, with feet involved first and the more proximal regions later, if at all. Weakness tends to involve the extensors of the middle and ring fingers first before spreading to other finger and thumb extensors and then wrist extensors. Weakness of the thenar muscles and, less commonly, the interossei may be present, although it is usually mild. Rarely, more proximal muscles may be involved, even up to the shoulder girdle ([13]).

Extensor bilateral neuropathy involving the hands, fingers, deltoids, biceps, and triceps may also occur ([11]).

The older literature suggests that even neck, bulbar, and extraocular muscles may be involved ([14]).

Weakness is also found in the lower limbs, where it is usually confined to foot dorsiflexion and toe extension. Occasionally, in severe intoxication, weakness may progress to quadriplegia ([15]). In severe cases of lead toxicity, wrist drop and foot drop have been well documented ([16,17,18]).

Prognosis for recovery is good, once the patient has been removed from exposure, depending on the severity of the weakness ([19]). In patients with severe weakness recovery may be slow and incomplete ([13]).

The most persuasive evidence in support of the contention that lead is a general neurotoxin comes from a recent study of 46 individuals in Latvia with long-term industrial lead exposure ([8]). All patients complained of pain and paresthesias that occurred in a distal and symmetrical distribution. Pain and vibration perception were both impaired, indicating involvement of both large and small fibers. No patient had weakness. Muscle stretch reflexes were diminished or absent distally. About half of the patients had autonomic involvement manifested as orthostatic hypotension, hyperhidrosis, and altered distal limb color and temperature. Thus, these patients had a much more typical toxic neuropathy affecting sensory more than motor functions and having loss of sensation in a distal and symmetrical distribution.

Likewise our study showed that there were not any electrophysiological changes in HTC workers who occupationally exposed to exhaust gas. However, in other cases mild slowing in nerve conduction velocity were reported even in asymptomatic lead workers. In individuals with predominantly motor findings, nerve conduction

velocity may not be altered even after significant occupational exposure ([11]). Catton et al. compared 19 neurologically asymptomatic lead-exposed workers with 17 hospital workers without known lead exposure. The patients and controls were not specifically age-matched but they were of the same age range and the mean age for the two groups was identical. They found that motor and sensory response amplitudes and maximal motor and sensory conduction velocities were the same in the two groups but that there was a slight, but statistically significant, segmental amplitude decrement in the peroneal nerve in the leg, suggesting an increased range of conduction velocities. There was no relationship between the severity of these changes and the blood lead level ([20]). The association between symptoms and exposure to chemicals is still controversial ([11]).

This study shows that highway patrol workers who are exposed to exhaust may develop polineuropathy regardless of normal electrophysiological test results. Hence, we strongly recommend highway patrol workers to have neurological examinations at least once a year and electrophysilogical tests more frequently.

CORRESPONDENCE TO

Burcu Tokmak Düzce University Medical Faculty Neurology Department Konuralp, DÜZCE Telephone: 903805414107 Fax: 903805414105 e-mail: b_ozdemirli@yahoo.com

References

1. Salvi SS, Frew A, Holgate S. Is diesel exhaust a cause for increasing allergies?. Clin Exp Allergy. 1999; 29:4-8.
2. Sydbom A, Blomberg A, Parnia S, Stenfors N, Sandstrom T, Dahlen SE. Health effects of diesel exhaust emissions. Eur Respir J. 2001;17:733-46.
3. Scheepers PT, Bos RP. Combustion of diesel fuel from a toxicological perspective. II. Toxicity. Int Arch Occup Environ Health. 1992;64:163-77.
4. Yoshino S, Sagai M. Enhancement of collagen-induced arthritis in mice by diesel exhaust particles. J Pharmacol Exp Ther. 1999;290(2):524-9.
5. Schwartz J, Dockery DW, Neas LM. Is daily mortality associated specifically with fine particles?. J Air Waste Manag Assoc. 1996 Oct;46(10):927-39.
6. Triebig G, Weltle D, Valentin H. Investigations on neurotoxicity of chemical substances at the workplace. . Int Arch Occup Environ Health. 1984;53(3):189-203.
7. He FS, Zhang SL, Li G, Zhang SC, Huang JX. An electroneurographic assessment of subclinical lead neurotoxicity. Int Arch Occup Environ Health. 1988;61(1-2):141-6.
8. O Rubens, I Logina, I Kravale, M Eglite, M Donaghy. Peripheral neuropathy in chronic occupational inorganic lead exposure: a clinical and electrophysiological study. J Neurol Neurosurg Psychiatry 2001;71:200-4.
9. Hursidic-Radulovic A, Cvitkovic J. Lead exposure in highway toll-booth workers. Arh Hig Rada Toksikol. 2003;54(2):133-140.
10. Strauss P, Orris P, Buckley L. A health survey of toll booth workers. Am J Ind Med 1992;22(3):379-384.
11. Christopher G.Goetz; Textbook of clinical neurology, second edition. Chapter 39, p 848
12. Kajiyama K, Doi R, Sawada J, Hazama T, Nakata S. A case of lead neuropathy--importance of subclinical entrapment of nerves in lead neuropathy. Rinsho Shinkeigaku. 1991 Jul; 31 (7): 725-9
13. Thomson RM, Parry GJ. Neuropathies associated with excessive exposure to lead. Muscle nerve. 2006 Jun: 33 (6): 732-41
14. Aub JC, Fairhall LT, Minot AS, Reznikoff P. Lead poisoning. Medicine 1925; 4: 1-250.
15. Oh SJ. Lead neuropathy: case report. Arch Phys Med Rehabil 1975; 56: 312-317
16. Imbus CE, Warner J, Smith E, Pegelow CH, Allen JP. Peripheral neuropathy in lead-intoxicated sickle cell patients. Muscle Nerve. 1978 Mar-Apr; 1 (2): 168-171
17. Barats MS, Gonick HC, Rothenberg S, Balabanian M, Manton WI. Severe lead-induced peripheral neuropathy in a dialysis patient. Am J Kidney Dis. 2000 May; 35 (5): 963-8.
18. Santana-Lopez S, Bistel-Gonzalez RA, Rodriguez-Garcia A, Castellanos JA. Paralysis of the radial nerve due to exposure to lead. Rev. Neurol. 2006 Feb; 16-28; 42 (4): 253-5
19. Windebank AJ. Heavy metals and neurological disease. In: Evans RW , Baskin DS , Yatsu FM , editors. Prognosis of neurological disorders. New York: Oxford University Press; 1992. p 571-576.
20. Catton MJ, Harrison MJG, Fullerton PM, Kazantzis G. Subclinical neuropathy in lead workers. Br Med J 1970; 2: 80-82

Author Information

Ayhan Ozturk
Assistant Professor, Medical Faculty Neurology Department, Düzce University

Burcu Tokmak
Resident, Medical Faculty Neurology Department, Düzce University

Abdurrahman Tokmak
Resident, Medical Faculty Otorhinolaryngology Department, Düzce University