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Peripheral Neuropathy in Workers Exposed to Nitromethane

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Background *Two workers from a headlight subassembly plant developed severe peripheral neuropathy. These workers had extensive, but brief (1–2 months) dermal and inhalational exposure to nitromethane, a solvent.*

Methods *Environmental sampling was performed for nitromethane and ethyl cyanoacrylate. Medical records, including electrodiagnostic studies, were reviewed. Literature on nitromethane, ethyl cyanoacrylate, and other exposures in the workplace was reviewed.*

Results *Electromyography and nerve conduction studies performed on these patients were consistent with a severe, axonal neuropathy. No etiology was discovered despite an extensive medical evaluation. Environmental sampling revealed exposure to nitromethane at the threshold limit value.*

Conclusions *The history of acute onset of severe peripheral neuropathy temporally associated with exposure to nitromethane is suggestive of a toxic neuropathy. While it cannot be definitively concluded that these two workers developed peripheral neuropathy secondary to exposures at work, occupational exposure to nitromethane appears to be the most likely etiology.* Am. J. Ind. Med. 40:107–113, 2001.

KEY WORDS: neuropathy; nitromethane; ethyl cyanoacrylate; methyl methacrylate

INTRODUCTION

In 1999, the National Institute for Occupational Safety and Health (NIOSH) received a request for technical assistance from the Occupational Safety and Health Administration (OSHA) to review medical information concerning sensorimotor polyneuropathy in two former employees of a headlight subassembly plant. OSHA conducted an industrial hygiene inspection at the plant based on a referral from a neurologist treating these employees for peripheral neuropathy. This paper describes the work histories as well as the clinical and electrophysiological evaluations of the workers concerned.

At the time of our investigation (several months after the employees became ill), the worksite employed approximately 20 workers. The employees worked at tables of four, where two employees glued a rubber gasket seal around the headlight using Loctite Prism 401® and two employees inspected the headlight and cleaned off excess glue using nitromethane. Loctite Prism 401® is a rapidly drying glue composed of 90–95% ethyl cyanoacrylate, 5–10% methyl methacrylate, and 0.1–0.5% hydroquinone. Nitromethane was sprayed onto the headlight from a small spray bottle then wiped off with a rag. No gloves were worn during this process. Personal protective equipment worn by all employees consisted of aprons and safety glasses. Work hours averaged 55–60 hr weekly, but could vary from fewer than 40 to over 90 hr per week. During an 8-hr day,

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approximately one 2-kg bottle of Loctite Prism 401® and 3/4 gallon of nitromethane were used in the plant. This increased to $1\frac{1}{2}$ bottles of Loctite Prism 401® and 1 gallon of nitromethane when the workday was 10 hr or longer.

CASE PRESENTATIONS

Case I

One month after beginning work in the plant, a 26-year-old woman, whose primary duty was to clean headlights with nitromethane, began to note weakness in her hands, legs, and feet. She quit work, but after 2 weeks she felt worse, sought care at the Emergency Department (ED), and was admitted to the hospital. The neurological exam at that time was significant for absent gastrocsoleus and brachioradialis reflexes, and for weakness that was more severe distally, particularly in the lower extremities. MRI of the cervical spine was normal. Multiple laboratory tests (including antinuclear antibody, rheumatoid factor, erythrocyte sedimentation rate, B12 and folate levels, heavy metal screening, thyroid function tests, liver function tests, electrolytes, human immunodeficiency virus, rapid plasma reagin, cryoglobulins, hepatitis panel, *Helicobacter pylori* titer, and complete blood count) yielded no abnormal findings. Serum immunoelectrophoresis showed an increased gamma component. Lumbar puncture revealed an elevated protein level of 108 mg/dL (normal = 15–45 mg/dL) and a normal cell count.

Electrodiagnostic testing performed during the hospitalization (about 4 weeks after ceasing employment) were interpreted as being consistent with Guillain-Barre syndrome (GBS). She was transferred to a tertiary care center where she was diagnosed with severe peripheral neuropathy rather than GBS. Because of the severity and the rapid onset it was thought to be secondary to a toxic exposure. Independent interpretation, without knowledge of the history, of her initial electrodiagnostic studies by one of us (M.J.G.) was axonal polyneuropathy consistent with toxin exposure. Her symptoms persisted and electrodiagnostic studies performed 8 months after onset of symptoms revealed evidence of a diffuse, symmetrical axonal polyneuropathy with demyelinating features. The needle electrode examination suggested changes compatible with nerve regeneration as well. (Tables I and II). Again, this was confirmed by an independent interpretation by one of the authors (M.J.G.). She reported slight improvement in symptoms at the time of our evaluation.

TABLE I. Nerve Conduction Data, Case I, 8 Months After the Onset of Symptoms.

Nerve/stimulation site	Amplitude (mV)	Latency (ms)	Conduction velocity (m/s)
Sensory studies			
Ulnar (L[a])			
Wrist	51.6	2.5	—
Below elbow	21.9	6.5	56
Above elbow	17.4	8.8	57
Sural (R[b]/L)	0/0	—[c]/1.3	—
Motor studies			
Ulnar (L)			
Wrist	2.8	4.5	—
Below elbow	1.5	9.7	43
Above elbow	2.4	12.7	43
Peroneal (L)			
EDB[d]—Ankle	0.3	5.5	—
Fibular head	0.3	7.4	53
Tibial (R/L)			
Ankle	NR[e]	NR	—
Popliteal fossa	NR	NR	—

[a] L, left.
[b] R, right.
[c] — Indicates not performed.
[d] EDB, extensor digitorum brevis.
[e] NR, no response.

Case II

Six weeks after beginning employment at the plant and approximately 4–5 months after the onset of illness in the previous case, a 23-year-old man, whose primary duty was to clean headlights with nitromethane, presented to the ED complaining of foot numbness for $1\frac{1}{2}$ weeks. A definitive diagnosis was not made. He again presented to the ED 10 days later complaining of pain and swelling in both legs and feet for 2 weeks. Physical exam was unremarkable. In the time between the two ED visits, his employment at the plant was terminated**.** Three days after his second ED visit he was admitted to a nearby hospital, where electrodiagnostic testing was done; results were interpreted as consistent with GBS. Independent review of the electrodiagnostic studies by (M.J.G.) found evidence for a polyradiculopathy or peripheral neuropathy in the lower extremities, while the upper extremity testing was normal. The patient was transferred to a tertiary care center where he was diagnosed with probable toxic neuropathy rather than GBS after reviewing his history and electrodiagnostic studies. Pertinent physical findings at that time included diminished lower extremity reflexes, slightly decreased muscle strength on dorsiflexion of both feet, and decreased sensation to pinprick up to the mid-shin.

Laboratory parameters (including antinuclear antibody, rheumatoid factor, erythrocyte sedimentation rate, B12 and folate levels, toxicological screening for drugs [barbiturates, amphetamines, benzodiazepines, cannabinoids, cocaine

TABLE II. Needle EMG Examination, Case I, 8 Months After the Onset of Symptoms.

Muscle (all left side)	Fibrillation	Positive sharp waves	Fasciculation	Polyphasic action potentials	Amplitude	Duration
Tibialis anterior	3+	3+	none	many	↑[b]	↑
Gastrocnemius	3+	3+	none	many	↑	↑
Vastus medialis	2+	2+	none	many	↑	↑
Abductor pollicus brevis	3+	3+	none	many	↑	↑
Abductor digitus minimus	3+	3+	none	many	↑	↑
Extensor indicus	2+	2+	none	many	↑	↑

[a]Maximum effort was documented in all muscle groups.
[b]↑ Indicates increased.

metabolites, opiates, propoxyphene and phencyclidine] and alcohol, thyroid function tests, liver function tests, electrolytes, serum protein electrophoresis, hemoglobin A_1C, human immunodeficiency virus, rapid plasma reagin, and complete blood count) were unremarkable except for an elevated creatinine phosphokinase that returned to normal levels spontaneously. Lumbar puncture was not performed.

His symptoms persisted and follow-up electrodiagnostic studies were done $3\frac{1}{2}$ weeks later. These studies demonstrated progressive denervation and severe axonal peripheral neuropathy (Tables III and IV). At the time of our investigation this individual reported significant, although incomplete, improvement. No further electrodiagnostic studies were performed.

Social, family, environmental, and past medical histories were noncontributory in both of these individuals.

ENVIRONMENTAL SAMPLING

As part of their evaluation, OSHA's investigator conducted air sampling for nitromethane and ethyl cyanoacrylate over 2 days. Personal breathing zone samples were collected on four workers for nitromethane and six workers for ethyl cyanoacrylate (Table V) No sampling was done for methyl methacrylate. Personal breathing zone concentrations of nitromethane for workers who were cleaning headlights ranged from 10 to 20 ppm as an 8-hr time-weighted average (TWA), with a mean of 12.75 ppm. The OSHA permissible exposure limit (PEL) for nitromethane is 100 ppm [Code of Federal Regulations, 1997] and the American Conference of Governmental Industrial Hygienists' (ACGIH ®) Threshold Limit Values (TLV ®) is 20 ppm [ACGIH, 2000]. Personal breathing zone concentrations of ethyl cyanoacrylate for workers who were gluing

TABLE III. Nerve Conduction Data, Case II, 6–7 Weeks After Onset of Symptoms.

Nerve/stimulation site	Amplitude (mV)	Latency (ms)	Conduction velocity (m/s)
Sensory studies			
Ulnar (R[a]/L[b])			
Digital	20/20	2.3/2.5	—[c]
Median (R/L)			
Digital	20/20	2.8/2.9	—
Sural (R/L)	—	—	—
Motor studies			
Median (R/L)			
APB[d]—wrist	1/1.5	4.6/3.1	—
APB—elbow	1/1.5	10.0/8.3	47/46
Ulnar (R/L)			
ADQ[e]—wrist	2/2	2.5/2.7	—
Below elbow	NR[f]	NR	—
Above elbow	2/2	8.5/9.2	53/48
Peroneal (R/L)			
EBD[g]—ankle	NR	NR	—
Fibular head	NR	NR	—
Knee	NR	NR	—
Tibial (R/L)			
Ankle	NR	NR	—
Popliteal fossa	NR	NR	—

[a]R, right.
[b]L, left.
[c]—, Indicates not performed.
[d]APB, abductor pollicis brevis.
[e]ADQ, abductor digiti quadratus.
[f]NR, no response.
[g]EDB, extensor digitorum brevis.

TABLE IV. Needle EMG Examination, Case II, 6–7 Weeks After Onset of Symptoms.

Muscle	Fibrillation	Positive sharp waves	Insertional activity	Polyphasic action potentials	Amplitude	Duration
Tibialis anterior (R[a]/L[b]	3+	3+	↑[c]	>10	1–2	5–10
Gastrocnemius (R and L)	3+	3+	↑	>10	1–2	5–10
Quadriceps (R and L)	2+	2+	↑	>10	1–2	5–10
Peroneus longus (R and L)	3+	3+	↑	>10	1–2	5–10
Flexor digitorum longus (R and L)	3+	3+	↑	>10	1–2	5–10
First dorsal interosseous (R and L)	3+	3+	↑	>10	1–2	5–10
Extensor digitorum brevis (R and L)	3+	3+	↑	>10	1–2	5–10
Pronator teres (R)	3+	3+	↑	>10	1–2	5–10
Brachioradialis (L)	3+	3+	↑	>10	1–2	5–10
Biceps (R)	0	0	Normal	>10	1–2	5–10
Deltoid (R and L)	0	0	Normal	>10	1–2	5–10

[a]R, right.
[b]L, left.
[c]↑ Indicates increased.

ranged from 0.04 to 0.16 ppm as an 8-hr TWA, with a mean of 0.09 ppm. There is no PEL for ethyl cyanoacrylate, but the TLV is 0.2 ppm.

DISCUSSION

Nitromethane

Nitromethane is used as a solvent, a fuel, in explosives, and as a chemical coating. The most common route of occupational exposure to nitromethane is via inhalation of vapors. NIOSH estimates that over 134,000 workers in the United States are exposed to nitromethane [NIOSH, 1990]. ACGIH lists the critical health effects of nitromethane as irritation, narcosis, liver, and thyroid toxicity, blood dyscrasias, and neuropathy [ACGIH, 2000]. Other than irritation and narcosis, these health effects have been found only in animal studies [ACGIH, 1991]. ACGIH cites studies done by the National Toxicology Program to assess neurotoxicity in rats and mice [NTP, 1997]. In these studies, rats and mice were exposed to nitromethane at a variety of concentrations for 6 hr per day, 5 days per week, from 16 days to 2 years. In the 16-day study of rats, all those exposed to 1500-ppm showed loss of coordination in the hindlimbs. Sciatic nerve degeneration was found in all rats exposed to 375 ppm and above. In the 13-week study, hindlimb paralysis was seen in all rats in the 1500-ppm group. Rats exposed to 375 ppm or greater had minimal to mild degeneration of the spinal cord and sciatic nerve. In contrast, the mice demonstrated no neurologic abnormalities in any of the studies, and there were no abnormalities in either species in 2-year studies.

TABLE V. Environmental Sampling Results, Personal Breathing Zone Samples

Substance	Results (8 hr time-weighted average) (parts per million (ppm))
Nitromethane	13
	20
	10
	18
Ethyl cyanoacrylate	0.16
	0.07
	0.04
	0.11
	0.11
	0.07

In one human case report, a 20-year-old female who had worked for 2 years using a mixture of trichlorotrifluoroethane (94%), methanol (6%), and nitromethane (0.25%) presented with Parkinsonism [Sandyk and Gillman, 1984]. There were no sensory abnormalities. A medical evaluation did not reveal a cause for the Parkinsonism, and she had no family history of that disorder. The authors concluded that in the absence of other potential etiologies, exposure to nitromethane could have been the cause of the Parkinsonism. Another case report describes a 19-year-old male who developed a primary, symmetric demyelinating polyneuropathy after a 2-month exposure to an industrial solvent composed primarily of 1-bromopropane, but also containing nitromethane and other components [Sclar, 1999]. The authors attributed the neuropathy to 1-bromopropane exposure.

Exposure to nitromethane has also been noted to interfere with the Jaffe reaction, the most commonly used colorimetric method for measuring serum creatinine, result-

ing in a false elevation of serum creatinine levels [Gabrielli and Hammett-Stabler, 1998; Mullins and Hammett-Stabler, 1998].

Ethyl Cyanoacrylate

Cyanoacrylates are the main ingredient in high-strength, fast-acting ''super'' glues. They also have numerous medical applications [Vinter et al., 1985]. There are several forms, including ethyl-2-cyanoacrylate and methyl-2-cyanoacrylate. Cyanoacrylates are known to be irritants and to cause asthma [Belsito, 1987; Bruze et al., 1995; Conde-Salazer et al., 1998] and allergic contact dermatitis [Nakazawa, 1990; Savonius et al., 1993; Chan et al., 1994]. A case of peripheral neuropathy, thought to be secondary to cyanoacrylate toxicity was described in a 1991 report [Hanft et al., 1991]. In that case, a 63-year-old male with a 20-year history of glue contact at work and home presented with pain and numbness of the hands and feet. Nerve conduction velocities showed decreased distal latencies. No other potential causes for peripheral neuropathy were identified, and the authors concluded this represented peripheral neuropathy caused by cyanoacrylates.

Methyl Methacrylate

Methyl methacrylate (MMA) is produced by the reaction of hydrogen cyanide with acetone. It is used in the production of acrylic sheeting, molding powders and resins, and surface coatings. It is also a component of surgical bone cement, dentures, and artificial fingernails [IARC, 1999]. It can be absorbed through the skin or inhaled.

Methyl methacrylate, which is chemically distinct from cyanoacrylate [Lemiere et al., 1997], has been reported to cause allergic contact dermatitis [Kassis et al., 1984; Hachman and Zalkind 1997] and asthma [Lozewicz et al., 1985; Nakazawa, 1990]. In one study, nerve conduction velocities were measured in 20 dental technicians who used MMA and reported mild neurological symptoms in their fingers. Nerve conduction abnormalities were found but were slight in degree, with the mean values within normal limits [Seppalainen and Rajaniemi, 1984]. Two case reports describe peripheral neuropathy in dental technicians with long histories of occupational exposure to MMA. One report describes a 36-year-old woman who had been a dental technician for 14 years, three of which were spent in a dental lab using MMA monomer in a variety of procedures [Sadoh et al., 1999]. She had an 11-month history of bilateral stocking-distribution paresthesias and numbness. An extensive medical evaluation was unremarkable except for electrodiagnostic studies that revealed the absence of sensory nerve action potentials (SNAPs) in the legs. The other report describes the case of a 58-year-old man who was a dental prosthetic technician who developed a generalized sensorimotor peripheral neuropathy of the axonal degeneration type [Donaghy et al., 1991]. He had a 30-year history of daily exposure to MMA. His symptoms began with the thumb and index finger of his dominant hand, which he used to mold the dentures, and progressed to mild leg weakness. Medical history and lab studies revealed no explanation for his symptoms. Electrodiagnostic studies were consistent with generalized neuropathy, predominantly involving axonal degeneration.

There are many causes of peripheral neuropathy — a partial list includes inflammatory disorders, metabolic disorders, alcohol abuse, hereditary conditions, vascular conditions, and occupational and environmental exposure to neurotoxic agents [Berger and Schaumburg, 1994; Bleeker, 1994]. The cause of approximately one-third of all polyneuropathies remains unknown despite a thorough evaluation; therefore, the absence of an alternative cause does not necessarily implicate a toxin [Bleecker, 2000]. Toxic neuropathies most commonly present with paresthesias of the hands and feet [Rosenberg, 1992; Bleeker, 2000] and are clinically indistinguishable from neuropathy secondary to other causes. Workplace exposures are not typically high enough to produce acute poisoning so the distinction comes from taking a detailed occupational and environmental history [Bleeker, 2000]. Axonopathies are the commonest form of toxic neuropathy, but there can be secondary demyelination, which results from retraction of myelin around axonal swelling [Rosenberg, 1992; Bleeker, 2000]. Primary demyelination is uncommon in toxic neuropathies. Recovery from a toxic neuropathy can be very slow (months to years) and may not be complete [Rosenberg, 1992; Bleeker, 2000]. In addition, in cases of neuropathy from certain toxins such as *n*-hexane and methyl-*n*-butyl ketone, both symptoms and electrodiagnostic findings continued to deteriorate for several months after removal from exposure [Bleeker, 2000].

While the air sampling did not reveal any concentrations above the current occupational exposure limits for the substances evaluated in our cases, one nitromethane sample had a concentration of 20 ppm, equal to the ACGIH TLV (an 8-hr TWA). However, not all workers will be protected from adverse health effects even though their exposures are maintained below occupational criteria, possibly because of individual susceptibility, a preexisting medical condition, or a hypersensitivity. Some substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled at the level set by the criterion.

Since these headlight factory workers average 55–60 hr per week, they may be exposed to this substance in cumulative amounts exceeding what would result from 40 hours at the TLV. The two workers with neuropathy wiped nitromethane off the headlights with a rag and did not wear

gloves, so skin absorption is a likely source of exposure. Ascertaining the degree of dermal absorption can be problematic. In some cases biological monitoring can provide a more comprehensive measure of exposure than environmental sampling, but there is no known biological marker for nitromethane exposure, and validated biological exposure indices (BEI) exist for relatively few substances. There are no BEIs for nitromethane, methyl methacrylate, or ethyl cyanoacrylate.

Published evidence regarding the occupational exposures described here as potential causes of peripheral neuropathy is limited. Although there is some evidence for nitromethane from animal studies, the results may not be relevant for humans [Hooper et al., 1992; Lowe, 1992]. The two human case reports of neurotoxicity from nitromethane describe different neurologic illnesses (neuropathy and Parkinsonism) and nitromethane was a minor constituent of the solvents to which the cases were exposed. Despite the limited information, however, occupational exposure appears likely as a cause for the peripheral neuropathy in these two workers. Neither had other causes identified. While they could represent idiopathic peripheral neuropathy, which comprises a large proportion of diagnosed neuropathy, we think this is unlikely for several reasons. These two workers were very young and without existing illness. Both developed peripheral neuropathies soon after beginning work in the same plant, in the same position. Both were exposed to a substance for which there is very limited evidence of neurotoxicity.

The company reported that no other workers are known to have experienced similar problems; however, turnover is high, with the average length of employment in the subassembly area being slightly less than one year. Therefore, workers may have left employment secondary to health problems that were not reported to the employer, perhaps because they were not recognized as being work-related. In fact, the company was not aware that these two individuals might have experienced work-related health effects until the time of our investigation.

It is not uncommon, and in fact is quite common, to see only a fraction of workers affected by exposure to a hazardous substance. For example, only a small portion of workers exposed to asthma-causing agents in the workplace actually develop asthma [Chan-Yeung, 1994]. It is possible that these two employees had work practices that may have increased their exposure, such as eating or drinking in the workplace, or that they had some unknown factor which may have increased their susceptibility to develop neuropathy.

While both of these workers had some degree of recovery, one was still severely disabled months after exposure ceased. As noted above, it is not uncommon for both symptoms and electrodiagnostic findings to deteriorate for several months after removal from exposure and recovery to be on the order of months to years, and in some cases complete recovery may not occur.

Nitromethane in some formulation is used by over 134,000 workers in the United States alone, and peripheral neuropathy has never been reported in association with its occupational use. But peripheral neuropathy is not uncommon in the general population, and is often idiopathic, therefore an association with nitromethane could easily have been overlooked, especially since there is very little in the human medical literature concerning nitromethane.

CONCLUSIONS

We present the cases of two workers who developed peripheral neuropathy within weeks of employment at a job where they had brief but significant dermal and inhalational exposure to nitromethane. The striking temporal association of occupational exposure followed by neuropathy in otherwise healthy, young workers supports an occupational exposure as the cause of these neuropathies. Nitromethane appears to be the exposure most relevant to these workers' illnesses because they had significant dermal and inhalational exposure to it. However, the possibility of a mixed exposure (nitromethane plus cyanoacrylates and/or methyl methacrylate) cannot be excluded because workers who cleaned the headlights worked at the same table as those who glued them; therefore, there was potentially some degree of inhalational exposure. The severity of these cases warrants further investigation and prospective study, particularly of dermal absorption, of these compounds as they relate to human exposure.

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